ISSN：0037—3796

# 神経化学

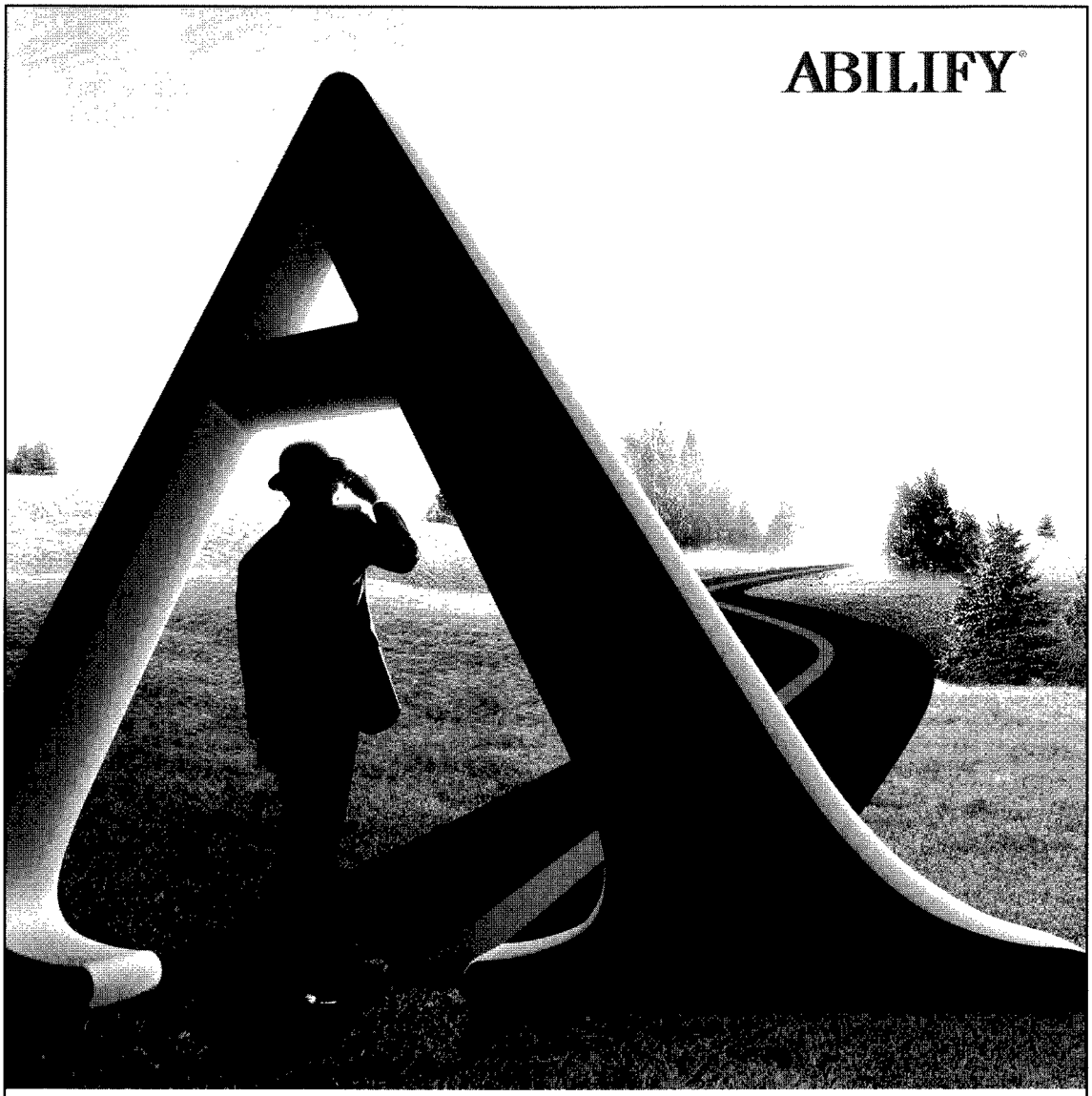
Bulletin of the Japanese Society for Neurochemistry

Vol.46(No.2,3),2007

## Neuro2007
## 第50回日本神経化学会（横浜）大会
## 第30回日本神経科学大会
## 第17回日本神経回路学会大会
## 合同大会

抄 録 号

平 成 19 年 8 月




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〈'07.7作成〉

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# ご挨拶

Neuro2007 にようこそ。

この会は、第 30 回日本神経科学大会、第 50 回日本神経化学会大会、第 17 回日本神経回路学会大会の合同大会で、脳・神経科学の広い分野での最新の研究を発表・議論します。

我が国における脳・神経科学が科学・技術の広い分野との協力と融合を進め、世界との繋がりをますます深めるようになることを目指して企画・準備してきました。

3 日間の会期中には、プレナリー講演 3 件、シンポジウム 40 件（194 演題）、一般口演 288 件、ポスター 1266 件、ランチョンセミナー 13 件と、多くの演題が発表されます。また、63 社から 108 小間の機器展示、12 社から書籍展示がなされます。

激しく討論し、刺激の中で研究構想を考え、新しい仲間を作り、旧交を温め、参加者の皆様の 3 日間が実り豊かなときになりますことを心からお祈りします。

第 30 回日本神経科学大会
大会長　田中　啓治
第 50 回日本神経化学会大会
大会長　高坂　新一
第 17 回日本神経回路学会大会
大会長　大森　隆司



# 大会委員一覧

## 大会長

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| 高坂　新一 | 国立精神・神経センター | 日本神経化学会 |
| 大森　隆司 | 玉川大学 | 日本神経回路学会 |

## 実行委員

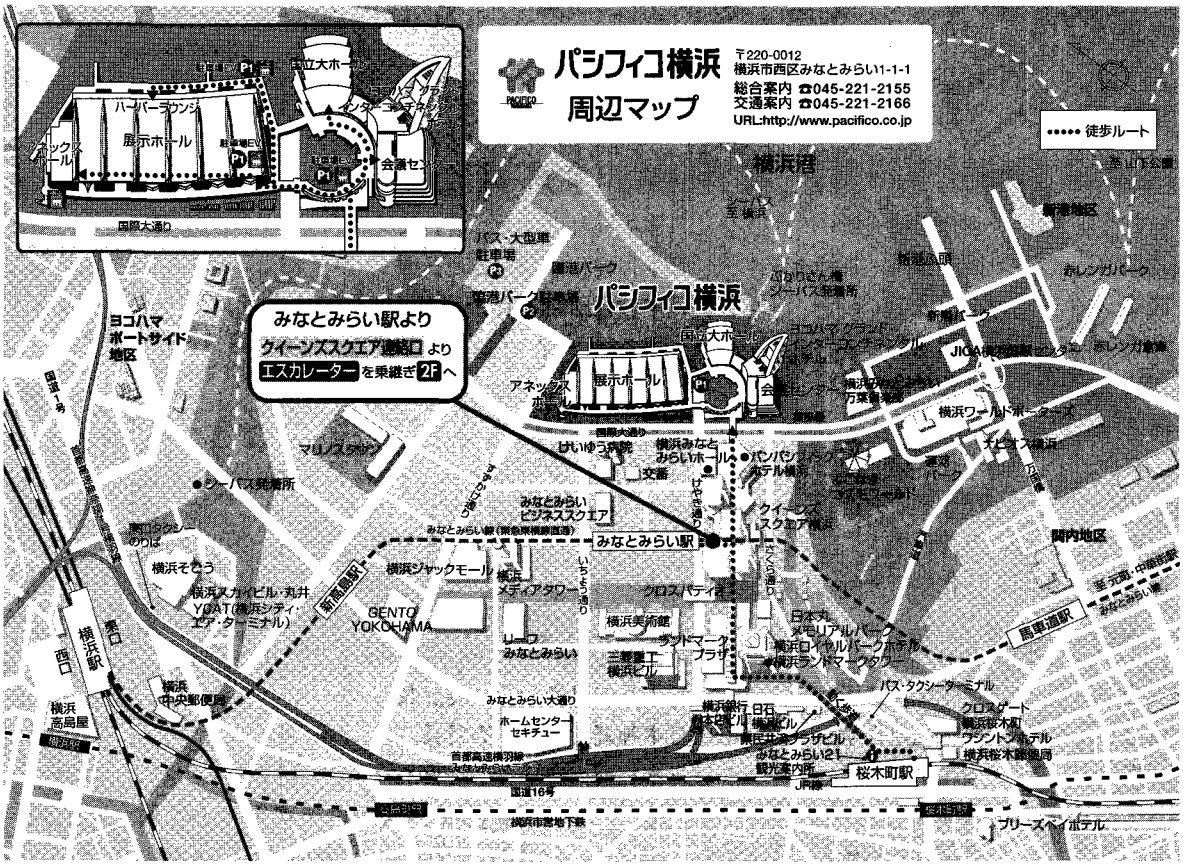
| | | |
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| 岡野　栄之 | 慶應義塾大学 | |
| 岡本　　仁 | 理化学研究所 | |
| 加藤　忠史 | 理化学研究所 | |
| 熊倉鴻之助 | 上智大学 | |
| 深井　朋樹 | 理化学研究所 | |

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| 乾　　敏郎 | 京都大学 | |
| 井上　和秀 | 九州大学 | |
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| 高松　　研 | 東邦大学 | |
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| 三浦　正幸 | 東京大学 | |
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| 吉川　武男 | 理化学研究所 | |
| Kathleen S Rockland | 理化学研究所 | |
| 和田　圭司 | 国立精神・神経センター | |

（50音順）

# パシフィコ横浜へのアクセス




## 交通のご案内

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 電車で | 渋谷駅 | 東急東横線:特急→みなとみらい線<br>(東急東横線・みなとみらい線 直通運転) | | 横浜駅 | 30分 | みなとみらい駅 | 徒歩 | 3分 |
| 電車で | 新宿駅 | JR湘南新宿ライン | 29分 | 横浜駅 | みなとみらい線 3分<br>(東急東横線直通) | 「クイーンズスクエア横浜連絡口」よりエスカレーターを乗継ぎ「2F」へ | | |
| 電車で | 東京駅 | JR東海道線 | 25分 | 横浜駅 | | | | |
| 電車で | 品川駅 | 京浜急行:快速特急 | 15分 | 横浜駅 | タクシー<br>(東口ポルタ地下2Fより) | | | 7分 |
| 電車・新幹線で | | JR横浜線 3分 | 菊名駅 | 東急東横線 6分<br>(みなとみらい線直通 みなとみらい駅まで10分) | JR京浜東北線 3分 | 桜木町駅 | 徒歩 | 12分 |
| 電車・新幹線で | | JR横浜線<br>(京浜東北線経由東神奈川駅乗り換え) | | | 15分 | | バス | 7分 |
| 電車・新幹線で | | 横浜市営地下鉄 | | | 15分 | | タクシー | 5分 |
| 飛行機で | 成田空港 | JR成田エクスプレス | 90分 | 横浜駅 | みなとみらい線 3分<br>(東急東横線直通) | みなとみらい駅 | 徒歩 | 3分 |
| 飛行機で | 成田空港 | リムジンバス<br>(パシフィコ横浜行きは120分) | 90分 | 横浜駅 | | 「クイーンズスクエア横浜連絡口」よりエスカレーターを乗継ぎ「2F」へ | | |
| 飛行機で | 羽田空港 | 京浜急行 | 24分 | YCAT(横浜シティ・エア・ターミナル)(横浜駅東口) | | | | |
| 飛行機で | 羽田空港 | リムジンバス | 30分 | YCAT(横浜シティ・エア・ターミナル)(横浜駅東口) | タクシー<br>(東口 ポルタ地下2Fより) | | | 7分 |

パシフィコ横浜

## お車ご利用の場合

| | | | | |
|---|---|---|---|---|
| [東京方面より] 首都高速 | 横羽線 横浜公園方面 | ▸▸▸ | 横羽線みなとみらいランプ 約3分 | ▸▸▸ |
| | 湾岸線(鶴見つばさ橋、ベイブリッジ経由)横浜方面 | ▸▸▸ | 横羽線みなとみらいランプ 約3分 | ▸▸▸ |
| [関西・中部方面より] 東名高速横浜町田IC | 保土ヶ谷バイパス 狩場方面 約20分 ▸ | 狩場IC ▸ | 高速神奈川3号 狩場線横浜方面 約10分 ▸ | 横羽線みなとみらいランプ 約3分 ▸ |

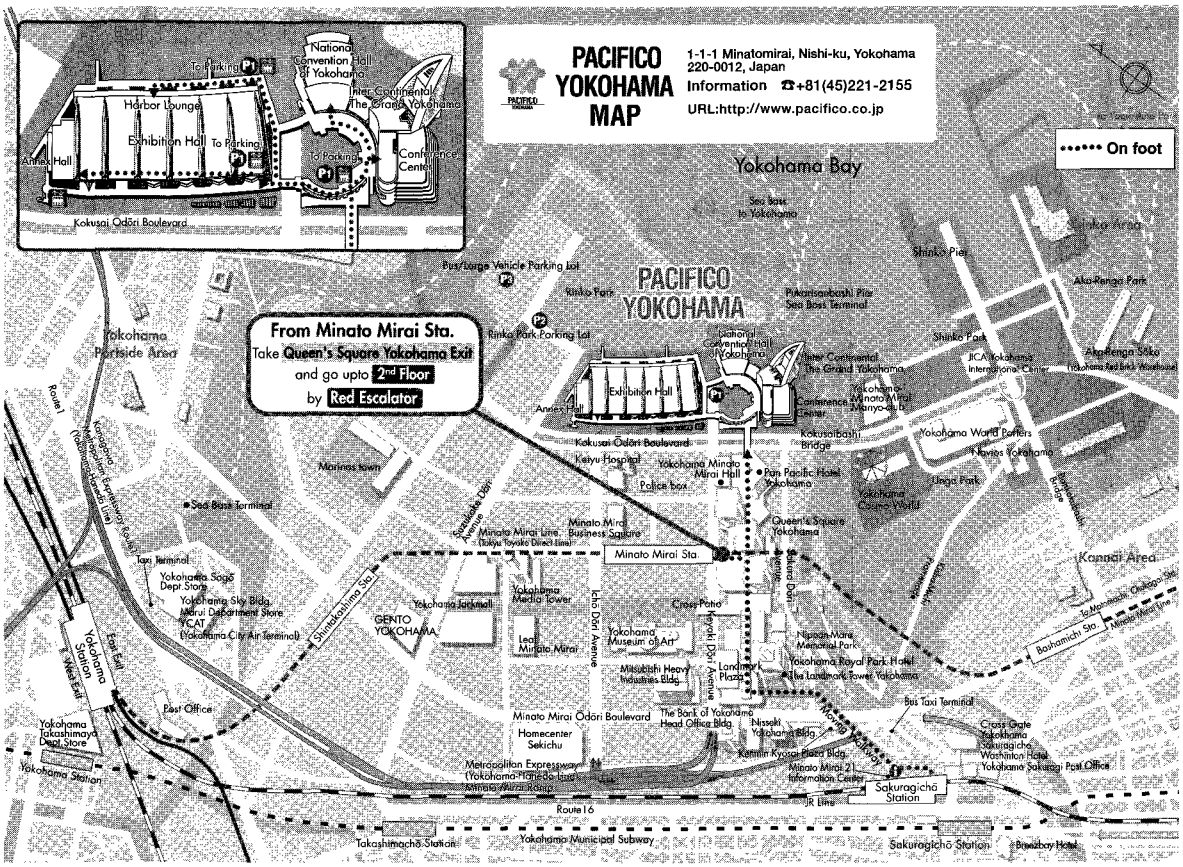
パシフィコ横浜

## 駐車場

| | | | | |
|---|---|---|---|---|
| P1 みなとみらい公共駐車場 ☎045-221-1301 | 1,200台(普通車) | 24時間利用可 | 料金(30分):280円 | *平日割引 8:00~23:00 最大1,300円 *深夜割引 0:00~6:00 半額 |
| P2 臨港パーク駐車場 ☎045-221-2175 | 100台(普通車) | 1 0:00~21:00 | 料金(1時間):500円 | |
| P3 バス・大型車駐車場 ☎045-221-1302 (お問い合せ 10:00~21:00) | 40台(バス・大型車) | 24時間利用可 (入出庫は7:00~22:00) | 料金(30分):500円 | *深夜割引 22:00~7:00 半額 |

再生紙を使用しています。 2006.8

# PACIFOCO YOKOHAMA VICINITY




## Traffic Information

**By Train**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Shibuya Sta. | Tokyu Toyoko Line : Limited Express (Direct link to Minato Mirai Line) | | Yokohama Sta. | 30min. | Minato Mirai Sta. | on foot | 3min. |
| Shinjuku Sta. | JR Shonan Shinjuku Line | 29min. | | Minato Mirai Line (Connecting from Tokyu Toyoko Line) | 3min. | Take "Queen's Square Yokohama Exit" and go upto "2nd Floor" by Red Escalator | |
| Tokyo Sta. | JR Tokaido Line | 25min. | | | | | |
| Shinagawa Sta. | Keikyu Express : Limited Express | 15min. | | by Taxi | | | 7min. |
| | JR Yokohama Line 3min. → Kikuna Sta. → Tokyu Toyoko Line 6min. (Connecting to Minato Mirai Line 10min. to Minato Mirai Sta.) | | | (From taxi pool at 2nd basement of Porta shopping mall on the east exit) | | | |
| | JR Yokohama Line (Change to Keihin-Tohoku Line at Higashikanagawa-Sta.) | 15min. | | JR Keihin-Tohoku Line | 3min. | on foot | 12min. |
| | Yokohama Subway | 15min. | | | | Sakuragicho Sta. by Bus | 7min. |
| | | | | | | by Taxi | 6min. |

**By Train & Shinkansen**

**By Air**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Narita Airport | JR Narita Express | 90min. | Yokohama Sta. YCAT Yokohama City Air Terminal Yokohama St. East | Minato Mirai Line (Connecting from Tokyu Toyoko Line) | 3min. | Minato Mirai Sta. Take "Queen's Square Yokohama Exit" and go upto "2nd Floor" by Red Escalator | on foot 3min. |
| | Airport Limousine Bus (120min. for PACIFICO YOKOHAMA) | 90min. | | | | | |
| Haneda Airport | Keikyu Express | 24min. | | | | | |
| | Airport Limousine Bus | 30min. | | by Taxi (From taxi pool at 2nd basement of Porta shopping mall on the east exit) | | | 7min. |

PACIFICO YOKOHAMA

## Driving To Pacifico

[From Tokyo]

| | | | |
|---|---|---|---|
| Metropolitan Expressway | Toward Yokohama Park, Yokohane Route | Minato Mirai Ramp,3min. | PACIFICO YOKOHAMA |
| | Toward Yokohama (over Bay bridge), Wangan Route | Minato Mirai Ramp,3min. | |

[From Kansai or Chubu]

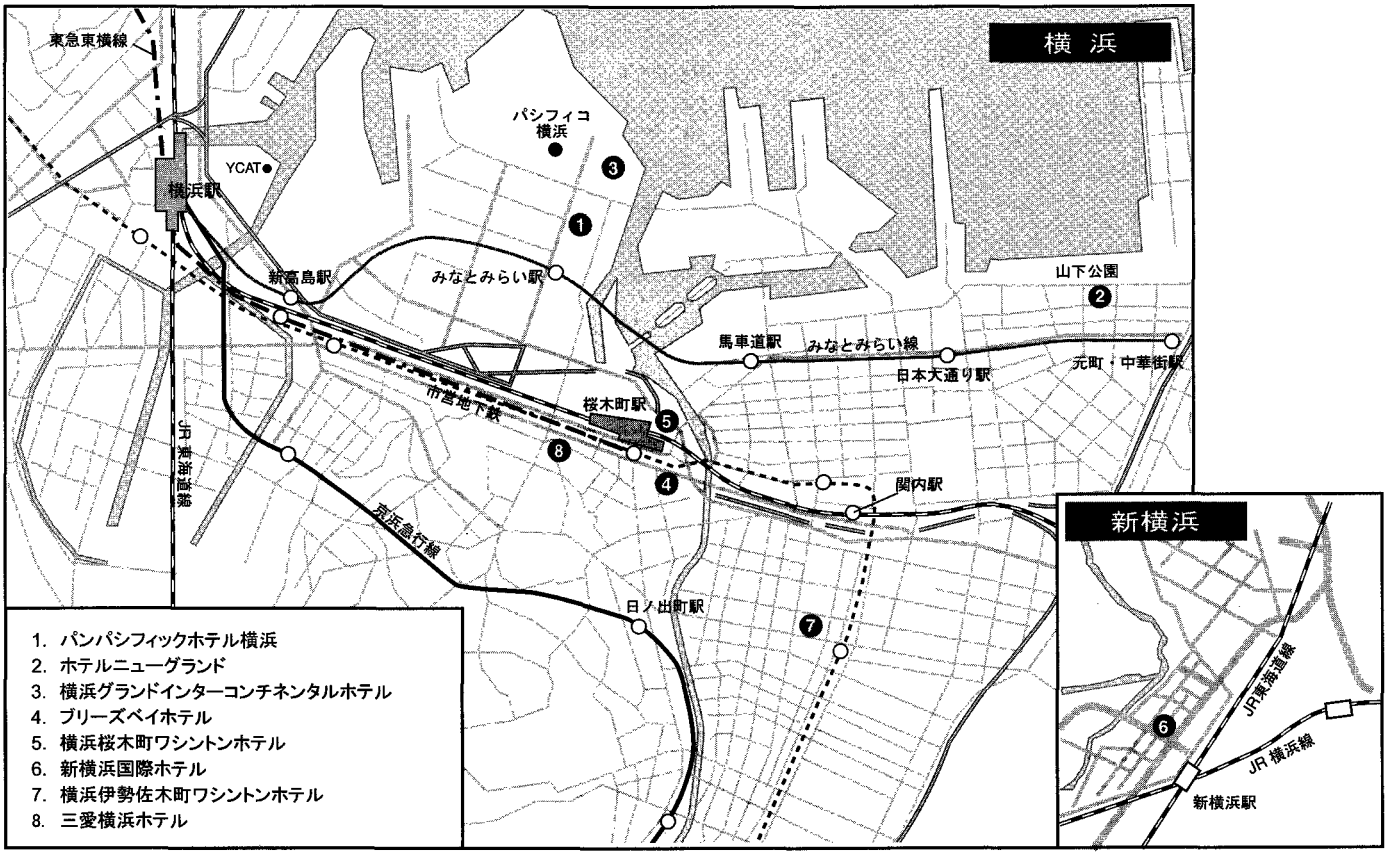
Tomei Expressway Yokohama Machida IC — Hodogaya Bypass,20min → Kariba IC → Toward Yokohama,Metropolitan Expressway,Kariba Route,10min → Minato Mirai Ramp,3min → PACIFICO YOKOHAMA

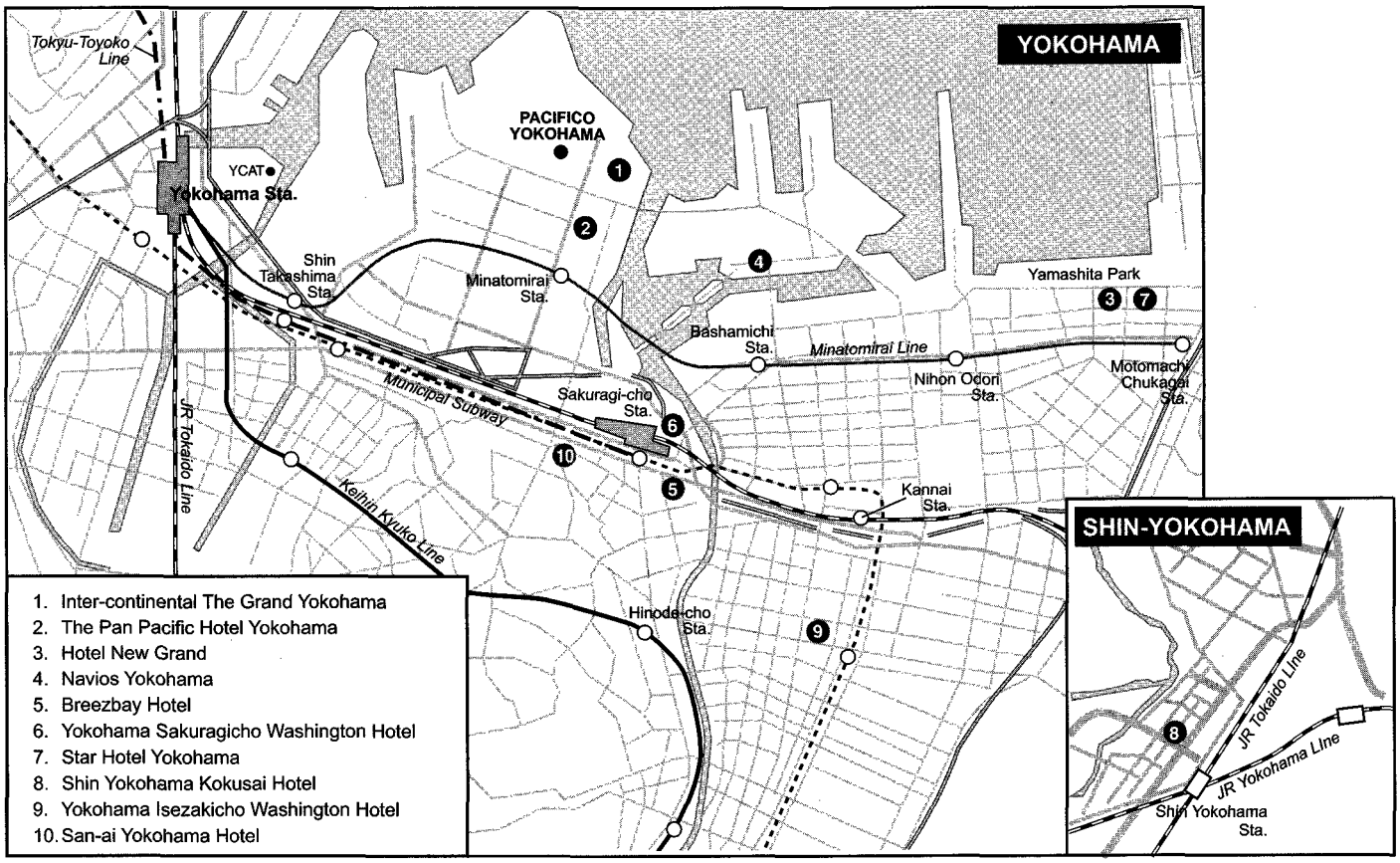
## Parking Lot

| | | | | |
|---|---|---|---|---|
| P1 Minato Mirai Public Parking Lot ☎045-221-1301 | Capacity: 1,200 (Standard-sized car) | Open 24 hours | Rates: ¥260/30min. | *Weekday Discount 8:00~23:00 ¥1,300 for max<br>*Late Night Discount 0:00~ 6:00 half-rate |
| P2 Rinko Park Parking Lot ☎045-221-2175 | Capacity: 100 (Standard-sized car) | 10:00~21:00 | Rates: ¥500/1hour | |
| P3 Bus/Large Vehicle Parking Lot ☎045-221-1302 | Capacity: 40 (Bus, Large Vehicle) | Open 24 hours (Loading and unloading: 7:00~22:00) | Rates: ¥500/30min. | *Late Night Discount 22:00~7:00 half-rate |

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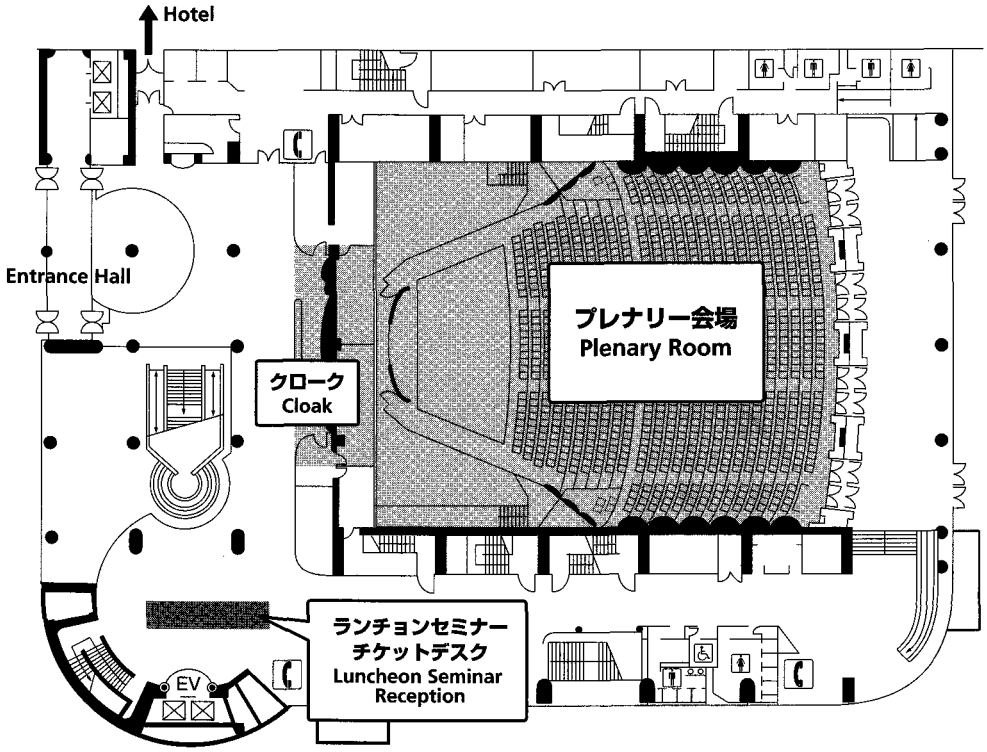
# 周辺ホテル案内　Hotel Map



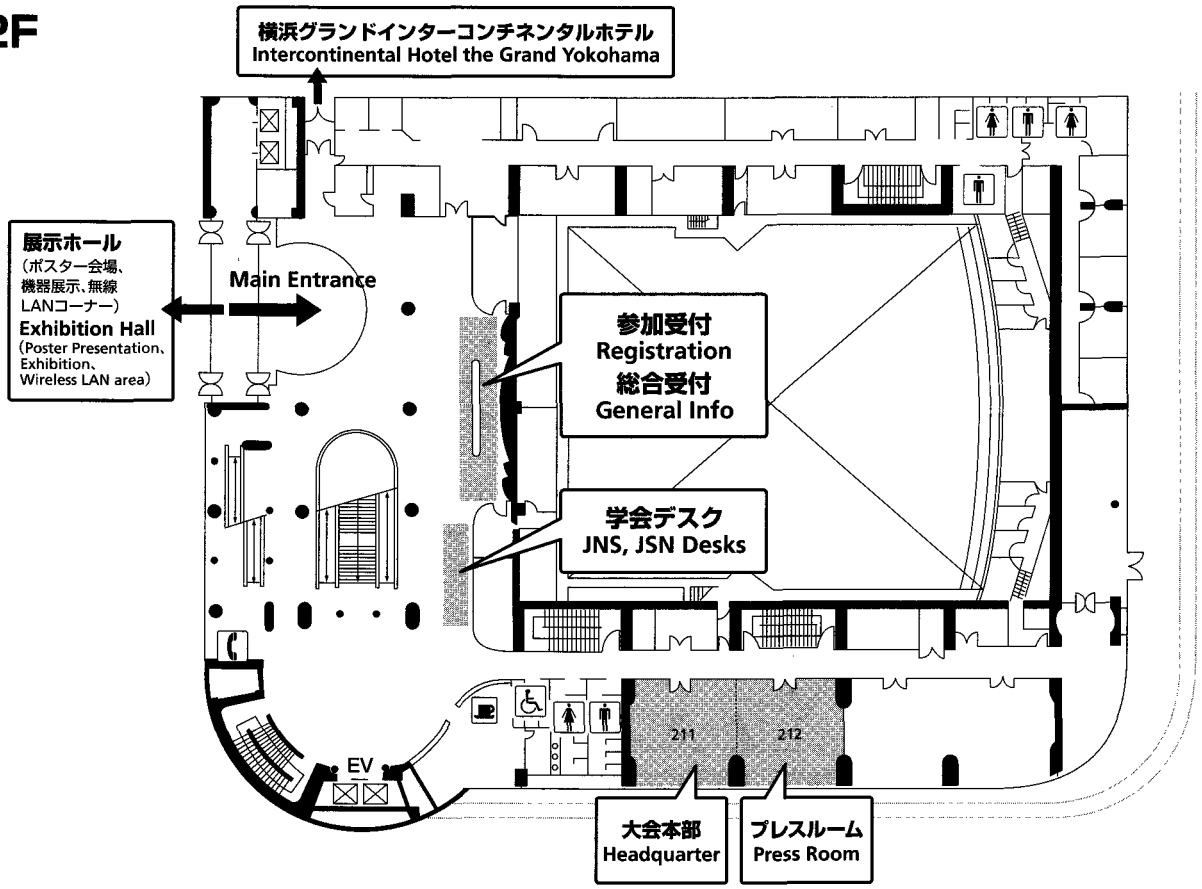



# 会場案内図 Floor Plans of Conference Venue

## 1F

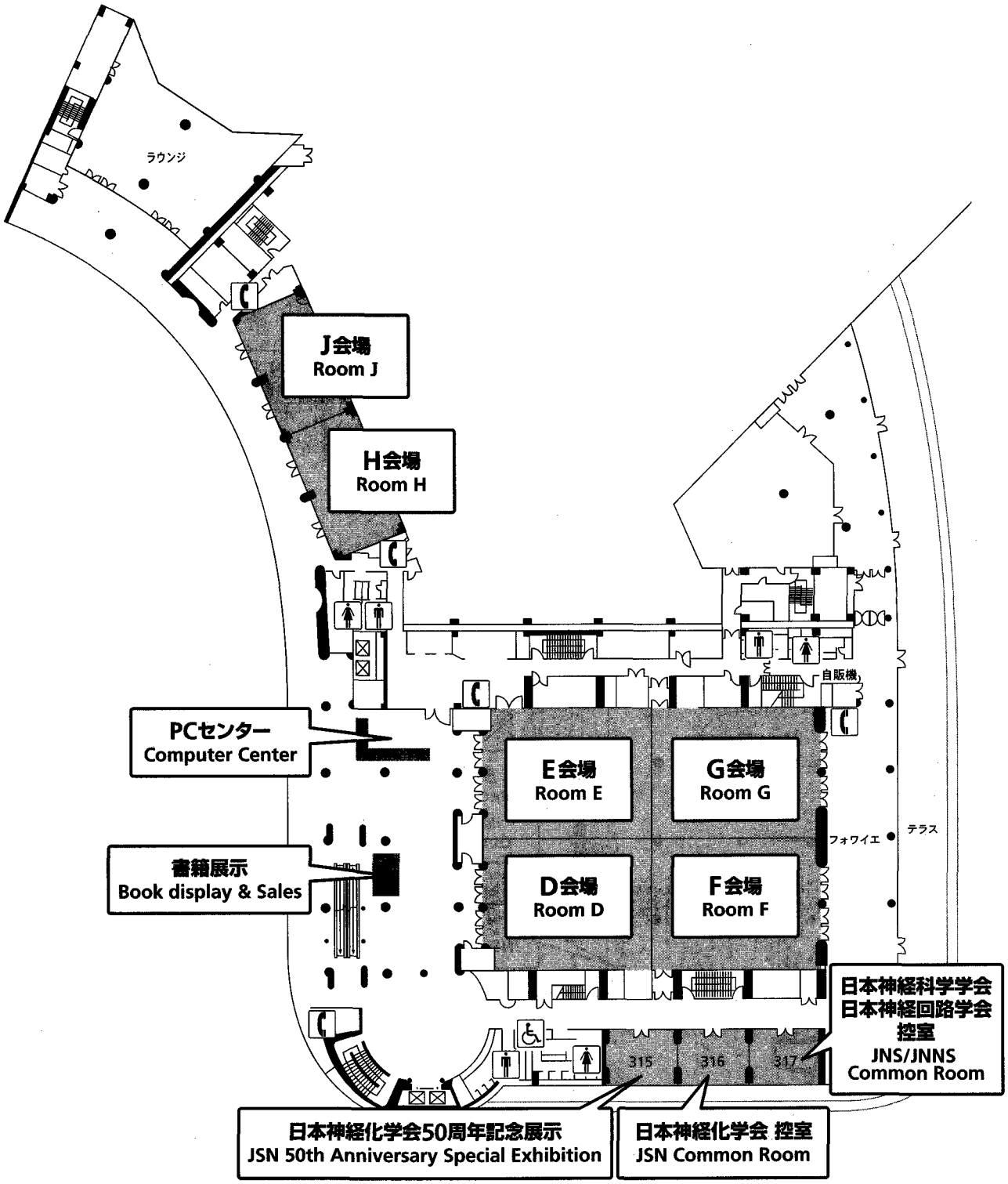



## 2F



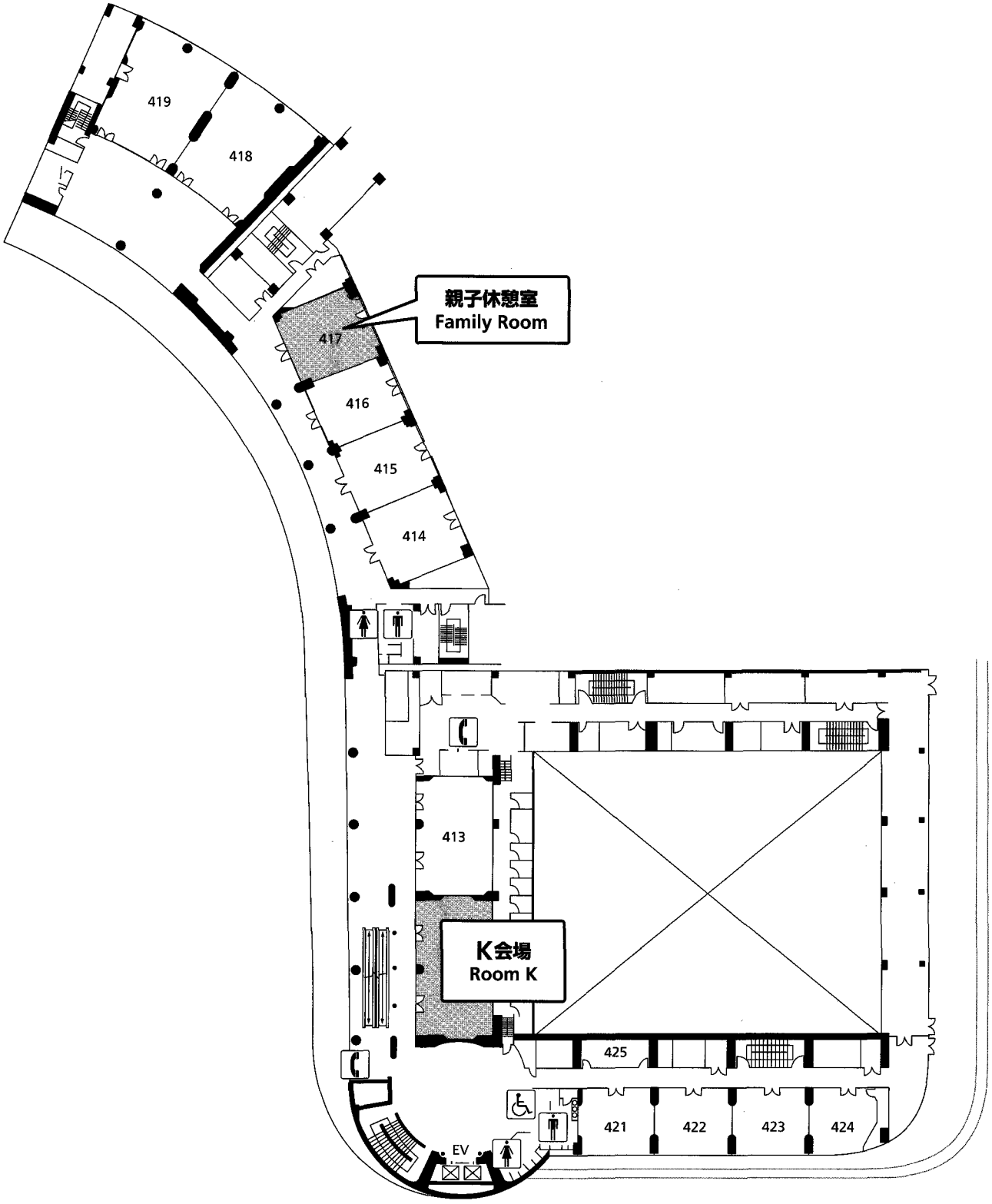
# 会場案内図　Floor Plans of Conference Venue

## 3F



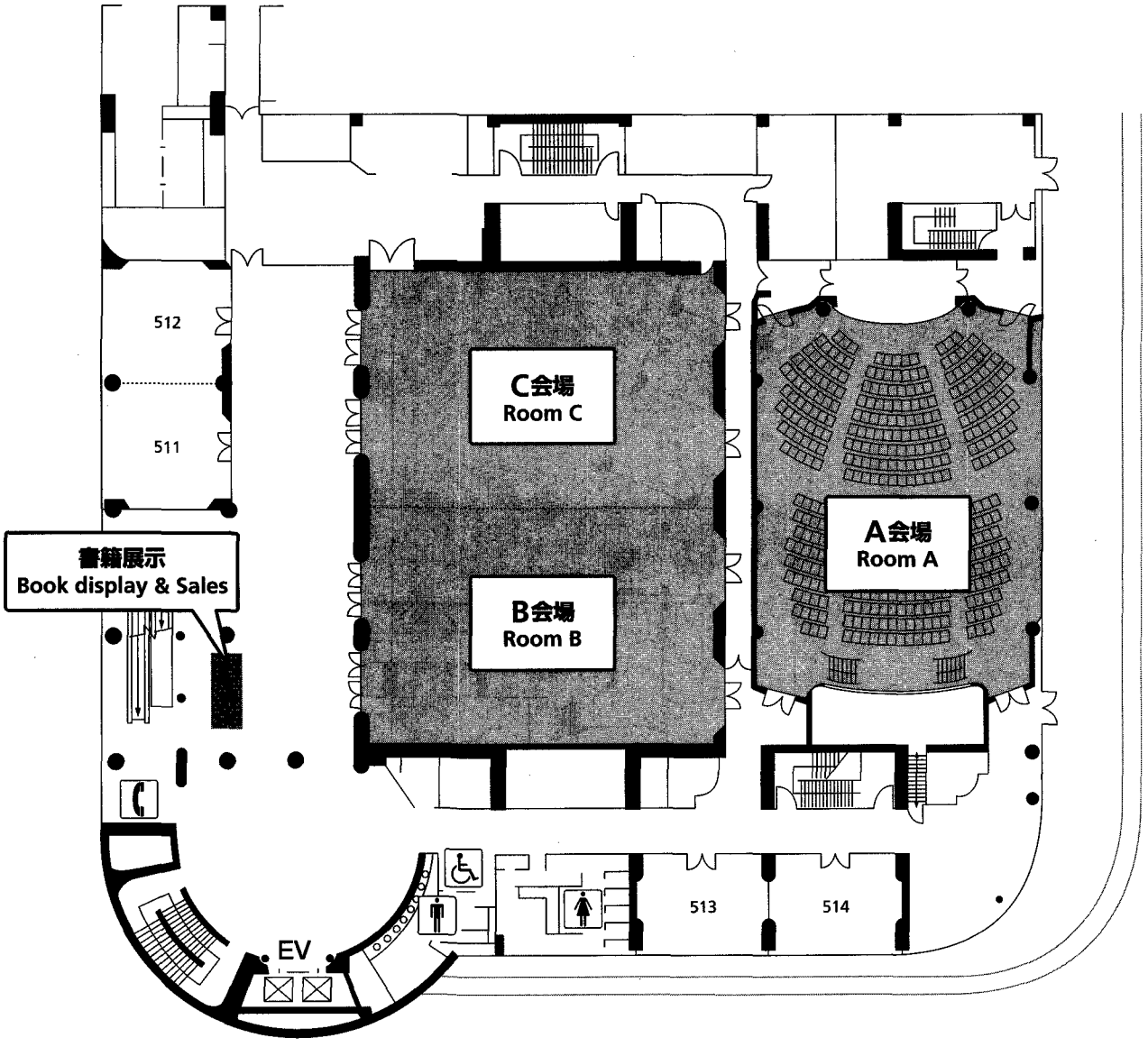
# 会場案内図 Floor Plans of Conference Venue

## 4F



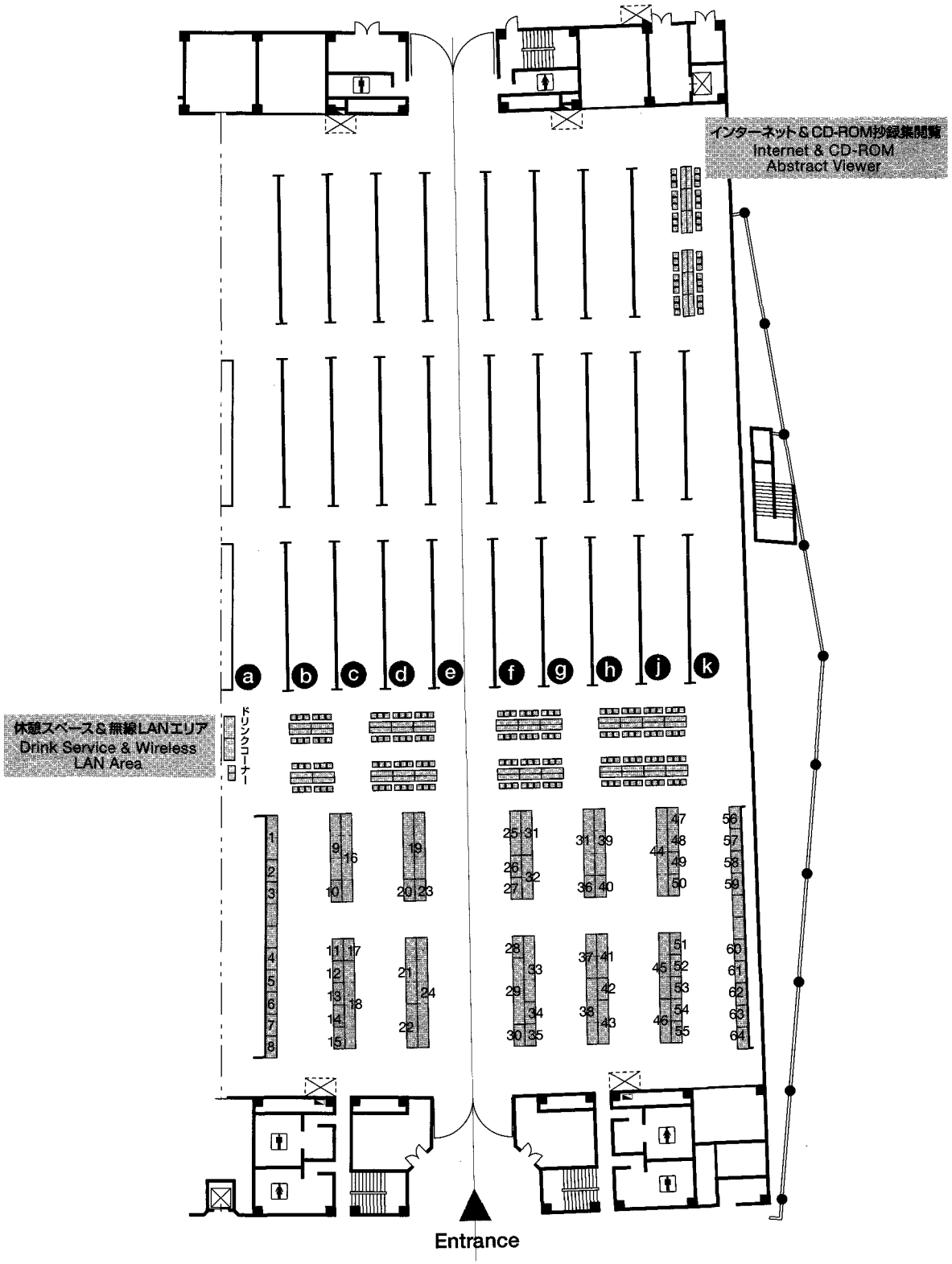
# 会場案内図　Floor Plans of Conference Venue

## 5F



# 会場案内図　Floor Plans of Conference Venue

## 展示ホール



# 展示案内

## 企業展示（小間番号順）

1 ナカライテスク株式会社
2 株式会社日伸理化
3 ブレインビジョン株式会社
4 日本バイナリー株式会社
5 三栄バイタルズ株式会社
6 有限会社シンテクノ
7 日本光電工業株式会社
8 エム・シー・メディカル株式会社
9 プライムテック株式会社
10 日本ミリポア株式会社
11 アドバンストシステムズ株式会社
12 ネッパジーン株式会社
13 フィッシャーバイオサイエンスジャパン株式会社
14 株式会社イーオス
15 ジョンソン・エンド・ジョンソン株式会社
16 カール ツァィス マイクロイメージング株式会社
17 住友ベークライト株式会社
18 ショーシン EM 株式会社
19 小原医科産業株式会社
20 株式会社ソルトン
21 株式会社インターメディカル
22 株式会社ニューロサイエンス
23 トミーデジタルバイオロジー株式会社
24 オリンパス株式会社
25 独立行政法人理化学研究所 脳科学総合研究センター
26 独立行政法人理化学研究所 ゲノム科学研究センター
27 独立行政法人理化学研究所 バイオリソースセンター
28 ワールドプレシジョンインスツルメンツ
29 株式会社フィジオテック
30 ハーバードアパレイタス
31 株式会社成茂科学器械研究所
32 メイワフォーシス株式会社
33 株式会社ニコンインステック
34 株式会社ミユキ技研
35 ログ・リサーチ
36 ユニーテック株式会社
37 アルファメッドサイエンス株式会社
38 株式会社グライナージャパン
39 室町機械株式会社
40 オメガウェーブ株式会社
41 株式会社ブレインサイエンス・イデア
42 株式会社ソフィア・サイエンティフィック
43 シグマ アルドリッチジャパン株式会社
44 バイオリサーチセンター株式会社
45 株式会社ユニークメディカル
46 株式会社キーエンス
47 ベックマン・コールター株式会社
48 株式会社ベリタス
49 株式会社東海ヒット
50 株式会社エイコム
51 ライカマイクロシステムズ株式会社
52 マイクロブライトフィールドジャパン株式会社
53 株式会社モンテシステム
54 日本チャールス・リバー株式会社
55 STEM バイオメソッド株式会社
56 住商ファーマインターナショナル株式会社
57 ミルテニーバイオテク株式会社
58 株式会社医薬分子設計研究所
59 堂阪イーエム株式会社
60 バリアン テクノロジーズ ジャパン リミテッド
61 アフィメトリクス・ジャパン株式会社
62 株式会社クレアクト・インターナショナル
63 株式会社コーナン・メディカル
64 株式会社島津製作所

## 書籍展示・販売（50 音順）

株式会社医学恵文堂
株式会社ウォルターズ・クルワー・ヘルス・ジャパン
大井書店
オックスフォード大学出版局株式会社
株式会社ガリバー静岡店
株式会社クバプロ（NPO 脳の世紀推進会議）
サイエンス
株式会社星和書店
株式会社ニホン・ミック
株式会社文進堂書店
丸善株式会社
ワイリー ブラックウエル

# 大会日程概要　Program Summary

### ◆プレナリーレクチャー　Plenary Lectures：プレナリー会場（Plenary Room）

9月10日(月)　September 10(Mon)　18:15～19:15
9月11日(火)　September 11(Tue)　18:15～19:15
9月12日(水)　September 12(Wed)　14:00～15:00

### ◆時実賞・塚原賞　受賞講演　Tokizane, Tsukahara Award Lectures：A会場（Room A）

9月10日(月)　September 10(Mon)　14:00～14:40　(Tokizane Award Lecture, SL1)
14:40～15:20　(Tsukahara Award Lecture, SL2)
15:20～16:00　(Tsukahara Award Lecture, SL3)

### ◆日本神経化学会若手奨励賞シンポジウム（JSN Young Scientists Lectureship Award Symposium）：A会場（Room A）

9月11日(火)　September 11(Tue)　14:00～16:00

### ◆シンポジウム Symposia：A会場～K会場（Room A～Room K）

9月10日(月)　September 10(Mon)　8:30～11:00
9月11日(火)　September 11(Tue)　8:30～11:00
9月12日(水)　September 12(Wed)　8:30～11:00
15:15～17:45

### ◆一般口演 Oral Sessions：A会場～K会場（Room A～Room K）

9月10日(月)　September 10(Mon)　14:00～18:00
9月11日(火)　September 11(Tue)　14:00～18:00

### ◆ポスター発表 Poster Sessions：展示ホール（Exhibition Hall）

<table>
<tr><th></th><th>掲示時間<br>Display Time</th><th>演題番号<br>Abst. No.</th><th>コアタイム<br>Core Time</th><th>撤去時間<br>Removal</th></tr>
<tr><td rowspan="2">9月10日(月)<br>Sept. 10(Mon)</td><td rowspan="2">9:00～17:00</td><td>奇数 Odd</td><td>11:00～12:00</td><td rowspan="6">17:00～17:30</td></tr>
<tr><td>偶数 Even</td><td>13:00～14:00</td></tr>
<tr><td rowspan="2">9月11日(火)<br>Sept. 11(Tue)</td><td rowspan="2">9:00～17:00</td><td>奇数 Odd</td><td>11:00～12:00</td></tr>
<tr><td>偶数 Even</td><td>13:00～14:00</td></tr>
<tr><td rowspan="2">9月12日(水)<br>Sept .12(Wed)</td><td rowspan="2">9:00～17:00</td><td>奇数 Odd</td><td>11:00～12:00</td></tr>
<tr><td>偶数 Even</td><td>13:00～14:00</td></tr>
</table>

# 大会日程概要　Program Summary

**◆ランチョンセミナー　Luncheon Seminars：A 会場～ H 会場（Room A ～ Room H）**

9月10日(月)　September 10(Mon)　12:00～12:50
9月11日(火)　September 11(Tue)　12:00～12:50
9月12日(水)　September 12(Wed)　12:00～12:50

**◆機器展示　Exhibition：展示ホール（Exhibition Hall）**

9月10日(月)　September 10(Mon)　9:00～17:00
9月11日(火)　September 11(Tue)　9:00～17:00
9月12日(水)　September 12(Wed)　9:00～17:00

**◆総会等**

日本神経科学学会総会(JNS Members' Business Meeting)：K会場(Room K)
9月10日(月)　September 10(Mon)　13:00～14:00
日本神経化学会評議員会(JSN Board Meeting)：A会場(Room A)
9月11日(火)　September 11(Tue)　11:30～12:00
日本神経化学会総会(JSN Members' Business Meeting)：A会場(Room A)
9月11日(火)　September 11(Tue)　12:00～13:00

**◆懇親会（NeuroSocial）インターコンチネンタルホテル３階「ボールルーム」**
**(Intercontinental Hotel the Yokohama Grand, 3F Ball Room)**

9月11日(火)　September 11(Tue)　19:30～

# 演題番号の見方　How to read Abstract numbers

## プレナリーレクチャー
Plenary Lectures

**PL2**

- 2: 発表日 Day

## 小澤賞、塚原賞
Ozawa Award Lecture, Tsukahara Award Lectures

**SL3**

- 3: 発表順 Order

## 日本神経化学会　若手奨励賞シンポジウム
JSN Young Scientists Lectureship Award Symposium

**NCS2P-A1**

- 2: 発表日 Day
- P: 午前・午後 am, pm
- A: 会場番号 Room
- 1: 発表順 Order

## シンポジウム
Symposia

**S1A-J2**

- 1: 発表日 Day
- A: 午前・午後 am, pm
- J: 会場番号 Room
- 2: 発表順 Order

## 一般口演
Oral Sessions

**O2P-K13**

- 2: 発表日 Day
- P: 午前・午後 am, pm
- K: 会場番号 Room
- 13: 発表順 Order

## ポスター
Poster Sessions

**P3-b17**

- 3: 発表日 Day
- b: 掲示板 Poster Panel
- 17: ポスター番号 Poster No.

## ランチョンセミナー
Luncheon Seminars

**LS1-C**

- 1: 発表日 Day
- C: 会場番号 Room

# 大会日程表

## ■ 大会第1日目　9月10日(月)

| 会場 | プレナリー会場 (メインホール) | A会場 (小ホール) | B会場 (501) | C会場 (502) | D会場 (301) | E会場 (302) | F会場 (303) | G会場 (304) | H会場 (311+312) | J会場 (313+314) | K会場 (411+412) | ポスター会場 (展示ホール) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 8:00 | | | | | | | | | | | | |
| 8:30 | | シンポジウム S1A-A<br>興奮—抑制バランス：シナプス・回路・システム<br>座長：Takao Hensch, Arthur Konnerth<br>P.115 | シンポジウム S1A-B<br>霊長類モデル研究は神経科学に何を寄与するか？<br>座長：Kathleen S. Rockland, 船橋新太郎<br>P.115 | シンポジウム S1A-C<br>社会的認知と意思決定：神経心理学者と神経科学者のクロストーク<br>座長：河村 満, 坂上雅道<br>P.116 | シンポジウム S1A-D<br>シナプス形成と機能獲得<br>座長：柚崎通介, 岡部繁男<br>P.116 | シンポジウム S1A-E<br>神経細胞死の制御の新展開<br>座長：三浦正幸<br>P.117 | シンポジウム S1A-F<br>エピジェネティクス異常に起因する精神発達障害疾患の理解<br>座長：久保田健夫, 伏木 信次<br>P.118 | シンポジウム S1A-G<br>扁桃体を中心とする情動系の形成と機能の制御メカニズム<br>座長：湯浅茂樹<br>P.118 | シンポジウム S1A-H<br>ニューロインフォマティクスの国際展開<br>座長：臼井支朗, 大森隆司<br>P.119 | シンポジウム S1A-J<br>親子関係の神経基盤<br>座長：小川園子, 菊水健史<br>P.119 | シンポジウム S1A-K<br>Notch シグナルと中枢神経系発生<br>座長：見学美根子, 後藤由季子<br>P.120 | 8:30 貼付<br>9:00 | 企業展示 |
| 9:00 | | | | | | | | | | | | |
| 10:00 | | | | | | | | | | | | |
| 11:00 | | | | | | | | | | | | 11:00 奇数コア |
| 12:00 | | 12:00 ランチョンセミナー LS1-A 成茂科学器械研究所 P.141 | ランチョンセミナー LS1-B エーザイ ファイザー P.141 | ランチョンセミナー LS1-C 大塚製薬 P.141 | ランチョンセミナー LS1-D 室町機械 Neuralynx P.142 | | ランチョンセミナー LS1-F カールツァイス マイクロイメージング P.142 | | | | | 12:00 |
| 13:00 | | 12:50 | | | | | | | | | 科学総会 | 13:00 偶数コア |
| 14:00 | | 14:00 時実賞 SL1 西道隆臣 P.114<br>14:40 塚原賞 SL2 銅谷賢治 P.114<br>15:20 塚原賞 SL3 宮脇敦史 P.114 | 口演 01P-B01 ～ 04 睡眠・生体リズム P.150<br>15:00 口演 01P-B05 ～ 08 体性運動 1 P.150<br>16:00 口演 01P-B09 ～ 12 体性運動 2 P.151<br>17:00 口演 01P-B13 ～ 16 体性運動 3 P.152 | 口演 01P-C01 ～ 04 眼球運動 P.152<br>口演 01P-C05 ～ 08 視覚 1 P.153<br>口演 01P-C09 ～ 12 視覚 2 P.153<br>口演 01P-C13 ～ 16 視覚 3 P.154 | 口演 01P-D01 ～ 04 回路網形成 1 P.154<br>口演 01P-D05 ～ 08 回路網形成 2 P.155<br>口演 01P-D09 ～ 12 回路網形成 3 P.156<br>口演 01P-D13 ～ 16 回路網形成 4 P.156 | 口演 01P-E01 ～ 04 イオンチャンネル 1 P.157<br>口演 01P-E05 ～ 08 イオンチャンネル 2 P.157<br>口演 01P-E09 ～ 12 イオンチャンネル 3 P.158<br>口演 01P-E13 ～ 16 イオンチャンネル 4 (J) P.158 | 口演 01P-F01 ～ 04 シナプス 1 P.159<br>口演 01P-F05 ～ 08 シナプス 2 P.160<br>口演 01P-F09 ～ 12 シナプス 3 P.160<br>口演 01P-F13 ～ 16 シナプス 4 P.161 | 口演 01P-G01 ～ 04 学習記憶 1 P.161<br>口演 01P-G05 ～ 08 学習記憶 2 P.162<br>口演 01P-G09 ～ 12 学習記憶 3 P.162<br>口演 01P-G13 ～ 16 言語とコミュニケーション P.163 | 口演 01P-H01 ～ 03 神経細胞死 P.164<br>口演 01P-H04 ～ 07 グリア 1 (J) P.164<br>口演 01P-H08 ～ 10 グリア 2 P.165<br>口演 01P-H11 ～ 14 グリア 3 P.165 | 口演 01P-J01 ～ 04 ポリグルタミン病、ALS、その他 P.166<br>口演 01P-J05 ～ 08 発達障害 P.166<br>口演 01P-J09 ～ 14 モデル動物 (J) P.167 | 口演 01P-K01 ～ 04 情報伝達 1 P.168<br>口演 01P-K05 ～ 08 情報伝達 2 P.169<br>口演 01P-K09 ～ 11 虚血 P.169 | 14:00 |
| 15:00 | | | | | | | | | | | | |
| 16:00 | | 16:00 口演 01P-A01 ～ 04 幹細胞 1 P.149 | | | | | | | | | | |
| 17:00 | | 17:00 口演 01P-A05 ～ 08 幹細胞 2 P.149 | | | | | | | | 17:30 | | 17:00 撤去<br>17:30 |
| 18:00 | 17:45 化学会 50 周年記念式典 | 18:00 | | | | | | | | | | |
| 19:00 | 18:15 プレナリーレクチャー PL1 Heinrich Betz P.113 | | | | | | | | | | | |
| 20:00 | 19:15 | | | | | | | | | | | |

(J)は日本語セッション

# Program at a Glance

## ■ Day 1 September 10 (Mon)

| 会場 | Plenary Room (Main Hall) | Room A (Small Auditorium) | Room B (501) | Room C (502) | Room D (301) | Room E (302) | Room F (303) | Room G (304) | Room H (311+312) | Room J (313+314) | Room K (411+412) | Poster Room (Exhibition Hall) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 8:00 | | | | | | | | | | | | |
| 8:30 | | Symposium S1A-A<br>Excitatory-Inhibitory Balance: Synapses, Circuits, Systems<br>Chair: T. Hensch, A. Konnerth<br>P.115 | Symposium S1A-B<br>What is Contributed by the Primate Model?<br>Chair: K. S. Rockland, S. Funahashi<br>P.115 | Symposium S1A-C<br>Social Cognition and Decision-making: Cross-talk between Neuropsychologists and Neuroscientists<br>Chair: M. Kawamura, M. Sakagami<br>P.116 | Symposium S1A-D<br>Synapse Formation and Functional Maturation<br>Chair: M. Yuzaki, S. Okabe<br>P.116 | Symposium S1A-E<br>Recent Advance of Regulatory Mechanisms of Neuronal Cell Death<br>Chair: M. Miura<br>P.117 | Symposium S1A-F<br>Understanding of Defects in Epigenetics in Neurodevelopmental Diseases<br>Chair: T. Kubota, S. Fushiki<br>P.118 | Symposium S1A-G<br>Amygdala and Related Emotional System-control Mechanisms of their Development and Functions<br>Chair: S. Yuasa<br>P.118 | Symposium S1A-H<br>International Collaborations on Neuro-informatics<br>Chair: S. Usui, T. Omori<br>P.119 | Symposium S1A-J<br>Neural Basis of Parent-Infant Relationship<br>Chair: S. Ogawa, T. Kikusui<br>P.119 | Symposium S1A-K<br>Notch Signaling in the Developing Central Nervous System<br>Chair: M. Kengaku, Y. Gotoh<br>P.120 | 8:30 Installation |
| 9:00 | | | | | | | | | | | | 9:00 |
| 10:00 | | | | | | | | | | | | |
| 11:00 | | 11:00 | | | | | | | | | | 11:00 Odd Core |
| 12:00 | | 12:00<br>Luncheon Seminar LS1-A<br>NARISHIGE SCIENTIFIC INSTRUMENT LAB.<br>P.141 | Luncheon Seminar LS1-B<br>Eisai<br>Pfizer Japan<br>P.141 | Luncheon Seminar LS1-C<br>Otsuka Pharmaceutical<br>P.141 | Luncheon Seminar LS1-D<br>MUROMACHI KIKAI<br>P.142 | | Luncheon Seminar LS1-F<br>Carl Zeiss MicroImaging<br>P.142 | | | | | 12:00 |
| 13:00 | | 12:50 | | | | | | | | | JNS Members' Business Meeting | 13:00 Even Core |
| 14:00 | | 14:00<br>Tokizane Prize SL1<br>T. Saido<br>P.114 | Oral Session O1P-B01 ~ 04<br>Sleep and Biological Rhythms<br>P.150 | Oral Session O1P-C01 ~ 04<br>Oculomotor System<br>P.152 | Oral Session O1P-D01 ~ 04<br>Network Formation 1<br>P.154 | Oral Session O1P-E01 ~ 04<br>Ion Channels 1<br>P.157 | Oral Session O1P-F01 ~ 04<br>Synapse 1<br>P.159 | Oral Session O1P-G01 ~ 04<br>Learning and Memory 1<br>P.161 | Oral Session O1P-H01 ~ 03<br>Neuronal Death<br>P.164 | Oral Session O1P-J01~04<br>Polyglutamine Diseases, ALS, Other<br>P.166 | | 14:00 |
| 15:00 | | 14:40<br>Tsukahara Prize SL2<br>K. Doya<br>P.114 | Oral Session O1P-B05 ~ 08<br>Somatomotor System 1<br>P.150 | Oral Session O1P-C05 ~ 08<br>Visual System 1<br>P.153 | Oral Session O1P-D05 ~ 08<br>Network Formation 2<br>P.155 | Oral Session O1P-E05 ~ 08<br>Ion Channels 2<br>P.157 | Oral Session O1P-F05 ~ 08<br>Synapse 2<br>P.160 | Oral Session O1P-G05 ~ 08<br>Learning and Memory 2<br>P.162 | Oral Session O1P-H04 ~ 07<br>Glia 1 (J)<br>P.164 | Oral Session O1P-J05 ~ 08<br>Developmental Disorders<br>P.166 | Oral Session O1P-K01 ~ 04<br>Signal Transduction 1<br>P.168 | |
| 16:00 | | 15:20<br>Tsukahara Prize SL3<br>A. Miyawaki<br>P.114 | | | | | | | | | | |
| 17:00 | | 16:00<br>Oral Session O1P-A01 ~ 04<br>Stem Cells 1<br>P.149 | Oral Session O1P-B09 ~ 12<br>Somatomotor System 2<br>P.151 | Oral Session O1P-C09 ~ 12<br>Visual System 2<br>P.153 | Oral Session O1P-D09 ~ 12<br>Network Formation 3<br>P.156 | Oral Session O1P-E09 ~ 12<br>Ion Channels 3<br>P.158 | Oral Session O1P-F09 ~ 12<br>Synapse 3<br>P.160 | Oral Session O1P-G09 ~ 12<br>Learning and Memory 3<br>P.162 | Oral Session O1P-H08 ~ 10<br>Glia 2<br>P.165 | Oral Session O1P-J09 ~ 14<br>Animal Models (J)<br>P.167 | Oral Session O1P-K05 ~ 08<br>Signal Transduction 2<br>P.169 | 17:00 Removal |
| 18:00 | 17:45 JSN 50th Anniversary Event | 17:00<br>Oral Session O1P-A05 ~ 08<br>Stem Cells 2<br>P.149 | Oral Session O1P-B13 ~ 16<br>Somatomotor System 3<br>P.152 | Oral Session O1P-C13 ~ 16<br>Visual System 3<br>P.154 | Oral Session O1P-D13 ~ 16<br>Network Formation 4<br>P.156 | Oral Session O1P-E13 ~ 16<br>Ion Channels 4 (J)<br>P.158 | Oral Session O1P-F13 ~ 16<br>Synapse 4<br>P.161 | Oral Session O1P-G13 ~ 16<br>Language and Communication<br>P.163 | Oral Session O1P-H11 ~ 14<br>Glia 3<br>P.165 | 17:30 | Oral Session O1P-K09 ~ 11<br>Ischemia<br>P.169 | 17:30 |
| 19:00 | 18:15 Plenary Lecture PL1<br>Heinrich Betz<br>P.113 | 18:00 | | | | | | | | | | |
| 20:00 | 19:15 | | | | | | | | | | | |

Exhibition

(J) Japanese Sessions

# 大会日程表

## ■ 大会第2日目　9月11日(火)

| 会場 | プレナリー会場 (メインホール) | A会場 (小ホール) | B会場 (501) | C会場 (502) | D会場 (301) | E会場 (302) | F会場 (303) | G会場 (304) | H会場 (311+312) | J会場 (313+314) | K会場 (411+412) | ポスター会場 (展示ホール) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 8:00 | | | | | | | | | | | | |
| 8:30 | | シンポジウム S2A-A<br>シナプスにおける情報伝達制御<br>座長：持田澄子<br>P.121 | シンポジウム S2A-B<br>神経活動とスパイン形態：アクチンダイナミズムとその制御因子<br>座長：白尾智明　森　望<br>P.121 | シンポジウム S2A-C<br>RNA 制御と神経疾患<br>座長：塩見春彦　岡野ジェイムス洋尚<br>P.122 | シンポジウム S2A-D<br>2光子励起法による in vivo 観察の最前線<br>座長：鍋倉淳一　河西春郎<br>P.123 | シンポジウム S2A-E<br>海馬ネットワークの最前線ー動的再構成とシナプス可塑性<br>座長：神谷温之　八尾　寛<br>P.123 | シンポジウム S2A-F<br>霊長類の社会関係認知<br>座長：中村克樹<br>P.124 | シンポジウム S2A-G<br>ミトコンドリア機能異常としての精神・神経疾患<br>座長：後藤雄一<br>P.125 | シンポジウム S2A-H<br>情動・痛み・本能・自律機能を結ぶ神経メカニズム<br>座長：加藤総夫　松井秀樹<br>P.125 | シンポジウム S2A-J<br>神経変性疾患における神経細胞機能障害<br>座長：岡澤　均　Steven Finkbeiner<br>P.126 | シンポジウム S2A-K<br>新世代神経行動学のストラテジー<br>座長：小川宏人　岡浩太郎<br>P.126 | 8:30 貼付 |
| 9:00 | | | | | | | | | | | | 9:00 |
| 10:00 | | | | | | | | | | | | |
| 11:00 | | 11:00 | | | | | | | | | | 11:00 奇数コア |
| 12:00 | | 11:30 化学評議員会<br>12:00 化学総会 | | | ランチョンセミナー LS2-D<br>ヤンセンファーマ<br>P.142 | ランチョンセミナー LS2-E<br>オリンパス<br>P.143 | ランチョンセミナー LS2-F<br>ライカマイクロシステムズ<br>P.143 | ランチョンセミナー LS2-G<br>田辺製薬<br>P.143 | ランチョンセミナー LS2-H<br>プレクソンフィジオテック<br>P.143 | | | 12:00 |
| 13:00 | | 13:00 | | | 12:50 | | | | | | | 13:00 偶数コア |
| 14:00 | | 14:00<br>日本神経化学会若手奨励賞シンポジウム<br>座長：津田正明　田代朋子<br>P.140 | 口演 O2P-B01 ～ 04<br>シナプス伝達 (J)<br>P.171<br>15:00<br>口演 O2P-B05 ～ 08<br>シナプス可塑性 1<br>P.171 | 口演 O2P-C01 ～ 04<br>視覚 4<br>P.173<br>口演 O2P-C05 ～ 08<br>視覚 5<br>P.173 | 口演 O2P-D01 ～ 04<br>幹細胞 3<br>P.175<br>口演 O2P-D05 ～ 07<br>幹細胞 4<br>P.176 | 口演 O2P-E01 ～ 04<br>パーキンソン病 1<br>P.177<br>口演 O2P-E05 ～ 07<br>パーキンソン病 2 (J)<br>P.178 | 口演 O2P-F01 ～ 04<br>認知 1<br>P.179<br>口演 O2P-F05 ～ 08<br>認知 2<br>P.180 | 口演 O2P-G01 ～ 04<br>脳イメージング<br>P.181<br>口演 O2P-G05 ～ 08<br>細胞イメージング<br>P.182 | 口演 O2P-H01 ～ 04<br>精神障害 (J)<br>P.184<br>口演 O2P-H05 ～07<br>統合失調症<br>P.184 | 口演 O2P-J01 ～ 04<br>遺伝子制御<br>P.186<br>口演 O2P-J05 ～ 08<br>化学感覚 1<br>P.186 | 口演 O2P-K01 ～ 04<br>方法<br>P.188<br>口演 O2P-K05 ～ 08<br>感覚運動系の可塑性<br>P.189 | 14:00 |
| 16:00 | | 16:00<br>口演 O2P-A01 ～ 03<br>回路網形成 5<br>P.170 | 口演 O2P-B09 ～ 12<br>シナプス可塑性 2<br>P.172 | 口演 O2P-C09 ～ 12<br>視覚 6<br>P.174 | 口演 O2P-D08 ～ 11<br>神経新生<br>P.176 | 口演 O2P-E08 ～ 13<br>大脳基底核<br>P.178 | 口演 O2P-F09 ～ 12<br>アルツハイマー病 1(J)<br>P.180 | 口演 O2P-G09 ～ 12<br>社会的行動 1<br>P.183 | 口演 O2P-H08 ～ 11<br>依存・乱用<br>P.185 | 口演 O2P-J09 ～ 12<br>化学感覚 2<br>P.187 | 口演 O2P-K09～12<br>ブレインマシン / コンピュータ・インターフェイス<br>P.189 | |
| 17:00 | | 17:00<br>口演 O2P-A04 ～ 07<br>回路網形成 6<br>P.170 | 口演 O2P-B13 ～ 16<br>シナプス可塑性 3<br>P.172 | 口演 O2P-C13 ～ 16<br>痛覚 (J)<br>P.174 | 口演 O2P-D12 ～ 15<br>栄養因子<br>P.177 | 17:30 | 口演 O2P-F13 ～ 16<br>アルツハイマー病 2<br>P.181 | 口演 O2P-G13 ～ 16<br>社会的行動 2<br>P.183 | 口演 O2P-H12 ～ 15<br>神経内分泌<br>P.185 | 口演 O2P-J13 ～ 16<br>受容体<br>P.188 | 口演 O2P-K13 ～ 16<br>学際領域 (J)<br>P.190 | 17:00 撤去<br>17:30 |
| 18:00 | 18:15<br>プレナリーレクチャー PL2<br>Michael E. Greenberg<br>P.113 | 18:00 | | | | | | | | | | |
| 19:00 | 19:15 | | | | | | | | | | | |
| 20:00 | | | | | | | | | | | | |

企業展示

懇親会
会場：インターコンチネンタルホテル 3F ボールルーム
19:30～21:30

(J)は日本語セッション

# Program at a Glance

## ■ Day 2 September 11 (Tue)

| 会場 | Plenary Room (Main Hall) | Room A (Small Auditorium) | Room B (501) | Room C (502) | Room D (301) | Room E (302) | Room F (303) | Room G (304) | Room H (311+312) | Room J (313+314) | Room K (411+412) | Poster Room (Exhibition Hall) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 8:00 | | | | | | | | | | | | |
| 8:30 | | Symposium S2A-A Signal Integration at Synapses Chair: S. Mochida P.121 | Symposium S2A-B Activity-dependent Post-synaptic Actin Remodeling: Its Dynamisms and Regulatory Molecules Chair: T. Shirao N. Mori P.121 | Symposium S2A-C RNA Metabolism: Its Involvement in Physiology and Neurologic Diseases Chair: H. Siomi H. J. Okano P.122 | Symposium S2A-D In vivo Imaging with Multiphoton Microscopy Chair: J. Nabekura H. Kasai P.123 | Symposium S2A-E The Hippocampal Network Forefront - Dynamic Reorganization and Synaptic Plasticity Chair: H. Kamiya H. Yawo P.123 | Symposium S2A-F Primate Social Cognition Chair: K. Nakamura P.124 | Symposium S2A-G Mitochondrial Dysfunction and Psycho-neurological Disorders Chair: Y. Goto P.125 | Symposium S2A-H Central Neuro-crossroads for Emotion, Pain, Instinct and Autonomic Functions Chair: F. Kato H. Matsui P.125 | Symposium S2A-J Neuronal Dysfunctions in Neuro-degenerative Diseases Chair: H. Okazawa S. Finkbeiner P.126 | Symposium S2A-K Strategy for New Generation of Neuroethology Chair: H. Ogawa K. Oka P.126 | 8:30 Installation |
| 9:00 | | | | | | | | | | | | 9:00 |
| 10:00 | | | | | | | | | | | | |
| 11:00 | | 11:00 | | | | | | | | | | 11:00 Odd Core |
| 12:00 | | 11:30 JSN Board Members' Meeting<br>12:00 JSN Members' Business Meeting | | | Luncheon Seminar LS2-D Janssen Pharmaceutical P.142 | Luncheon Seminar LS2-E OLYMPUS P.143 | Luncheon Seminar LS2-F Leica Microsystems P.143 | Luncheon Seminar LS2-G TANABE SEIYAKU P.143 | Luncheon Seminar LS2-H PLEXON PHYSIO-TECH P.143 | | | 12:00 |
| 13:00 | | 13:00 | | | 12:50 | | | | | | | 13:00 Even Core |
| 14:00 | | 14:00 JSN Young Scientists Lectureship Award Chair: M. Tsuda T. Tashiro P.140 | Oral Session O2P-B01 ~ 04 Synaptic Transmission (J) P.171<br>Oral Session O2P-B05 ~ 08 Synaptic Plasticity 1 P.171 | Oral Session O2P-C01 ~ 04 Visual System 4 P.173<br>Oral Session O2P-C05 ~ 08 Visual System 5 P.173 | Oral Session O2P-D01 ~ 04 Stem Cells 3 P.175<br>Oral Session O2P-D05 ~ 07 Stem Cells 4 P.176 | Oral Session O2P-E01 ~ 04 Parkinson's Disease 1 P.177<br>Oral Session O2P-E05 ~ 07 Parkinson's Disease 2 (J) P.178 | Oral Session O2P-F01 ~ 04 Cognition 1 P.179<br>Oral Session O2P-F05 ~ 08 Cognition 2 P.180 | Oral Session O2P-G01 ~ 04 Brain Imaging P.181<br>Oral Session O2P-G05 ~ 08 Cellular Imaging P.182 | Oral Session O2P-H01 ~ 04 Psychiatric Disorders (J) P.184<br>Oral Session O2P-H05 ~ 07 Schizophrenia P.184 | Oral Session O2P-J01 ~ 04 Gene Regulation P.186<br>Oral Session O2P-J05 ~ 08 Chemical Senses 1 P.186 | Oral Session O2P-K01 ~ 04 Methodology P.188<br>Oral Session O2P-K05 ~ 08 Sensori-Motor Plasticity P.189 | 14:00 |
| 15:00 | | | | | | | | | | | | |
| 16:00 | | 16:00 Oral Session O2P-A01 ~ 03 Network Formation 5 P.170 | Oral Session O2P-B09 ~ 12 Synaptic Plasticity 2 P.172 | Oral Session O2P-C09 ~ 12 Visual System 6 P.174 | Oral Session O2P-D08 ~ 11 Neurogenesis P.176 | Oral Session O2P-E08 ~ 13 Basal Ganglia P.178 | Oral Session O2P-F09 ~ 12 Alzheimer's Disease 1 (J) P.180 | Oral Session O2P-G09 ~ 12 Social Behavior 1 P.183 | Oral Session O2P-H08 ~ 11 Addiction and Abuse P.185 | Oral Session O2P-J09 ~ 12 Chemical Senses 2 P.187 | Oral Session O2P-K09 ~ 12 BMI/BCI P.189 | |
| 17:00 | | 17:00 Oral Session O2P-A04 ~ 07 Network Formation 6 P.170 | Oral Session O2P-B13 ~ 16 Synaptic Plasticity 3 P.172 | Oral Session O2P-C13 ~ 16 Pain (J) P.174 | Oral Session O2P-D12 ~ 15 Trophic Factors P.177 | 17:30 | Oral Session O2P-F13 ~ 16 Alzheimer's Disease 2 P.181 | Oral Session O2P-G13 ~ 16 Social Behavior 2 P.183 | Oral Session O2P-H12 ~ 15 Neuroendocrine System P.185 | Oral Session O2P-J13 ~ 16 Receptors P.188 | Oral Session O2P-K13 ~ 16 Interdisciplinary Areas (J) P.190 | 17:00 Removal<br>17:30 |
| 18:00 | 18:15 Plenary Lecture PL2 Michael E. Greenberg P.113 | 18:00 | | | | | | | | | | |
| 19:00 | 19:15 | | | | | | | | | | | |
| 20:00 | | | | | | | | | | | | |

Banquet
Intercontinental Hotel 3F
"Ball Room"
19:30~21:30

Exhibition

(J) Japanese Sessions

# 大会日程表

## ■ 大会第3日目　9月12日(水)

| 会場 | プレナリー会場 (メインホール) | A会場 (小ホール) | B会場 (501) | C会場 (502) | D会場 (301) | E会場 (302) | F会場 (303) | G会場 (304) | H会場 (311+312) | J会場 (313+314) | K会場 (411+412) | ポスター会場 (展示ホール) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 8:00 | | | | | | | | | | | | |
| 8:30 | | シンポジウム S3A-A<br>学習・記憶形成制御の分子基盤<br>座長：喜田　聡　井ノ口馨<br>P.128 | シンポジウム S3A-B<br>幹細胞の運命決定と分化可塑性制御のメカニズム<br>座長：中島欽一　岡野栄之<br>P.128 | シンポジウム S3A-C<br>神経発達・機能を制御するタンパク・膜相互作用ー神経回路から病態へー<br>座長：小島正己　尾藤晴彦<br>P.129 | シンポジウム S3A-D<br>精神疾患の分子機構<br>座長：功刀　浩<br>P.130 | シンポジウム S3A-E<br>小脳の神経伝達可塑性とその展望<br>座長：Thomas Knöpfel　永雄総一<br>P.130 | シンポジウム S3A-F<br>行動学習の脳内機構ーシステムレベルにおける実験と計算モデルの融合<br>座長：鮫島和行<br>P.131 | シンポジウム S3A-G<br>脳神経回路機能障害におけるミクログリアの多面性<br>座長：井上和秀　高坂新一<br>P.132 | シンポジウム S3A-H<br>疼痛処理の分子病理<br>座長：Bryan C. Hains<br>P.132 | シンポジウム S3A-J<br>美と快のイメージング<br>座長：本田　学　外池光雄<br>P.133 | シンポジウム S3A-K<br>鳥の歌制御システムにおける可塑性の分子機構<br>座長：坂口博信<br>P.133 | 8:30 貼付 |
| 9:00 | | | | | | | | | | | | 9:00 |
| 10:00 | | | | | | | | | | | | |
| 11:00 | | | | | | | | | | | | 11:00 奇数コア |
| 12:00 | | | | | 12:00 ランチョンセミナー LS3-D<br>ニコンインステック<br>P.144 | | ランチョンセミナー LS3-F<br>アフィメトリクス・ジャパン<br>P.144 | ランチョンセミナー LS3-G<br>科学技術振興機構<br>P.145 | | | | 12:00 |
| 13:00 | | | | | 12:50 | | | | | | | 13:00 偶数コア |
| 14:00 | 14:00 プレナリーレクチャー PL3<br>Earl K. Miller<br>P.113 | | | | | | | | | | | 14:00 |
| 15:00 | 15:00 | 15:15 シンポジウム S3P-A<br>アルツハイマー病研究のフロントライン<br>座長：西道隆臣　樋口真人<br>P.134 | シンポジウム S3P-B<br>知覚，意思決定，感情，行動における潜在的脳情報処理研究と応用<br>座長：下條信輔<br>P.134 | シンポジウム S3P-C<br>トニック $GABA_A$ コンダクタンスのメカニズムと機能的意義<br>座長：Alexey Semyanov　福田敦夫<br>P.135 | シンポジウム S3P-D<br>細胞内外シグナルの連携による神経細胞移動の制御<br>座長：五嶋良郎　仲嶋一範<br>P.135 | シンポジウム S3P-E<br>自閉症の臨床から脳科学への接近<br>座長：加我牧子　内野茂夫<br>P.136 | シンポジウム S3P-F<br>神経回路による情報処理の新しい概念ーニューロン説を超えてー<br>座長：宮川博義　坪川　宏<br>P.136 | シンポジウム S3P-G<br>脳機能発現と維持におけるアストロサイトの役割<br>座長：工藤佳久　小泉修一<br>P.137 | シンポジウム S3P-H<br>幻肢痛，幻肢覚における脳機能再構築<br>座長：吉峰俊樹　伊佐　正<br>P.138 | シンポジウム S3P-J<br>ハイスループットの遺伝子改変マウスの作成と表現型解析<br>座長：宮川　剛　愛場　篤<br>P.138 | シンポジウム S3P-K<br>脳の認知制御神経回路のダイナミクスから<br>座長：Scott Makeig　山口陽子<br>P.139 | 17:00 撤去 |
| 17:30 | | | | | | | | | | | | |
| 18:00 | | 17:45 | | | | | | | | | | |
| 19:00 | | | | | | | | | | | | |
| 20:00 | | | | | | | | | | | | |

企業展示

(J)は日本語セッション

# Program at a Glance

## ■ Day 3 September 12 (Wed)

| 会場 | Plenary Room (Main Hall) | Room A (Small Auditorium) | Room B (501) | Room C (502) | Room D (301) | Room E (302) | Room F (303) | Room G (304) | Room H (311+312) | Room J (313+314) | Room K (411+412) | Poster Room (Exhibition Hall) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 8:00 | | | | | | | | | | | | |
| 8:30 | | Symposium S3A-A<br>Understanding Mechanisms of Learning and Memory from Molecule to Behavior<br>Chair: S. Kida, K. Inokuchi<br>P.128 | Symposium S3A-B<br>Cell Fate Decision and Plasticity of Stem Cells<br>Chair: K. Nakashima, H. Okano<br>P.128 | Symposium S3A-C<br>Dynamic Protein-Lipid Interaction: Key Roles in Neuronal Development and in Diseases<br>Chair: M. Kojima, H. Bito<br>P.129 | Symposium S3A-D<br>Molecular Mechanism of Mental Disorders<br>Chair: H. Kunugi<br>P.130 | Symposium S3A-E<br>Synaptic Plasticity in the Cerebellum and Beyond<br>Chair: T. Knöpfel, S. Nagao<br>P.130 | Symposium S3A-F<br>Brain Mechanisms of Action Learning - Integrating Experimentation and Computational Modelling at the System Level<br>Chair: K. Samejima<br>P.131 | Symposium S3A-G<br>Multiple Function of Microglia in Brain Neurocircuit Disorder<br>Chair: K. Inoue, S. Kohsaka<br>P.132 | Symposium S3A-H<br>Molecular Pathology of Pain Processing<br>Chair: B. C. Hains<br>P.132 | Symposium S3A-J<br>Neuroimaging of Beauty and Pleasure<br>Chair: M. Honda, M. Tonoike<br>P.133 | Symposium S3A-K<br>Molecular Basis of Functional Plasticity in Birdsong Control System<br>Chair: H. Sakaguchi<br>P.133 | 8:30<br>Installation |
| 9:00 | | | | | | | | | | | | 9:00 |
| 10:00 | | | | | | | | | | | | |
| 11:00 | | 11:00 | | | | | | | | | | 11:00<br>Odd Core |
| 12:00 | | | | | 12:00<br>Luncheon Seminar<br>LS3-D<br>NIKON INSTECH<br>P.144 | | Luncheon Seminar<br>LS3-F<br>Affymetrix Japan<br>P.144 | Luncheon Seminar<br>LS3-G<br>JST RISTEX<br>P.145 | | | | 12:00 |
| 13:00 | | | | | 12:50 | | | | | | | 13:00<br>Even Core |
| 14:00 | 14:00<br>Plenary Lecture PL3<br>Earl K. Miller<br>P.113 | | | | | | | | | | | 14:00 |
| 15:00 | 15:00 | 15:15 | | | | | | | | | | |
| 16:00 | | Symposium S3P-A<br>Frontiers of Alzheimer Research<br>Chair: T. Saido, M. Higuchi<br>P.134 | Symposium S3P-B<br>Implicit Processing in Perception, Decision, Emotion and Action<br>Chair: S. Shimojo<br>P.134 | Symposium S3P-C<br>Mechanisms and Functional Significance of Tonic $GABA_A$ Receptor Mediated Conductance<br>Chair: A. Semyanov, A. Fukuda<br>P.135 | Symposium S3P-D<br>Regulation of Neuronal Migration by an Interplay between Intracellular and Extracellular Signals<br>Chair: Y. Goshima, K. Nakajima<br>P.135 | Symposium S3P-E<br>Autism-Clinical Research to Brain Science<br>Chair: M. Kaga, S. Uchino<br>P.136 | Symposium S3P-F<br>New Ideas on the Information Processing by Neural Networks - Beyond the Neuron Doctrine -<br>Chair: H. Miyakawa, H. Tsubokawa<br>P.136 | Symposium S3P-G<br>Roles of Astrocytes in the Brain Function and its Maintenance<br>Chair: Y. Kudo, S. Koizumi<br>P.137 | Symposium S3P-H<br>Cerebral Reorganization in the Phantom Limb Pain and Sensation<br>Chair: T. Yoshimine, T. Isa<br>P.138 | Symposium S3P-J<br>High Throughput Generation and Phenotyping of Genetically-engineered Mice<br>Chair: T. Miyakawa, A. Aiba<br>P.138 | Symposium S3P-K<br>EEG Rhythms and Cognitive Control Cognitive Control and Network Dynamics in the Brain<br>Chair: S. Makeig, Y. Yamaguchi<br>P.139 | 17:00<br>Removal |
| 17:00 | | | | | | | | | | | | 17:30 |
| 18:00 | | 17:45 | | | | | | | | | | |
| 19:00 | | | | | | | | | | | | |
| 20:00 | | | | | | | | | | | | |

Exhibition

(J) Japanese Sessions

# 参加者へのご案内

## 1. 参加受付について

### ◆事前参加登録がお済みの方へ

あらかじめ送付しております参加証（ネームカード）にご所属とお名前をご記入の上、必ずご着用ください。ネームカードホルダーは受付付近の記名台にご用意しております。参加証（ネームカード）のない方の入場は、固くお断りいたします。

### ◆当日参加登録をされる方へ

本大会に当日参加登録される方は、パシフィコ横浜会議センター2階「当日参加受付」にてご登録ください。お支払いは全て現金にてお願いいたします。

・受付時間

9月10日（月）7：30～18：00
9月11日（火）7：30～18：00
9月12日（水）7：30～17：00

・参加費と引き換えに①参加証（ネームカード）、②プログラム集、③CD-ROM抄録集をお渡しいたします。会場内では、必ず参加証（ネームカード）をご着用ください。

・参加費は下記の通りです。

| | | | |
|---|---|---|---|
| 会　員 | 一　般 | 15,000円 | |
| | 大学院生 | 3,000円 | 学生証をご提示ください |
| 非会員 | 一　般 | 17,000円 | |
| | 大学院生 | 6,000円 | 学生証をご提示ください |
| | 学部学生 | 無　料 | 学生証をご提示ください |

・筆頭著者発表を行わない学部学生の参加費は無料です。筆頭著者発表を行う学部学生の皆様には事前参加登録の際に参加費をお支払いいただきました。

### ◆懇親会（NeuroSocial）

今大会の懇親会は、先端で活躍されている研究者、およびこれからそれぞれの分野で活躍しようとする若手研究者（学部学生・院生・ポスドク含む）が集い、飲み物を片手に自由に交流できる会として開催します。プレナリー講演者やシンポジウムの演者の先生方にも参加を呼びかけますので、著名な研究者との会話のチャンスでもあります。懇親会の中で、日本神経科学学会奨励賞（受賞者は89頁をご覧ください）および日本神経化学会若手奨励賞（受賞者は90頁をご覧ください）の授賞式を行います。多くの皆様の参加をお待ちしています。

・日時と場所

9月11日（火）19：30～
インターコンチネンタルホテル グランド横浜　3階「ボールルーム」

・懇親会費（当日）

| | |
|---|---|
| 一　般 | 4,000円 |
| 大学院生 | 3,000円 |
| 学部学生 | 2,000円 |

・参加登録

懇親会参加を希望される方は、パシフィコ横浜会議センター2階当日参加受付にてご登録ください。

また、懇親会開催直前（19:00～20:00）は会場前に懇親会参加受付を設けますのでご登録ください。定員になり次第締め切らせていただきます。

## 2. 英文抄録

日本神経科学学会の会員として演題登録された方の英文抄録はNeuroscience Research（Elsevier）のSupplementに掲載されます。筆頭著者は掲載費として4,000円をお支払いいただきました。掲載費をお支払いいただいた方には、**9月下旬にご登録の住所に郵送**されます。大会会期中の引き換えは出来ませんので、ご了承ください。

神経科学学会の会員として演題登録された方以外で、Neuroscience Research Supplementを購入ご希望の方は大会会場にてお申し込みください（4,000円）。9月下旬にお申し込みの住所に郵送させていただきます。

## 3. 大会受付での認定医研修単位認定について

本大会への参加は、下記学会の専門医・認定医研修単位取得の対象となります。専門医・認定医の先生方はぜひご利用ください。

・日本神経学会
・日本精神神経学会
・日本小児神経学会
・日本老年精神医学会
・日本良導絡自律神経学会

日本精神神経学会の「専門医資格更新にかかる研修ポイント」の取得は、大会の総合窓口にて「精神科専門医認定試験合格者証（ポイントカード）」を提示してください。係員が専用パソコンにてポイント数を入力いたします。

それ以外の学会については、Neuro2007の参加証および領収書を各自で各学会に提出し、単位申請してください。詳細についてはそれぞれの学会にお問い合わせください。

## 4. ランチョンセミナーについて

ランチョンセミナーにつきましては、お弁当数に限りがありますので、整理券を開催日の朝（参加受付時間から、先着順）にランチョンセミナーデスク（1階ロビー）にて発券いたします。発券は、1人1日1枚となります。発券には参加証（ネームカード）のご提示が必要となります。

## 5. 呼び出し、伝言

会場内でのスライドおよび館内放送での呼び出しは行いません。展示会場内の会員連絡板をご利用ください。

## 6. クローク

パシフィコ横浜会議センター1階にクロークを設けますので、ご利用ください。なお、貴重品・傘などはお預かりできませんのでご了承ください。

《開設時間》

9月10日（月）7：30～20：00
9月11日（火）7：30～21：00
9月12日（水）7：30～18：00

## 7. 機器展示・書籍販売

展示ホールにおいて機器展示を、会議センターにおいて書籍販売を行います。

9月10日（月）9：00～17：00
9月11日（火）9：00～17：00
9月12日（水）9：00～17：00

## 8. インターネットコーナー

展示ホールの休憩コーナーにて無線LANおよび電源がご利用いただけます。また、ポスター会場奥の休憩コーナーでは、抄録検索システムが利用可能なパソコン5台、インターネット接続可能なLANケーブルおよび電源をご用意しています。

## 9. ドリンクコーナーについて

会期中（10日～12日）展示ホールの休憩コーナーでフリードリンクのサービスが行われます。

## 10. お子様連れの参加者の方へ

本大会では「託児室」と「親子休憩室」を設置いたします。
「託児室」には保育スタッフが常駐し、保護者の方が大会に参加される間、お子様をお預かりいたします。利用には事前予約が必要です。
「親子休憩室」は、お子様と一緒にお食事や休憩を取りたい方が無料で自由にご利用いただけます。ご利用の際は必ず保護者の方がご同伴ください。

●託児室

託児室は、全国ベビーシッター協会正会員の株式会社アルファ・コーポレーションに委託しております。マットや玩具などを備え、専門の保育スタッフがケアにあたります。

対象：0歳（3ヶ月）～小学6年生まで
開室時間：
9月10日（月）8:00～19:30
9月11日（火）8:00～19:30
9月12日（水）8:00～18:00
料金：200円／30分（当日会場にてお支払いください。おむつなどの実費は除きます。）
開設場所：セキュリティのため、ご利用の方にのみお知らせします。
**受付締切：平成19年9月3日（月）**
締切日以降のご予約は、余裕があれば対応いたしますのでご相談ください。

利用申し込み方法：
以下の項目をメールにお書き添えの上、アルファ・コーポレーションまで直接お申込みください。
メールアドレス：yoyaku@alpha-co.com
サブジェクト：「Neuro2007　託児室予約」
1）所属学会、会員番号（非会員の方は必要ありません）
2）保護者氏名、所属、連絡先（含む携帯電話番号）
3）子供の人数、名前、年齢、性別
4）保育希望日時
5）保育上の注意点（アレルギー等）、希望事項（散歩等）
お申込み後、ご予約確認のメールと共に「利用規約」と「申込書」を返信いたします。申込書は記入、押印の上、当日託児室までお持ちください。

※ご質問時のメールサブジェクトは「Neuro2007　託児室予約＋（ご用件）」としてください。
※ご予約内容の変更は、必ずアルファ・コーポレーションまでご連絡ください。
※お電話でのお申込みは　0120-086-720　（月～金　9:30-17:30）
万が一の事故・怪我の場合は、株式会社アルファ・コーポレーションが加入している保険の適用範囲で補償されます。学会及び大会事務局は一切の責任を負いません。

お申し込み・お問い合わせ：
株式会社アルファ・コーポレーション
担当：小滝（コタキ）
E-mail：yoyaku@alpha-co.com
Tel：03-5772-1222
ホームページ URL：http://www.alpha-co.com/

昼食について：
食物アレルギー、衛生面などへの配慮から、原則として準備しません。昼食時間帯の保育を希望する方は、予約時にご相談ください。なお、託児室とは別に親子休憩室（飲食可）を用意しておりますのでご利用ください。

●親子休憩室
マットや玩具、ついたてなどをご用意しております。お子様とご一緒のお食事や休憩、授乳など、ご自由にご利用ください。事前予約は不要です。親子休憩室に保育スタッフはおりません。ご利用の際は必ず保護者が付き添い、責任を持って安全に留意してください。
開室日時：9月10日（月）8:00～19:30
9月11日（火）8:00～19:30
9月12日（水）8:00～18:00
開設場所：パシフィコ横浜会議センター内
利用料金：無料
その他：親子休憩室での万が一の事故や怪我の場合、委託会社による保険は適用されません。また学会及び大会事務局は一切の責任を負いません。

## 11. 公開講座　高校生のための神経科学入門

8月9日（木）に理化学研究所脳科学総合研究センターに於いて同センターと共催で行いました。

## 12. 関連行事

### ◆ 理事会

日本神経化学会
日時：9月9日（日）13:00～17:00　会場：4階414会議室

日本神経科学学会
日時：9月9日（日）15:30～20:30　会場：4階417会議室

### ◆ 評議員会

日本神経化学会
日時：9月10日（日）11:30～12:00　会場：5階小ホール（A会場）

### ◆ 総会

第30回日本神経科学大会
日時：9月10日（月）13:00～14:00　会場：4階411+412会議室（K会場）
総会議事に引き続き、各賞授賞式を行います。

1. 時実利彦記念賞授賞式（受賞者は89ページをご覧ください）
2. Travel Award授賞式（受賞者は90ページをご覧ください）

第50回日本神経化学会大会
日時：9月11日（火）12:00～13:00　会場：5階A会場

## 13. 各種委員会

9月9日（日）

| 会合名 | 開催時間 | 会場 |
|---|---|---|
| （科学）　NSR優秀論文賞選考委員会 | 12:00～13:00 | 4階　417会議室 |
| （化学）　日本神経化学会　理事会 | 13:00～17:00 | 4階　414会議室 |
| （科学）　NSR編集委員会 | 13:30～15:00 | 4階　417会議室 |
| （科学）　日本神経科学学会　理事会 | 15:30～20:30 | 4階　417会議室 |

9月10日（月）

| 会合名 | 開催時間 | 会場 |
|---|---|---|
| （化学）　出版・広告委員会 | 11:00～13:00 | 4階　414会議室 |
| （化学）　国際対応委員会 | 11:00～13:00 | 4階　415会議室 |
| （化学）　脳研究推進委員会 | 11:00～13:00 | 4階　418会議室 |
| （科学）　Neuroscience2008実行委員会 | 12:00～13:00 | 4階　416会議室 |
| 実験動物使用者会議 | 12:00～13:00 | 3階　H会場 |
| （科学）　時実賞運営委員会会食会 | 12:00～13:00 | 4階　422会議室 |
| （科学）　男女共同参画推進委員会 | 12:00～13:00 | 4階　423会議室 |
| （化学）　将来計画委員会 | 14:00～16:00 | 4階　414会議室 |
| （化学）　シンポジウム企画委員会 | 14:00～16:00 | 4階　418会議室 |

9月11日（火）

| 会合名 | 開催時間 | 会場 |
|---|---|---|
| （化学） 化学会 評議員会 | 11:30～12:00 | 5階 A会場 |
| （科学） 神経科学ニュース編集小委員会 | 12:00～13:00 | 4階 416会議室 |
| （科学） 動物実験倫理委員会 | 12:00～13:00 | 4階 414会議室 |
| 成茂基金昼食会 | 12:00～13:00 | 4階 415会議室 |
| （化学） 奨励賞選考委員会 | 16:00～19:00 | 4階 415会議室 |

9月12日（火）

| 会合名 | 開催時間 | 会場 |
|---|---|---|
| （科学） Neuroscience2008プログラム委員会 | 12:00～13:00 | 4階 415会議室 |
| NPO脳の世紀推進会議 | 12:00～13:00 | 4階 416会議室 |
| （科学） 将来計画委員会 | 12:00～13:00 | 4階 418会議室 |
| （科学） Neuroscience2009実行委員会 | 12:00～13:00 | 4階 422会議室 |

## 14. プレスルーム

開設日時：9月10日（月）～12日（水） 8:00～19:00 場所：2階 212会議室

### ◆ プレスルームの趣旨および取材条件

日本神経科学大会、日本神経化学会大会、日本神経回路学会大会の社会への広報活動として、報道機関に対する積極的な情報発信を行います。そのために、大会の認める範囲で、来場した記者の会場内での円滑な取材活動と、さらに興味のある発表者を呼んでのインタビュー等の充実した取材活動を支援すべく、学会場に取材用のプレスルームを確保します。

取材のために来場した報道関係者は、プレス受付にて記者証（フリーランスライターは身分証明書と過去に媒体掲載の記事数点のコピー）を提示確認の上で、プレス登録をして頂きます。会場内にては、大会事務局で交付したプレスバッチまたは腕章を装着して取材活動を行って頂きます。

報道関係者には、対象となる研究発表者をプレスルームに招いて取材して頂きます。プレスルーム内では、発表者の了解の上で撮影・録音・インタビューをお願いします。口演会場およびポスター会場での、撮影・録音・インタビューは原則として認めません。また、取材情報の整理、起稿入稿などの作業を行う場としてプレスルームを提供します。

## 15. サテライト企画のご案内

本大会では、以下のサテライト企画が開催されますので、ご案内いたします。

### ◆サテライトシンポジウム

**1. 第2回日韓神経科学シンポジウム“神経科学の最先端”**

日時：9月13日（木）9：30～20：00

場所：5階 小ホール

主催：日本神経科学学会、韓国脳神経科学学会、理研脳科学総合研究センター

オーガナイザー:津本忠治（理研脳科学総合研究センター)、伊藤正男（理研脳科学総合研究センター)、
Yoo-Hun Suh（ソウル国立大学）
参加費：無料

**2. 第2回 MCCS-Asia シンポジウム　高次脳機能の解明：モデル動物の進展 II**
日時：9月8日（土）17：00～20：00　ポスターセッション
9月9日（日）9：00～18：00　オーラルセッション
場所：3階　301+302 会議室
主催：理化学研究所脳科学総合研究センター先端技術開発グループ、
Molecular and Cellular Cognition Society (MCCS)-Asia
オーガナイザー：糸原重美（理研脳科学総合研究センター)、三品昌美（東京大学医学系研究科)、
和田圭司（国立精神・神経センター神経研究所)、
宮脇敦史（理研脳科学総合研究センター)、
Paul Chapman（Glaxo Smith Kline)、
Sumantra Chattarji（National Centre for Biological Sciences, TIFR）
参加費：無料

**3. 再生医療の実現化プロジェクト　第4回成果発表会（公開シンポジウム）**
**～幹細胞研究の最前線：リーディングプロジェクトの研究から～**
日時：9月9日（日）　14：00～17：30
場所：5階　502、511+512
主催：再生医療の実現化プロジェクト　成果発表会実行委員会
共催：文部科学省 http://www.pacifico.co.jp/promoter/facilities/conference/pop_5f.html
参加費：無料
対象：企業・大学の研究者

## 16. 抄録検索システムについて

すべての演題の発表者、所属、発表時間・会場、抄録などについて項目別に検索し、日程表を作成することができる抄録検索システムを含むCD-ROMを大会プログラムに同封いたします。
また、同等の抄録検索システムが大会ホームページ及び神経科学学会ホームページからダウンロードできます。
以下のURLからダウンロードしてお使いください。

http://www.convention.jp/neuro2007/

推奨動作環境

■ Windows
- OS： Windows 2000(Service Pack 4), XP(Service Pack 2), Vista
- CPU： Pentium III 500MHz 以上
- メモリ： 256MB 以上
- ディスプレイ解像度： XGA(1024 × 768)以上

■ Mac OS X
- OS： PowerPC 搭載機では Mac OS X 10.3.9 以上
  Intel プロセッサ搭載機では Mac OS X 10.4.5 以上
- CPU： PowerPC G3, G4, G5 または Intel プロセッサ
- メモリ：256MB 以上
- ディスプレイ解像度：XGA(1024 × 768)以上

# 座長へのご案内

・座長の方は、15 分前までに各会場の次座長席へお越しの上、進行係に声を掛けてください。
・発表時間、質疑応答時間を厳守し、円滑な運営にご協力をお願いいたします。

# シンポジスト・一般口演発表者へのご案内

## ■ 発表言語

・シンポジウム：英語
・一般口演：英語または日本語（演題登録時に選択した言語でお願いいたします。）
・シンポジウム・一般口演とも、討論は英語・日本語どちらでも結構です。

## ■ 発表時間

・シンポジウムは座長の指示に従って下さい。
・一般口演は発表 10 分、討論 5 分です（座長の指示に従ってください）。

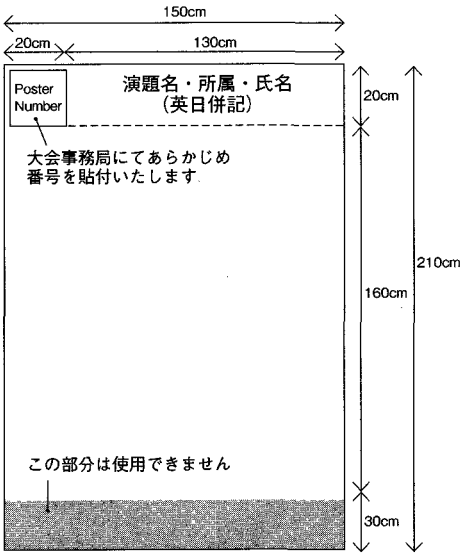
## ■発表は各自お持込みの PC を使用した PC プレゼンテーションに限ります

・本大会の発表は、PC プレゼンテーションに限らせて頂きます。各自ノートパソコンをご用意ください。
・Windows2000 または Mac OS8.5 以上が動作し、外部モニター出力端子を備えた PC を使用してください。
・SONY の VAIO、Mac 等、モニター出力端子の形状が特殊な機種をご使用の場合は、Dsub-15 ピン用の変換アダプターを持参して下さい。
・バッテリー切れに備え、必ず発表会場に AC アダプターをご持参ください。
・発表中にスクリーンセーバーや省電力モードにならないように設定して下さい。
・発表の 45 分前までに PC センター（3 階ホワイエ）にて出力をチェックしてください。
・発表時の操作は、演壇上のモニターとリモートマウスを用いて、発表者自身が行います。
・演壇上には時計機をご用意いたしますので、時間内に完結できるようご協力下さい。
・プレゼンテーションデータに静止画・動画・グラフ等のデータをリンクさせている場合は、そのデータも必ず保存していただき、事前に動作確認を行ってください。
・スピーカの用意はございません。必要な場合は事前に事務局へ御連絡ください。
・音声の使用はできません。
・発表データの配色については、色盲・色弱の参加者へのご配慮をお願いいたします。

＜ D-sub 15pin ＞ 

# ポスター発表者へのご案内

・ポスター展示は、展示ホールで行います。まず展示ホール入口にある、ポスター受付にて受付をお願いいたします。
・ポスターはご自身の演題番号の位置に、貼り付け時間内に、ご自身で貼り付けて下さい。ポスターパネル番号が、あらかじめ貼付パネルに表示してあります。演題番号をご確認の上、正しいパネルに貼り付けしていただくようご注意下さい。
・画鋲はポスター受付にてお渡しいたします。糊・セロテープは使用できません。
・ポスターを貼付できるスペースは、右図をご覧ください。
・演題名、発表者名、所属を、英語・日本語表記でご準備いただき、ポスター貼付スペースの上端部に貼付してください。
・ポスターの発表内容（図面・説明）は、すべて英語で作成してください。




・発表者の方には、ポスター受付で発表者の腕章をお渡ししますので、討論の際には胸につけて、下記のコアタイム中は各ポスターパネルの前で参加者の質疑に応じてください。
・撤去時間後も貼付されているポスターは事務局で即日処分させていただきますのでご了承ください。

| | 貼付け時間 | 演題番号 | コアタイム | 撤去時間 |
|---|---|---|---|---|
| 9月10日(月) | 8:30 ～ 9:00 | 奇数 | 11:00 ～ 12:00 | 17:00 ～ 17:30 |
| | | 偶数 | 13:00 ～ 14:00 | |
| 9月11日(火) | 8:30 ～ 9:00 | 奇数 | 11:00 ～ 12:00 | |
| | | 偶数 | 13:00 ～ 14:00 | |
| 9月12日(水) | 8:30 ～ 9:00 | 奇数 | 11:00 ～ 12:00 | |
| | | 偶数 | 13:00 ～ 14:00 | |

## ■ 発表資料作成に関するお願い

男性学会参加者の20人に1人は色盲・色弱です。発表資料作成の際は以下の点にご配慮いただきますよう、皆様のご理解とご協力をお願いいたします。

1. 2重染色やDNAチップの画像は、緑と赤ではなく、緑と赤紫（マゼンタ）で表示する。
2. 3重以上の染色は、全色の重ね合わせだけでなく重要な2色だけの組み合わせも緑と赤紫で表示する。（あるいは各チャンネルの図を別々に表示する。）
3. グラフや解説図では、離れた2ヶ所の色を照合するのが非常に難しいので、色分けされた各項目の内容を別に凡例で示すのはなく、図中に直接書き込む。また各項目は、色だけでなく線種やシンボルの形、網掛け（ハッチング）などでも区別する。
4. 赤は鮮やかな明るい色に見えないので、暗い背景に赤い文字を使わない。また、青い細かな字は色覚に関わらず非常に読みづらい。なるべく白や黄色、オレンジ等を使う。

詳しくは http://www.nig.ac.jp/color/ をご覧ください。

# Information for participants

## 1. Registration

### ◆ Participants with advance registration

The conference pass (name card) has been sent to you by mail. Fill in your name and affiliation on it, and wear it throughout Neuro2007. A name holder will be available at reception. Those without a conference pass (name card) are not admitted to any conference sites.

### ◆ Participants with on-site registration

Go to 2F of Pacifico Yokohama Conference Center for on-site registration. Only cash payment is accepted.

- On-site registration desk opens:
  - September 10 (Monday) 7:30-18:00
  - September 11 (Tuesday) 7:30-18:00
  - September 12 (Wednesday) 7:30-17:00
- You will receive 1) a conference pass (name card), 2) program booklet, and 3) CD-ROM (abstracts) upon payment. Please make sure you wear your name card at all conference sites.
- Registration fees

| | | | |
|---|---|---|---|
| Member | Regular | JPY 15,000 | |
| | Graduate student | JPY 3,000 | with student ID card |
| Non-member | Regular | JPY 17,000 | |
| | Graduate student | JPY 6,000 | with student ID card |
| | Undergrad student | Free | with student ID card |

- Undergraduate students who are not first/presenting authors participate free of charge. Undergraduate students who are first/presenting authors have paid the registration fee at the pre-registration.

### ◆ Banquet (NeuroSocial)

This banquet offers the opportunity for enthusiastic graduate, undergaduate students and post docs to meet with leading researchers in a friendly atmosphere. You may even have a chance to discuss with the plenary lecturers and symposium speakers there. Do not miss this opportunity.

- Date

  September 11 (Tuesday) 19:30 -
  Intercontinental Hotel The Grand Yokohama "Ball Room" (3F)

- Banquet fee (on-site)

| | |
|---|---|
| Regular | JPY 4,000 |
| Graduate student | JPY 3,000 |
| Undergraduate student | JPY 2,000 |

- Banquet registration

Banquet on-site registration is accepted at 2F Conference Center of Pacifico Yokohama. Another registration desk will open between 19:00-20:00 at the banquet site. On-site registration is closed when it reaches the number limit.

## 2. Neuroscience Research Supplement

Abstract registered by Japan Neuroscience Society (JNS) members are published as a Supplement to Neuroscience Research (Elsevier). The first/presenting authors were requested to pay additional JPY4,000. The volume of the English abstracts will be sent to a payer's registered address by mail in late September. The volumes are not available at the conference sites.

Others who wish to buy the volume of Neuroscience Research (other than abstract presenters who are members of JNS) may apply for it at the conference site (JPY4,000). It will be sent to the provided address by mail in late September.

## 3. Luncheon seminars

A luncheon voucher is available on the morning of the seminar day at Luncheon seminar desk (1F Lobby) in order of arrival when the reception desk opens. As the number of lunches is limited, present your conference pass (name card) to pick up your voucher there. One participant is provided with one voucher per day.

## 4. Cloak room

A cloak room is available on 1F Pacifico Yokohama Conference Center. Valuables and umbrellas cannot be kept there.

<Cloak dates and hours>

| | |
|---|---|
| September 10 (Monday) | 7:30-20:00 |
| September 11 (Tuesday) | 7:30-21:00 |
| September 12 (Wednesday) | 7:30-18:00 |

## 5. Exhibition of equipments/products and book sales

The exhibition is held in the Exhibition Hall and the book sales in the Conference Center.

<Exhibit dates and hours>

| | |
|---|---|
| September 10 (Monday) | 9:00-17:00 |
| September 11 (Tuesday) | 9:00-17:00 |
| September 12 (Wednesday) | 9:00-17:00 |

## 6. Internet access corner

Wireless LAN and AC power are available at the Rest Area in the Exhibition Hall. There are also 5 personal computers with the Abstract Viewer and several LAN cables at the back side of the Poster area.

## 7. Drink corner

Free drink is provided at the Rest Area in the Exhibition Hall.

## 8. Nursery service and Resting room for family

### Day nursery room

To participants accompanying children

Day nursery and child room will be available at the meeting. At day nursery, professional staff will take care of children when their parents participate in the meeting. Reservation is required.

Child room with toys is also available free of charge for parents to have meals and take some rest with their children. Parent(s) must accompany their child(ren) for its use.

## Day nursery room

We have entrusted ALPHA Corporation Inc. with childcare service. This is an official member of All-Japan Babysitter Association. Professional nursery staff will take care of children with mats and toys.

Nursery Room Hours:

| | |
|---|---|
| September 10 (Monday) | 8:00-19:30 |
| September 11 (Tuesday) | 8:00-19:30 |
| September 12 (Wednesday) | 8:00-18:00 |

Place: For security purpose, the room location will be informed only to its users.

Age: from 0 (over 3 months) to 6$^{th}$ grade student of elementary school

Charge: 200yen/30minutes (Payment is made on site. Actual costs (e.g. diaper) are extra.

Deadline of application: September 3 (Monday), 2007
We may be able to accept an application after the deadline, but only if there is extra capacity.

How to apply:
Send email to ALPHA Corporation directly, providing information items 1-5 below.
Email address: yoyaku@alpha-co.com
Subject: Neuro2007 Day nursery reservation
1) Society that you belong to and membership number (not necessary to a non-member)
2) Name of delegate, affiliation, and contact address and telephone number (including cell phone)
3) Number of children, names, ages, and sexes
4) Day and time of requested nursery service
5) Notes (e.g. allergy) and requests (a walk, etc)

Then ALPHA Corporation will send you confirmation email along with the guidelines and rules of its use and an application form. The application with your seal (or signature) will be submitted directly to the nursery room on the day when you need nursery service.

- When you make inquiries by email, write "Neuro2007 Day Nursery Reservation" as the subject of email.
- When you make a change of your reservation, contact ALPHA Corporation directly.
- Reservation is also accepted by phone at 0120-086-720 (Monday to Friday, 9:30-17:30)

Contact: Please contact the following for detailed information and application.
Day Nursery Service: ALPHA Corporation Inc.
Attn. : Ms. Kotaki

TEL: +81-3-5772-1222(from overseas) / (03)5772-1222
E-mail: yoyaku@alpha-co.com
URL: http://www.alpha-co.com/english/index.html

Lunch: Giving careful considerations to food allergy and hygienic conditions, lunch is not provided at the nursery room. Consult with ALPHA Corporation if you request nursery service during the lunch break. A child room is also available where you can have lunch with your child(ren).

Note: In case of accidents and/or injuries, compensation is covered by insurance that ALPHA Corporation holds. Neuro2007 and the Secretariat are not responsible for any accidents and/or injuries caused in the nursery room.

### Family room

There will be mats, toys and screens. Parents may take some rest with children and/or breastfeed them. No reservation is necessary. As no nursery staff is provided in the family room, parents are required to accompany children and are responsible for safety in the room.

Family Room Hours:

| | |
|---|---|
| September 10 (Monday) | 8:00-19:30 |
| September 11 (Tuesday) | 8:00-19:30 |
| September 12 (Wednesday) | 8:00-18:00 |

Location: at the conference room in Pacifico Yokohama

Charge: Free

Note: In case of accidents and/or injuries at the family room, no insurance is available. Neuro2007 and the Secretariat are not responsible for any accidents and/or injuries caused in the family room.

## 9. Abstract Viewer

The CD-ROM enclosed to the program booklet enables you to make your own schedule to search authors, affiliations, presenting time and place of abstracts. This program is also downloadable from Neuro2007 or Japan Neuroscience Society website.

URL : http://www.convention.jp/neuro2007/

Recommended operating systems and versions:

■ Windows

- OS : Windows 2000 (Service Pack 4), XP (Service Pack 2), Vista
- CPU : Pentium III 500MHz or above
- RAM : 256MB or above
- Resolution : XGA (1024 × 768) or above

■ Mac OS X

- OS :
  Mac OS X 10.3.9 or above for machines with PowerPC
  Mac OS X 10.4.5 or above for machines with Intel Processor
- CPU : PowerPC G3, G4, G5 or Intel Processor
- RAM : 256MB or above
- Resolution : XGA (1024 × 768) or above

# Information for Session Chairpersons

Chairpersons are requested to take their place in the seat for the next chairperson at least 15 minutes before their session and to make their presence known to the MC.

Please adhere strictly to the time allocated for presentations and questions in order to ensure the smooth running of the conference.

# Information for Speakers in Symposia and Oral Sessions

## Language

Symposia: English
Oral Sessions: English or Japanese (please use the language selected at the time of registration)
Discussions following both symposium and oral presentations may be in either English or Japanese.

## Presentation time

The time allocated for symposium presentations is at the discretion of the chairperson. Please follow his/her instructions.
The time allocated for general oral presentations is 15 minutes (a 10-minute presentation followed by 5 minutes for discussion).

## Presentation aim

Presentations must be given with your own laptop computer, which you should bring with you.
The operating system for your laptop should be Windows 98 or newer, or Mac OS 8.5 or newer.
Your laptop must have external monitor output.
If your laptop's external monitor output is not via a Dsub-15 pin, please bring your own adaptor for connecting to a Dsub-15.
Be sure to bring a power cable and adapter to the meeting room in case your computer's battery runs low.
All energy-conserving functions such as screen-savers, sleep/power-saving modes and suchlike must be disabled on all laptop computers to be used during presentations.
Please check that your presentation outputs correctly at the Computer Center at least 45 minutes before your session.
You will operate your presentation by using a remote mouse and a monitor at the speaker's podium.
A time indicator will be provided on the speaker's podium. Please adhere strictly to your allocated time to ensure the smooth progression of the conference.
Should your presentation file include links to still images, moving images, graphs, or similar, please be sure to save these also and check in advance that they operate correctly.
Speakers are not provided. Should you require speakers, please contact the Secretariat in advance.
Audio cannot be used.
Please take the needs of colorblind participants into account when deciding your color scheme.

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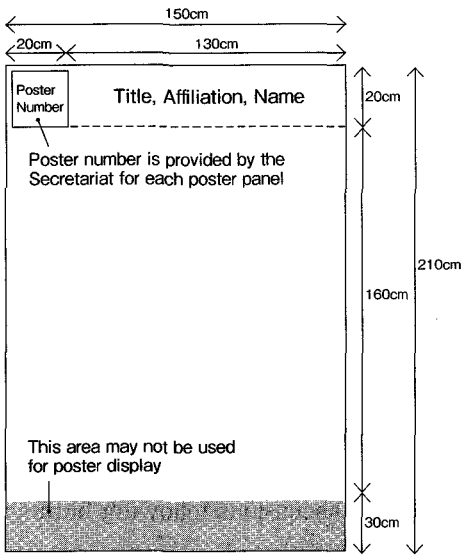


# Information for Poster Presenters

Poster Sessions will be held at the Exhibition Hall. Please report to the Poster Registration Desk at the entrance of the Exhibition Hall before displaying your poster.

Display your poster yourself in the designated panel between 8:30 and 9:00. Your presentation number will be indicated on the panel. Please use the pushpins provided at each panel. Glue and tape may not be used.

Please see right figure for the poster display space.




Please indicate your presentation title, name, and affiliation at the top of your poster. The entire content of your poster presentation (graphics and explanations) must be in English.

Presenter's ribbons will be distributed at the Poster Registration Desk. Presenters are required to wear them in a clearly visible position and to stand in front of their poster to answer questions from participants during each presentation and discussion session.

Posters should be removed during the following time period.

September 10th: 17:00-17:30
September 11th: 17:00-17:30
September 12th: 17:00-17:30

Posters on display beyond the designated time will be removed and discarded within the day.

## Requests concerning the preparation of presentation documents

One in 20 male conference participants is colorblind or has difficulty differentiating colors. Please take the following points into consideration when preparing your presentation documents. We appreciate your understanding and cooperation on this point.

1. Display images of double staining or DNA chips in green and magenta rather than in green and red.
2. For triple or more staining, show not only the overall montage, but also important two-channel combinations in green and magenta. (Alternatively, display a separate image for each channel.)
3. Colorblind participants find it extremely difficult to match the colors in two separate locations in graphs or explanatory figures. Rather than indicating the meanings of the different colors in a separate legend, include them directly in the figures. Alternatively, distinguish them by using different types of line, forms of symbol, hatching, or other means rather than color.
4. As red does not appear as a bright, vivid color, do not use red lines against a dark background. Fine blue writing is also extremely difficult to read regardless of colorblindness. Use white, yellow, or orange wherever possible.
   See http://jfly.iam.u-tokyo.ac.jp/color/ for more detailed information.

# 各賞受賞者　List of the Awardees

## 時実利彦記念賞　Tokizane Award

**■西道　隆臣（Takaomi C. Saido）**

理化学研究所脳科学総合研究センター
Laboratory for Proteolytic Neuroscience, RIKEN Brain Science Institute
Session No: SL1
Title: アミロイドβペプチド代謝とアルツハイマー病
Metabolism of amyloid β peptide and Alzheimer's disease

## 塚原仲晃記念賞　Tsukahara Award

**■銅谷　賢治（Kenji Doya）**

沖縄科学技術大学院大学先行研究
Okinawa Institute of Science and Technology, ATR Computational Neuroscience Laboratories
Session No: SL2
Title: 強化学習の脳機構の探求
In Pursuit of the Brain Mechanism of Reinforcement Learning

**■宮脇　敦史（Atsushi Miyawaki）**

理化学研究所脳科学総合研究センター
RIKEN Brain Science Institute
Session No: SL3
Title: 脳神経系の構造・機能を可視化する蛍光タンパク質技術
Innovations in fluorescence imaging of brain functions using fluorescent proteins

## 日本神経科学学会奨励賞　JNS Young Investigator Award

**■池田　　弘（Hiroshi Ikeda）**

福井大学
脊髄後角におけるシナプス可塑性の機序

**■磯村　宣和（Yoshikazu Isomura）**

理化学研究所
大脳皮質・海馬ネットワーク活動のインビボおよびインビトロ解析

**■後藤　幸織（Yukiori Goto）**

University of Pittsburgh
目標達成行動を担う側坐核におけるドーパミン依存性の辺縁系および皮質情報の統合

**■神野　尚三（Shozo Jinno）**

九州大学
海馬 GABA 作動性ニューロンのステレオロジー定量解析

**■坪井　貴史（Takashi Tsuboi）**

理化学研究所
ホルモン分泌顆粒ドッキング機能の分子メカニズムの解明

（50 音順；敬称略）

# 日本神経化学会平成19年度若手奨励賞
# JSN Young Scientists Lectureship Award

**■近藤　慎一 (Shinichi Kondo)**

宮崎大学医学部　解剖学講座分子細胞生物学分野

脳組織における細胞種特異的な小胞体ストレス応答機構

Cell type-specific ER stress response in the brain

**■定方　哲史 (Tetsushi Sadakata)**

独立行政法人理化学研究所　分子神経形成研究チーム

自閉症の発症に関連する遺伝子 CAP2 の解析

Analysis of autism-related gene CAP2/CADPS2

**■津田　　誠 (Makoto Tsuda)**

九州大学大学院薬学研究院薬理学専攻分野

脊髄ミクログリアを介する神経因性疼痛の発症維持メカニズム

Mechanisms underlying neuropathic pain through spinal microglia

# Travel Award (in alphabetical order)

**■ Reyaz Waris Ansari (India)**

Developmental Toxicology Division,Industrial Toxicology Research Centre, Lucknow, India

Session No: P2-g09

Title: Neurobehavioral and neurochemical changes caused by dichlorvos: Role of forced swim stress

**■ Saeid Azizollahi (Iran)**

Department of Veterinary Medicine, Shahid Bahonar University, Iran

Session No: P1-a01

Title: Effects univibrissae rearing on response properties of barrel cortical neurons in C-Fiber-depleted rats

**■ Jin Bao (Germany)**

Department of Membrane Biophysics, Max-Plank Institute for Biophysical Chemistry, Goettingen, Germany

Session No: P2-b07

Title: Information content of synapses with realistic short-term dynamics

**■ Anna M. Barron (Australia)**

School of Psychiatry & Clinical Neurosciences, The University of Western Australia, Perth, Australia

Session No: O2P-F15

Title: Human chorionic gonadotropin increases exploratory behaviour and impairs episodic-like memory in PS1M146V mice.

**■ Weiyan Cai (P.R.China)**

Department of Physiology, Nanjing Medical University, Nanjing, China

Session No: P3-j47

Title: Time dependent progesterone-nouroprotection against ischemic brain damages

**■ Hong Cao (P.R.China)**

Institute of Neurobiology, Fudan University, Shanghai, China

Session No: O1P-K06

Title: Involvement of ERK cascades in the Anterior Cingulate Cortex in Pain-related Negative Emotion in Rat

**■ Sraboni Chaudhury (India)**

Department of Anatomy, All India Institute of Medical Sciences, New Delhi, India

Session No: P3-g22

Title: Altered levels of BDNF protein following prenatal auditory stimulation

**■ Hee Soon Choi (Korea)**

Department of Pharmacology, College of Medicine, Seoul National University, Seoul, Korea

Session No: O2P-D06

Title: Effects of the monomeric, oligomeric and fibrillar A$\beta$42 peptideson the proliferation and differentiation of adult neural stemcells from SVZ

**■ Yulin Dong (P.R.China)**

Department of Anatomy, The Fourth Military Medical University,xi'an

Session No: P2-f26

Title: c-fos expression in the catecholaminergic neurons of the brainstem after eccentric rotation of rats

**■ Vasiliy V. Fedorin (Russia)**

Department of Physiology, Moscow State University, Moscow, Russia

Session No: P1-a02

Title: Low concentration of nicotine triggers a novel regulatory pathway in neuromuscular synapses

**■ Erkang Fei (P.R.China)**

School of Life Sciences, University of Science & Technology of China, Hefei, China

Session No: P1-j13

Title: SUMO-1 modification increases human SOD1 stability and aggregation

**■ Xiaowei Fei (P.R.China)**

Department of Physiology and Biophysics, School of Life Sciences, Fudan University, Shanghai, China

Session No: P3-c13

Title: Modulation by brain natriuretic peptide of rectify outward K+ current on mouse Schwann cells

**■ Abbas Haghparast (Iran)**

Neuroscience Research Center, Shaheed Beheshti Medical University, Tehran, Iran

Session No: O2P-H09

Title: Chronic administration of nicotine retards the development of morphine dependency and tolerance in mice

**■ Huang Jing (P.R.China)**

Department of Anatomy, the Fourth Military Medical University, xi'an, China

Session No: P2-c34

Title: Colocalization of neuronal markers and cell-cycle markers in GABAergic neuron progenitors in the mouse spinal cord

■ **Ashu Johri (India)**

Developmental Toxicology Division, Industrial Toxicology Research Centre, India

Session No: O2P-J04

Title: Imprinting of the cerebral cytochrome P450s and developmental neurotoxicity of lindane

■ **Roger J. Jou (U.S.A.)**

Department of Psychiatry & Investigative Medicine Program, Yale University, New Haven, USA

Session No: O2P-G04

Title: Abnormalities in white matter structure in autism spectrum disorders detected by diffusion tensor imaging

■ **Sharika K.M. (India)**

Systems and Cognitive Department, National Brain Research Centre, Manesar, India

Session No: P3-h02

Title: Predictive control of saccadic eye movements during errors

■ **Rajpal S Kashyap Singh (India)**

Central India Institute Of Medical Sciences, Research Lab, Nagpur, Maharastra, India

Session No: P3-j39

Title: Identification and characterization of protein marker(s) for diagnosis and prognosis of acute Ischemic stroke patients.

■ **Abbas Khani (Iran)**

Neuroscience Research Center, Shaheed Beheshti Medical University, Tehran, Iran

Session No: O2P-H10

Title: Nicotine and morphine interactions; new protocol for morphine dependency in mice

■ **Minji Kim (Korea)**

Department of Pharmacology, College of Medicine, Seoul National University, Seoul, Korea

Session No: P1-c20

Title: Minocycline attenuates neuronal cell death and improves cognitive impairment in Alzheimer's disease models

■ **Tae-Don Kim (Korea)**

Department of Life Science, POSTECH, Pohang, Korea

Session No: P2-g24

Title: Rhythmic control of AANAT translation by hnRNP Q in circadian melatonin production

■ **Vladimir V. Klinshov (Russia)**

Nonlinear dynamics department, Institute of Applied Physics, Nizhny Novgorod, Russia

Session No: O1P-G07

Title: Dynamical model of working memory system

■ **Jaewon Ko (Korea)**

Department of Biological Sciences, Korea Advanced Institute of Science and Technology, Daejeon, South Korea

Session No: P1-a04

Title: Regulation of the differentiation of excitatory synapses by the SALM family of cell adhesion-like molecules

■ **Ji Hyun Ko (Canada)**

Department of Neurology, McGill University, Montreal, Canada

Session No: O2P-G01

Title: The contribution of the dorsolateral prefrontal cortex to executive function

■ **Xue Liu (P.R.China)**

Department of Biomedical Engineering, Shanghai Jiao Tong University, Shanghai, China

Session No: O2P-C06

Title: Contribution of the GABAergic Pathway to the Correlated Activities of Chicken Retinal Ganglion Cells

■ **Chun Wai Ma (Hong Kong)**

Department of Physiology, The University of Hong Kong

Session No: P1-e46

Title: Modifications in perineuronal nets of rat vestibular nuclear neurons during postnatal development and in injury

■ **Panchanan Maiti (India)**

Applied Physiology Division, Defence Institute of Physiology and Allied sciences, Delhi, India

Session No: P3-g23

Title: Hypobaric hypoxia induces neuronal damage and impair spatial memory in rat brain

■ **Ka Pak Ng (Hong Kong)**

Department of Physiology, The University of Hong Kong, Hong Kong, China

Session No: P1-e47

Title: Responsiveness of Thalamic Neurons to Rotational and Translational Motions in the Horizontal Plane

■ **Vasudevan Prabhakaran (India)**

Neurochemistry Laboratory, Department of Neurological Sciences, Christian Medical College and Hospital

Session No: P2-b43

Title: N-glycans govern innate immune response in neurocysticercosis

■ **Rajat Puri (India)**

Department of Biological Sciences and Bioengineering, Indian Institute of Technology, Kanpur, India

Session No: P3-c12

Title: Neurofibrillary tangles in Lafora disease: Functional link between laforin and tau proteins

■ **Arjun R. (India)**

Systems and Cognitive Department, National Brain Research Centre, Manesar, India

Session No: O1P-C02

Title: Neural Control of Saccadic Decision-making

■ **Leila Satarian (Iran)**

Department of Physiology, School of Medical Sciences, Tarbiat Modares University, Tehran, Iran

Session No: P2-j14

Title: Epinephrine inhibits analgesic tolerance to intrathecal morphine while increases the expression of CaM kinase II $\alpha$

■ **Sonali Sengupta (India)**

Department of Biological Sciences and Bioengineering, Indian Institute of Technology, Kanpur, India

Session No: P1-j14

Title: Role of satellite III repeats in stress response and neurodegeneration

■ **Kim Seonghan (Korea)**

Department of Pharmacology, College of Medicine, Seoul National University, Seoul, Korea

Session No: P3-j04

Title: Microglial migration is up-regulated by alpha-synuclein-mediated induction of CD44 and MT1-MMP in Parkinson's disease

■ **Kavita Seth (India)**

Developmental Toxicology Division, Industrial Toxicology Research Centre, Lucknow, India

Session No: O2P-D07

Title: Enhanced Survival of Neural Progenitor Cells Derived TH Positive Neurons under Surveillance of Olfactory Ensheating Cells on Exposure to 6-Hydoxydopamine.

■ **Madhu Singh (India)**

Developmental Toxicology Division, Industrial Toxicology Research Centre, Lucknow, India

Session No: P3-j05

Title: Genetic Polymorphism in susceptibility genes for PD

■ **Nishi Srivastava (India)**

Devlopmental Toxicology Division, Industrial Toxicology Research Centre, India

Session No: P2-c33

Title: Stem cell transplantation in rat model cognitive dysfunction: Neurobehavioral, Neurochemical and Immunohistochemical assessment

■ **Erika Toth (Israel)**

Neurobiology Department, Weizmann Institute of Sciences, Rehovot, Israel

Session No: P1-j48

Title: The Effect of Subconvulsive Electrical Stimulation of the Nucleus Accumbens on Depressive Behavior

■ **Wan-Ting Tseng (Taiwan)**

Institude of Zoology, National Taiwan University, Taiwan, Taipei

Session No: P2-f25

Title: On selectivity of laser-heat irradiation in activation of cutaneous nociceptors

■ **Anusara Vattanajun (Thailand)**

Department of Physiology, Phramongkutklao College of Medicine, Bangkok, Thailand

Session No: P1-f30

Title: Plasticity for functional restoration of forelimb movements following neonatal unilateral decortication.

■ **Lev V. Vdovin (Russia)**

Nonlinear Dynamics Department, Institute of Applied Physics of the RAS, Nizhny Novgorod , Russia

Session No: P2-c10

Title: Map-based model of neural activity with chaotic spike-bursting oscillations

■ **Suyambu Kesava Vijayan Ramaswamy (India)**

Indian Institute of Chemical Biology , Kolkata . India

Session No: P2-k25

Title: A Fast In-silico Protocol for Identification of Anti Epileptic Drug Leads

■ **Oraphan Wanakhachornkrai (Thailand)**

Interdepartment of Physiology, Chulalongkorn University

Session No: P1-c19

Title: Effects of N-(2-propylpentanoyl) urea on impairment of learning and memory induced by common carotid artery occlusion in mice

■ **Hongfeng Wang (P.R.China)**

School of Life Sciences, University of Science & Technology of China, Hefei, China

Session No: P1-j15

Title: p45 targets the polyglutamine disease protein ataxin-3 to the proteasome

■ **Mingmei Wu (P.R.China)**

Institute of Neurosciences, The Fourth Military Medical University, Xi'an, PR China

Session No: P1-d37

Title: NEUROPROTECTIVE EFFECTS OF TRANSPLANTED OLFACTORY ENSHEATHING CELLS ON AXOTOMIZED RETINAL GANGLION CELLS IN ADULT RATS

■ **Hamed Yazdanpanah (Iran)**

Department of Physiology & Pharmacology, Kashan University of Medical Sciences, Kashan, Iran

Session No: P1-k20

Title: A study on impulsivity and choice between delayed reinforcers in T-maze

■ **Wei Zhang (P.R.China)**

Institute of Neuroscience, Chinese Academy of Sciences, Shanghai, China

Session No: P3-k09

Title: Investigate the Function of Neural Circuits in Drosophila Using Light-activating Ion Channels

■ **Yi Zhang (P.R.China)**

Department of Biochemistry, The University of Hong Kong, Hong Kong, China

Session No: P2-d04

Title: Heparanase upregulation in astrocytes and macrophages recruited to the injured spinal cord

# 開催概要

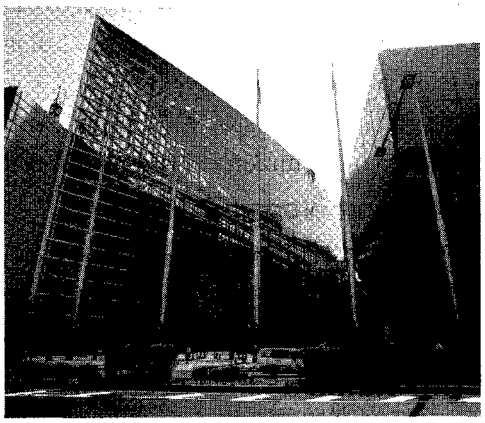
# Neuroscience 2008

The 31st Annual Meeting of the Japan Neuroscience Society

## 第31回日本神経科学大会

**2008年7月9日(水)-11日(金) 東京国際フォーラム**

**大会長：岡野 栄之** 慶應義塾大学医学部生理学 教授



### ◎プログラム概要(予定)

## I. Plenary Lecture

**Professor, Robert C. Malenka**
Department of Psychiatry and Behavioral Sciences
Stanford University School of Medicine

**Professor, Nicole Le Douarin**
Professeur Honoraire au College de France
Secretaire Perpetuelle de l'Academie des Sciences

**Professor, Giacomo Rizzolatti**
Section of Physiology
Department of Neurosciences
Faculty of Medicine and Surgery
University of Parma

**Professor, Sangram S. Sisodia**
Thomas Reynolds Sr. Family Professor of Neurosciences
Director, The Center for Molecular Neurobiology
The University of Chicago

## II. 特別講演

**宮下 保司 先生**
東京大学大学院医学系研究科統合生理学 教授

**田中 啓二 先生**
財団法人東京都医学研究機構 東京都臨床医学総合研究所 所長代行

**藤吉 好則 先生**
京都大学大学院理学研究科 教授

**III. シンポジウム** ― 企画及び公募(36セッション程度) 予定

**IV. 一般口演・ポスターセッション** ― 募集開始:2007年12月　募集締切:2008年2月予定

**V. 機器展示・書籍展示** ― 募集中

**VI. 共催セミナー** ― 共催シンポジウム・ランチョンセミナー・モーニングセミナー募集中

**VII. 市民公開講座** ― 「高校生のための神経科学入門」2008年7月5日(土) 日本科学未来館

### ◎主催事務局

日本神経科学学会
〒113-0033　東京都文京区本郷7丁目2-2 本郷ビル9F
TEL:03-3813-0272　FAX:03-3813-0296

### ◎大会事務局

株式会社コングレ
〒102-8481　東京都千代田区麹町5-1 弘済会館ビル
TEL:03-5216-5318　FAX:03-5216-5552

URL http://www.jnss.org/neurosci2008/　E-mail:neurosci2008@congre.co.jp

# Meeting Outline

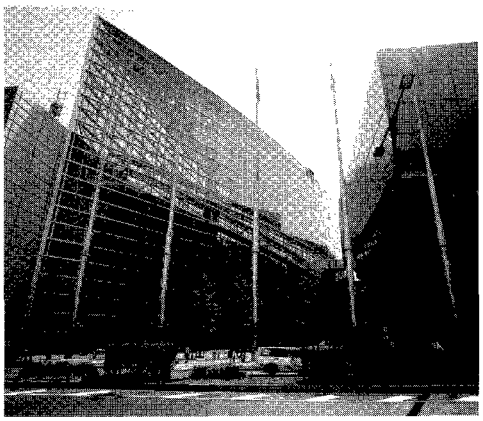
# Neuroscience 2008

The 31st Annual Meeting of the Japan Neuroscience Society

**July 9 (Wed.) - 11 (Fri.) Tokyo International Forum**

**President : Hideyuki Okano, PhD**

Department of Physiology, Keio University, School of Medicine



## Program (tentative)

### I. Plenary Lecture

**Professor, Robert C. Malenka**
Department of Psychiatry and Behavioral Sciences
Stanford University School of Medicine

**Professor, Nicole Le Douarin**
Professeur Honoraire au College de France
Secretaire Perpetuelle de l'Academie des Sciences

**Professor, Giacomo Rizzolatti**
Section of Physiology
Department of Neurosciences
Faculty of Medicine and Surgery
University of Parma

**Professor, Sangram S. Sisodia**
Thomas Reynolds Sr. Family Professor of Neurosciences
Director, The Center for Molecular Neurobiology
The University of Chicago

### II. Special Lecture

**Yasushi Miyashita, PhD**
Department of Physiology, The University of Tokyo School of Medicine

**Keiji Tanaka, PhD**
The Tokyo Metropolitan Institute of Medical Science

**Yoshinori Fujiyoshi, PhD**
Graduate School of Science, Kyoto University

### III. Symposium
Call for symposia: planned (approx. 36 sessions)

### IV. Oral Presentations / Poster Sessions
Call for abstracts open: December, 2007
Deadline: February, 2007 (tentative)

### V. Equipment & Publications Exhibition
Application submission now open

### VI. Sponsored Seminars
Sponsorship application: Now open for Sponsored Symposium, Morning and Lancheon Seminar

### VII. Open Forum
"Introduction of Neuroscience for High School Students"
Saturday, July 5, 2008 at the National Museum of Emerging Science and Innovation

**Travel Awards Available**

**Host Secretariat**

Japan Neuroscience Society
Hongo Building 9F, 7-2-2 Hongo, Bunkyo-ku, Tokyo, 113-0033, Japan
TEL: +81-3-3813-0272 FAX: +81-3-3813-0296

**Secretariat for the 31st Annual Meeting of the Japan Neuroscience Society**

Congress Corporation
Kohsai-kaikan Bldg., 5-1 Kojimachi, Chiyoda-ku, Tokyo 102-8481, Japan
TEL: +81-3-5216-5318 FAX: +81-3-5216-5552

URL http://www.jnss.org/neurosci2008/ E-mail:neurosci2008@congre.co.jp

# 学会からのお知らせ

## 1．評議員会

**日　時：9月11日（火）11時30分～12時00分**

**場　所：5階　第1会場「小ホール（503）」**

日本神経化学会2007年度評議員会議題

「今後の神経化学会大会のあり方について」

## 2．総会

**日　時：9月11日（火）12時00分～13時00分**

**場　所：5階　第1会場「小ホール（503）」**

日本神経化学会2007年度総会議題

1. 平成19年度理事改選結果報告
2. 理事長報告
3. 庶務報告
4. 会計報告
5. 出版・広報報告
6. 各種委員会報告
   - ①将来計画委員会
   - ②脳研究推進委員会
   - ③国際対応委員会
   - ④出版・広報委員会
   - ⑤研究助成金等候補者選考委員会
   - ⑥シンポジウム企画委員会
   - ⑦連合大会委員会
7. 平成19年度奨励賞選考結果報告
8. プログラム編成報告
9. 平成18年度決算の件
10. 同監査報告
11. 平成20年度予算の件
12. 評議員選任の件
13. 名誉会員選任の件
14. 次期大会の件
15. その他

# 日本神経化学会
## 2007年度第1回理事会議事録

**日　時**：2007年4月4日（水）13：00～17：00
**会　場**：（財）国際医学情報センター　2階　第2会議室
**出　席**：遠山正彌（理事長）、池中一裕（副理事長、国際対応委員長）、田代朋子（庶務担当）、井上和秀（会計担当）、柳澤勝彦（出版・広報担当）、米田幸雄、木山博資、仲嶋一範、東田陽博、西川徹、和中明生、岡野栄之、塩坂貞夫、今泉和則、神庭重信（以上、理事）、三木直正（監事）
御子柴克彦（将来計画委員長）、辻省次（脳研究推進委員長）、津田正明（奨励賞選考委員長）、高坂新一（第50回大会長）
**委任状**：佐藤真、島田昌一、鍋島俊隆（以上、理事）宮本英七（監事）
**欠　席**：武田雅俊（理事）

### 議　題

### 【報告事項】

### 1．2006年度第2回理事会議事録承認の件（資料P.1～P.4）
遠山正彌理事長より議場に諮ったところ、一同異議なく承認した。

### 2．選挙結果報告（資料P.5～P.11）
（1）理事改選結果について
平成17年2月2日に開票された理事選挙結果について、田代朋子選挙管理委員から報告がなされた。
当選した馬場明道氏辞退のため、次点の神庭重信氏が繰り上げ当選した。なお、同点の取り扱いについて、細則では「年令順とする」とだけ定められており、昇順・降順の規定がされていないが、昇順と判断し、神庭重信氏を繰り上げ当選とした。他の次点者については、補充理事の就任要請を行う。
「昇順」の取り扱いについては、細則等で規定するよう田代朋子選挙管理委員より提案がなされた。

（2）科研費審査員候補者選出選挙の件
平成17年2月2日に開票された科研費審査員候補者選出選挙結果について、田代朋子選挙管理委員から報告がなされた。
今回より、日本学術振興会（以下、学振と記す）が審査員選出方法を変更した。日本神経化学会（以下JSNと記す）が推薦する候補者が学振に登録され、その中から学振が選出するという手法が取られるため、登録数は多い方が望ましいとの観点から、今回の選挙で選出された候補者の上位20名を推薦することとした。今回の変更に伴い、候補者選出選挙の方法も見直す必要があり、理事会で検討してほしいと田代朋子選挙管理委員より提案がなされた。

### 3．役職理事・委員長選出
（1）理事長・副理事長選出の件
2007年度理事（補充理事除く）12名による投票により、理事長に高坂新一理事（国立精神・神経センター神経研究所所長）、副理事長に池中一裕理事（自然科学研究機構生理学研究所）が選出された（任期は2009年2月末日迄）。

（2）補充理事選出の件
得票数、専門分野および地域性等を考慮し、2007年度理事（補充理事除く）12名による投票を行い、下記の通り補充理事候補を選出した。（敬称略：順不同）
辻　　省次　（東京大学・院）
和田　圭司　（国立精神・神経センター神経研究所）
高橋　正身　（北里大学・医・代謝・蛋白学）

（3）担当理事選任の件

協議の結果、各担当理事（任期2年）を以下の通りに選任した。（敬称略）

庶務担当：　米田　幸雄
会計担当：　木山　博資
出版広報担当：仲嶋　一範

（4）委員会委員長選任の件

協議の結果、各委員会の委員長（任期2年）を以下の通りに選任した。（敬称略）

将来計画委員長：　米田　幸雄
出版広報委員長：　仲嶋　一範
シンポジウム企画委員長：　東田　陽博
国際対応委員長：　和中　明生
研究助成金等候補者選考委員長：神庭　重信
脳研究推進委員長：　辻　省次
奨励賞選考委員長：　津田　正明
連合大会委員長：　池中　一裕（副理事長兼務）

また、各委員長から委員の推薦を受けることと決した。

## 4．庶務報告（資料P.12）

田代朋子庶務担当理事より、庶務報告が行われた。

4名の評議員より定年を機に退会したい旨の申し出がある件につき、定年から70歳までの評議員に対し、「功労会員」のような新たな会員種別を設けてはどうか、また、定年に伴い退会される評議員について、感謝の意を表すようなことができないかとの意見が出され、今期の理事会で検討することとなった。

## 5．会計報告

井上和秀会計担当理事より、2006年度決算書について報告があった。

・収入の部：予算を下回っているが、会費の納入が計画より少なかったためである。しかし、対予算徴収率を見ると、評議員が93%、正会員が85%、学生会員が83%となっており、それほどの落ち込みではないと思われる。
・支出の部：学会誌作成費が若干予算を超過したが、鍋島俊隆第49回大会長より余剰金100万円の寄付がなされたため、大会運営補助費が0となっている。

## 6．出版・広報報告

柳澤勝彦出版広報担当理事より、学会機関誌の編集・発行、学会ホームページの運営、会員検索システムの構築、広報活動について、詳細は委員会報告にて説明する旨の報告がなされた。

## 7．委員会報告

（1）将来計画委員会

御子柴克彦委員長より、次の報告がなされた。

JSNと日本神経科学学会との合同大会をどう行うべきか議論がなされた。合同大会は、お互いの特徴（特にJSNの特徴）が薄れてしまう傾向にあるため、これからは1対1の連合ではなく、複数のゆるやかな連合で大会を開催するべきではないか。JSNは必ずしも規模を大きくする必要はなく、専門的に若手を教育する場となるのがよいのではないかとの意見が出された。

また、国際化の問題について、JSNは世界的にも認められている組織であり、それをアピールしていく必要もあるとの意見が出された。

（2）出版・広報委員会

・学会機関誌の編集・発行（柳澤勝彦委員長）

『神経化学』2007年第1号を4/5に発送。その他、従来と変更なしとの報告がなされた。

・会員検索システムの構築（仲嶋一範理事）

E-mail配信は、71%程度同意を得られており、すでに機能しているが、会員検索システムについては、個人情報管理の問題があり、充分機能しているとはいえないとの報告がなされた。

・学会HPの管理・運営（島田昌一理事）

学会ホームページの運営を学会事務局への移管が完了したとの報告がなされた。英語版ホームページについては、基本的な情報は英訳できているが、外国人の入会を見込んでホームページを作成する必要があり、外国人会員に対する規約が設けられていないため、今後対応が必要とのことであった。また、一般市民や中高生・学部学生を対象とした広報活動もホームページで行っていきたいとし、広報活動の原稿を現在準備中である旨報告がなされた。

・広報活動（木山博資理事）

ホームページ上に「神経化学トピックス」としてコンテンツを掲載する企画を進めているが、版権管理等、コンテンツの運営を出版・広報委員会で行っていくべきについて、理事会の判断を仰ぎたいとの意見が出された。

ISNのホームページにてJSNの学会活動が紹介されており、ISNのホームページからJSNのホームページへリンクが設定されているとの報告がなされた。

また、50周年記念事業に際し、他委員会と共になにか活動すべきかについて協議してはどうかとの意見が木山博資理事より出された。

（3）脳研究推進委員会

辻省次委員長より、将来計画委員会と合同で脳研究のあり方について、幅広い議論ができた。

会員アンケートについては、結果がまとまり次第最終報告する旨報告がなされた。

（4）国際対応委員会

池中一裕委員長より、ISNの理事選で田代理事がノミネートされており、ISN会員はぜひ投票願いたいとの報告がなされた。

（5）研究助成金等候補者選考委員会

佐藤真委員長欠席につき、報告なし。

（6）シンポジウム企画委員会

東田陽博委員長より、昨年の活動として、50周年大会の招待講演者をProf. Heinrich Betzに決定した。今年の活動として、2/28に「日本神経化学会2007冬の学校」を金沢大学で行い、15名程度の参加者があったとの報告がなされた。

（7）奨励賞選考委員会

津田正明委員長より、応募者が少ないため、基本的には自主的な応募を待つが、状況によっては大会の座長に推薦を依頼することも予想されるとの報告がなされた。また、最優秀奨励賞の英語名について、奨励賞選考委員会で検討することとなった。

## 8．第50回（2007年度）大会の件

髙坂新一理事長より次の報告がなされた。

本年度は9月10日から12日までパシフィコ横浜にて3学会合同大会となっている。9月9日に理事会、9月10日に50周年記念式典およびプレナリー講演、9月11日に評議員会および総会が行われる予定である。その他、大会の流れについて説明がなされた。

田代前庶務担当理事より、50周年記念ブースについて報告がなされ、準備委員として熊倉委員、高橋委員、馬場委員、木山委員が就任することが報告され、さらに仲嶋委員を加え、準備を継続していくこととした。

50周年記念号発行については、協議の結果、予算面も確認しながら、一括購入する方向で調整することとなった。また、スポンサーをつけ無料配布をする案も出され、今後検討していくこととなった。

日本神経科学学会との関係、JSNのあり方について議論がなされ、髙坂理事長より、日本神経科学学会との関係については、将来計画委員会等で今後も十分議論すべきとの意見が出された。また、神経内科系の評議員を増やすべく、次回の理事会で協議することとなった。

科研費審査において、JSN会員が多く採択されるよう対策を講じる必要があるとの意見も出された。

## 9．その他

遠山理事長より、退任の挨拶がなされた。

**【審議事項】**

## 1．2006年度奨励賞受賞者の評議員推薦の件

奨励賞受賞者については、自動的に評議員となることが確認され、3名が承認された。

## 2．新評議員推薦の件（参考資料：現評議員名簿）

2名の候補者のうち、1名の推薦を承認、1名を次回の理事会にて再審査することとなった。

## 3．名誉会員推薦の件（参考資料：現名誉会員名簿）

次回理事会において候補者を審査し、候補者を総会へ推薦することが承認された。

## 4．第51回（2008年度）大会について

武田理事欠席のため、武田理事よりメールにて、以下の報告がなされた。

・2008年9月11日から9月13日まで富山市で行う
・日本生物学的精神医学会との合同で開催する

日本神経精神薬理学会を含めた3者連合で行われることが確認され、髙坂理事長より、開催地について日本神経精神薬理学会 山脇成人大会長へ最終確認することとなった。

また、武田理事よりJSNの発表を全て英語で行ってはどうかとの提案がなされ、武田理事が将来計画委員会、連合大会委員会と協議することとなった。

## 5．第52回（2009年度）大会について

田代前庶務担当理事が大会長として承認され、日本生物学的精神医学会と日本神経精神薬理学会に高坂理事長から報告することとなった。また、第53回（2010年）については、JSNの独自性を出すため、日本神経科学学会に「ゆるやかな連合」での開催を提案することとなった。

## 6．評議員再任の見直し、退会について

退会希望の評議員について協議がなされ、慰留に努めることとなった。また、功労会員についても議論がなされ、次回総会において70歳以上の評議員については、功労会員として推薦することを確認し、定年退職・定年退官した70歳未満の評議員についても、功労会員として慰留していくことと決した。

## 7．その他

・英語版学会ホームページの件について、翻訳の専門家に監修を依頼し、それに伴う費用は会計担当理事と相談する旨確認がなされ、ホームページ上の「神経化学トピックス」については、出版・広報委員会内に必要に応じてワーキンググループを立ち上げ、活動することとなった。
・賛助会員を積極的に募るよう努めていく旨意見が出された。
・外国人の入会について、発展途上国の方と先進国の方との会費が同額でよいのか等学会費の問題が提起され、庶務担当理事を中心として検討することとなった。
・柳澤前出版・広報委員長より、学会誌も翻訳されていないため対応が必要であることと、アジア圏での会員を増やしていくよう意見が出された。
・JSNの独自性を対外的にどうアピールするかについて、活発な議論が交わされ、今後も重要なテーマとして扱っていくこととされた。

以上を以て予定した全ての議事を終了し、本年度第1回理事会を閉じた。

# 日本神経化学会
## 2007年度第2回理事会議事録

**日　時**：2007年5月31日（木）13：30～17：30
**会　場**：慶應義塾大学医学部総合医科学研究棟5階　会議室5
**出　席**：髙坂新一（理事長、第50回大会長）、池中一裕（副理事長、連合大会委員長）、米田幸雄（庶務担当、将来計画委員長）、木山博資（会計担当）、仲嶋一範（出版・広報担当）、御子柴克彦（理事）、塩坂貞夫（理事）、今泉和則（理事）、武田雅俊（理事、第51回大会長）、東田陽博（理事、シンポジウム企画委員長）、辻省次（理事、脳研究推進委員長）、和田圭司（理事）、高橋正身（理事）、宮本英七（監事）、田代朋子（第50回大会記念ブース担当、第52回大会長、奨励賞選考委員）
**委任状**：岡野栄之（理事）、和中明生（国際対応委員長）、三木直正（監事）
**欠　席**：神庭重信（理事、助成金等候補者選考委員長）、津田正明（奨励賞選考委員長）

### 議　題

### 【報告事項】

### 1．2006年度第2回理事会議事録承認の件（資料P.1～P.5）
髙坂新一理事長より議場に諮ったところ、一同異議なく承認した。

### 2．理事長報告
補充理事委嘱について、評議員へ文書で確認したところ、過半数が賛成であったことが報告された。また、各委員会委員長より委員の推薦がなされ（別添資料）、承認された。ただし、奨励賞選考委員会については、「日本神経化学会奨励賞内規」に基づき、後日、委員長選出手続きを行うことが確認された。

### 3．庶務報告（資料P.6～P.9）
米田幸雄庶務担当理事より、庶務報告が行われた。
・年会費を長期滞納している会員に対し、どのような対応を取るか審議することとなった。
・退会を希望している評議員の慰留について、報告がなされた。
・物故会員について報告がなされ、総会にて黙祷をささげることが確認された。
・Neurochemistry International誌50周年記念号の準備について進捗状況が報告され、池中一裕副理事長よりJ. Neurochemistry誌50周年記念号の準備について、進捗状況が報告された。また、買い上げについても検討され、財政面を考慮しながら引き続き業者と交渉していくこととなった。

### 4．会計報告（資料P.10）
木山博資会計担当理事より、名誉会員3名から2007年度年会費として誤入金があり、うち2名については返金したが、1名については「返金不要」との申し出があったことが報告された。

### 5．出版・広報報告（資料P.11）
仲嶋一範出版広報担当理事より、学会誌の編集・発行について説明がなされた。その他、学会ホームページの運営、会員検索システムの構築、広報活動については、委員会報告にて説明する旨の報告がなされた。

### 6．委員会報告
（1）将来計画委員会
米田幸雄委員長より、大会期間中に委員会を開くことが報告された。また、大会・学会のあり方について、若手や臨床系研究者等へアピールしていくことが重要ではないかとの意見が出された。
また、髙坂新一理事長より、第53回大会に関し、次回理事会で方向性をつけてほしい旨意見がなされ、脳研究推進委員会と共に具体策を検討することとなった。

（2）出版・広報委員会

次の通り報告がなされた。

・会員検索システムの構築（仲嶋一範委員長）

E-mail 配信は、全会員の約 73%の同意を得られ、会員検索システムについては、約 66%の同意を得られており、少しずつ増えてはいる。回答があった会員に限ると、それぞれ約 94%、85%の同意が得られていることから、今後は未回答の会員（全会員の約 22%）からいかに意思表示をしていただくかが課題となる。また、会員検索システムに各会員の自主的判断で研究室ホームページの URL を掲載できる欄を新たに設けてはどうかと提案が出され、了承された。

・学会ホームページの管理・運営（仲嶋一範委員長）

企業等から学会ホームページ及び学会誌への広告掲載依頼があった場合の対応について検討していただきたいとの意見がなされ、審議することとなった。

・広報活動（今泉和則委員）

今年の活動として次の通り報告がなされた。

①一般向け広報活動：ホームページ上に「神経化学トピックス」としてコンテンツを掲載する企画が前委員会で決定されたので、運用へ動き出したい。

②ISN に対する広報活動：ISN のホームページから日本神経化学会（以下、JSN と記す）のホームページへリンクが設定されているが、役員名簿しか閲覧できないのが現状であるため、今後はより充実した情報を掲載するよう検討が必要である。

③JSN の歴史や概要に関する紹介文の掲載：大会時に催される 50 周年記念ブースの内容を盛り込み、ホームページ上に掲載することを企画している。また、田代朋子 50 周年記念ブース担当より、大会終了後に同ブースで使用した資料のうち、掲載可能なものに関しては提供されることとなった。

（3）シンポジウム企画委員会

東田陽博委員長より、2007 年は幕張で開催するとの報告がなされ、2008 年については、内容に関して具体的な項目が挙がってきてから検討していくとのことであった。

（4）国際対応委員会

池中一裕副委員より報告がなされ、ISN の理事選で田代朋子氏が当選されたとの報告がなされた。

（5）研究助成金等候補者選考委員会

神庭重信委員長が欠席のため、第 27 回猿橋賞推薦結果について文書で報告された。

（6）脳研究推進委員会

辻省次委員長より、いかに学会を若手や臨床系研究者等へアピールしていくかについて、将来計画委員会と共に議論していくとの報告がなされた。

（7）奨励賞選考委員会

田代朋子委員より応募者数について報告がなされた。

応募者が少ないため、評議員に推薦を依頼することとなった。前理事会で決定された大会の座長への推薦依頼は、池中一裕副理事長が行うことと決した。

（8）連合大会委員会

池中一裕委員長より報告がなされ、ゆるやかな連合を継続していくため、様々な学会に窓口を設けてもらい、話し合いの場を持ちたいとの意見が出された。また、各学会の組合のような受け皿となる組織がほしいとの意見も出された。差し当たり、高坂新一理事長より日本生物学的精神医学会や日本神経精神薬理学会へ、学会の窓口を設けるよう依頼済みであることが確認された。

## 8．第50回（2007年度）大会について（資料1）

和田圭司実行委員長より、プログラムが確定した旨報告され、セッション・演題数等について説明がなされた。

髙坂新一第50回大会長よりスケジュールや会場について説明がなされた。また、50周年記念ブースにおけるキャッチフレーズについても提案がなされ、「討論を大切にする学会」、「専門店を目指す学会」、「学会員を大切にする手作りの学会」等が挙げられた。

田代朋子50周年記念ブース担当より、50周年記念ブースの準備状況について報告され、展示方法や特集号の買い取りについて議論がなされた。BlackwellとElsevierに展示ブースを無料提供し、バーターとして記念号等の無料提供を受けてはどうかとの提案がなされ、交渉していくこととなった。

**【審議事項】**

## 1．新評議員の推薦について（資料P.13）

4名の候補者のうち、3名の推薦を承認し、1名に対し審査を継続することとなった。

## 2．名誉会員の推薦について（資料P.14）

2名の候補者の推薦を承認し、総会へ推薦することが承認された。

なお、2名のうち宮本英七氏については、監事職満了時の総会にて推薦することが確認された。

## 3．年会費長期未納者に対する取扱いについて（資料P.15）

年会費を3年以上納入していない会員に対しては、退会処分を実行することとなった。次回理事会にて長期未納者リストを基に協議することとし、その際、具体的な手続きについても検討することとなった。

## 4．学会誌及び学会ホームページにおける広告掲載について

企業広告の掲載について審議がなされ、学会誌には記事中のみ掲載可能とし、掲載料1ページ5万円とすることとした。また、学会ホームページにて告知を行い、広告を募ることとなった。一方、学会ホームページへの広告掲載については、当面行わないことと決した。

## 5．第51回（2008年度）大会について（資料P.16～P.17）

武田雅俊第51回大会長より、準備状況について報告がなされた。使用言語について議論され、「シンポジウムは全て英語、オーラルプレゼンテーションは日本語・英語どちらでも使用可（ポスター、スライドは英語）」とされた。また、ディスカッションは日本語で行うのが適当ではないかとの提案がなされ、今後将来計画委員会で検討することとなった。また、会費についても議論がなされ、具体的な金額については引き続き検討することとなった。

## 6．第52回（2009年度）大会について

田代朋子第52回大会長より報告がなされた。どの学会と合同開催するかについて議論がなされ、日本生物物理学会が候補に挙がり、次の理事会までに田代大会長が打診することとなった。

## 7．評議員の退会希望について（資料P.18～P.21）

7名の評議員から退会希望の申し出があり、4名については承認、3名については功労会員として慰留することと決した。また、評議員のあり方について議論がなされ、定数を増やしてはどうかとの意見が出されたが、当面は変更しないこととなった。

## 8．学会誌の価格について（資料P.22）

第50回大会においては、抄録号の販売は行わないことが確認された。販売を求められた場合は、JSNへの入会を促すこととされた。

## 9．第 50 回大会における名誉会員の表彰について（資料 P.23）

記念式典において感謝状を贈呈することが決定した。記念品についても議論がなされ、今後も引き続き検討していくこととなった。

## 10．国立大学教育研究評価委員会専門委員候補者の推薦について（資料 P.24 ～ P.30）

理事が評議員の中から推薦し、それを踏まえ髙坂新一理事長と池中一裕副理事長で検討することとなった。

## 11．その他

・名誉会員からの入金について

会計報告にて挙げられた誤入金の件につき、「返金不要」との申し出があった 1 名分の取扱いについて審議され、寄付として申し受けることに決した。

・第 50 回大会の記念行事特別予算について

特別展示、特集号や記念品等 記念行事にかかる経費について、特別事業積立金から上限を 100 万円として予算化することを審議し、承認された。

以上を以て予定した全ての議事を終了し、本年度第 2 回理事会を閉じた。

# MEMO

# MEMO

平成19年8月

日本神経化学会
会員各位

日本神経化学会
理事長　髙坂　新一

# ＊第50回日本神経化学会・総会の開催について＊

謹　啓

時下ますますご清祥のこととお慶び申し上げます。

さて、下記の通り日本神経化学会総会を開催致したくご案内申し上げます。

尚、総会に欠席される方は委任状（ハガキ）に必要事項をご記入の上、事務局宛にご送付下さるか、学会当日、受付までご提出下さいますようお願い申し上げます。

敬　白

記

日　時　：　平成19年9月11日（火）　12：00～13：00

場　所　：　5階　第1会場「小ホール（503）」

議　題　：（予定）

1. 2007年度理事改選結果報告
2. 理事長報告
3. 庶務報告
4. 会計報告
5. 出版・広報報告
6. 各種委員会報告
7. 2007年度奨励賞選考結果報告
8. プログラム編成報告
9. 2006年度決算の件
10. 同監査報告
11. 2008年度予算の件
12. 評議員選任の件
13. 名誉会員選任の件
14. 次期大会の件
15. その他

（切り離してご投函ください）

平成19年
日本神経化学会
総会

委任状

（ご欠席の方はご記入ください）

平成19年日本神経化学会総会の議決権の行使に関する一切の権限を

代理人＿＿＿＿＿＿＿＿に委任します。

平成19年　月　日

所属

氏名　　㊞

郵便はがき

料金受取人払郵便

新宿局
承認
6881

差出有効期限
平成20年8月
9日まで

160-8792

376

東京都新宿区信濃町35番地
信濃町煉瓦館

(財)国際医学情報センター内

日本神経化学会 行

# プログラム Program

## プレナリーレクチャー
Plenary Lectures

## 時実利彦記念賞受賞講演
Tokizane Award Lecture

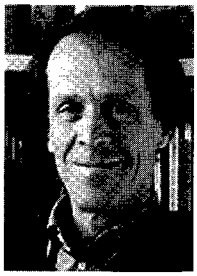
## 塚原仲晃記念賞受賞講演
Tsukahara Award Lectures

## シンポジウム
Symposia

## 日本神経化学会　若手奨励賞シンポジウム
JSN Young Scientists Lectureship Award Symposium

## ランチョンセミナー
Luncheon Seminars

# プレナリーレクチャー Plenary Lectures



## PL1 Glycine neurotransmission: from molecules to mice and disease

September 10 (Mon) 18:15-19:15 Plenary Room

Heinrich Betz
Department of Neurochemistry, Max-Planck-Institute for Brain Research, Frankfurt, Germany

Chairperson：御子柴克彦（Katsuhiko Mikoshiba）



## PL2 Signaling networks that control synapse development and cognitive function

September 11 (Tue) 18:15-19:15 Plenary Room

Michael E. Greenberg
Neurobiology Program, Children's Hospital Boston, and Harvard Medical School, Boston, USA

Chairperson：田中 啓治（Keiji Tanaka）



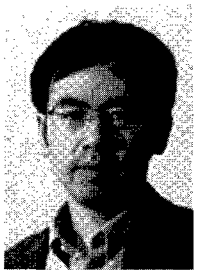
## PL3 Frontal lobe mechanisms for rules and control

September 12 (Wed) 14:00-15:00 Plenary Room

Earl K. Miller
The Picower Institute for Learning and Memory, RIKEN-MIT Neuroscience Research Center and Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, USA

Chairperson：大森 隆司（Takashi Omori）

# 時実利彦記念賞受賞講演　Tokizane Award Lecture



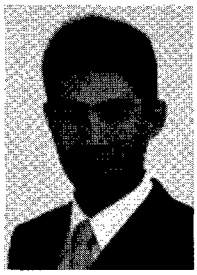
## SL1　アミロイドβペプチド代謝とアルツハイマー病
## Metabolism of amyloid β peptide and Alzheimer's disease

September 10 (Mon)　14:00-14:40　Room A

西道　隆臣（Takaomi C. Saido）
理化学研究所　脳科学総合研究センター

Chairperson：西野　仁雄（Hitoo Nishino）

# 塚原仲晃記念賞受賞講演　Tsukahara Award Lectures

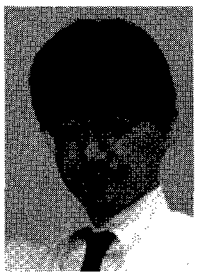


## SL2　強化学習の脳機構の探求
## In pursuit of the brain mechanism of reinforcement learning

September 10 (Mon)　14:40-15:20　Room A

銅谷　賢治（Kenji Doya）
沖縄科学技術大学院大学先行研究

Chairperson：川人　光男（Mitsuo Kawato）



## SL3　脳神経系の構造・機能を可視化する蛍光タンパク質技術
## Innovations in fluorescence imaging of brain functions using fluorescent proteins

September 10 (Mon)　15:20-16:00　Room A

宮脇　敦史（Atsushi Miyawaki）
理化学研究所　脳科学総合研究センター

Chairperson：岡野　栄之（Hideyuki Okano）

## Symposium 1A-A 8:30-11:00 Room A

### 興奮—抑制バランス：シナプス・回路・システム
### Excitatory-Inhibitory Balance: Synapses, Circuits, Systems

Chairpersons Takao Hensch(理化学研究所脳科学総合研究センター)
Arthur Konnerth(Munich Tech)

S1A-A1 8:30
皮質局所神経回路における興奮・抑制結合の多様性
Distinct excitatory and inhibitory subnetworks of the cortical microcircuit
川口 泰雄(Yasuo Kawaguchi)
自然科学研究機構・生理研・大脳神経回路論

S1A-A2 9:00
Disynaptic inhibition between neocortical pyramidal cells
Henry Markram
EPFL Brain Mind Institute, Lausanne, Switzerland

S1A-A3 9:30
視覚野における新しいタイプの錐体細胞間抑制性結合
A novel form of inhibitory connections between nearby neocortical pyramidal cells
小松由紀夫(Yukio Komatsu)
名古屋大学・環境医学研究所・視覚神経科学

S1A-A4 10:00
Neuronal signaling patterns in the developing mouse visual cortex in vivo
Arthur Konnerth
Institute of Neuroscience, Technical University Munich, Germany

S1A-A5 10:30
Inhibitory circuit control of critical period plasticity
Takao K. Hensch
RIKEN BSI

## Symposium 1A-B 8:30-11:00 Room B

### 霊長類モデル研究は神経科学に何を寄与するか？
### What is Contributed by the Primate Model?

Chairpersons Kathleen S. Rockland(理化学研究所脳センター)
船橋新太郎（Shintaro Funahashi）（京都大学人間・環境学研究科）

S1A-B1 8:30
注意欠陥 / 多動性障害（ADHD）の霊長類モデル
Primate model of attention deficit/hyperactivity disorders (ADHD)
船橋新太郎[1](Shintaro Funahashi)，清水 慶子[2](Keiko Shimizu)
[1]京都大学院・人間環境学・認知行動科学，[2]京都大・霊長類研

S1A-B2 9:00
急性および日内性精神疲労による行動と脳活動の変化：サルを用いた PET 実験
Changes in behavior and brain activity by acute and diurnal mental fatigue: PET study with macaque monkeys
尾上 浩隆[1](Hirotaka Onoe)，尾上 嘉代[1](Kayo Onoe)，塚田 秀夫[2](Hideo Tsukada)，
渡辺 恭良[1](Yasuyoshi Watanabe)
[1]（独）理化学研究所 分子イメージング研究プログラム，[2]浜松ホトニクス株式会社 PETセンター

S1A-B3 9:30
Effects of early experience on visual performance
Yoichi Sugita
Neuroscience Research Institute, AIST, Tsukuba, Japan

S1A-B4 10:00
サル側頭皮質の2領域における遺伝子発現プロフィール
Gene expression profiles in two areas of monkey temporal cortex
一戸　紀孝(Noritaka Ichinohe), Marie Wintzer, 宮下　俊雄(Toshio Miyashita), Kathleen Rockland
理研・脳総研・脳皮質

S1A-B5 10:30
Nonhuman primate models of normal social behavior, neurodevelopmental disorders and functional reorganization following early brain Damage
David G Amaral
The M.I.N.D. Institute, UC Davis, Sacramento, CA. USA

## Symposium 1A-C　8:30-11:00　Room C

## 社会的認知と意思決定：神経心理学者と神経科学者のクロストーク
## Social Cognition and Decision-making: Cross-talk between Neuropsychologists and Neuroscientists

Chairpersons　河村　満 (Mitsuru Kawamura)（昭和大学神経内科）
坂上　雅道 (Masamichi Sakagami)（玉川大学学術研究所）

S1A-C1 8:40
パーキンソン病における意思決定の障害
Impaired decision making in individuals with Parkinson's disease
三村　將(Masaru Mimura)
昭和大・医・精神

S1A-C2 9:05
パーキンソン病患者の社会的認知と意思決定
Impaired recognition of the facial expressions in patients with Parkinson's disease
小山　慎一[1](Shinichi Koyama), 河村　満[2](Mitsuru Kawamura), 三村　將[2](Masaru Mimura),
坂上　雅道[3](Masamichi Sakagami), 鈴木　敦命[4](Atsunobu Suzuki), Hackjin Kim[5], Peter Bossaerts[5]
[1]千葉大学院・工学, [2]昭和大学・医・神経内科, [3]玉川大学, [4]イリノイ大学, [5]カリフォルニア工科大学

S1A-C3 9:30
表情認知の多次元性に対する新たな神経心理学的証拠
Novel neuropsychological evidence for multidimensionality of facial expression recognition
鈴木　敦命[1](Atsunobu Suzuki)
[1]イリノイ大・ベックマン研究所, [2]東京大・ジェロントロジー寄付研究部門

S1A-C4 9:55
Functionally and temporally dissociable neural processes underlying social decision-making on faces
Hackjin Kim[1], Mitsuru Kawamura[2], Masamichi Sakagami[3], Masaru Mimura[2], Shinichi Koyama[2], Atsunobu Suzuki[4], Peter Bossaerts[1]
[1]Division of Humanities and Social Sciences, Caltech, Pasadena, USA., [2]Showa Univ., [3]Tamagawa Univ., [4]University of Illinois

S1A-C5 10:20
Human Imagination in Financial Markets with Insiders
Peter L. Bossaerts[1,2], Antoine Bruguier[1], Steve Quartz[1]
[1]Computational and Neural Systems, California Institute of Technology, Pasadena (CA), USA, [2]Swiss Finance Institute

## Symposium 1A-D　8:30-11:00　Room D

## シナプス形成と機能獲得
## Synapse Formation and Functional Maturation

Chairpersons　柚崎　通介 (Michisuke Yuzaki)（慶應義塾大学医学部生理学）
岡部　繁男 (Shigeo Okabe)（東京医科歯科大学）

S1A-D1 8:30
海馬錐体細胞と介在細胞におけるグルタミン酸作動性シナプス後部の分子構築と動態の相違
Differential organization and remodeling of the glutamatergic postsynaptic specializations between excitatory and inhibitory neurons
岡部　繁男(Shigeo Okabe)
東京医科歯科大院・医歯学・細胞生物

S1A-D2 シナプスを創る分子
9:05 Finding the presynaptic organizers
梅森　久視(Hisashi Umemori)
ミシガン大学医学部

S1A-D3 Cbln ファミリー分子—シナプス前部より放出されシナプス形成と可塑性を誘導する新しい分子群の解析
9:40 Cbln family proteins-characterization of anterogradely transported molecules regulating synapse formation and plasticity
柚崎　通介(Michisuke Yuzaki)
慶應大・医・生理

S1A-D4 Development and plasticity of functional odor inputs into the olfactory bulb
10:15 Venkatesh N Murthy, Dinu F Albeanu, William J Tyler, Tomokazu F Sato, Edward R Soucy, Markus Meister
Dept. of Molecular & Cellular Biology, Harvard University, Cambridge, USA.

**Symposium 1A-E** 8:30-11:00 Room E

## 神経細胞死の制御の新展開
## Recent Advance of Regulatory Mechanisms of Neuronal Cell Death

Chairperson　三浦　正幸（Masayuki Miura）（東京大学大学院薬学系研究科）

演題番号（*）は口演からの繰込

S1A-E1 神経発生における Apaf-1 依存性カスパーゼ活性の生理機能
8:30 Physiologcal roles of Apaf-1-mediated caspase signaling in neural development
三浦　正幸(Masayuki Miura)
東京大院・薬・遺伝

S1A-E2 鳥類胚の頚髄で起こる早期の運動神経細胞死によって、胸髄の肋間筋を支配する運動神経に相当する細胞群が取り除かれる。
8:55 Early motoneuron death in the cervical cord of the avian embryo occurs to eliminate a specific subgroup of motoneurons.
八木沼洋行(Hiroyuki Yaginuma)
福島医大・医・神経解剖発生

S1A-E3 神経栄養因子シグナル不全により誘導されるカスパーゼ非依存性神経細胞死
9:20 Caspase-independent neuronal death triggered by abolition of neurotrophic signals
榎本　秀樹(Hideki Enomoto)
理化学研究所　発生・再生科学総合研究センター

S1A-E4* Pael-R transgenic mouse crossed with parkin null mouse displays persistent endoplasmic
9:45 reticulum stress, reduction in complex I activity and dopaminergic neuronal death
Ryosuke Takahashi[1], Hua-Qin Wang[1], Yuzuru Imai[2], Haruhisa Inoue[1], Ayane Kataoka[3], Sachiko Iita[3], Nobuyuki Nukina[3]
[1]Department of Neurology, Kyoto University Graduate School of Medicine, [2]Tohoku University, [3]RIKEN

S1A-E5* プロサイモシンα1：ネクローシスからアポトーシスへの細胞死モードスイッチ因子の同定
10:10 Identification of prothymosin-alpha1, the necrosis-apoptosis switch molecule under the in vitro ischemia condition
植田　弘師(Hiroshi Ueda), 藤田　亮介(Ryousuke Fujita), 吉田　明(Akira Yoshida),
松永　隼人(Hayato Matsunaga), 植田　睦美(Mutsumi Ueda)
長崎大・院医歯薬学総合・分子薬理

S1A-E6 低酸素脳虚血負荷による海馬錐体神経細胞死へのオートファジーの関わりについて
10:35 Participation of autophagy in pyramidal neuron death of the hippocampus after hypoxic ischemic brain injury
内山 安男(Yasuo Uchiyama)
大阪大・医・神経生物学

## Symposium 1A-F 8:30-11:00 Room F

### エピジェネティクス異常に起因する精神発達障害疾患の理解
### Understanding of Defects in Epigenetics in Neurodevelopmental Diseases

Chairpersons 久保田健夫 (Takeo Kubota) (山梨大学大学院医学工学総合研究部環境遺伝医学講座)
伏木 信次 (Shinji Fushiki) (京都府立医科大学大学院医学研究科分子病態病理学)

S1A-F1 ゲノム刷り込み異常に起因するてんかん性疾患 (Angelman 症候群)
8:45 An epileptic disease with genomic imprinting disorder: Angelman syndrome
斉藤 伸治(Shinji Saitoh)
北海道大学・医・小児科

S1A-F2 クロマチン再構成異常に起因する重度精神遅滞 (ATR-X 症候群)
9:15 A severe mental retardation disease with defect in chromatin remodling: ATR-X syndrome
和田 敬仁(Takahito Wada)
信州大学・医・遺伝医学予防医学

S1A-F3 ヒストン蛋白メチルトランスフェラーゼ異常に起因する脳性巨人症 (Sotos 症候群)
9:45 Cerebral gigantism with histone metyl transferase abnormality : Sotos syndrome
松本 直通(Naomichi Matsumoto)
横浜市大院・医・環境分子医科学

S1A-F4 精神発達障害におけるエピジェネティクスのオーバービュー
10:15 Overview of epigenetic disorders in neurodevelopmental diseases
久保田健夫(Takeo Kubota), 楊 春妹(Chunshu Yang), 五月女雅樹(Masaki Soutome),
遠藤 和志(Kazushi Endoh)
山梨大院・医・環境遺伝医学

S1A-F5 精神発達障害のエピジェネティクス研究に期待するもの
10:45 What we do for neurodevelopmental diseases with epigenetic disorders
伏木 信次(Shinji Fushiki), 矢追 毅(Takeshi Yaoi), 中村 惠子(Keiko Nakamura), 伊東 恭子(Kyoko Itoh)
京都府立医科大学院・分子病態病理学

## Symposium 1A-G 8:30-11:00 Room G

### 扁桃体を中心とする情動系の形成と機能の制御メカニズム
### Amygdala and Related Emotional System-control Mechanisms of their Development and Functions

Chairperson 湯浅 茂樹 (Shigeki Yuasa) (国立精神・神経センター神経研究所微細構造研究部)

S1A-G1 扁桃体と情動神経回路の発生
8:40 The development of amygdala and emotional neural circuit
湯浅 茂樹(Shigeki Yuasa), 相馬 美歩(Miho Soma)
国立精神・神経センター・神経研・微細構造

S1A-G2 NMDA 受容体のチロシンリン酸化による扁桃体機能の制御
9:10 Regulation of amygdala functions by NMDAR tyrosine phosphorylation
中澤 敬信(Takanobu Nakazawa), Mina Delawary, 山本 雅(Tadashi Yamamoto)
東京大・医科研

S1A-G3 9:40 ジストロフィン欠損マウスにおける扁桃体 GABA シナプスの損傷と情動行動の亢進
Impaired amygdala GABAergic synapses and enhanced emotional behavior in dystrophin-deficient mdx mice
関口　正幸(Masayuki Sekiguchi)
国立精神神経セ・神経研究所・四部

S1A-G4 10:10 Modulation of hippocampal synaptic plasticities by the activities of the amygdala
Norio Matsuki, Yuji Ikegaya
Lab of Chem Pharmacol, Grad Sch of Pharmaceut Sci, University of Tokyo, Tokyo, Japan

## Symposium 1A-H　8:30-11:00　Room H

### ニューロインフォマティクスの国際展開
### International Collaborations on Neuroinformatics

Chairpersons　臼井　支朗（Shiro Usui）（理化学研究所ニューロインフォマティクス技術開発チーム）
大森　隆司（Takashi Omori）（玉川大学学術研究所）

S1A-H1 8:50 マウス小脳発達トランスクリプトームデータベース
Cerebellar development transcriptome database
古市　貞一(Teiichi Furuichi)
理研・脳センター・分子神経形成

S1A-H2 9:20 Large scale analysis of gene expression in CNS: a rapidly expanding collaborative landscape
Robert W. Williams
Dept. of Anatomy & Neurobiology, University of Tennessee, Memphis TN 38120, USA

S1A-H3 9:50 ニューロングリアプラットフォーム：細胞・局所回路網レベルのニューロインフォマティクス
Neuron-Glia Platform: Neuroinformatics at the cellular and local network level.
宮川　博義(Hiroyoshi Miyakawa)
東京薬科大学・生命科学部・脳神経機能学

S1A-H4 10:20 Large scale simulations of cortical neuronal networks
M Diesmann, M_O Gewaltig, A Morrison, H E Plesser
RIKEN Brain Science Institute

## Symposium 1A-J　8:30-11:00　Room J

### 親子関係の神経基盤
### Neural Basis of Parent-Infant Relationship

Chairpersons　小川　園子（Sonoko Ogawa）（筑波大学大学院人間総合科学研究科行動神経内分泌学研究室）
菊水　健史（Takefumi Kikusui）（東京大学大学院農学生命科学研究科動物行動学研究室）

S1A-J1 8:30 早期離乳ストレスによる神経行動学的変異
Neurobehavioral influences by early weaning stress
菊水　健史(Takefumi Kikusui)
東京大学院・農・動物行動

S1A-J2 9:05 マウス養育行動を制御する MPOA 内シグナル伝達経路の解析
Intracellular signaling mechanism regulating mouse parental behavior in the medial preoptic area
黒田　公美[1](Kumi_O Kuroda), Michael_J Meaney[2], 加藤　忠史[1](Tadafumi Kato)
[1]理研・脳・精神疾患, [2]McGill大学

S1A-J3 マウスの情動・社会行動の発達における親仔関係の役割
9:40 Roles of parent-offspring relationship in the development of emotional and social behaviors in mice
小川 園子[1](Sonoko Ogawa), 津田夢芽子[1](Mumeko Tsuda), 富原 一哉[2](Kazuya Tomihara)
[1]筑波大学大学院・人間総合科学研究科・感性認知脳科学専攻・行動神経内分泌学, [2]鹿児島大学・法文学部

S1A-J4 ヒトの母子相互交渉の霊長類的起源および進化的改変
10:15 Origins of and evolutionary modifications in the mother-infant interaction in humans
竹下 秀子(Hideko Takeshita)
滋賀県立大学・人間文化学部

## Symposium 1A-K 8:30-11:00 Room K

## Notch シグナルと中枢神経系発生
## Notch Signaling in the Developing Central Nervous System

Chairpersons 見学美根子 (Mineko Kengaku) (理化学研究所脳科学総合研究センター)
後藤由季子 (Yukiko Gotoh) (東京大学分子細胞生物学研究所)

演題番号 (*) は口演からの繰込

S1A-K1 Oscillatory versus persistent Hes1 expression promotes proliferation and differentiation of neural progenitors
8:35
Ryoichiro Kageyama, Hiromi Shimojo, Toshiyuki Ohtsuka
Institute for Virus Research, Kyoto University, Kyoto, Japan

S1A-K2 マウス大脳のニューロン分化細胞決定における Notch リガンド Dll1 の機能
9:00 Differential expression of the Notch ligand Dll1 among neural precursors is a critical determinant of neurogenesis in the developing mouse telencephalon
後藤由季子(Yukiko Gotoh), 川口 大地(Daichi Kawaguchi)
東京大・分生研

S1A-K3 Differential Notch/CBF1 signaling in telencephalic neural stem cells and neuroblasts
9:25
Nicholas Gaiano[1], Ken-ichi Mizutani[1], Keejung Yoon[2], Louis Dang[1], Akinori Tokunaga[1]
[1]Department of Neurology, Johns Hopkins University, Baltimore, Maryland, USA, [2]Center for Biomedical Sciences, National Institute of Health, Seoul, Korea

S1A-K4* RBP-J による大脳皮質神経細胞移動の調節
9:50 The regulation of cortical neuronal migration by RBP-J
谷垣 健二[1](Kenji Tanigaki), 村木 一枝[1](Kazue Muraki), 本庶 佑[2](Tasuku Honjo)
[1]滋賀県立成人病センター 研究所, [2]京都大学大学院大学 免疫ゲノム医学

S1A-K5 中枢神経系生後発生における DNER-Notch シグナルの機能
10:08 DNER-Notch signaling in the postnatal CNS
見学美根子(Mineko Kengaku), 金 清華(Qinghua Jin), 藤島 和人(Kazuto Fujishima)
理化学研究所・脳科学総合研究センター・神経細胞極性研究チーム

S1A-K6 Hes1 遺伝子の新しい発現の調節機構
10:26 Novel regulatory mechanism of Hes1 gene expression
岡野 栄之[1](Hideyuki Okano), 深見 伸一[1,2](Shin-ichi Fukami)
[1]慶大・医・生理学, [2]独立行政法人科学技術振興機構・戦略的創造研究推進事業

## Symposium 2A-A 8:30-11:00 Room A

### シナプスにおける情報伝達制御
### Signal Integration at Synapses

Chairperson 持田 澄子 (Sumiko Mochida) (東京医科大学細胞生理学講座)

S2A-A1
8:30
**シナプスにおける情報伝達制御**
**Signal Integration at Synapses**
持田 澄子(Sumiko Mochida)
東京医大・細胞生理

S2A-A2
8:33
**神経ペプチドであるノシセプチンによる海馬長期増強と記憶形成の調節**
**The neuropeptide nociceptin regulates hippocampal long-term potentiation (LTP) and memory formation**
真鍋 俊也[1,2](Toshiya Manabe)
[1]東京大・医科学研究所・神経ネットワーク, [2]CREST, JST

S2A-A3
9:07
**中枢神経系における内因性カンナビノイド放出と逆行性シナプス抑圧のメカニズム**
**Mechanisms of endocannabinoid release and retrograde synaptic suppression in the CNS**
狩野 方伸[1](Masanobu Kano), 橋本谷祐輝[2](Yuki Hashimotodani), 少作 隆子[3](Takako Ohno-Shosaku), 前島 隆司[4](Takashi Maejima)
[1]大阪大院・医・細胞神経科学, [2]大阪大院・医・神経生理学, [3]金沢大院・医・機能障害, [4]生理研・発達生理学

S2A-A4
9:41
**Inhibition of neurotransmitter release by group III metabotropic glutamate receptors**
Heinrich Betz, Astrid Scheschonka, Chuansheng Zhang, Anina Moritz
Dept. of Neurochemistry, Max-Planck-Institute for Brain Research, Frankfurt, Germany

S2A-A5
10:15
**Calcium channels & synaptic plasticity**
W Catterall[1], A Few[1], N Lautermilch[1], T Scheuer[1], S Mochida[2]
[1]Pharmacology, U Washington, Seattle, [2]Physiology, Tokyo Med College

## Symposium 2A-B 8:30-11:00 Room B

### 神経活動とスパイン形態:アクチンダイナミズムとその制御因子
### Activity-dependent Post-Synaptic Actin Remodeling: Its Dynamisms and Regulatory Molecules

Chairpersons 白尾 智明 (Tomoaki Shirao) (群馬大学医学部高次細胞機能学)
森 望 (Nozomu Mori) (長崎大学大学院医歯薬学総合研究科)

S2A-B1
8:30
**ドレブリン依存性スパイン関連蛋白集積のグルタメート受容体活性による制御について**
**Glutamate receptor activity regulates drebrin-dependent accumulation of spine resident proteins**
白尾 智明(Tomoaki Shirao)
群馬大院・医・高次細胞機能

S2A-B2
9:00
**終脳特異的細胞接着分子テレンセファリンによる樹状突起フィロポディア形成の分子機構 —ERM ファミリーアクチン結合蛋白質の関与—**
**Involvement of ERM family actin-binding proteins in telencephalin-induced dendritic filopodia formation**
吉原 良浩(Yoshihiro Yoshihara), 松野 仁美(Hitomi Matsuno), 古谷 裕(Yutaka Furutani)
理研・脳センター・シナプス分子機構

S2A-B3
9:30
Modulation of excitatory synapse maturation by assembly of a postsynaptic protein complex containing PSD-95 and neuroligins
Alaa El-Husseini
Department of Psychiatry, University of British Columbia, BC, Canada

S2A-B4
10:00
脳由来神経栄養因子前駆体とシナプス退縮
Precorsor BDNF is a novel regulator of synapse degeneration
小島　正己[1,2](Masami Kojima)，清末　和之[3](Kazuyuki Kiyosue)
[1]産業技術総合研究所・セルエンジニアリング研究部門，[2]SORST, JST，[3]産業技術総合研究所・脳神経情報研究部門

S2A-B5
10:30
Shc 系アダプター分子によるスパイン形成制御
Involvement of Shc-related phospho-tyrosine signal adaptors in the dendritic spine formation
森　　望(Nozomu Mori)，山口（白石）陽子(Yoko Shiraishi-Yamaguchi)
長崎大院・医歯薬総合研究科

## Symposium 2A-C　8:30-11:00　Room C

## RNA 制御と神経疾患
## RNA Metabolism: Its Involvement in Physiology and Neurologic Diseases

Chairpersons　塩見　春彦（Haruhiko Siomi）（徳島大学ゲノム機能研究センター）
岡野ジェイムス洋尚（Hirotaka James Okano）（慶應義塾大学医学部生理学）

S2A-C1
8:30
神経系の発生における RNA 結合タンパク質の役割
A role of RNA binding proteins in neuronal differentiation
岡野ジェイムス洋尚(Hirotaka J. Okano)
慶應大・医・生理

S2A-C2
9:00
ナンセンス変異依存 mRNA 分解機構による神経疾患の臨床表現型の修飾メカニズム
Nonsense mediated mRNA decay as a modifier of neurologic disease traits and phenotypes
井上　　健[1](Ken Inoue)，James_R Lupski[2]
[1]国立精神・神経センター　神経研究所　疾病２部，[2]ベイラー医科大学　分子人類遺伝学

S2A-C3
9:30
RNA と自閉症
Is RNA involved in autistic behaviors?
内匠　　透(Toru Takumi)
大阪バイオサイエンス研究所

S2A-C4
10:00
In vivo application of RNA interference
Takanori Yokota
Department of Neurology, Tokyo Medical and Dental University, Japan

S2A-C5
10:30
脆弱 X 症候群の原因は RNA サイレンシング機構の異常か？
Connection between RNA silencing and Fragile X Syndrome
塩見　春彦(Haruhiko Siomi)
徳島大学・ゲノム機能研究センター

## Symposium 2A-D　8:30-11:00　Room D

### 2光子励起法による in vivo 観察の最前線
### In vivo Imaging with Multiphoton Microscopy

Chairpersons　鍋倉　淳一 (Junichi Nabekura) (生理学研究所)
河西　春郎 (Haruo Kasai) (東京大学大学院医学系研究科)

S2A-D1
8:30
**大脳皮質障害後の神経細胞微細構造およびグリアの経時的観察**
**Long term observation of fine structural plasticity of neurons and glias in damaged cerebral cortex of living mice**
和氣　弘明(Hiroaki Wake), 根本　知己(Tomomi Nemoto), 鍋倉　淳一(Junichi Nabekura)
自然科学研究機構　生理学研究所　発達性理学系　生体恒常機能発達機構研究部門

S2A-D2
8:55
**樹状突起の興奮性を介した体性感覚受容野の成熟**
**Dendritic excitability maturates somatosensory receptive field.**
駒井　章治(Shoji Komai)
奈良先端科学技術大学院大学・バイオサイエンス研究科

S2A-D3
9:20
**2光子顕微鏡を用いた、マウス大脳新皮質神経細胞の樹状突起におけるグルタミン酸受容体の in vivo 機能マッピング**
**Functional mapping of glutamate receptors in dendrites of mouse neocortical neurons in vivo using two-photon microscope**
野口　潤[1](Jun Noguchi), 宮崎　崇史[1](Takashi Miyazaki), 長岡　陽[1](Akira Nagaoka), Graham C.R. Ellis-Davies[2], 松崎　政紀[1](Masanori Matsuzaki), 河西　春郎[1](Haruo Kasai)
[1]東京大院・医・疾患生命工学センター, [2]ドレクセル大学

S2A-D4
9:45
**The functional microarchitecture of the mouse barrel cortex**
Takashi Sato[1], Noah Gray[1], Zach Mainen[2], Karel Svoboda[1]
[1]Janelia Farm Research Campus, [2]CSHL

S2A-D5
10:10
**二光子励起カルシウムイメージングによる単一細胞レベルの機能マッピング**
**Single-cell resolution functional mapping with two-photon calcium imaging in vivo**
大木　研一(Kenichi Ohki)
ハーバード大院・医・神経生物

## Symposium 2A-E　8:30-11:00　Room E

### 海馬ネットワークの最前線 － 動的再構成とシナプス可塑性
### The Hippocampal Network Forefront - Dynamic Reorganization and Synaptic Plasticity

Chairpersons　神谷　温之 (Haruyuki Kamiya) (北海道大学医学研究科)
八尾　寛 (Hiromu Yawo) (東北大学大学院生命科学研究科)

S2A-E1
8:30
**カルシウムストアと海馬プレシナプス可塑性**
**Calcium store and presynaptic plasticity in the hippocampus**
神谷　温之(Haruyuki Kamiya)
北海道大院・医・神経生物学

S2A-E2
8:55
**海馬苔状線維による神経回路と行動の制御**
**Regulation of neural circuit and behavior by hippocampal mossy fibers**
小林　克典(Katsunori Kobayashi)
日本医科大学・薬理

S2A-E3
9:20
**海馬苔状線維による機能的神経回路の形成**
**Development of functional networks by hippocampal mossy fibers**
小山　隆太(Ryuta Koyama)
東京大学大学院薬学系研究科

S2A-E4 9:45
**成体海馬ニューロン新生**
**Adult hippocampal neurogenesis**
久恒　辰博(Tatsuhiro Hisatsune)
東京大院・新領域・先端生命

S2A-E5 10:10
**海馬歯状回の苔状細胞特異的に NMDA 型受容体を欠損させたマウスの機能解析**
**Characterization of dentate mossy cell-restricted NMDA receptor knockout mice**
中沢　一俊(Kazutoshi Nakazawa), 神出誠一郎(Seiichiro Jinde), Catherine J. Cravens, Juan E. Belforte
米国・国立精神衛生研究所

S2A-E6 10:35
**海馬プレシナプスにおける小胞ダイナミクスとその可塑性**
**Vesicular dynamics and its plasticity in the hippocampal synapse**
八尾　寛[1](Hiromu Yawo), 須山　成朝[1](Shigetomo Suyama), 引間　卓弥[1](Takuya Hikima),
杉山　友香[1](Yuka Sugiyama), 阪上　洋行[2](Hiroyuki Sakagami), 石塚　徹[1](Toru Ishizuka)
[1]東北大院・生命・脳機能解析, [2]東北大院・医・細胞生物

## Symposium 2A-F　8:30-11:00　Room F

**霊長類の社会関係認知**
**Primate Social Cognition**

Chairperson　中村　克樹 (Katsuki Nakamura) (国立精神・神経センター神経研究所)

演題番号 (*) は口演からの繰込

S2A-F1* 8:30
**顔表情に識別的に応答するサル視床枕ニューロン**
**The monkey pulvinar neurons differentially response to emotional face**
西条　寿夫[1,2](Hisao Nishijo), Rafael S. Maior[1,2], 小野　武年[1,2](Taketoshi Ono), 堀　悦郎[1,2](Etsuro Hori)
[1]富山大・院・医学薬学研究部・システム情動科学, [2]科学技術振興機構　CREST

S2A-F2 8:50
**Neuronal responses to species-specific emotional expression in the monkey amygdala**
中村　克樹[1,2](Katsuki Nakamura), Koji Kuraoka[1,2]
[1]国立精神・神経センター神経研究所, [2]CREST, JST

S2A-F3 9:10
**More than just a pretty face: the importance of expression, gaze, and identity in the temporal lobe of the macaque**
Kari L. Hoffman
Department of Psychology, York University, Toronto, Canada

S2A-F4* 9:40
**対戦ゲーム中のサル前頭連合野ニューロン活動**
**Primate prefrontal neuronal activity during a competitive video game**
細川　貴之(Takayuki Hosokawa), 渡邊　正孝(Masataka Watanabe)
東京都神経科学総合研究所

S2A-F5 10:00
**頭頂葉の社会環境適応的神経活動表現**
**Dynamic social adaptation of parietal neurons in primates**
藤井　直敬(Naotaka Fujii), 日原さやか(Sayaka Hihara), 入來　篤史(Atsushi Iriki)
理化学研究所 脳科学総合研究センター 象徴概念発達研究チーム

S2A-F6 10:30
**Mirror neurons and their role in the macaque social cognition**
Pier Francesco Ferrari
Dipartimento di Biologia Evolutiva e Funzionale, Università di Parma

## Symposium 2A-G　8:30-11:00　Room G

### ミトコンドリア機能異常としての精神・神経疾患
### Mitochondrial Dysfunction and Psycho-neurological Disorders

Chairperson　後藤　雄一（Yu-ichi Goto）（国立精神・神経センター神経研究所疾病研究第二部）

S2A-G1
8:40
Mitochondrial dysfunction and aging
Naoaki Ishii
Department of Molecular Life Science, Tokai University School of Medicine, Kanagawa, Japan

S2A-G2
9:20
ミトコンドリア異常とパーキンソン病
Mitochondrial dysfucntion in Parkinson's disease
服部　信孝(Nobutaka Hattori)
順天堂大学・医・脳神経内科

S2A-G3
9:50
ミトコンドリア異常と神経・筋疾患
Mitochondrial dysfunction inducing neuromuscular disorders known as mitochondrial encephalomyopathies
後藤　雄一(Yu-ichi Goto)
国立精神神経センター神経研・疾病研究2部

S2A-G4
10:20
ミトコンドリア異常と精神疾患
Mitochondrial dysfunction in mental disorders
笠原　和起(Takaoki Kasahara)，加藤　忠史(Tadafumi Kato)
理研・脳科学総合研究センター・精神疾患動態研究チーム

## Symposium 2A-H　8:30-11:00　Room H

### 情動・痛み・本能・自律機能を結ぶ神経メカニズム
### Central Neuro-crossroads for Emotion, Pain, Instinct and Autonomic Functions

Chairpersons　加藤　総夫（Fusao Kato）（東京慈恵会医科大学総合医科学研究センター神経生理学）
松井　秀樹（Hideki Matsui）（岡山大学大学院細胞生理学）

S2A-H1
8:35
Central Extended Amygdala（扁桃体中心核延長領域）—痛みによる不快情動生成のキーコンポーネント—
Central extended amygdala as a key component for pain-induced aversion
南　雅文(Masabumi Minami)
北海道大院・薬・薬理

S2A-H2
9:10
痛みの情動の記憶は扁桃体中心核シナプスに書き込まれる
Emotional memory of chronic pain engraved on the central amygdala synapses
加藤　総夫[1](Fusao Kato)，池田　亮[2](Ryo Ikeda)，高橋由香里[1](Yukari Takahashi)
[1]慈恵医大・神経生理，[2]慈恵医大・整形外科

S2A-H3
9:45
情動反応における NPW/NPB システムの役割
A role of the NPW/NPB system in regulation of emotional responses
桜井　武(Takeshi Sakurai)
筑波大学基礎医学系

S2A-H4
10:20
オキシトシンによる情動不安の制御機構
Regulatory Mechanism of Anxiety by Oxytocin
松井　秀樹(Hideki Matsui)，沖本　直輝(Naoki Okimoto)，富澤　一仁(Kazuhito Tomizawa)
岡山大学院・医歯薬総合・細胞生理

## Symposium 2A-J　8:30-11:00　Room J

# 神経変性疾患における神経細胞機能障害
# Neuronal Dysfunctions in Neurodegenerative Diseases

Chairpersons　岡澤　均 (Hitoshi Okazawa) (東京医科歯科大学神経病理)
Steven Finkbeiner (Department of Neurology and Physiology, UCSF)

演題番号 (*) は口演からの繰込

S2A-J1
8:30
**Mechanisms of polyglutamine-dependent neurodegeneration revealed by automated imaging and structural approaches**
Steven M Finkbeiner
Department of Neurology and Physiology, University of California San Francisco

S2A-J2*
9:00
**蛍光相関分光法による細胞内ポリグルタミン蛋白質オリゴマーの検出**
**Polyglutamine protein oligomer formation in cells characterized by fluorescence correlation spectroscopy**
永井　義隆[1](Yoshitaka Nagai), 高橋　保夫[2,3](Yasuo Takahashi), 岡本　佑馬[1](Yuma Okamoto),
ポピエルヘレナ明子[1](H Akiko Popiel), 藤掛　伸宏[1](Nobuhiro Fujikake), 金城　政孝[3](Masataka Kinjo),
戸田　達史[1](Tatsushi Toda)
[1]大阪大院・医・臨床遺伝学, [2]オリンパス, [3]北海道大・電子研

S2A-J3*
9:15
**可溶性ポリグルタミンオリゴマーは細胞毒性を惹起する**
**Soluble polyglutamine oligomer formation precedes inclusion body formation and increases the cytotoxicity**
高橋　俊昭[1](Toshiaki Takahashi), 菊池　信矢[2](Shinya Kikuchi), 堅田　慎一[2](Shinichi Katada),
小野寺　理[2](Osamu Onodera), 西澤　正豊[2](Masatoyo Nishizawa)
[1]新潟大学医学部保健学科基礎生体情報学講座, [2]新潟大学脳研究所神経内科

S2A-J4
9:30
**プロテオミクス解析によって明らかになった新しいポリグルタミン病態としてのゲノトキシックストレス**
**Proteome analysis of soluble nuclear proteins links genotoxic stress to the polyglutamine disease pathology**
岡澤　均(Hitoshi Okazawa)
東京医科歯科大・難治研・神経病理

S2A-J5
10:00
**第 16 番染色体長腕に連鎖する新しい常染色体優性遺伝性小脳失調症**
**A new autosomal dominant cerebellar ataxia linked to chromosome 16q22.1 (16q-ADCA)**
水澤　英洋(Hidehiro Mizusawa)
東京医科歯科大学

S2A-J6
10:30
**ポリグルタミン病における可逆性神経機能障害**
**Reversible dysfunctioin in polyglutamine disease**
祖父江　元(Gen Sobue)
名古屋大学院・医・神経内科

## Symposium 2A-K　8:30-11:00　Room K

# 新世代神経行動学のストラテジー
# Strategy for New Generation of Neuroethology

Chairpersons　小川　宏人 (Hiroto Ogawa) (埼玉医科大学医学基礎部門生物学教室)
岡　浩太郎 (Kotaro Oka) (慶應義塾大学理工学部生命情報学科)

S2A-K1
8:30
**線虫 *C. elegans* の「脳」による情報処理とは？～忌避行動可塑性の統合的解析から～**
**Integrative functional analysis of the "brain" of the nematode *C. elegans* for odor-avoidance**
木村幸太郎[1,2,3](Kotaro Kimura)
[1]国立遺伝研・構造セ, [2]総研大・遺伝学専攻, [3]科技構・さきがけ

S2A-K2
9:00
シロアリにおける社会行動・カースト分化の分子基盤
The molecular basis for social behavior and caste differentiation in termites.
三浦　徹(Toru Miura)
北海道大・院・地球環境

S2A-K3
9:30
ゼブラフィッシュのロコモーションの制御に関わる脊髄神経回路の分子的基盤
Development and function of spinal locomotor circuits in zebrafish
東島　眞一(Shin-ichi Higashijima)，木村有希子(Yukiko Kimura)，佐藤　千恵(Chie Satou)
岡崎統合バイオ＆生理学研究所

S2A-K4
10:00
カラスの社会性を支える認知基盤 - 個体認知と観察学習 -
Cognitive basis of social behaviour in the crow: understanding 'who' is doing 'what'
伊澤　栄一(Ei-Ichi Izawa)
慶應義塾大学・文・心理

S2A-K5
10:30
昆虫神経細胞の樹状突起形態に潜む感覚情報抽出アルゴリズム
Dendritic design implements algorithm for extraction of sensory information in insect sensory interneuron
小川　宏人(Hiroto Ogawa)
埼玉医大・医・生物

## Symposium 3A-A 8:30-11:00 Room A

### 学習・記憶形成制御の分子基盤
### Understanding Mechanisms of Learning and Memory from Molecule to Behavior

Chairpersons 喜田 聡 (Satoshi Kida) (東京農業大学応用生物科学部)
井ノ口 馨 (Kaoru Inokuchi) (三菱化学生命科学研究所記憶形成精神疾患研究グループ)

S3A-A1
8:30
**転写調節因子群による記憶形成制御**
**Mechanisms of memory formation by transcription factors**
喜田 聡(Satoshi Kida)
東京農大・応生科・バイオ

S3A-A2
8:50
**学習・記憶機構解明を目的とした、遺伝子改変マウスの電気生理・行動学的解析**
**Electrophysiological and behavioral analysis of gene-targeted mice to understand mechanisms of learning and memory**
城山 優治[1](Yuji Kiyama), 真鍋 俊也[1,2](Toshiya Manabe)
[1]東京大・医科学研究所・神経ネットワーク, [2]科学技術振興機構 戦略的創造研究推進事業

S3A-A3
9:10
**Reconfiguration of synaptic network and enhancement of memory by magnesium ion**
Guosong Liu[1,2]
[1]Center for Learning and Memory, School of Medicine, Tsinghua Univerisity, Beijing, China, [2]Center for Learning and Memory, University of Texas, Austin, USA

S3A-A4
9:40
**Symptomatic treatment for Alzheimer's disease: enhancing cholinergic function.**
Paul F. Chapman, Darrel J Pemberton, Woei Shin Chen, Zeenat Atcha, Fong Kuan Wong
GSK Centre for Cognitive and Neurodegenerative Disorders, Singapore, Singapore

S3A-A5
10:10
**Molecular and cellular cognition: unraveling mechanisms of learning and memory**
Alcino J Silva
Department of Neurobiology, UCLA, Los Angeles, USA

S3A-A6
10:40
**恐怖記憶形成の分子機構**
**Molecular mechanism underlying maintenance of fear memory**
井ノ口 馨[1,2](Kaoru Inokuchi)
[1]三菱化学生命科学研究所、記憶形成・精神疾患研究グループ, [2]CREST、科学技術振興機構

## Symposium 3A-B 8:30-11:00 Room B

### 幹細胞の運命決定と分化可塑性制御のメカニズム
### Cell Fate Decision and Plasticity of Stem Cells

Chairpersons 中島 欽一 (Kin-ichi Nakashima) (奈良先端大学)
岡野 栄之 (Hideyuki Okano) (慶應義塾大学医学部生理学)

S3A-B1
8:30
**神経系細胞の分化・可塑性制御とエピジェネティクス**
**Epigenetic mechanism regulating fate specification and plasticity of neural cells**
中島 欽一[1](Kinichi Nakashima), 高塚絵里子[1](Eriko Takatsuka), 山下 徹[2,3](Toru Yamashita), Jenny Hsieh[4], Fred H. Gage[5], 波平 昌一[1](Masakazu Namihira), 岡野 栄之[2](Hideyuki Okano), 澤本 和延[3,6](Kazunobu Sawamoto), 神山 淳[1](Jun Kohyama)
[1]奈良先端大・バイオ・分子神経分化制御, [2]慶應大・医・生理学, [3]慶応大・医・ブリジストン神経発生・再生学寄付講座, [4]テキサス大サウスウェスタン校・メディカルセンター・分子生物学, [5]ソーク研究所・遺伝学, [6]名古屋市立大・院医・再生医学

S3A-B2
9:00
**神経前駆細胞の分化・未分化制御シグナルに関する考察**
**Signals regulating the differentiation vs. maintenance of neural progenitor cells**
砂堀　毅彦(Takehiko Sunabori)，岡野　栄之(Hideyuki Okano)
慶大・医・生理

S3A-B3
9:30
**多能性細胞からの神経分化とその制御機構**
**Regulatory mechanisms of neural differentiation from pluripotent cells**
笹井　芳樹(Yoshiki Sasai)
理研・発生再生センター・細胞分化器官発生

S3A-B4
10:00
**神経幹細胞における sox2 転写制御機構**
**Molecular mechanism of sox2 expression in neural stem cells**
近藤　　亨(Toru Kondo)
理化学研究所・発生再生研・分化転換

S3A-B5
10:30
**より ES 細胞に近い人工万能幹（iPS）細胞の樹立**
**Generation of high quality iPS cells**
高橋　和利[1](Kazutoshi Takahashi)，沖田　圭介[1](Keisuke Okita)，中川　誠人[1](Masato Nakagawa)，
青井　貴之[1](Takashi Aoi)，一阪　朋子[1,2](Tomoko Ichisaka)，山中　伸弥[1,2](Shinya Yamanaka)
[1]京都大・再生研，[2]科学技術振興機構，CREST



## Symposium 3A-C　8:30-11:00　Room C

**神経発達・機能を制御するタンパク・膜相互作用 - 神経回路から病態へ -**
**Dynamic Protein-Lipid Interaction: Key Roles in Neuronal Development and in Diseases**

Chairpersons　小島　正己（Masami Kojima）（産業技術総合研究所セルエンジニアリング研究部門）
　　尾藤　晴彦（Haruhiko Bito）（東京大学大学院脳神経医学専攻神経生化学分野）

S3A-C1
8:30
**脳由来神経栄養因子（BDNF）により誘導される神経細胞のコレステロール合成とプレシナプス蛋白質のラフトへの局在**
**BDNF stimulates neuronal cholesterol biosynthesis and accumulates presynaptic proteins in lipid rafts**
鈴木　辰吾[1,2,3](Shingo Suzuki)，清末　和之[4](Kazuyuki Kiyosue)，狭間　俊介[1,5](Shunsuke Hazama)，
小倉　明彦[5](Akihiko Ogura)，原　とも子[1](Tomoko Hara)，小清水久嗣[1](Hisatsugu Koshimizu)，
小島　正己[1,3](Masami Kojima)
[1]産総研・セルエンジニア，[2]北海道大学・薬学院，[3]SORST/JST，[4]産総研・脳神経・脳遺伝子，[5]阪大院・生命機能・神経可塑性生理学

S3A-C2
8:50
**ラフトアンカー型 CaM キナーゼ、CLICK-III による樹状突起形成制御**
**Regulation of dendritogenesis via a lipid raft-associated $Ca^{2+}$/calmodulin-dependent protein kinase CLICK-III/CaMKI$\gamma$**
竹本ー木村さやか(Sayaka Takemoto-Kimura)，石原ー上田奈津実(Natsumi Ishihara-Ageta)，
野中　美応(Mio Nonaka)，奥野　浩行(Hiroyuki Okuno)，尾藤　晴彦(Haruhiko Bito)
東京大学院・医・神経生化

S3A-C3
9:10
**成長円錐ガイダンスを制御する非対称性 $Ca^{2+}$ シグナルと膜輸送**
**Asymmetric $Ca^{2+}$ signaling and membrane dynamics mediate growth cone guidance**
上口　裕之(Hiroyuki Kamiguchi)
理化学研究所　脳科学総合研究センター

S3A-C4
9:35
**ラフトとアミロイドβタンパク質の産生**
**Lipid rafts and Abeta production**
森島　真帆(Maho Morishima-Kawashima)
北海道大学 大学院薬学研究院 神経病理・病態生化学（IBL）

S3A-C5 神経細胞におけるアポリポ蛋白Eアイソフォーム依存的コレステロール代謝調節
10:00 Apolipoprotein E-isoform-specific modulation of cholesterol metabolism in neurons
道川　誠(Makoto Michikawa)
国立長寿医療センター・研究所・アルツハイマー病研究部

S3A-C6 細胞膜上でラフトが促進するシグナル変換：1分子追跡による研究
10:25 Raft-facilitated signal transduction in the plasma membrane
楠見　明弘(Akihiro Kusumi)
京都大学大学院医学研究科/再生医科学研究所附属ナノ再生医工学研究センター

## Symposium 3A-D　8:30-11:00　Room D

## 精神疾患の分子機構
## Molecular Mechanism of Mental Disorders

Chairperson　功刀　浩 (Hiroshi Kunugi) (国立精神・神経センター神経研究所疾病研究第三部)

演題番号 (*) は口演からの繰込

S3A-D1* 統合失調症モデルとしての変異型DISC1トランスジェニックマウスの作製と解析
8:30 Production and analyses of mutant DISC1 transgenic mice: an animal model of schizophrenia
疋田　貴俊[1,2](Takatoshi Hikida), Hanna Jaaro-Peled[3], Stephanie Tankou[3],
森田　正彦[3](Masahiko Morita), Mikhail Pletnikov[3], 喜田　聡[4](Satoshi Kida), 森　進[5](Susumu Mori),
澤　明[2,3](Akira Sawa)
[1]大阪バイオサイエンス研・システムズ生物学, [2]ジョンズホプキンス大・医・神経科学, [3]ジョンズホプキンス大・医・精神医学, [4]東京農大・応用生物・バイオサイエンス, [5]ジョンズホプキンス大・医・放射線医学

S3A-D2 統合失調症の分子カスケード
8:45 Molecular pathogenesis of schizophrenia: genes, or pathways?
山田　和男(Kazuo Yamada)
理化学研究所・脳科学総合研究センター・分子精神科学研究チーム

S3A-D3 双極性障害の分子基盤
9:15 Molecular basis of bipolar disorder
加藤　忠史(Tadafumi Kato)
理研・脳センター・精神疾患動態

S3A-D4 気分障害におけるグルココルチコイドと脳由来神経栄養因子
9:45 Glucocorticoid and brain-derived neurotrophic factor in mood disorders
功刀　浩(Hiroshi Kunugi), 沼川　忠広(Tadahiro Numakawa)
国立精神セ・神経研・疾病三部

S3A-D5 精神疾患の分子イメージング
10:15 Molecular imaging of mental disorders
須原　哲也(Tetsuya Suhara)
放射線医学総合研究所

## Symposium 3A-E　8:30-11:00　Room E

## 小脳の神経伝達可塑性とその展望
## Synaptic Plasticity in the Cerebellum and Beyond

Chairpersons　Thomas Knöpfel(理化学研究所脳科学総合研究センター神経回路ダイナミックス研究チーム)
永雄　総一 (Soichi Nagao) (理化学研究所脳科学総合研究センター運動学習制御研究チーム)

S3A-E1 Arc is required for the late phase of cerebellar long-term synaptic depression
8:35
David J Linden, Jason D. Shepherd, Paul F. Worley
Dept. of Neuroscience, Johns Hopkins University, Baltimore, USA

S3A-E2 9:00
**Decoding Purkinje cell simple spike trains: Patterns and pauses**
Erik De Schutter
Okinawa Institute of Science and Technology, Japan

S3A-E3 9:25
**Cerebellar somatosensory mossy fibre input - Synaptic integration and synaptic plasticity.**
Carl-Fredrik Ekerot, Henrik Jorntell
Department of Experimental Medical Science, Lund University, Lund, Sweden

S3A-E4 10:05
**マウス瞬目条件反射学習の二段階学習**
**Two-stage learning in eyeblink conditioning in mice**
糸原　重美(Shigeyoshi Itohara), Hye-Soo Kim, Jin-Sung Park
理研・脳センター・行動遺伝

S3A-E5 10:30
**小脳シナプス可塑性の超微形態基盤**
**Cerebellar plasticity at ultrastructural level**
重本　隆一(Ryuichi Shigemoto)
自然科学研究機構生理学研究所



## Symposium 3A-F　8:30-11:00　Room F

## 行動学習の脳内機構－システムレベルにおける実験と計算モデルの融合
## Brain Mechanisms of Action Learning - Integrating Experimentation and Computational Modelling at the System Level

Chairperson　鮫島　和行 (Kazuyuki Samejima)（玉川大学脳科学研究所）

S3A-F1 8:30
**線条体の行動価値と皮質 - 基底核回路の強化学習モデル**
**Action value in the striatum and reinforcement learning model of cortico-basal ganglia network**
鮫島　和行[1](Kazuyuki Samejima), 上田　康雅[2](Yasumasa Ueda), 銅谷　賢治[3](Kenji Doya),
木村　實[2](Minoru Kimura)
[1]玉川大・脳科学研, [2]京都府立医大・神経生理, [3]沖縄科技大院

S3A-F2 9:05
**強化学習の複数のアルゴリズムと神経回路**
**Multiple pathways and algorithms for reinforcement leaning**
銅谷　賢治[1,2](Kenji Doya)
[1]沖縄科学技術大学院大学先行研究, [2]ATR脳情報研究所

S3A-F3 9:40
**The anterior cingulate cortex in learning and reward-guided decision making**
Matthew F S Rushworth[1,2], Timothy EJ Behrens[1,2], Mark E Walton[1]
[1]Department of Experimental Psychology, University of Oxford, Oxford, England, UK, [2]FMRIB, University of Oxford, Oxford, England, UK

S3A-F4 10:25
**前頭前野における行動価値の予測誤差へのトップダウン注意**
**Top-down attention to prediction errors of action values in the prefrontal cortex**
松元　健二(Kenji Matsumoto), 松元まどか(Madoka Matsumoto), 阿部　央(Hiroshi Abe),
田中　啓治(Keiji Tanaka)
理研脳センター・認知機能

## Symposium 3A-G　8:30-11:00　Room G

### 脳神経回路機能障害におけるミクログリアの多面性
### Multiple Function of Microglia in Brain Neurocircuit Disorder

Chairpersons　井上　和秀（Kazuhide Inoue）（九州大学大学院薬学研究院）
高坂　新一（Shinichi Kohsaka）（国立精神・神経センター神経研究所）

S3A-G1
8:30
**損傷神経細胞とミクログリアの微妙な関係**
**Kiss of microglia - Is it sweet or bitter-**
木山　博資(Hiroshi Kiyama)，前田　光代(Mitsuyo Maeda)
大阪市大院・医・機能細胞形態

S3A-G2
9:05
**Microglial activation by TLR ligands: usual and unusual suspects**
Uwe-Karsten Hanisch
Institute of Neuropathology, University of Goettingen, Goettingen, Germany

S3A-G3
9:40
**細胞外ＡＴＰによるミクログリア遊走能の調節機構**
**Molecular mechanisms of ATP-induced microglial chemotaxis**
大澤　圭子[1](Keiko Ohsawa)，中村　泰子[1](Yasuko Nakamura)，鈴木　恵里[1](Eri Suzuki)，
井上　和秀[2](Kazuhide Inoue)，高坂　新一[1](Shinichi Kohsaka)
[1]国立精神・神経センター　神経研　代謝，[2]九州大院　薬学　薬効解析学

S3A-G4
10:15
**疼痛シグナル伝達のミクログリアによる変調作用**
**Microglial modulation of pain signalling**
井上　和秀(Kazuhide Inoue)
九大院・薬・薬理

## Symposium 3A-H　8:30-11:00　Room H

### 疼痛処理の分子病理
### Molecular Pathology of Pain Processing

Chairperson　Bryan C. Hains(Department of Neurology, Yale University)

S3A-H1
8:30
**Molecular-biophysical correlates in the pathological sensory neuraxis after spinal cord injury**
Bryan C. Hains
Department of Neurology, Yale University, New Haven CT, USA

S3A-H2
9:05
**ニューロパチックペインモデルの DRG 及び脊髄における分子変化**
**Molecular changes in DRG and spinal cord in the model of neuropathic pain**
野口　光一(Koichi Noguchi)
兵庫医科大学解剖学第二

S3A-H3
9:40
**慢性疼痛下における脳内アストロサイトの機能変化に伴う情動変化**
**Chronic pain induces the changes in glial function associated with emotional dysfunction**
成田　　年(Minoru Narita)，葛巻　直子(Naoko Kuzumaki)，新倉　慶一(Keiichi Niikura)，
鈴木　　勉(Tsutomu Suzuki)
星薬科大学　薬品毒性学教室

S3A-H4
10:15
**末梢神経損傷に伴う脊髄内 TRP チャネルの可塑的変化**
**Plastic modification of TRP channel activities in the spinal dorsal horn following peripheral nerve injury**
吉村　　恵(Megumu Yoshimura)，古江　秀昌(Hidemasa Furue)
九州大院・医・統合生理

## Symposium 3A-J　8:30-11:00　Room J

# 美と快のイメージング
# Neuroimaging of Beauty and Pleasure

Chairpersons　本田　学（Manabu Honda）（国立精神・神経センター）
外池　光雄（Mitsuo Tonoike）（千葉大学工学部メディカル情報工学講座　医用情報教育研究分野）

S3A-J1
8:30
### 香道における聞香期間中の推論過程と前頭前野機能
### Prefrontal cortex manipulates reasoning process during Koh-Do performances
Dilshat Abla[1]，藤井　直敬[2](Naotaka Fujii)，工藤　紀子[1](Noriko Kudo)，日原さやか[2](Sayaka Hihara)，
入來　篤史[2](Atsushi Iriki)，岡ノ谷一夫[1](Kazuo Okanoya)
[1]理研・脳・生物言語研究チーム，[2]理研・脳・象徴概念発達研究チーム

S3A-J2
9:05
### 味覚における美学：脳機能イメージングからの洞察
### Aesthetics in gustation: insight from recent neuroimaging studies
檀　一平太(Ippeita Dan)
食品総合研究所・食認知科学

S3A-J3
9:40
### 美と欲望の神経相関
### Neural correlates of beauty and desire
川畑　秀明(Hideaki Kawabata)
鹿児島大・教育・心理

S3A-J4
10:15
### 美と快のイメージング：ケーススタディ・ハイパーソニック・エフェクト
### Imaging beauty and pleasure: A case study of the hypersonic effect
本田　学[1](Manabu Honda)，森本　雅子[1](Masako Morimoto)，八木　玲子[1](Reiko Yagi)，
仁科　エミ[3](Emi Nishina)，河合　徳枝[2](Norie Kawai)，大橋　力[2](Tsutomu Oohashi)
[1]国立精神・神経セ・神経研・疾病研究第七部，[2]国際科学振興財団，[3]メディア教育開発センター

## Symposium 3A-K　8:30-11:00　Room K

# 鳥の歌制御システムにおける可塑性の分子機構
# Molecular Basis of Functional Plasticity in Birdsong Control System

Chairperson　坂口　博信（Hironobu Sakaguchi）（獨協医科大学）

S3A-K1
8:30
### 鳥類歌制御系発達の分子的基盤
### Molecular basis of the avian song system development
松永　英治[1,2](Eiji Matsunaga)，岡ノ谷一夫[1](Kazuo Okanoya)
[1]理化学研究所　脳科学総合研究センター　生物言語研究チーム，[2]日本学術振興会　東京都千代田区一番町８番地

S3A-K2
9:05
### Synaptic plasticity related to song learning and maintenance
Neal A Hessler，Jian Wang
RIKEN Brain Science Institute, Wako-shi, Japan

S3A-K3
9:45
### 発声によって誘導される遺伝子群の脳内動態
### Dynamic cascades of vocalizing-driven gene regulation
和多　和宏(Kazuhiro Wada)
北大院・理・先端生命科学・先端生命制御科学

S3A-K4
10:20
### Neural mechanisms of birdsong memory
Johan J. Bolhuis
Behavioural Biology, Utrecht University, Utrecht, The Netherlands

## Symposium 3P-A　15:15-17:45　Room A

# アルツハイマー病研究のフロントライン
# Frontiers of Alzheimer Research

Chairpersons　西道　隆臣 (Takaomi Saido) (理化学研究所脳科学総合研究センター神経蛋白制御研究チーム)
樋口　真人 (Makoto Higuchi) (放射線医学総合研究所 分子イメージング研究センター)

S3P-A1
15:15
### アルツハイマー病モデルマウスにおける脳アミロイドと神経炎症の可視化ならびに治療制御
### Visualization and therapeutic regulation of brain amyloidosis and neuroinflammation in Alzheimer's disease
樋口　真人(Makoto Higuchi)
放射線医学総合研究所・分子イメージング研究センター

S3P-A2
15:45
### アルツハイマー病血漿マーカーの探索
### Exploration of plasma biomarkers for Alzheimer's disease in coupling with biochemical and cognitive-psucological approach
小海　康夫[1](Yasuo Kokai)，相馬　仁[1](Hitoshi Sohma)，村上　新治[2](Shinji Murakami)
[1]札幌医科大学医学部分子機能解析部門，[2]札幌医科大学保健医療学部作業療法学科

S3P-A3
16:15
### タウ蛋白凝集中間体の探索
### Searching tau aggregation intermediates
佐原　成彦(Naruhiko Sahara)，前田　純宏(Sumihiro Maeda)，高島　明彦(Akihiko Takashima)
理化学研究所・脳科学総合研究センター・アルツハイマー病研究チーム

S3P-A4
16:45
### 線虫モデルを用いたタウオパチー神経変性機構の解析
### C. elegans model of Tauopathy
宮坂　知宏(Tomohiro Miyasaka)
東京大院・医・神経病理

S3P-A5
17:15
### γセクレターゼ複合体の構造活性相関
### Structure and function of γ-secretase complex
富田　泰輔(Taisuke Tomita)
東京大院・薬・臨床薬学

## Symposium 3P-B　15:15-17:45　Room B

# 知覚，意思決定，感情，行動における潜在的脳情報処理研究と応用
# Implicit Processing in Perception, Decision, Emotion and Action

Chairperson　下條　信輔 (Shinsuke Shimojo) (California Institute of Technology)

S3P-B1
15:15
### Effects of "invisible" stimuli on plasticity
Takeo Watanabe[1,5], Yuka Sasaki[2,3,4], Yoshiaki Tsushima[1]
[1]Dept. of Psychology, Boston University, Boston, MA, USA, [2]Martinos Center, MGH, Charlestown, MA, USA, [3]Dept. of Radiology, Harvard Medical School, Boston, MA, USA, [4]ERATO Shimojo Implicit Brain Function Project, Pasadena, CA, USA, [5]Program in Neuroscience, Boston University, Boston, MA, USA

S3P-B2
15:45
### 運動予測が裏切られたとき，なにが起こるか？ - 引き手錯覚と停止エスカレータ効果を考察する -
### What happens when motor prediction is betrayed?- Tugged hand illusion and stopped escalator effect -
五味　裕章[1,2](Hiroaki Gomi)
[1]NTT CS研，[2]JST-ERATO下條潜在脳機能プロジェクト

S3P-B3
16:15
### Implicit brain responses during insight
Joy Bhattacharya[1]
[1]Department of Psychology, Goldsmiths, University of London, London, UK, [2]Commission for Scientific Visualization, Austrian Academy of Sciences, Vienna A1220, Austria

S3P-B4 16:45
報酬探索行動の調節とドパミンの役割
Modulation of reward seeking behavior and the role of dopamine
廣中　直行(Naoyuki Hironaka)，田中　智子(Tomoko Tanaka)，板坂　典郎(Michio Itasaka)，
高野　祐治(Yuji Takano)，高橋　伸彰(Nobuaki Takahashi)，小泉美和子(Miwako Koizumi)
JST ERATO 下條潜在脳機能プロジェクト

S3P-B5 17:15
Consumer neuroscience: bringing neuroscience to the "real world "
Christian Scheier
decode Marketingberatung GmbH, Hamburg, Germany

**Symposium 3P-C** 15:15-17:45 Room C

## トニック $GABA_A$ コンダクタンスのメカニズムと機能的意義
## Mechanisms and Functional Significance of Tonic $GABA_A$ Receptor Mediated Conductance

Chairpersons Alexey Semyanov(理化学研究所脳科学総合研究センター)
福田　敦夫（Atsuo Fukuda)（浜松医科大学生理学第一）

S3P-C1 15:25
Function and adaptive plasticity of tonic GABA(A) receptor mediated inhibition in the hippocampus
Matthew C Walker
Institute of Neurology, University College London, London, UK

S3P-C2 16:00
大脳新皮質トニック $GABA_A$ コンダクタンスの発達に伴うモーダルシフト
Modal shift of the tonic $GABA_A$ receptor-mediated conductance during neocortical development
福田　敦夫(Atsuo Fukuda)
浜松医大・医・生理学第一

S3P-C3 16:35
Regulation of adult neurogenesis by GABA
Hongjun Song, Shaoyu Ge, Eyleen Goh, Kurt Sailor, Yasuji Kitabatake, Guo-li Ming
Institute for Cell Engineering, Department of Neurology, Johns Hopkins University, Baltimore, USA

S3P-C4 17:10
Shunting tonic conductance synchronizes neuronal networks
Alexey Semyanov[1], Leonid Savtchenko[2], Inseon Song[1]
[1]RIKEN Brain Science Institute, Wako-shi, Japan, [2]Physical Department, Dniepropetrovsk National University, Ukraine

**Symposium 3P-D** 15:15-17:45 Room D

## 細胞内外シグナルの連携による神経細胞移動の制御
## Regulation of Neuronal Migration by an Interplay between Intracellular and Extracellular Signals

Chairpersons 五嶋　良郎（Yoshio Goshima)（横浜市立大学医学部）
仲嶋　一範（Kazunori Nakajima)（慶應義塾大学医学部）

S3P-D1 15:15
LL5βによる細胞移動と大脳皮質形成における radial migration の制御について
Phosphoinositide-sensitive LL5β enhances lamellipodium formation through Filamin A, inhibits FILIP activity and is essential for radial migration
佐藤　　真[1,2](Makoto Sato)
[1]福井大・医・形態機能医科学組織細胞形態学・神経科学領域，[2]福井大学生命科学複合研究教育センター

S3P-D2 15:45
発生期大脳皮質における神経細胞移動と層形成の制御
Regulation of neuronal migration and layer formation in the developing cerebral cortex
仲嶋　一範[1,2](Kazunori Nakajima)
[1]慶應・医・解剖，[2]慈恵医大・DNA研・分子神経

S3P-D3 16:15
CRMP ファミリー分子は神経軸索、シナプス成熟および細胞移動を制御する
CRMP family proteins that regulate axon guidance, synapse maturation and cell migration
五嶋　良郎(Yoshio Goshima)
横浜市立大学・医・分子薬理神経生物学

S3P-D4 16:45
Nkx2.1 specifies cortical interneuron fate by directly activating Lhx6
Stewart A Anderson, Tonggong Du
Weill Cornell Medical College, New York, USA

**Symposium 3P-E** 15:15-17:45 Room E

## 自閉症の臨床から脳科学への接近
## Autism- Clinical Research to Brain Science

Chairpersons 加我　牧子 (Makiko Kaga)(国立精神・神経センター精神保健研究所知的障害部)
内野　茂夫 (Shigeo Uchino)(国立精神・神経センター神経研究所代謝研究部)

S3P-E1 15:15
自閉症：臨床医学から神経科学へ
Autism: Clinical medicine to neuroscience
稲垣　真澄(Masumi Inagaki), 加我　牧子(Makiko Kaga)
国立精神・神経センター・精研・知的障害部

S3P-E2 15:45
自閉症スペクトラム障害の神経画像
Neuroimaging in autism spectrum disorders
笠井　清登(Kiyoto Kasai), 川久保友紀(Yuki Kawakubo), 桑原　斉(Hitoshi Kuwabara),
山末　英典(Hidenori Yamasue)
東京大院・医・精神医学

S3P-E3 16:15
自閉症モデルとしての臨床症候群
Clinical syndromes as models of autism
橋本　俊顕(Toshiaki Hashimoto)
鳴門教育大学・障害児教育

S3P-E4 16:45
臨床医学から分子生物学へ
Clinical medicine to molecular biology
難波　栄二(Eiji Nanba)
鳥取大・生命機能研究支援センター・遺伝子探索分野

S3P-E5 17:15
モデルとしての実験動物群
Experimental animals as models of autism
成田　正明(Masaaki Narita)
三重大学院・医・発生再生

**Symposium 3P-F** 15:15-17:45 Room F

## 神経回路による情報処理の新しい概念 ーニューロン説を超えてー
## New Ideas on the Information Processing by Neural Networks - Beyond the Neuron Doctrine -

Chairpersons 宮川　博義 (Hiroyoshi Miyakawa)(東京薬科大学生命科学部脳神経機能学研究室)
坪川　宏 (Hiroshi Tsubokawa)(東北大学大学院情報科学研究科応用情報科学専攻生命フラクチュオマティクス論)

S3P-F1 15:20
Critical role of electrical coupling in generating population oscillations in neocortex, at frequencies from <1 Hz to >100 Hz
Roger D. Traub
Department of Physiology, Pharmacology and Neurology, SUNY Downstate Medical Center, USA

S3P-F2 15:50
Coding by action potential waveform in the cortex
Hugh P. C. Robinson[1], Gonzalo G. De Polavieja[2], Mikko Juusola[3]
[1]Department of Physiology, Development and Neuroscience, University of Cambridge, UK, [2]Dept of Theoretical Physics, Universidad Autonoma de Madrid, Spain, [3]Dept of Biomedical Sciences, University of Sheffield, UK

S3P-F3 16:20
Is electric field a way for neuronal communication? Steep decrease of dendritc membrane resistivity controls neuronal responses to extracellular electric fields
Masashi Inoue[1], Tohru Aonishi[2]
[1]Tokyo Univ of Pharm and Life Sci, [2]Tokyo Inst of Tech

S3P-F4 16:45
電場電位はニューロンの情報伝達手段か？ - 理論的側面 -
Is electric field a way for neuronal communication? Steep decrease of dendritc membrane resistivity controls neuronal responses to extracellular electric fields -Theoretical aspect-
青西　亨(Tohru Aonishi)
東工大院・総合理工学・知能システム科学

S3P-F5 17:10
An attempt of modeling signal transduction system in neurons: Computer simulation of PKC translocation wave along dendrites of cerebellar Purkinje cells
Norihiro Katayama, Jio Bao, Akihiro Karashima, Mitsuyuki Nakao, Hiroshi Tsubokawa
Graduate School of Information Sciences, Tohoku University, Japan



## Symposium 3P-G　15:15-17:45　Room G

## 脳機能発現と維持におけるアストロサイトの役割
## Roles of Astrocytes in the Brain Function and its Maintenance

Chairpersons　工藤　佳久（Yoshihisa Kudo）（東京薬科大学　名誉教授）
小泉　修一（Schuichi Koizumi）（山梨大学大学院医学工学総合研究部）

演題番号（*）は口演からの繰込

S3P-G1 15:15
アストロサイトに発現する GPCR の機能解析
Functional role of GPCRs expressed in astrocytes
和田　圭司[1,2](Keiji Wada), 古田　晶子[1](Akiko Furuta), 和田恵津子[1,2](Etsuko Wada)
[1]国立精神・神経センター, [2]科学技術振興機構CREST

S3P-G2 15:39
リン酸化によるグリア細胞の機能制御
Regulation of glial functions by protein phosphorylation
高橋　正身(Masami Takahashi), 板倉　誠(Makoto Itakura), 安田　圭一(Keiichi Yasuda),
菅谷津貴子(Tsukiko Sugaya)
北里大・医・生化学

S3P-G3 16:03
アストロサイトにおけるアミノ酸代謝異常と精神神経疾患
Disruption of amino acid metabolism in astrocyte and neurological disorders
榎戸　靖(Yasushi Enokido)
東京医科歯科大学

S3P-G4 16:27
中枢神経外傷における reactive astrocyte の新しい役割
A dual role for reactive astrocytes after spinal cord injury
岡田　誠司[1](Seiji Okada), 中村　雅也[2](Masaya Nakamura), 戸山　芳昭[2](Yoshiaki Toyama),
岩本　幸英[3](Yukihide Iwamoto), 岡野　栄之[4](Hideyuki Okano)
[1]九州大院・医・SSP幹細胞ユニット, [2]慶應大・医・整形外科, [3]九州大院・医・整形外科, [4]慶應大・医・生理学

S3P-G5* 16:51
BMP 受容体シグナルが制御するアストロサイトの増殖
BMP receptor signaling differentially regulates astrocytic proliferation.
山田　真久[1](Masahisa Yamada), 岸田　治夫[1](Haruo Kishida), 北村　尚士[1](Naohito Kitamura),
三品　裕司[2](Yuji Mishina), 新家　瑠奈[1](Runa Araya)
[1]理研・脳センター・山田研究ユニット, [2]NIH-NIEHS

S3P-G6 The two-photon microscopic analysis of cortical spreading depression
17:15 高野 隆弘[1](Takahiro Takano), Guo-Feng Tian[1], Anker J. Hansen[2], Maiken Nedergaard[1]
[1]University of Rochester, Medical Center, [2]Discovery, Novo Nordisk, Maaloev, Denmark

## Symposium 3P-H 15:15-17:45 Room H

## 幻肢痛、幻肢覚における脳機能再構築
## Cerebral Reorganization in the Phantom Limb Pain and Sensation

Chairpersons 吉峰 俊樹 (Toshiki Yoshimine) (大阪大学大学院脳神経外科)
伊佐 正 (Tadashi Isa) (自然科学研究機構生理学研究所)

S3P-H1 各種神経イメージング手法を用いたヒト痛覚認知機構の解明
15:15 Analysis of pain cognition by neuroimaging study
柿木 隆介(Ryusuke Kakigi)
自然科学研究機構・生理研・統合生理

S3P-H2 大脳一次運動野刺激による大脳皮質再構築の検討
15:50 Analysis of cortical reorganization (CR) of the patients with phantom-limb pain and the mechanism of motor cortex stimulation
齋藤 洋一(Youichi Saitoh), 柳澤 琢史(Takufumi Yanagisawa), 押野 悟(Satoru Oshino),
平田 雅之(Masayuki Hirata), 後藤 哲(Tetsu Goto), 細見 晃一(Koichi Hosomi), 谷 直樹(Naoki Tani),
吉峰 俊樹(Toshiki Yoshimine)
大阪大学大学院医学系研究科 脳神経外科

S3P-H3 幻肢痛に対する視床知覚中継核刺激
16:25 Thalamic sensory relay nucleus stimulation and phantom limb pain
片山 容一(Yoichi Katayama)
日本大学・医・脳神経外科

S3P-H4 Cerebral reorganization by adaptable robot hand
17:00 Hiroshi Yokoi[1], Ryu Katoh[1], Alejandro Arieta Hernandez[1], Tamaki Miyamoto[2], Katsunori Ikoma[2], Takashi Onishi[3], Wenwei Yu[4], Tamio Arai[1]
[1]Departiment of Precision Engineering, The University of Tokyo, Tokyo, Japan, [2]School of Medicine, Hokkaido University, [3]Radiology, National Center of Neurology and Psychiatry, [4]Department of Medical System Engineering, Chiba University

## Symposium 3P-J 15:15-17:45 Room J

## ハイスループットの遺伝子改変マウスの作成と表現型解析
## High Throughput Generation and Phenotyping of Genetically-engineered Mice

Chairpersons 宮川 剛 (Tsuyoshi Miyakawa) (藤田保健衛生大学総合医科学研究所)
饗場 篤 (Atsu Aiba) (神戸大学大学院分子細胞生物学講座細胞生物学分野)

S3P-J1 遺伝子改変マウスの体系的な脳行動表現型解析と精神神経疾患研究
15:15 Impact of brain-behavior phenotypying of genetically-engineered mice on research of neuropsychiatric disorders
宮川 剛(Tsuyoshi Miyakawa)
京都大院・医・先端技術センター

S3P-J2 C57BL/6ES 細胞株 RENKA を用いたコンディショナルノックアウトマウスの系統的作成
15:40 Systematic generation of conditional knockout mice using C57BL/6 ES cell RENKA
崎村 建司(Kenji Sakimura), 阿部 学(Manabu Abe), 山崎 真弥(Maya Yamazaki),
薄井 宏(Hiroshi Usui)
新潟大学・脳研究所・細胞神経生物学

S3P-J3 16:00
標準化されたマウス表現型解析基盤と表現型データベース
Standardized mouse phenotyping platform and integrative database system
桝屋　啓志(Hiroshi Masuya)
理研GSC・ゲノム機能情報

S3P-J4 16:20
The genes to cognition programme
Noboru H. Komiyama, Seth_GN Grant
Wellcome Trust Sanger Institute

S3P-J5 16:50
mGluR1 シグナルの遺伝学的解析及びプロテオミクス解析
Genetic and proteomic approaches to mGluR1-signaling
饗場　篤(Atsu Aiba)
神戸大・院医・分子遺伝

S3P-J6 17:10
The organizing principles of real-time memory encoding: Neural clique assemblies and universal neural codes
Joe Z Tsien
Center for Systems Neurobiology Departments of Pharmacology and Biomedical Engineering, Boston University, Boston, USA



## Symposium 3P-K　15:15-17:45　Room K

## 脳の認知制御　神経回路のダイナミクスから
## EEG Rhythms and Cognitive Control Cognitive Control and Network Dynamics in the Brain

Chairpersons Scott Makeig(Swartz Center for Computational Neuroscience, University of California San Diego)
山口　陽子（Yoko Yamaguchi）(理化学研究所脳科学総合研究センター創発知能ダイナミクス研究チーム)

S3P-K1 15:15
情動に関する中枢神経説と末梢神経説～情動過程の流れ～
Central and peripheral theories of emotion ～ flow of emotional process ～
堀　悦郎[1,2](Etsuro Hori), 田積　徹[2,3](Toru Tazumi), 小野　武年[1,2](Taketoshi Ono),
西条　寿夫[1,2](Hisao Nishijo)
[1]富山大・院・医学薬学研究部・システム情動科学, [2]科学技術振興機構, CREST, [3]聖泉大学・人間学部

S3P-K2 15:55
Dynamical properties of whole-head EEG synchronization: spontaneous and evoked activity
Cees van Leeuwen
Laboratory for Perceptual Dynamics, RIKEN BSI, Wako-shi, Japan

S3P-K3 16:30
認知制御の神経機構に関する仮説：複数の周波数帯のリズム活動が同期して形成される神経回路
A hypothesis of oscillation networks with multiple frequency synchronization for cognitive control
山口　陽子[1](Yoko Yamaguchi), 北城　圭一[1](Keiichi Kitajo), 水原　啓暁[1,2](Hiroaki Mizuhara),
佐藤　直行[1](Naoyuki Sato), 尾崎　隆[1](Takashi Ozaki)
[1]理化学研究所脳科学総合研究センター　創発知能ダイナミクス研究チーム, [2]京都大学大学院情報学研究科知能情報学専攻

S3P-K4 17:05
Coordination of oscillatory and non-oscillatory modes of brain activity during active cognition in humans
Scott Makeig
University of California San Diego

## NCS2P-A 14:00-16:00 Room A

### 日本神経化学会 若手奨励賞シンポジウム
### JSN Young Scientists Lectureship Award Symposium

Chairpersons 津田 正明 (Masaaki Tsuda)
田代 朋子 (Tomoko Tashiro)

NCS2P-A1 **脳組織における細胞種特異的な小胞体ストレス応答機構**
Cell type-specific ER stress response in the brain
近藤 慎一(Shinichi Kondo)
宮崎大学医学部解剖学講座分子細胞生物学分野

NCS2P-A2 **自閉症の発症に関連する遺伝子 CAP2 の解析**
Analysis of autism-related gene CAP2/CADPS2
定方 哲史(Tetsushi Sadakata)
理化学研究所分子神経形成研究チーム

NCS2P-A3 **脊髄ミクログリアを介する神経因性疼痛の発症維持メカニズム**
Mechanisms underlying neuropathic pain through spinal microglia
津田 誠(Makoto Tsuda)
九州大学大学院薬学研究院薬理学専攻分野

# ランチョンセミナー　Luncheon Seminars

## LS1-A　株式会社成茂科学器械研究所　NARISHIGE SCIENTIFIC INSTRUMENT LAB.

**September 10 (Mon)　12:00～12:50　Room A**

### グリア細胞の電気生理学的研究
### Electrophysiological studies on glial cells

座長：工藤　佳久（東京薬科大学生命科学部）
Yoshihisa Kudo (School of Life Science,Tokyo University of Pharmacy and Life Science)

(J)　ニューロンとグリアの相互作用
Interactions between neurons and glial cells
加藤　宏司（山形大学医学部神経機能統御学分野）
Hiroshi Kato (Department of Neurophysiology,Yamagata University Faculty of Medicine)

## LS1-B　エーザイ株式会社　Eisai Co., Ltd.
## ファイザー株式会社　Pfizer Japan Inc.

**September 10 (Mon)　12:00～12:50　Room B**

座長：貫名　信行（独立行政法人理化学研究所　脳科学総合研究センター 病因遺伝子研究グループ
グループディレクター）
Nobuyuki Nukina (RIKEN Brain Science Institute)

(J)　アルツハイマー病の分子病理学
Molecular Pathology of Alzheimer's disease
岩坪　　威（東京大学大学院　医学系研究科　神経病理学分野　教授
薬学系研究科　臨床薬学教室　教授）
Takeshi Iwatsubo (Tokyo University)

## LS1-C　大塚製薬株式会社　Otsuka Pharmaceutical Co., Ltd.

**September 10 (Mon)　12:00～12:50　Room C**

座長：西川　　徹（東京医科歯科大学大学院　精神行動医科学　教授）
Toru Nishikawa (Professor, Section of Psychiatry and Behavioral Sciences, Tokyo Medical and Dental University Graduate School )

(J)　受容体プロファイルから見た理想的な抗精神病薬～統合失調症の病態生理を含めて～
Ideal antipsychotic agents in terms of receptor profile - implication in pathophysiology of schizophrenia
岩田　仲生（藤田保健衛生大学医学部　精神医学教室　教授）
Nakao Iwata (Professor, Department of Psychiatry, Fujita Health University School of Medicine)



(J)…Japanese presentation

## LS1-D　室町機械株式会社　MUROMACHI KIKAI CO., LTD.
## Neuralynx, Inc.　Neuralynx, Inc.

**September 10 (Mon)　12:00～12:50　Room D**

### 多電極脳活動記録：技術と応用
### Multi-electrode recording: technologies and applications

座長：藤井　直敬（理化学研究所　脳科学総合研究センター　象徴概念発達研究チーム）
Naotaka Fujii (Laboratory for Symbolic Cognitive Development, RIKEN BSI)

(E)　社会的知性解明への挑戦
How to reveal neural mechanism of social intelligence.
藤井　直敬（理化学研究所　脳科学総合研究センター　象徴概念発達研究チーム）
Naotaka Fujii (Laboratory for Symbolic Cognitive Development, RIKEN BSI)

(E)　無線式神経活動記録手法とリアルタイム実験制御
Digital telemetry system and real-time experimental controls
Casey Stengel (Neuralynx, Inc.)

## LS1-F　カール　ツァイス　マイクロイメージング　Carl Zeiss MicroImaging

**September 10 (Mon)　12:00～12:50　Room F**

### 共焦点レーザ走査顕微鏡による神経微細構造のイメージング—ZEISS のソフトウェア革新"ZEN"—
### Imaging Neuronal Morphology by Confocal Laser Scanning Microscopy - Innovation of New ZEISS Software "ZEN" -

座長：石館　文善（バイオリサーチマーケティング　ディビジョン）
Fumiyoshi Ishidate (Bio Research Marketing Division)

(J)　レーザー走査顕微鏡による神経の微細構造イメージング
Imaging Neuronal Morphology by Laser Scanning Microscopy
岡部　繁男（東京大学大学院医学系研究科神経細胞生物学分野）
Shigeo Okabe (Department of Cellular Neurobiology, Graduate School of Medicine, University of Tokyo)

(E)　レーザスキャン顕微鏡の新世代：新しいナビゲーションソフトウエアのご紹介
ZEN - Efficient Navigation. The New Software for the Laser Scanning Microscopes from Carl Zeiss.
Bernhard Zimmermann (Carl Zeiss MicroImaging GmbH, Jena, Germany)

## LS2-D　ヤンセン ファーマ株式会社　Janssen Pharmaceutical K.K.

**September 11 (Tue)　12:00～12:50　Room D**

座長：鍋島　俊隆（名城大学大学院薬学研究科臨床薬学専攻病態解析学コース薬品作用学教室　教授）
Toshitaka Nabeshima (Professor and Chairman, Department of Chemical Pharmacology, Graduate School of Pharmaceutical Sciences, Meijo University)

(E)　Individualization and Management of Antiepileptic Drug Therapy
Emilio Perucca (Professor of Clinical Pharmacology and Director, Laboratories for Diagnostics and Applied Biological Research, Institute of Neurology and Clinical Pharmacology Unit, University of Pavia, Italy)

(J) …Japanese presentation　(E) …English presentation

## LS2-E　オリンパス株式会社　OLYMPUS CORPORATION

**September 11 (Tue)　12:00～12:50　Room E**

座長：加藤　誠（オリンパス（株）ＭＩＳ事業部　ＭＩＳマーケティング部）
MAKOTO KATO（OLYMPUS　CORPORATION　MIS Marketing Department）

（J）大脳シナプスの２光子顕微鏡による研究
Two-photon imaging and stimulation of the synapses in the brain.
河西　春郎（東京大学　大学院医学系研究科　疾患生命工学センター）
Haruo Kasai（Center for Disease Biology and Integrative Medicine, Graduate School of Medicine, The University of Tokyo）

## LS2-F　ライカ　マイクロシステムズ株式会社　Leica Microsystems K.K.

**September 11 (Tue)　12:00～12:50　Room F**

座長：鈴木　博（顕微鏡プロダクトマーケティング部　企画）
Hiroshi Suzuki（Microscope　Product Marketing　Planning）

（E）光学走査顕微鏡、共焦点顕微鏡に関するブレークスルー技術
Breakthrough Technologies for light scanning or confocal microscope
Irmtraud Steinmetz（International Marketing Division）

## LS2-G　田辺製薬株式会社　TANABE SEIYAKU CO., LTD.

**September 11 (Tue)　12:00～12:50　Room G**

座長：吉良　潤一（九州大学大学院医学研究院神経内科）
JUN-ICHI KIRA（Department of Neurology, Neurological Institute Graduate School of Medical Sciences, Kyushu University）

（J）自己免疫性小脳失調症の診断と治療 - IVIg は有効か？ -
Diagnosis and treatment of autoimmune cerebellar ataxia - IVIg is effective? -
南里　和紀（東京医科大学八王子医療センター神経内科）
Kazunori Nanri（Department of Neurology, Tokyo Medical University Hachioji Medical Center）

## LS2-H　プレクソン　インコーポレーション　PLEXON Incorporated
## 株式会社フィジオテック　Physio-Tech Co.,Ltd.

**September 11 (Tue)　12:00～12:50　Room H**

**電気生理学研究による情報表現・認知との連関**
**Neuronal Representation of Information. －Electrophysiological Approach for Cognition－**

座長：谷藤　学（独立行政法人理化学研究所脳科学総合研究センター、脳を知る領域認知脳科学研究グループ、脳統合機能研究チーム　チームリーダー）
Manabu Tanifuji（Brain Science Institute, Understanding the Brain　Cognitive Brain Science Group　Laboratory for Integrative Neural Systems）

（E）大脳辺縁系ニューロンはどのような行動情報を表現しているか？
What behavioral information does neuronal activity in the limbic system convey?
堀　悦郎（富山大学大学院医学薬学研究部システム情動科学 准教授）
Etsuro Hori（System Emotional Science　Graduate School of Medicine and Pharmaceutical Science for Research University of Toyama）

(J)…Japanese presentation　(E)…English presentation

(E) マカクザルTE野においてポピュレーションコーディングによる物体の視覚表現を探る
～高密度多電極アレイ記録法によるアプローチ
Dense multiple electrode recording method to study the population coding of visual object representation in macaque area TE.
宮川　尚久（独立行政法人理化学研究所脳科学総合研究センター、脳を知る領域認知脳科学研究グループ、脳統合機能研究チーム　研究員）
Naohisa Miyakawa (Brain Science Institute, Understanding the Brain Cognitive Brain Science Group Laboratory for Integrative Neural Systems)

## LS3-D　株式会社ニコンインステック　NIKON INSTECH CO.,LTD.

**September 12 (Wed) 12:00～12:50 Room D**

### 生細胞のリアルタイムイメージング　－タンパク質からシグナル分子まで－
### Realtime imaging in "living" cells -From proteins to signaling molecules-

座長：及川　義朗（(株) ニコンインステック　AS部）
Yoshiro Oikawa (Application Support Department, NIKON INSTECH CO., LTD.)

(J) IP 3受容体の細胞内トラフィッキング
Intracellular trafficking of Inositol 1,4,5,-Trisphosphate Receptors (IP,Rs)
坂内　博子（理研 脳科学総合研究センター 発生神経生物研究チーム）
Hiroko Bannai

(J) 新規IP3センサーを用いた細胞内のIP3とカルシウム濃度の同時測定
IP3 and calcium in mammalian cells
松浦　　徹（理研 脳科学総合研究センター 発生神経生物研究チーム）
Toru Matsuura

(J) 今後の新しい展開に向けて
Towards further development of the signaling field
御子柴克彦（理研 脳科学総合研究センター 発生発達研究グループ）
Katsuhiko Mikoshiba

## LS3-F　アフィメトリクス・ジャパン株式会社　Affymetrix Japan K.K.

**September 12 (Wed) 12:00～12:50 Room F**

(E) GeneChip® リシークエンシングシステムを応用した神経変性疾患の網羅的遺伝子解析
The application of GeneChip® resequencing system to the comprehensive gene analysis of neurodegenerative diseases
高橋　祐二（東京大学医学部附属病院神経内科　助教授）
Yuji Takahashi (Department of Neurology, Graduate School of Medicine, The University of Tokyo)

(J) ゲノム解析における GeneChip® Array の活用
An introduction to GeneChip® Arrays for genome analysis
渋谷　健太（アフィメトリクス・ジャパン株式会社）
Kenta Shibuya (Affymetrix Japan K.K.)

## LS3-G　(独) 科学技術振興機構　社会技術研究開発センター　JST RISTEX

**September 12 (Wed)　12:00～12:50　Room G**

### 脳機能イメージングにおける倫理を考える
### Ethics of Neuroimaging

座長：佐倉　統（東京大学情報学環）
Osamu Sakura (Interfaculty Initiative in Information Studies, University of Tokyo)

(J)　脳研究支援施設における倫理・安全審査のありかた
－ ATR 脳活動イメージングセンタでの経験から－
IRB Management at ATR Brain Activity Imaging Center
正木　信夫（(株) ATR-Promotions 脳活動イメージングセンタ事業部）
Shinobu Masaki (Brain Activity Imaging Center, ATR-Promotions Inc.)

(J)　神経科学は新たな障害観を生むか？－学際バリアフリー研究から見た神経倫理－
Do New Views of Disabilities Emerge from Neuroscience? : Neuroethics in Interdisciplinary Barrier-Free Research
近藤　武夫（東京大学先端科学技術研究センター バリアフリー分野）
Takeo Kondo (Research Center for Advanced Science and Technology, The University of Tokyo)

(J)　海外における脳機能イメージング研究の倫理動向と日本の展望
Ethics of Neuroimaging: International situation and perspectives in Japan
福士　珠美（(独) 科学技術振興機構　社会技術研究開発センター）
Tamami Fukushi (JST RISTEX)



(J)…Japanese presentation

# プログラム Program

## 一般口演
Oral Sessions

## ポスター発表
Poster Sessions

## Oral Session 1P-A 16:00-17:00 Room A

### 幹細胞 1
### Stem Cells 1

Chairperson 小野 勝彦 (Katsuhiko Ono) (自然科学研究機構生理学研究所分子神経生理部門)

O1P-A01
16:00
**神経前駆細胞の分裂と分化のシグナル連鎖**
**A role for glycogen synthase kinase 3β and β-catenin in coordinating proliferation and differentiation of telencephalic neural precursor cells**
清水 健史[1](Takeshi Shimizu), 鹿川 哲史[1](Tetsushi Kagawa), 井上 俊洋[1](Toshihiro Inoue),
高田 慎治[2](Shinji Takada), 田賀 哲也[1](Tetsuya Taga)
[1]熊本大 発医研 転写制御, [2]統合バイオサイエンス、岡崎

O1P-A02
16:15
**単一細胞の網羅的遺伝子発現プロファイルにより明らかとなった哺乳類神経前駆細胞の性質**
**Properties of mammalian neural progenitor cells as revealed by genome-wide single cell gene expression profiles**
川口 綾乃[1](Ayano Kawaguchi), 井川 知子[1](Tomoko Ikawa), 粕川 雄也[2](Takeya Kasukawa),
上田 泰己[2](Hiroki Ueda), 栗本 一基[3](Kazuki Kurimoto), 斎藤 通紀[3](Mitinori Saitou),
松崎 文雄[1,4](Fumio Matsuzaki)
[1]理研・CDB・非対称細胞分裂, [2]理研・CDB・機能ゲノミクス, [3]理研・CDB・哺乳類生殖細胞, [4]CREST・科学技術振興機構

O1P-A03
16:30
**ヒストン脱アセチル化酵素 SIRT1 の核内移行と神経分化**
**Nuclear translocation of histone deacetylase SIRT1 affects neuronal differentiation**
久原 真[1,2](Shin Hisahara), 千葉 進[2](Susumu Chiba), 松本 博之[2](Hiroyuki Matsumoto),
佐藤 真[3](Makoto Sato), 下濱 俊[2](Shun Shimohma), 堀尾 嘉幸[1](Yoshiyuki Horio)
[1]札幌医大・医・薬理, [2]札幌医大・医・神経内科, [3]福井大・医・形態機能医科学

O1P-A04
16:45
**ショウジョウバエ脳において、Tailless は細胞周期遺伝子を促進し Prospero を抑制することによって、神経幹細胞の自己複製を維持する**
**Tailless maintains neural stem cell renewal by activating cell-cycle genes and repressing Prospero in the *Drosophila* brain**
来栖 光彦[1](Mitsuhiko Kurusu), 丸山 泰[2](Yasushi Maruyama), 岡部 正隆[3](Masataka Okabe),
鈴木えみ子[1](Emiko Suzuki), 古久保 - 徳永克男[2](Katsuo Furukubo-Tokunaga)
[1]国立遺伝学研究所, [2]筑波大学大学院 生命環境科学研究科, [3]東京慈恵会医科大学 DNA医学研究所



## Oral Session 1P-A 17:00-18:00 Room A

### 幹細胞 2
### Stem Cells 2

Chairperson 鹿川 哲史 (Tetsushi Kagawa) (熊本大学発生医学研究センター転写制御分野)

O1P-A05
17:00
**RNA 結合蛋白質 Musashi の哺乳類中枢・末梢神経系の発生・再生過程における解析**
***In vivo* and *in vitro* analysis of Musashi family in mammalian CNS and PNS development and regeneration**
芝田 晋介[1](Shinsuke Shibata), 榊原 伸一[2](Shin-ichi Sakakibara), 岡野ジェイムス洋尚[1](Hirotaka J. Okano),
岡野 栄之[1,3](Hideyuki Okano)
[1]慶應・医・生理, [2]獨協医大・解剖, [3]科技団・戦略

O1P-A06
17:15
**シナプス形成を終えたマウス網膜水平細胞の増殖**
**Differentiated horizontal interneurons can proliferate after synaptogenesis**
味岡 逸樹[1](Itsuki Ajioka), Rodrigo Martins[1], Ildar Bayazitov[1], Dianna Johnson[2], Stacy Donovan[1],
Stanislav Zakharenko[1], Michael Dyer[1,2]
[1]セントジュード小児研究病院・発生神経生物学, [2]テネシー州立大・医・眼科

O1P-A07 正常成体脊髄に存在するグリア前駆細胞の探索
17:30 Exploring the glial progenitor cells in the intact adult spinal cord
北田 容章(Masaaki Kitada), 糸数 裕(Yutaka Itokazu), 井出 千束(Chizuka Ide),
出澤 真理(Mari Dezawa)
京都大院・医・機能微細形態

O1P-A08 後脳の Olig2 前駆細胞は鰓運動 / 内臓運動 (branchiomotor/visceromotor) ニューロンにも分化する
17:45 Paradoxial neuron differentiation in the hindbrain: Olig2+ cells also differentiate into branchiomotor/visceromotor neurons.
小野 勝彦[1](Katsuhiko Ono), 池田 和代[2](Kazuyo Ikeda), 竹林 浩秀[1](Hirohide Takebayashi),
渡辺 啓介[1](Keisuke Watanabe), 池中 一裕[1](Kazuhiro Ikenaka)
[1]生理学研究所・分子神経生理部門, [2]香川大学医学部

**Oral Session 1P-B** 14:00-15:00 Room B

## 睡眠・生体リズム
## Sleep and Biological Rhythms

Chairperson 香山 雪彦 (Yukihiko Kayama) (福島県立医科大学神経生理学講座)

O1P-B01 Conditional Tg マウスにみられる CRH のレム睡眠促進作用
14:00 REM sleep enhancement in CNS-specific CRH-overexpressing mice
木村昌由美(M Kimura), P Mueller-Preuss, E Wiesner, C Flachskamm, A Lu, JM Deussing
マックス・プランク精神医学研究所

O1P-B02 増殖神経幹細胞のサーカディアンリズムと細胞周期の関係
14:15 Cell cycle in proliferating embryonic stem cell in relation to circadian rhythm
磯部 芳明[1](Yoshiaki Isobe), 津田 洋幸[2](Hiroyuki Tsuda)
[1]名古屋市大・医・脳神経生理, [2]名古屋市大院・医・分子毒性

O1P-B03 レム睡眠期に海馬と扁桃体で観測されるシータ波の同期現象
14:30 Synchronization of theta activity in hippocampus and amygdala during REM sleep
辛島 彰洋(Akihiro Karashima), 林 海緒(Mio Hayashi), 嶋崎 統彦(Motohiko Shimazaki),
片山 統裕(Norihiro Katayama), 中尾 光之(Mitsuyuki Nakao)
東北大・院・情報・応用情報

O1P-B04 マウスの覚醒ニューロン、睡眠ニューロンが示す睡眠・覚醒移行期における活動変化の順序
14:45 A temporal sequence of changes in firing among the wake- and sleep-promoting neurons at the transition between sleep and wakefulness in mice
高橋 和巳[1](Kazumi Takahashi), 小山 純正[2](Yoshimasa Koyama), 香山 雪彦[1](Yukihiko Kayama),
Jian-Sheng Lin[3], 酒井 一弥[3](Kazuya Sakai)
[1]公立大学法人福島県立医科大学・医・神経生理学講座, [2]福島大学・共生システム理工学類, [3]クロード・ベルナール大学

**Oral Session 1P-B** 15:00-16:00 Room B

## 体性運動 1
## Somatomotor System 1

Chairperson 関 和彦 (Kazuhiko Seki) (自然科学研究機構生理学研究所)

O1P-B05 随意運動の制御における筋感覚入力の役割
15:00 Modulation of spinal unit responses evoked from muscle afferents in a monkey performing an instructed delay task.
関 和彦[1,2](kazuhiko seki), 武井 智彦[1,3](Tomohiko Takei)
[1]生理研・ 認知行動発達, [2]総研大・ 生命科学, [3]京大・ 院・ 人間環境

O1P-B06 精密把持遂行時の皮質－手指筋間コヒーレンスの空間的分布
15:15 Spatial distribution of cortico-muscular coupling during precision grip in monkeys
伊佐　正[1,2,3](Tadashi Isa)，西村　幸男[1,2](Yukio Nishimura)
[1]自然科学研究機構生理学研究所，[2]科学技術振興機構CREST，[3]総研大・生命科学

O1P-B07 手の空間位置知覚における視覚優位性は後部頭頂皮質で計算される
15:30 Activity in posterior parietal cortex mediates the visual dominance over kinesthesia
羽倉　信宏[1,2](Nobuhiro Hagura)，武井　智彦[1](Tomohiko Takei)，廣瀬　智士[1,3](Satoshi Hirose)，
荒牧　勇[3,4,5](Yu Aramaki)，松村　道一[1](Michikazu Matumura)，定藤　規弘[4](Norihiro Sadato)，
内藤　栄一[1,3,5](Eiichi Naito)
[1]京都大院・人間・環境学，[2]日本学術振興会，[3]ATR脳情報研究所，[4]大学共同利用機関法人　自然科学研究機構生理学研究所，[5]独立行政法人　情報通信研究機構

O1P-B08 軌道計画を行わない到達運動学習・制御モデル
15:45 Motor control-learning model for arm' s reaching movements without desired trajectory
神原　裕行(Hiroyuki Kambara)，金　敬植(Kyoungsik Kim)，辛　徳(Duk Shin)，
佐藤　誠(Makoto Sato)，小池　康晴(Yasuharu Koike)
東京工業大学・精密工学研究所

**Oral Session 1P-B** 16:00-17:00 Room B

## 体性運動 2
## Somatomotor System 2

Chairperson　金子　武嗣（Takeshi Kaneko）（京都大学医学部高次脳形態学講座）

O1P-B09 プルキンエ細胞単一軸索形態とアルドラーゼC発現パタンから見た小脳皮質－小脳核間のトポグラッフィクな構築
16:00 Cortico-nuclear topographic organization in terms of morphology of single Purkinje cell axons and aldolase C expression in the rat cerebellum
杉原　泉(Izumi Sugihara)，篠田　義一(Yoshikazu Shinoda)
東京医科歯科大学・大学院・システム神経生理学

O1P-B10 基底核～脳幹投射系による歩行制御の神経機構
16:15 Basal ganglia modulation of fictive locomotion in decerebrate cats
高草木　薫[1](Kaoru Takakusaki)，太田　亮[2](Ryo Ohta)
[1]旭川医科大学・生理，[2]旭川医科大学・耳鼻科

O1P-B11 ラット線条体パッチ・マトリックス構造と視床線条体ニューロンの投射様式
16:30 Single cell tracing of thalamostriatal projection neurons with reference to patch and matrix compartments of rat striatum
雲財　知[1](Tomo Unzai)，藤山　文乃[1](Fumino Fujiyama)，金子　武嗣[1,2](Takeshi Kaneko)
[1]京都大院・医・高次脳形態学，[2]科学技術振興機構戦略的創造研究推進事業

O1P-B12 ラット大脳皮質運動関連領野へ投射する単一視床ニューロン軸索分岐の形態学的解析
16:45 Single-axon tracing study of thalamocortical projections arising from the motor nuclei of the rat
倉本恵梨子[1](Eriko Kuramoto)，古田　貴寛[1](Takahiro Furuta)，中村　公一[1,2](Kouichi Nakamura)，
日置　寛之[1](Hiroyuki Hioki)，雲財　知[1](Tomo Unzai)，金子　武嗣[1,2](Takeshi Kaneko)
[1]京都大院・医・高次脳形態，[2]科学技術振興機構　戦略的創造研究推進事業

## Oral Session 1P-B　17:00-18:00　Room B

### 体性運動 3
### Somatomotor System 3

Chairperson　丹治　順 (Jun Tanji) (玉川大学脳科学研究所)

O1P-B13
17:00
**無意味動作模倣における動的な視覚運動マッチングに関わる神経機構**
**Neural correlates of dynamic visuomotor matching for meaningless gesture imitation**
小川　健二[1](Kenji Ogawa), 乾　敏郎[1,2](Toshio Inui)
[1]JST ERATO浅田共創知能システムプロジェクト, [2]京都大学大学院情報学研究科

O1P-B14
17:15
**遅延到達運動課題中の MEG 脳活動計測**
**MEG activity during a delayed visuomotor reaching task**
相澤　寛(Hiroshi Aizawa), 蔵田　潔(Kiyoshi Kurata)
弘前大・医・生理学第二

O1P-B15
17:30
**運動前野は動作概念から運動企画への情報変換過程に関与する**
**Premotor cortex (PM) is involved in converting a concept for action into a motor plan**
星　英司[1](Eiji Hoshi), 中山　義久[2](Yoshihisa Nakayama), 丹治　順[1,2](Jun Tanji)
[1]玉川大・脳科学研究所, [2]玉川大・院・工学研究科

O1P-B16
17:45
**Inhibition of human motor cortex excitability during stop signal task**
Reda B. Gad[1], Tatsuya Mima[1], Toshihiko Aso[1], Mitsunari Abe[1], Masahiro Nakatsuka[1], Dina F. Mansour[1], Hamdy N. El Tallawy[2], Nageh F. Kamel[2], Takashi Nagamine[1], Hidenao Fukuyama[1]
[1]Human Brain Research Center, Kyoto University, Graduate School of Medicine, Kyoto, Japan, [2]Department of Neurology, Assiut University Hospital, Egypt

## Oral Session 1P-C　14:00-15:00　Room C

### 眼球運動
### Oculomotor System

Chairperson　河野　憲二 (Kenji Kawano) (京都大学大学院医学研究科)

O1P-C01
14:00
**ランダムに見えるが背後にダイナミクスを持つ時系列を予測するヒトの視覚系**
**Human vision can predict semi-random but linear process.**
鹿内　学(Manabu Shikauchi), 石井　信(Shin Ishii), 柴田　智広(Tomohiro Shibata)
奈良先端科学技術大学院大学・情報科学研究科・論理生命学講座

O1P-C02
14:15
**Neural control of saccadic decision-making**
Arjun R., S Chokandre, Aditya Murthy
NBRC, India

O1P-C03
14:30
**単眼性の運動視成分がない視覚刺激によって誘発される追従眼球運動**
**Ocular following responses elicited by binocular first-order motion in primates**
林　隆介(Ryusuke Hayashi), 三浦健一郎(Kenichiro Miura), 田端　宏充(Hiromitsu Tabata), 河野　憲二(Kenji Kawano)
京都大学大学院・医・認知行動脳科学

O1P-C04
14:45
**ニホンザル盲視モデルにおける残存視覚に対応した神経活動**
**Neural correlate of residual vision in the animal model of blindsight**
吉田　正俊[1,2](Masatoshi Yoshida), 高浦　加奈[1,2](Kana Takaura), 伊佐　正[1,2,3](Tadashi Isa)
[1]生理学研究所・認知行動発達, [2]総研大・生命科学, [3]科学技術振興機構・戦略的創造研究推進事業(CREST)

**Oral Session 1P-C** 15:00-16:00 Room C

## 視覚 1
## Visual System 1

Chairperson 大澤 五住 (Izumi Ohzawa) (大阪大学)

O1P-C05 15:00
**下側頭葉皮質細胞による刺激ー報酬連合の速やかな学習**
**Quick learning of stimulus-reward association by neurons in the monkey inferior temporal areas**
最上 嗣生(Tsuguo Mogami), 田中 啓治(Keiji Tanaka)
理化学研究所・BSI

O1P-C06 15:15
**大脳皮質視覚野における単一視床ニューロンの軸索分枝**
**Axonal arborizations of single thalamocortical neurons in the rat visual cortex**
中村 悠[1](Hisashi Nakamura), 日置 寛之[1](Hiroyuki Hioki), 中村 公一[1,2](Kouichi Nakamura), 古田 貴寛[1](Takahiro Furuta), 金子 武嗣[1,2](Takeshi Kaneko)
[1]京都大院・医・高次脳形態学教室, [2]CREST

O1P-C07 15:30
**高次受容野としての周辺抑制の空間構造と情報表現**
**Form representation via center-surround organizations of V1 neurons revealed by high-order receptive field analysis.**
田中 宏喜(Hiroki Tanaka), 大澤 五住(Izumi Ohzawa)
大阪大学・生命機能・脳神経工学

O1P-C08 15:45
**注意の誘引と復帰抑制における一次視覚野の機能の解明**
**Role of striatal visual pathway in facilitatory and inhibitory effect on visual attention**
池田 琢朗[1,2](Takuro Ikeda), 伊佐 正[1,2,3](Tadashi Isa)
[1]生理学研究所・発達生理学研究系, [2]戦略的創造研究推進事業, [3]総合研究大学院大学

**Oral Session 1P-C** 16:00-17:00 Room C

## 視覚 2
## Visual System 2

Chairperson 谷藤 学 (Manabu Tanifuji) (理化学研究所)

O1P-C09 16:00
**タスクスイッチ中の大脳皮質 MT 野の活動：II. サルの判断とニューロン活動との相関**
**Responses of MT neurons during task switching: II. Correlation between neuronal responses and perceptual judgments.**
宇賀 貴紀(Takanori Uka), 佐々木 亮(Ryo Sasaki)
順天堂大学・医・生理学第一

O1P-C10 16:15
**タスクスイッチ中の大脳皮質 MT 野の活動：I. サル及びニューロンの切り替えの定量化**
**Responses of MT neurons during task switching: I. Psychophysical and neuronal switch ratio.**
佐々木 亮(Ryo Sasaki), 宇賀 貴紀(Takanori Uka)
順天堂大・医・生理学第一

O1P-C11 16:30
**サル下側頭皮質の陰影方向選択性ニューロンの活動**
**Neural responses selective for the orientation of the luminance gradient in the inferior temporal cortex of the monkey**
小松 英彦[1](Hidehiko Komatsu), 安田 正治[1,2](Masaharu Yasuda), 郷田 直一[1,2](Naokazu Goda), 坂野 拓[1,2](Taku Banno)
[1]生理学研究所感覚認知情報部門, [2]総合研究大学院大学

O1P-C12 マカク TE 野の複数カラムの活動は視角度に影響されない物体表現を符号化しうる。
16:45 Multiple column activity in macaque area TE can encode object identity across view angles.
宮川 尚久[1](Naohisa Miyakawa), David Blake[2], Michael Merzenich[3], 谷藤 学[1](Manabu Tanifuji)
[1]理研・脳センター・脳統合機能, [2]Dept. Neurol., Medical College of Georgia, [3]Keck Center, UCSF

## Oral Session 1P-C 17:00-18:00 Room C

### 視覚 3
### Visual System 3

Chairperson 藤田 一郎 (Ichiro Fujita) (大阪大学大学院生命機能研究科)

O1P-C13 片眼遮蔽時に NMDA 受容体を阻害すると片眼遮蔽の効果は持続しない
17:00 NMDA receptor blockade during monocular deprivation prevents the maintenance of ocular dominance shift produced by the deprivation
稲葉 美枝[1](Mie Inaba), 吉村由美子[2](Yumiko Yoshimura), 山田 和政[2](Kazumasa Yamada),
小松由紀夫[2](Yukio Komatsu)
[1]京都府立医科大院・医・発達小児科学, [2]名古屋大学・環境医学研究所・視覚神経科学

O1P-C14 ラット視覚野における ERK 活性とその細胞内局在の視覚入力依存的制御
17:15 Regulation of extracellular signal-regulated kinase activity and subcellular localization by visual input in rat visual cortex
高村 明孝[1](Hironori Takamura), 一坂 吏志[2](Satoshi Ichisaka), 林 千尋[2](Chihiro Hayashi),
牧 廣利[2](Hirotoshi Maki), 畠 義郎[1](Yoshio Hata)
[1]鳥取大・院・医・生体高次機能, [2]鳥取大・医・生命・神経生物

O1P-C15 網膜内網状層の側状抑制におけるアマクリン細胞間の電気シナプスの働き
17:30 Cellular components in electrical synapses between retinal amacrine cells contributing to lateral interactions
日高 聰(Soh Hidaka)
藤田保健衛生大・医・生理

O1P-C16 マカクザル V1 と TE の第 III 層錐体細胞の電気生理学的性質の比較
17:45 Comparison of electrophysiological properties of layer III pyramidal neurons between cortical areas V1 and TE of the macaque
石川 理子[1](Ayako Ishikawa), 丸山 敦子[2](Atsuko Maruyama), 細山 大輔[1](Daisuke Hosoyoma),
吉村由美子[2,3](Yumiko Yoshimura), 田村 弘[1,2](Hiroshi Tamura), 佐藤 宏道[4](Hiromichi Sato),
藤田 一郎[1,2](Ichiro Fujita)
[1]大阪大学院・生命機能, [2]科学技術振興機構・CREST, [3]名古屋大・環境医学研・視覚神経科学, [4]大阪大学・医学系研究科

## Oral Session 1P-D 14:00-15:00 Room D

### 回路網形成 1
### Network Formation 1

Chairperson 平田たつみ (Tatsumi Hirata) (国立遺伝学研究所)

O1P-D01 匂い地図を生み出す分子基盤
14:00 Molecular logics to generate the olfactory sensory map
竹内 春樹(Haruki Takeuchi), 芹沢 尚(Shou Serizawa), 坂野 仁(Hitoshi Sakano)
東京大院・理・生化

O1P-D02 嗅覚受容体に依存した転写制御と糸球マップ形成
14:15 Odorant receptor-dependent transcriptional regulation for the olfactory glomerular mapping
今井 猛(Takeshi Imai), 山崎 崇裕(Takahiro Yamazaki), 坂野 仁(Hitoshi Sakano)
東京大院・理・生化

O1P-D03 嗅索道標細胞 (lot 細胞) に対する Sema3F の反発効果
14:30 Repulsive effect of Sema3F on lot cells, the guidepost neurons in the lateral olfactory tract
伊藤 圭祐(Keisuke Ito), 川崎 能彦(Takahiko Kawasaki), 平田たつみ(Tatsumi Hirata)
国立遺伝学研究所・脳機能

O1P-D04 外来遺伝子導入と分子マーカーによるマウス扁桃体発生メカニズムの解析
14:45 Development of the mouse amygdala as revealed by labeling with EGFP gene transfer and molecular markers
相馬 美歩[1](Miho Soma), 伊東 義真[2](Yoshimasa Ito), 相澤 秀紀[3](Hidenori Aizawa),
田中 光一[2](Kohichi Tanaka), 湯浅 茂樹[1](Shigeki Yuasa)
[1]国立精神・神経センター・神経研・微細構造, [2]東京医科歯科大・生命情報・分子神経科学, [3]理化学研究所 脳科学総合研究センター

**Oral Session 1P-D** 15:00-16:00 Room D

## 回路網形成 2
## Network Formation 2

Chairperson 大島登志男 (Toshio Ohshima) (早稲田大学先進理工学部生命医科学科)

O1P-D05 Cdk5 は大脳皮質神経細胞の放射状移動時の多極性 - 双極性変換に必要である
15:00 Cdk5 is required for multipolar-to-bipolar transition during radial neuronal migration in the cerebral cortex
大島登志男[1,2](Toshio Ohshima), 平澤 基之[3](Motoyuki Hirasawa), 田畑 秀典[4](Hidenori Tabata),
岩里 琢治[5](Takuji Iwasato), 糸原 重美[5](Shigeyoshi Itohara), 仲嶋 一範[4](Kazunori Nakajima),
Ashok Kulkarni[3], 御子柴克彦[1](Katsuhiko Mikoshiba)
[1]理研・脳センター・発生神経生物, [2]早稲田大学・先進理工・生命医科, [3]米国国立衛生研究所, [4]慶応・医・解剖, [5]理研・脳センター・行動遺伝

O1P-D06 大脳皮質層形成を制御するマウス Dab1 は核 - 細胞質間をシャトリングする
15:15 Mouse Dab1, an essential component in layer formation of cerebral cortex, shuttles between the nucleus and the cytoplasm
本田 岳夫(Takao Honda), 仲嶋 一範(Kazunori Nakajima)
慶應・医・解剖

O1P-D07 移動中大脳皮質介在神経細胞の CXCR4 依存的「さまよい時間」
15:30 CXCR4-dependent "wandering period" of migrating cortical interneurons
田中 (原田) 大介[1](Daisuke H. Tanaka), 柳田 光俊[1](Mitsutoshi Yanagida), 山嵜 絵海[1](Emi Yamasaki),
Yan Zhu[1], 三上 栄[2](Sakae Mikami), 長澤 丘司[2](Takashi Nagasawa), 柳川右千夫[3](Yuchio Yanagawa),
小幡 邦彦[4](Kunihiko Obata), 村上富士夫[1](Fujio Murakami)
[1]大阪大院・生命機能, [2]京都大・再生研・生体システム制御, [3]群馬大院・医, [4]神経回路メカニズム研究グループ・BSI・理研

O1P-D08 大脳皮質形成過程における NMDA 受容体を介した細胞移動の分子基盤
15:45 Molecular basis of NMDA receptor-mediated neuronal migration in the early stage of mouse cortical development
内野 茂夫[1](Shigeo Uchino), 福村 怜子[2](Reiko Fukumura), 田畑 秀典[3](Hidenori Tabata),
平澤 孝枝[1](Takae Hirasawa), 服部功太郎[2](Kotaro Hattori), 湯浅 茂樹[2](Shigeki Yuasa),
仲嶋 一範[3,4](Kazunori Nakajima), 高坂 新一[1](Shinichi Kohsaka)
[1]国立精神神経センター・神経研究所・代謝, [2]国立精神神経センター・神経研究所・微細, [3]慶応大・医・解剖, [4]東京慈恵会医大・DNA医学研究所・分子神経生物

## Oral Session 1P-D　16:00-17:00　Room D

### 回路網形成 3
### Network Formation 3

Chairperson　能瀬　聡直（Akinao Nose）（東京大学大学院新領域創成科学研究所）

O1P-D09
16:00
**Wnt4 は阻害効果を介してシナプス特異性を決定する**
**Wnt4 is as a local repulsive cue that determines synaptic target specificity**
能瀬　聡直[1,2](Akinao Nose)，稲木美紀子[2](Mikiko Inaki)，油谷　浩幸[3](Hiroyuki Aburatani)
[1]東京大院・新領域・複雑理工，[2]東京大院・理・物理，[3]東京大・先端研

O1P-D10
16:15
**ニューロンの受容領域を決定・維持するリン酸化シグナルネットワーク**
**The kinase signaling network regulating establishment and maintenance of dendritic fields**
榎本　和生[1,2](Kazuo Emoto)，Yuh-Nung Jan[3]
[1]国立遺伝学研究所・神経形態，[2]JSTさきがけ，[3]カリフォルニア大学サンフランシスコ校

O1P-D11
16:30
**神経成長円錐のフィロポディアは自律的に右ねじ方向に回転運動する**
**Filopodia of nerve growth cones autonomously rotate in the direction of the right-handed screw**
玉田　篤史[1,2](Atsushi Tamada)，河瀬　　聡[1](Satoshi Kawase)，村上富士夫[2,3](Fujio Murakami)，
上口　裕之[1](Hiroyuki Kamiguchi)
[1]理研・BSI・神経成長，[2]基礎生物学研究所・行動制御，[3]大阪大・生命機能・脳神経工学

O1P-D12
16:45
**PTPδ変異マウスにおける嗅内野海馬投射の検討**
**Analysis of entorhino-hippocampal projection in PTPδ mutant mice**
中村　史雄[1](Fumio Nakamura)，五嶋　良郎[1](Yoshio Goshima)，上谷　則子[2](Noriko Uetani)，
岩倉洋一郎[2](Yoichiro Iwakura)，Stephen_M. Strittmatter[3]
[1]横浜市立大・医・分子薬理，[2]東京大学医科学研究所・細胞機能，[3]Yale大学医学部神経学教室

## Oral Session 1P-D　17:00-18:00　Room D

### 回路網形成 4
### Network Formation 4

Chairperson　中村　史雄（Fumio Nakamura）（横浜市立大学医学部分子薬理）

O1P-D13
17:00
**CRMP-2、Slp1 と Rab27B は TrkB の順行性輸送を仲介している**
**CRMP-2, Slp1 and Rab27B mediate anterograde transport of TrkB**
有村奈利子(Nariko Arimura)，島田　明子(Akiko Shimada)，貝淵　弘三(Kozo Kaibuchi)
名大院・医・神経情報薬理

O1P-D14
17:15
**MARCKS は膜脂質ラフトにおいて神経突起の形成・伸長に関与する**
**MARCKS participates in neurite initiation and outgrowth through the association with lipid rafts**
山口　大輝[1](hiroki yamaguchi)，白石　光也[2](Mitsuya Shiraishi)，田辺　淳弘[1](Atsuhiro Tanabe)，
佐々木泰治[1](Yasuharu Sasaki)
[1]北里大・薬・薬理，[2]鹿児島大・獣医・薬理

O1P-D15
17:30
**UEV-3（ユビキチン結合酵素 E2 variant）はシナプス形成において p38MAP キナーゼ経路で働いている**
**UEV-3, a ubiquitin conjugating enzyme E2 variant, functions in the p38 MAP kinase pathway for synaptogenesis**
中田　勝紀[1](Katsunori Nakata)，丸山　一郎[1](Ichiro Maruyama)，Yishi Jin[2]
[1]沖縄科学技術研究基盤整備機構・分子神経科学ユニット，[2]Division of Biological Sciences and HHMI, University of California, San Diego

O1P-D16 17:45
E3 ユビキチンライゲース Nedd4 による樹状突起伸長の制御機構
Regulation of dendritic development by E3 ubiquitin ligase Nedd4
川辺 浩志[1](Hiroshi Kawabe), Jeong-Seop Rhee[1], 桂林秀太郎[2,3](Shutaro Katsurabayashi), Antje Neeb[1], 海川 正人[4](Masato Umikawa), 苅谷 研一[4](Ken-ichi Kariya), Christian Rosenmund[2], Nils Brose[1]
[1]Max-Planck-Institute for Experimental Medicine, [2]Baylor College of Medicine, [3]崇城大学薬学部, [4]琉球大学大学院医学系研究科細胞生物学分野

## Oral Session 1P-E 14:00-15:00 Room E

## イオンチャンネル 1
## Ion Channels 1

Chairperson 古市 貞一 (Teiichi Furuichi) (理化学研究所脳科学総合研究センター神経形成)

O1P-E01 14:00
海馬領域における Kv4.2 カリウムチャンネルサブユニットの局在は活動性依存性である
Redistribution of Kv4.2 potassium channels in the hippocampus induced by acute neuronal excitation *in vivo*
神野 尚三[1](Shozo Jinno), 石塚 智[2](Satoru Ishizuka), Andreas Jeromin[3], 小坂 俊夫[1](Toshio Kosaka)
[1]九州大院・医・神経形態, [2]九州工業大院・生命体工学・脳情報, [3]テキサス大・神経科学研

O1P-E02 14:15
Nav1.1 欠損マウスにおけるパルブアルブミン陽性インターニューロンの異常とてんかん
Nav1.1 predominantly localizes to axons of parvalbumin-positive inhibitory interneurons: a circuit basis for epileptic seizures in Nav1.1-deficient mice
荻原 郁夫[1](I Ogiwara), 宮本 浩行[2](H Miyamoto), 森田 規之[3](N Morita), N Atapour[2], 真崎 恵美[1](E Mazaki), 井上 育代[1](I Inoue), 柳川右千夫[5](Y Yanagawa), 小幡 邦彦[4](K Obata), 古市 貞一[3](T Furuichi), ヘンシュ貴雄[2](Takao K Hensch), 山川 和弘[1](K Yamakawa)
[1]理研・脳センター・神経遺伝, [2]理研・脳センター・神経回路発達, [3]理研・脳センター・分子神経形成, [4]理研・脳センター・神経回路メカニズム研究グループ, [5]群馬大学大学院・医学系研究科・遺伝発達行動学、SORST・JST

O1P-E03 14:30
中枢ニューロンにおける高閾値型電位依存性 $Ca^{2+}$ チャネルの容量性 $Ca^{2+}$ 流入への寄与
High-threshold voltage-gated $Ca^{2+}$ channels may mediate capacitive $Ca^{2+}$ entry in central neurons
田端 俊英(Toshihide Tabata), 狩野 方伸(Masanobu Kano)
大阪大院・医・細胞神経科学

O1P-E04 14:45
ニューロンの発火率が入力電流に応じて増加するのはなぜか？
Why does the neuronal firing rate increase with a greater injected current?
和田 克己(Katsumi Wada), 阪口 豊(Yutaka Sakaguchi)
電気通信大学院・情報システム学・人間情報学

## Oral Session 1P-E 15:00-16:00 Room E

## イオンチャンネル 2
## Ion Channels 2

Chairperson 岡田 真人 (Masato Okada) (東京大学大学院新領域創成科学研究科)

O1P-E05 15:00
Bral1 はランビエ絞輪周囲の ECM 複合体形成に必須である
Bral1, brain-specific link protein, is essential for stabilizing extracellular matrix at the node of Ranvier in the CNS.
別宮 洋子(Yoko Bekku), 二宮 善文(Yoshifumi Ninomiya), 大橋 俊孝(Toshitaka Oohashi)
岡山大院・医歯薬・分子医化学

O1P-E06 15:15
バランスされた入力は神経発火の不規則性を発火率変動から切り離して一定に保つ
Balanced inputs "clamps" firing irregularity decoupled from rate fluctuations on any timescale
三浦 佳二[1,2,3](Keiji Miura), 坪 泰宏[3](Yasuhiro Tsubo), 深井 朋樹[1,3](Tomoki Fukai), 岡田 真人[1,3](Masato Okada)
[1]東京大・新領域・複雑理工, [2]学振特別研究員, [3]理化学研究所

O1P-E07 膜特性の不均一性は海馬CA1錐体細胞の樹状突起における信号伝播を向上させる
15:30 Non-uniformity of membrane property improves dendritic signal transfer in hippocampal CA1 pyramidal neuron
大森 敏明[1,2](Toshiaki Omori), 青西 亨[2,3](Toru Aonishi), 岡田 真人[2,4](Masato Okada)
[1]日本学術振興会特別研究員PD, [2]独立行政法人理化学研究所・脳科学総合研究センター, [3]東工大院・総合理工・知能システム科学, [4]東大院・新領域・複雑理工学

O1P-E08 スパイク時刻を正確に予測可能なニューロンモデルの提案
15:45 A simple model can predict the firing times of a cortical neuron
小林 亮太[1](Ryota Kobayashi), 坪 泰宏[2](Yasuhiro Tsubo), 深井 朋樹[2](Tomoki Fukai),
篠本 滋[1](Shigeru Shinomoto)
[1]京都大学 理 物理, [2]理化学研究所脳科学総合研究センター

**Oral Session 1P-E** 16:00-17:00 Room E

## イオンチャンネル 3
## Ion Channels 3

Chairperson 的崎 尚 (Takashi Matozaki) (群馬大学生体調節研究所バイオシグナル分野)

O1P-E09 受容体型膜蛋白質CD47とSHPS-1のトランスエンドサイトーシスとその分子メカニズム
16:00 Trans-endocytosis of CD47 and SHPS-1 and its molecular mechanism
大西 浩史(Hiroshi Ohnishi), 草苅 伸也(Shinya Kusakari), 金 鋒杰(Feng-Jie Jin),
村田 考啓(Takaaki Murata), 篠原 将彦(Masahiko Shinohara), 村田 陽二(Yoji Murata),
岡澤 秀樹(Hideki Okazawa), 的崎 尚(Takashi Matozaki)
群馬大学・生体調節研究所・バイオシグナル分野

O1P-E10 インポーチン$\alpha$および$\beta$の脳内発現分布
16:15 Importin-$\alpha$ and $\beta$ expression in the neonatal and adult mouse brain
細川 康二(Koji Hosokawa), 西 真弓(Mayumi Nishi), 河田 光博(Mitsuhiro Kawata)
京都府立医科大学 解剖学・生体構造科学

O1P-E11 網膜双極細胞におけるTRPM1の機能解析
16:30 The functional analysis of TRPM1 in retinal bipolar cells
小池千恵子[1](Chieko Koike), 佐貫理佳子[1](Rikako Sanuki), 宮田健太郎[2](Kentaro Miyata),
子安 俊行[2](Toshiyuki Koyasu), 三好 智満[3](Tomomitsu Miyoshi), 澤井 元[3](Hajime Sawai),
近藤 峰生[2](Mineo Kondo), 臼倉 治郎[4](Jiro Usukura), 古川 貴久[1](Takahisa Furukawa)
[1](財)大阪バイオ研・発生, [2]名大・医・眼科, [3]阪大院・医・システム生理, [4]名大・先端技術共同研究センター

O1P-E12 バクテリア由来ナトリウムチャネルの電位センサードメインの側方可動性
16:45 Lateral mobility of voltage sensor domain of bacterial sodium channels
名倉 仁[1](Hitoshi Nagura), 入江 克雅[2](Katsumasa Irie), 今井 友也[1](Tomoya Imai),
下村 拓史[1](Takushi Shimomura), 髭 俊秀[1](Toshihide Hige), 藤吉 好則[1](Yoshinori Fujiyoshi)
[1]京都大学院・理・生物物理, [2]理研 播磨研究所

**Oral Session 1P-E** 17:00-18:00 Room E

## イオンチャンネル 4 (J)
## Ion Channels 4 (J)

This session is held in Japanese.

Chairperson 加藤 英之 (Hideyuki Cateau) (理化学研究所脳科学総合研究センター)

O1P-E13 位相応答の形が多様である神経細胞集団の同期解析
17:00 Synchronization of excitatory neurons with strongly heterogeneous phase response
坪 泰宏[1,2](Yasuhiro Tsubo), 寺前順之介[1](Jun-nosuke Teramae), 深井 朋樹[1](Tomoki Fukai)
[1]理研BSI, [2]都神経研・統合生理

O1P-E14 17:15
Nefiracetam による NMDA 受容体賦活作用は Protein kinase C を介する
Nefiracetam potentiates NMDA receptor function via protein kinase C activation
森口　茂樹[1](Shigeki Moriguchi)，塩田　倫史[1](Norifumi Shioda)，楢橋　敏夫[2](Toshio Narahashi)，
福永　浩司[1](Kohji Fukunaga)
[1]東北大院・薬・薬理，[2]ノースウエスタン大・医

O1P-E15 17:30
バースト細胞の層状ネットワークが示す多様な活動伝播とその計算論的可能性
Computational studies of activity propagations through feed-forward networks of burst neurons
寺前順之介[1](Jun-nosuke Teramae)，加藤　英之[1](Hideyuki Cateau)，Alex Reyes[2]，
深井　朋樹[1](Tomoki Fukai)
[1]理化学研究所・脳科学総合研究センター，[2]Center for Neuroscience・ニューヨーク大学

O1P-E16 17:45
P2X7 受容体を介したアデノシンの細胞内取り込み
Adenosine uptake *via* P2X7 receptors
奥田　浩人[1](Hiroto Okuda)，長澤　一樹[1,2](Kazuki Nagasawa)，Raymond A Swanson[2]，
藤本　貞毅[1](Sadaki Fujimoto)
[1]京都薬大・衛生化学，[2]Dept. Neurol., UCSF, CA, USA

## Oral Session 1P-F　14:00-15:00　Room F

## シナプス 1
## Synapse 1

Chairperson　古川　貴久（Takahisa Furukawa）（財団法人大阪バイオサイエンス研究所）

O1P-F01 14:00
シナプス活性を制御するユビキチンリガーゼ Kspot
Kspot, a novel ubiquitin ligase, regulates synaptic activity
多田　敬典[1](Hirobumi Tada)，岡野ジェイムス洋尚[1,2](Hirotaka J. Okano)，高木　博[3](Hiroshi Takagi)，
松本　雅記[4,5](Masaki Matsumoto)，矢尾　育子[3](Ikuko Yao)，雑賀　徹[4,5](Toru Saiga)，
中山　敬一[4,5](Keiichi I.. Nakayama)，鹿島　晴雄[6](Haruo Kashima)，瀬藤　光利[3,7,8](Mitsutoshi Setou)，
岡野　栄之[1,2](Hideyuki Okano)
[1]慶應大・医・生理学，[2]SORST-JST，[3]九州大・生医研・分子発現制御学分野，[4]三菱化学生命科学研究所，
[5]CREST-JST，[6]慶應大・医・精神神経，[7]PREST-JST，[8]自然科学研究所機構岡崎統合バイオサイエンスセンター

O1P-F02 14:15
GluR6 の SUMO 化によるシナプス伝達の制御
SUMOylation regulates kainate receptor-mediated synaptic transmission
西宗　敦史(Atsushi Nishimune)，Stephane Martin，Jack R. Mellor，Jeremy M. Henley
MRC Centre for Synaptic Plasticity, Dept of Anatomy, University of Bristol, Bristol, UK

O1P-F03 14:30
IQ-ArfGEF/BRAG1 はシナプス後肥厚部において PSD-95 と複合体を形成する Arf6 グアニンヌクレオチド交換因子である
IQ-ArfGEF/BRAG1 is a guanine nucleotide exchange factor for Arf6 that localizes and interacts with PSD-95 at excitatory synapses.
阪上　洋行[1](Hiroyuki Sakagami)，三田　将司[1,2](Masashi Sanda)，深谷　昌弘[3](Masahiro Fukaya)，
福永　浩司[2](Kohji Fukunaga)，渡辺　雅彦[3](Masahiko Watanabe)，近藤　尚武[1](Hisatake Kondo)
[1]東北大院・医・細胞生物，[2]東北大院・薬・薬理，[3]北海道大院・医・解剖発生

O1P-F04 14:45
網膜視細胞のリボンシナプスに局在し Agrin/Perlecan にホモロジーのある新規タンパク質 pikachurin の同定と機能解析
Cloning and functional analysis of Pikachurin: a novel Agrin/Perlecan-related protein localized at the active zone of retinal photoreceptor ribbon synapse
古川　貴久[1](Takahisa Furukawa)，佐藤　茂[1,2](Shigeru Sato)，谷　昭子[1](Akiko Tani)，
宮田健太郎[3](Kentaro Miyata)，小安　俊行[3](Toshiyuki Koyasu)，近藤　峰生[3](Mineo Kondo)，
臼倉　治郎[4](Jiro Usukura)
[1]財団法人大阪バイオサイエンス研究所発生生物学部門，[2]阪大・医・眼科，[3]名大・医・眼科，[4]名大・先端技術共同研究センター

## Oral Session 1P-F　15:00-16:00　Room F

### シナプス 2
### Synapse 2

Chairperson　多田　敬典 (Hirobumi Tada) (慶應義塾大学医学部生理学)

O1P-F05
15:00
**前脳基底核アセチルコリン性ニューロンにおける興奮性シナプス伝達に関与するカルシウムチャネルとD1 型受容体を介するシナプス前抑制の生後発達変化**
**Developmental change in the calcium channel subtypes involved in the excitatory transmission and D1-like receptor-mediated presynaptic inhibition in the rat basal forebrain cholinergic neurons**
籾山　俊彦(Toshihiko Momiyama)
生理研・脳形態解析

O1P-F06
15:15
**マウス背側蝸牛神経核に存在する Fusiform cells における、$GABA_B$ 受容体を介したシナプスナプス前抑制とシナプス後抑制**
**Presynaptic and postsynaptic inhibition by $GABA_B$ receptor in fusiform cells in mice dorsal cochlear nucleus**
入江　智彦(Tomohiko Irie), 大森　治紀(Harunori Ohmori)
京都大院・医・生理学第一

O1P-F07
15:30
**大脳皮質視覚野における抑制性神経回路の発達**
**Development of pyramidal cell inhibition in visual cortex**
吉村由美子[1,2](Yumiko Yoshimura), 任　　鳴[1](Ming Ren), 堀部　尚子[1](Shoko Horibe),
小松由紀夫[1,2](Yukio Komatsu)
[1]名古屋大・環境医学研・視覚神経科学, [2]Crest, 科学技術振興機構

O1P-F08
15:45
**神経筋接合部シナプス後部の分化におけるラミニンの機能**
**A role for laminin in postsynaptic differentiation at the neuromuscular junction**
西宗　裕史[1](Hiroshi Nishimune), Jeff H. Miner[2], Joshua R. Sanes[3]
[1]カンザス大学・医・解剖＋細胞生物, [2]ワシントン大学・医・腎臓科, [3]ハーバード大学・分子細胞生物

## Oral Session 1P-F　16:00-17:00　Room F

### シナプス 3
### Synapse 3

Chairperson　松田　恵子 (Keiko Matsuda) (慶應義塾大学医学部生理学)

O1P-F09
16:00
**紫外及び2光子レーザー uncaging 刺激によるシナプス伝達特性の実験的及び数理的解析**
**Analysis of synaptic transmissions by UV and IR laser photolysis**
小島比呂志[1](Hiroshi Kojima), 丸尾　知彦[2](Tomohiko Maruo), 勝又　詩織[3](Shiori Katsumata),
東條　　諭[1](Satoshi Tojoh), 中里　陽子[1](Youko Nakazato), 塚田　　稔[1](Minoru Tsukada),
岡部　繁男[2](Shigeo Okabe), 酒井　　宏[3](Ko Sakai)
[1]玉川大学・工・知能情報システム, [2]東京医科歯科大学・細胞生物学, [3]筑波大学大学院・システム情報工学研究科

O1P-F10
16:15
**中枢神経系のグルタミン酸作動性シナプスにおいてシナプス前部 NMDA 受容体は電位依存性カルシウムチャネルを抑制し、シナプス伝達を制御する**
**Presynaptic NMDA receptors regulates synaptic transmission by inhibiting voltage-gated calcium channel at a central glutamatergic synapse**
鷹合　秀輝[1,2](Hideki Takago), 大島　知子[2](Tomoko Oshima), 喜多村　健[1](Ken Kitamura),
高橋　智幸[2](Tomoyuki Takahashi)
[1]東京医科歯科大学大学院・医歯学総合・耳鼻咽喉科学, [2]東京大院・医・神経生理

O1P-F11 16:30
カルモジュリン依存的前シナプス性カルシウム電流不活性化の生後発達変化
Developmental changes in calmodulin-dependent presynaptic calcium current inactivation at a calyx-type synapse
山下　貴之(Takayuki Yamashita)，中村　健(Takeshi Nakamura)，齋藤　直人(Naoto Saitoh)，
高橋　智幸(Tomoyuki Takahashi)
東京大院・医・神経生理

O1P-F12 16:45
細胞内構造の異なる２つのアルカドリン変異体のシナプス再構成における役割
The role of Arcadlin isoforms in synaptic remodeling
安田　新[1](Shin Yasuda)，杉浦　弘子[1](Hiroko Sugiura)，前野-引地愉香[1](Yuka Maeno-Hikichi)，
竹宮　孝子[1](Takako Takemiya)，田中　秀和[1,2](Hidekazu Tanaka)，山形　要人[1](Kanato Yamagata)
[1]東京都神経研・神経薬理，[2]大阪大院・医・薬理

**Oral Session 1P-F** 17:00-18:00 Room F

## シナプス４ Synapse 4

Chairperson　山形　要人 (Kanato Yamagata)（東京都神経科学総合研究所）

O1P-F13 17:00
CaMKIIβとの相互作用を介したArcのスパイン局在制御機構
Regulation of Arc localization at synapses via interaction with CaMKIIβ
奥野　浩行[1](Hiroyuki Okuno)，成瀬　紘也[1](Hiroya Naruse)，川島　尚之[1](Takashi Kawashima)，
藤井　哉[1](Hajime Fujii)，Shoaib Chowdhury[2]，Paul Worley[2]，尾藤　晴彦[1](Haruhiko Bito)
[1]東京大院・医・神経生化学，[2]ジョンズ・ホプキンス大・医・神経科学

O1P-F14 17:15
６量体Cbln1リガンドの小脳PSD分画への結合様式の解明。
Characterization of Cbln1 binding to the PSD fraction of the cerebellum: implication for specific binding proteins for Cbln1 in Purkinje cells
松田　恵子[1](Keiko Matsuda)，近藤　哲朗[2](Tetsuro Kondo)，飯島　崇利[1](Takatoshi Iijima)，
石田　綾[1](Aya Ishida)，柚崎　通介[1](Michisuke Yuzaki)
[1]慶應義塾大学・医・生理学，[2]産業技術研究所・脳神経情報

O1P-F15 17:30
LRR膜貫通分子Lrfn2/SALM1は興奮性シナプスの形成や機能発現に重要である
Lrfn2/SALM1, a synaptic leucine-rich repeat-containing transmembrane protein regulates synapse formation and function
守村　直子[1](Naoko Morimura)，片山　圭一[1](Kei-ichi Katayama)，山口　和彦[2](Kazuhiko Yamaguchi)，
太田　摩耶[1](Maya Ota)，神谷　明子[1](Akiko Kamiya)，有賀　純[1](Jun Aruga)
[1]理化学研究所・脳センター・比較神経発生，[2]理化学研究所・脳センター・記憶学習

O1P-F16 17:45
ニワトリにおける両耳間音圧差抽出の神経機構
Neural mechanisms for calculation of the interaural intensity difference in the chicken auditory brainstem
佐藤　達雄(Tatsuo Sato)
京都大院・医・神経生物

**Oral Session 1P-G** 14:00-15:00 Room G

## 学習記憶 1 Learning and Memory 1

Chairperson　倉田　耕治 (Koji Kurata)（琉球大学工学部機械システム工学科）

O1P-G01 14:00
行動により活性化した神経細胞におけるAMPA受容体動態の可視化
Imaging of AMPA receptor dynamics in behaviorally activated neurons
松尾　直毅(Naoki Matsuo)，Mark Mayford
スクリプス研究所

O1P-G02 報酬関連細胞を用いた海馬空間学習モデル
14:15 Hippocampal model for spatial learning by using reward related cells
松本　惇平(Jumpei Matsumoto)，牧野　悌也(Yoshinari Makino)，三浦　治己(Haruki Miura)，
矢野　雅文(Masafumi Yano)
東北大学電気通信研究所

O1P-G03 方向細胞とグリッド細胞の反ヘブ結合による自己組織モデル
14:30 A learning model of head-direction cells and grid cells by VQ layers connected via anti-Hebbian synapses
大城　尚紀(Naoki Oshiro)，倉田　耕治(Koji Kurata)
琉球大学・工・機械システム工学科

O1P-G04 Dentate Gyrus theta phase precession learned by enthorhinal grid cells
14:45
Colin Molter，Yoko Yamaguchi
Lab.for Dynamics of Emergent Intelligence, RIKEN-Brain Science Institute

**Oral Session 1P-G** 15:00-16:00 Room G

## 学習記憶 2
## Learning and Memory 2

Chairperson　櫻井　芳雄（Yoshio Sakurai）（京都大学大学院文学研究科心理学研究室）

O1P-G05 小鳥の脳神経核 HVC における歌要素系列の集団符号化
15:00 Population coding of song element sequence in the songbird brain nucleus HVC
西川　　淳[1](Jun Nishikawa)，岡田　真人[1,2](Masato Okada)，岡ノ谷一夫[1](Kazuo Okanoya)
[1]独立行政法人理化学研究所　脳科学総合研究センター，[2]東京大学大学院　新領域創成科学研究科

O1P-G06 単一神経細胞の樹状突起の有する潜在的計算能力は、高次機能にどのように関わり得るか：短期記憶のネットワークモデルを例とした理論的検討
15:15 Impact of dendritic branches on the function of an entire neuronal network
森田　賢治[1](Kenji Morita)，岡田　真人[2](Masato Okada)，合原　一幸[3,4](Kazuyuki Aihara)
[1]理化学研究所・脳科学総合研究センター，[2]東京大学大学院・複雑理工学専攻，[3]東京大学・生産技術研究所，
[4]ERATO・合原複雑数理モデルプロジェクト

O1P-G07 Dynamical model of working memory system
15:30
Vladimir V. Klinshov，Vladimir I Nekorkin
Nonlinear dynamics department, Institute of Applied Physics, Nizhny Novgorod, Russia

O1P-G08 行動中のラット海馬における錐体細胞の細胞体と樹状突起での空間情報表現
15:45 Spatial information of hippocampal pyramidal cells along the dendrosomatic axis of behaving rats
高橋　　晋[1](Susumu Takahashi)，櫻井　芳雄[1,2](Yoshio Sakurai)
[1]科学技術振興機構・CREST，[2]京大院・文・心理

**Oral Session 1P-G** 16:00-17:00 Room G

## 学習記憶 3
## Learning and Memory 3

Chairperson　小林　　康（Yasushi Kobayashi）（大阪大学大学院生命機能研究科）

O1P-G09 前頭前野は長期よりも短期間維持された文脈性恐怖記憶の消去学習に関与する
16:00 Medial prefrontal cortex is involved in extinction learning for recent but not remote contextual fear
圖子田　康(Ko Zushida)，和田　圭司(Keiji Wada)，関口　正幸(Masayuki Sekiguchi)
国立精神・神経センター神経研究所疾病研究第四部

O1P-G10 輝度コントラスト差による知覚的曖昧性と報酬関連脳活動
16:15 Perceptual ambiguity by luminance contrast and reward predictive activity in the brain
竹村 浩昌[1](Hiromasa Takemura), 奥田 次郎[2](Jiro Okuda), 鮫島 和行[2](Kazuyuki Samejima),
坂上 雅道[2](Masamichi Sakagami)
[1]東京大学大学院総合文化研究科広域科学専攻生命環境科学系, [2]玉川大学学術研究所

O1P-G11 サル脚橋被蓋核ニューロンの一過性応答の解析
16:30 Phasic responses of the pedunculopontine tegmental nucleus in primates
岡田 研一[1](Ken-Ichi Okada), 井上 由香[2](Yuka Inoue), 伊佐 正[2](Tadashi Isa),
小林 康[1,3](Yasushi Kobayashi)
[1]大阪大院・生命機能研究科, [2]生理学研究所・発達生理学研究系・認知行動発達機構研究部門, [3]ATR脳情報研究所

O1P-G12 脚橋被蓋核による報酬予測誤差計算機構
16:45 Different groups of pedunculopontine tegmental nucleus neurons signal the expected and actual task reward
小林 康[1,2](Yasushi Kobayashi), 岡田 研一[1](Ken-ichi Okada)
[1]大阪大院・生命機能研究科, [2]ATR脳情報研究所

## Oral Session 1P-G 17:00-18:00 Room G

## 言語とコミュニケーション Language and Communication

Chairperson 酒井 邦嘉 (Kuniyoshi L. Sakai) (東京大学大学院総合文化研究科相関基礎科学系)

O1P-G13 乳幼児向け発話における親の脳活動と個人差の検討
17:00 Processing of infant-directed speech in parents: An fMRI study
松田 佳尚[1](Yoshitaka Matsuda), 上野 賢一[2](Ken'ichi Ueno), R. Allen Waggoner[3],
Donna Erickson[4], 志村 洋子[5](Yoko Shimura), 田中 啓治[3](Keiji Tanaka), 程 康[2,3](Kang Cheng),
馬塚れい子[1,6](Reiko Mazuka)
[1]理研・脳・言語発達, [2]理研・脳・fMRI支援ユニット, [3]理研・脳・認知機能表現, [4]昭和音大・音楽, [5]埼玉大・教育, [6]デューク大・心理神経科学

O1P-G14 刺激の規則性、親密度、調和性の聴覚および視覚誘発脳磁図反応への影響
17:15 Effects of regularity, familiarity and congruity of stimuli on auditory and visually evoked magnetic fields
原田 暢善[1](Nobuyoshi Harada), 岩木 直[1](Sunao Iwaki), 外池 光雄[1,2](Mitsuo Tonoike)
[1]産業技術総合研究所、関西センター、人間福祉医工学部門, [2]千葉大学工学部メディカルシステム工学科

O1P-G15 脳腫瘍患者における選択的統語処理障害
17:30 Selective impairment of syntactic processing in patients with brain tumor
金野 竜太[1,2,3](Ryuta Kinno), 村垣 善浩[4](Yoshihiro Muragaki), 堀 智勝[4](Tomokatsu Hori),
丸山 隆志[4](Takashi Maruyama), 河村 満[2,3](Mitsuru Kawamura), 酒井 邦嘉[1,2](Kuniyoshi L. Saka)
[1]東京大院・総合文化・相関基礎科学, [2]戦略的創造研究推進事業, [3]昭和大学医学部 神経内科, [4]東京女子医科大学 脳神経外科

O1P-G16 英語正字法と音韻間のフォニクス規則学習時における成績向上の前頭葉脳活動による予測
17:45 Prefrontal activations predict future performance improvement in learning phonics rules of English orthography and phonology
武藤 牧子[1,2](Makiko Muto), 酒井 邦嘉[1,2](Kuniyoshi L Sakai)
[1]東京大学総合文化研究科, [2]戦略的創造研究推進事業, 科学技術振興機構

## Oral Session 1P-H　14:00-14:45　Room H

### 神経細胞死 Neuronal Death

Chairperson　桃井　隆（Takashi Momoi）（国立精神・神経センター神経研究所）

O1P-H01
14:00
**Apaf-1 欠損マウスにおける嗅神経の軸索走行異常**
**Involvement of caspase activity in the pathfinding of olfactory sensory neurons**
大澤志津江[1](Shizue Ohsawa)，濱田　俊[2](Shun Hamada)，吉田　裕樹[3](Hiroki Yoshida)，
八木　健[4](Takeshi Yagi)，三浦　正幸[1](Masayuki Miura)
[1]東京大院・薬・遺伝，[2]福岡女子大・人間環境・栄養健康科学・人体生理学，[3]佐賀大・医・分子生命，[4]阪大院・生命機能

O1P-H02
14:15
**アストロサイト NO 誘発アポトーシスにおける MAP キナーゼ活性化と酸化ストレスの関与**
**Involvement of oxidative stress and MAP kinases in NO-induced apoptosis in cultured astrocytes**
北尾　達哉[1](Tatsuya Kitao)，川崎　俊之[1](Toshiyuki Kawasaki)，池原　亜樹[1](Aki Ikehara)，
吾郷由希夫[1](Yukio Ago)，馬場　明道[2](Akemichi Baba)，松田　敏夫[1,3](Toshio Matsuda)
[1]大阪大院・薬・複合薬物動態，[2]大阪大院・薬・神経薬理，[3]大阪大院・医・子どものこころの分子統御機構研究センター

O1P-H03
14:30
**酸化ストレスによるグリセロアルデヒド３リン酸脱水素酵素のアミロイド様凝集には活性中心システイン残基が重要である**
**Critical role of the active site cysteine in oxidative stress-induced aggregation of GAPDH**
中嶋　秀満(Hidemitsu Nakajima)，天野　渉(Wataru Amano)，東　泰孝(Yasu-Taka Azuma)，
竹内　正吉(Tadayoshi Takeuchi)
大阪府立大学・生命環境・応用薬理

## Oral Session 1P-H　15:00-16:00　Room H

### グリア 1 (J) Glia 1 (J)

This session is held in Japanese

Chairperson　馬場　広子（Hiroko Baba）（東京薬科大学薬学部）

O1P-H04
15:00
**リアルタイムイメージングを用いた培養海馬組織切片におけるミクログリア活性化機構の解析**
**Real time imaging study on injury-induced microglial activation in the hippocampal slice culture**
岡村　敏行[1](Toshiyuki Okamura)，片山　貴博[1](Takahiro Katayama)，松田　知己[2](Tomoki Matsuda)，
永井　健治[2](Takeharu Nagai)，木村　宏[3](Hiroshi Kimura)，内野　茂夫[4](Shigeo Uchino)，
高坂　新一[4](Shinichi Kohsaka)，南　雅文[1](Masabumi Minami)
[1]北大院・薬・薬理学，[2]北大電子研・ナノシステム生理学，[3]京大院医・先端領域融合医学・先端画像解析，[4]国立精神神経センター神経研・代謝

O1P-H05
15:15
**パーキンソン病誘発物質は Astrocyte においてギャップ結合による細胞間伝達を亢進させる**
**Parkinsonism-causing toxins enhance gap junction in astrocytes**
河嵜　麻実[1](Asami Kawasaki)，林　奉権[2](Tomonori Hayashi)，中地　啓[2](Kei Nakachi)，
古武弥一郎[1](Yaichiro Kotake)，太田　茂[1](Shigeru Ohta)
[1]広島大院・医歯薬学総合研究科，[2]放射線影響研究所

O1P-H06
15:30
**神経細胞傷害後のアストロサイトとミクログリアでの異なったケモカイン産生誘導**
**Neuronal injury induces the expression of multiple chemokines in astrocytes and microglia in organotypic cortico-striatal slice cultures**
片山　貴博(Takahiro Katayama)，岡村　敏行(Toshiyuki Okamura)，大日方千紘(Chihiro Obinata)，
上原　孝(Takashi Uehara)，南　雅文(Masabumi Minami)
北海道大院・薬・薬理

O1P-H07 脳内亜鉛によるミクログリアの活性化
15:45 Zinc induces microglial activation
東　洋一郎[1](Youichirou Higashi), Sang Won Suh[2], Raymond A Swanson[2],
長澤　一樹[1](Kazuki Nagasawa), 藤本　貞毅[1](Sadaki Fujimoto)
[1]京都薬大・衛生化学, [2]Dept. Neurol., UCSF, CA, USA

**Oral Session 1P-H** 16:00-16:45 Room H

## グリア 2
## Glia 2

Chairperson　小泉　修一 (Schuichi Koizumi) (山梨大学大学院医学工学総合研究部薬理)

O1P-H08 アストロサイトに発現する TRPV4 が神経細胞との機能連関に関わる可能性
16:00 Implication that TRPV4 is involved in the communication between astrocytes and neurons
柴崎　貢志[1,2,3](Koji Shibasaki), 富永　真琴[1,2,3](Makoto Tominaga)
[1]岡崎統合バイオ・細胞生理, [2]生理学研究所, [3]総合研究大学院大学・生命科学研究科

O1P-H09 血管内皮細胞 $PGE_2$ によるアストロサイトを介した神経細胞障害の制御機構
16:15 The regulatory mechanism of neuronal damage by endothelial PGE2 via astrocytes
竹宮　孝子[1,2](Takako Takemiya), 松村　潔[3](Kiyoshi Matsumura), 杉浦　弘子[2](Hiroko Sugiura),
前原　通代[4](Michiyo Maehara), 安田　新[2](Shin Yasuda), 植松　智[5](Satoshi Uematsu),
審良　静男[5](Shizuo Akira), 山形　要人[2](Kanato Yamagata)
[1]東京女子医大・医・総研研究部, [2]東京都神経研・神経薬理, [3]大阪工業大学・情報科学, [4]東京女子医大・中央検査部, [5]大阪大学・微研・自然免疫

O1P-H10 アストロサイトにおけるニコチンアミドの輸送特性の解明
16:30 Transport characteristics for nicotinamide in astrocytes
鈴木　恵理(Eri Suzuki), 長澤　一樹(Kazuki Nagasawa), 藤本　貞毅(Sadaki Fujimoto)
京都薬大・衛生化学

**Oral Session 1P-H** 17:00-18:00 Room H

## グリア 3
## Glia 3

Chairperson　池中　一裕 (Kazuhiro Ikenaka) (自然科学研究機構生理学研究所)

O1P-H11 FcRγ/Fyn シグナリングと再ミエリン化
17:00 Restoration of FcRγ/Fyn signaling repairs central nervous system demyelination
山本　雅浩[1,2](Masahiro Yamamoto), 清和　千佳[1](Chika Seiwa), 福武　昌人[1,2](Masato Fukutake),
植木　俊之[1,2](Toshiyuki Ueki), 阿相　皓晃[1](Hiroaki Asou)
[1]都老人研・グリア, [2]（株）ツムラ・ツムラ研究所

O1P-H12 髄鞘形成における LIF の新たな役割
17:15 A novel function of leukemia inhibitory factor in myelination
石橋　智子[1,2](Tomoko Ishibashi), Philip R. Lee[1], 馬場　広子[2](Hiroko Baba), R. Douglas Fields[1]
[1]米国立衛生研究所, [2]東京薬科大学・薬・機能形態

O1P-H13 ミクログリアの貪食能を規定する新規分子 UDP
17:30 UDP, a novel mediator of microglial phagocytosis
小泉　修一[1,2](Schuichi Koizumi), 最上由香里[2](Yukari Shigemoto-Mogami), 多田　薫[2](Kaoru Nasu-Tada),
篠崎　陽一[2,3](Yoichi Shinozaki), 大澤　圭子[4](Keiko Ohsawa), 津田　誠[3](Makoto Tsuda),
高坂　新一[4](Shinichi Kohsaka), 井上　和秀[3](Kazuhide Inoue)
[1]山梨大院・医・薬理, [2]国立衛研・薬理, [3]九州大院・薬・薬理, [4]国立精神・神経センター神経研究所・代謝

O1P-H14 17:45
軸索切断に伴う synaptic stripping に先行した迷走神経背側運動ニューロンにおける神経伝達の抑制
Inhibition of synaptic transmission precedes synaptic stripping in vagal motoneurons after axotomy
山田　純[1](Jun Yamada)，林　良憲[1](Yoshinori Hayashi)，井上　和秀[2](Kazuhide Inoue)，
高坂　新一[3](Shinichi Kohsaka)，中西　博[1](Hiroshi Nakanishi)
[1]九州大院・歯・加齢口腔，[2]九州大・院・薬効解析学，[3]国立精神・神経センター

## Oral Session 1P-J　14:00-15:00　Room J

### ポリグルタミン病、ALS、その他
### Polyglutamine Diseases, ALS, Other

Chairperson　平野　牧人 (Makito Hirano)（奈良県立医科大学神経内科）

O1P-J01 14:00
HGF の遺伝子治療はハンチントン病トランスジェニックマウスの行動と神経病理学的変化を改善する
Gene transfer of hepatocyte growth factor (HGF) attenuates behavioral and neuropathological abnormalities in a transgenic mouse model of Huntington's disease
船越　洋[1](Hiroshi Funakoshi)，宮澤　大介[1](Daisuke Miyazawa)，大谷　若菜[1](Wakana Ohya)，
Robert S. Coffin[2]，野々口直助[3](Naosuke Nonoguchi)，宮武　伸一[3](Shin-ichi Miyatake)，
中村　敏一[1](Toshikazu Nakamura)
[1]大阪大院・医・分子再生医学，[2]ロンドン大学，[3]大阪医大

O1P-J02 14:15
High-throughput RNAi screening for modifiers of mutant Huntingtin aggregation in *Drosophila melanogaster*
Joanna Doumanis[1], Koji Wada[1], Adrian Moore[2], Nobuyuki Nukina[1]
[1]Lab for Structural Neuropathology, RIKEN BSI, Wako, Japan, [2]Moore Research Unit, RIKEN BSI

O1P-J03 14:30
低分子量 G タンパク質 Rab5 活性化因子 ALS2 は Rac1 が活性化するマクロピノサイトーシスの過程で新規の Rac1 エフェクターとして働く
The Rab5 activator ALS2/alsin acts as a novel Rac1 effector via Rac1-activated macropinocytosis
國田　竜太(Ryota Kunita)，大友　麻子(Asako Otomo)，水村　光(Hikaru Mizumura)，
鈴木宇都宮恭子(Kyoko Suzuki-Utsunomiya)，秦野　伸二(Shinji Hadano)，池田　穣衛(Joh-E Ikeda)
東海大学・医・基礎医学系

O1P-J04 14:45
Triple A 症候群の原因となる ALADIN 変異は失調症原因蛋白である aprataxin の核内輸送を障害する
Mutant ALADIN responsible for triple A syndrome impairs nuclear import of aprataxin, a DNA repair protein causative for ataxia.
平野　牧人[1](Makito Hirano)，降矢　芳子[1](Yoshiko Furiya)，浅井　宏英[1](Hirohide Asai)，
桐山　敬生[1](Takao Kiriyama)，安井　明[2](Akira Yasui)，上野　聡[1](Satoshi Ueno)
[1]奈良医大　神経内科，[2]東北大　加齢医学研

## Oral Session 1P-J　15:00-16:00　Room J

### 発達障害
### Developmental Disorders

Chairperson　山川　和弘 (Kazuhiro Yamakawa)（理化学研究所脳科学総合研究センター神経遺伝）

O1P-J05 15:00
Slitrk1 および Slitrk2 ノックアウトマウスの表現型解析
Analysis of Slitrk1- and Slitrk2-deficient mice
片山　圭一[1](Kei-ichi Katayama)，山田　一之[2](Kazuyuki Yamada)，井上　貴史[1](Takashi Inoue)，
太田　摩耶[1](Maya Ota)，有賀　純[1](Jun Aruga)
[1]理研・BSI・比較神経発生研究チーム，[2]理研・BSI・動物実験支援ユニット

O1P-J06 15:15
ダウン症候群モデルマウスの脳における変動タンパク質の同定
Altered expression of proteins in the brain of mouse model for Down syndrome
石原　慶一(Keiichi Ishihara)，天野　賢治(Kenji Amano)，柴崎　典子(Noriko Shibazaki)，
山川　和弘(Kazuhiro Yamakawa)
理化学研究所・脳研・神経遺伝

O1P-J07
15:30
**Wig ラット脳のプロテオーム解析：注意欠陥多動性障害（ADHD）研究のモデル**
**Proteome analysis of Wig rat brain: a model for attention-deficit hyperactivity disorder (ADHD) research**
増尾　好則[1](Yoshinori Masuo)，平野　美里[1](Misato Hirano)，柴藤　淳子[1](Junko Shibato)，
岩橋　　均[2](Hitoshi Iwahashi)，澤　　洋文[3](Hirofumi Sawa)，長嶋　和郎[4](Kazuo Nagashima)，
Randeep Rakwal[1]
[1]産総研・ヒューマンストレスシグナル研究センター，[2]産総研・ヒューマンストレスシグナル研究センター，[3]北海道大・人獣共通感染症リサーチセンター，[4]北海道大・医・分子細胞病理学

O1P-J08
15:45
**発達経過に著しい差異を認めたレット症候群双子例のエピジェネティクス解析**
**Genetic and epigenetic analyses in monozygotic twins with Rett syndrome with extreme differences in early development**
Chunshu Yang[1]，遠藤　和志[1](Kazushi Endoh)，五月女雅樹[1](Masaki Soutome)，
安達　　登[2](Noboru Adachi)，佐々木征行[3](Masayuki Sasaki)，久保田健夫[1](Takeo Kubota)
[1]山梨大院・医工総合・環境遺伝医学，[2]山梨大院・医工総合・環境遺伝医学，[3]国立精神・神経センター武蔵病院・小児神経科

**Oral Session 1P-J** 16:00-17:30 Room J

## モデル動物 (J)
## Animal Models (J)

This session is held in Japanese

Chairperson　大石　勝隆（Katsutaka Oishi）（産業技術総合研究所生物機能生物時計）

O1P-J09
16:00
**zone1 及び classII 除去マウスにおける糸球地図の形成と匂い認識**
**Glomerular map formation and odor perception in the zone1- and class II-depleted mutant mice**
小早川令子[1](Reiko Kobayakawa)，小早川　高[1](Ko Kobayakawa)，森　　憲作[2](Kensaku Mori)，
坂野　　仁[1](Hitoshi Sakano)
[1]東京大学・院理・生化，[2]東京大学・院医・機能生物学専攻・細胞分子生理学

O1P-J10
16:15
**注意欠陥多動性障害様の行動異常を示す ENU 誘発突然変異マウスは NR1 の細胞内 C0 ドメインにミスセンス変異を持つ**
**An ENU-induced mouse mutant with ADHD-related behavioral abnormality carries a missense mutation in the intracellular C0 domain of NR1**
和田由美子[1](Yumiko Wada)，古瀬　民生[1](Tamio Furuse)，桝屋　啓志[1](Hiroshi Masuya)，
三浦　郁生[1](Ikuo Miura)，串田　知子[1](Tomoko Kushida)，渋川　陽子[1](Yoko Shibukawa)，
山田　郁子[1](Ikuko Yamada)，瀬筒　秀樹[1](Hideki Sezutsu)，金田　秀貴[1](Hideki Kaneda)，
小林喜美男[1](Kimio Kobayashi)，妹尾　直樹[2](Naoki Senoo)，神田　知之[2](Tomoyuki Kanda)，
廣瀬　　了[2](Ryo Hirose)，土岐眞一郎[2](Shinichiro Toki)，中西　康介[2](Kousuke Nakanishi)，
権藤　洋一[1](Yoichi Gondo)，野田　哲生[1](Tetsuo Noda)，湯浅　茂樹[3](Shigeki Yuasa)，
若菜　茂晴[1](Shigeharu Wakana)，城石　俊彦[1](Toshihiko Shiroishi)
[1]理研・GSC・ゲノム機能情報，[2]協和発酵工業株式会社，[3]国立精神神経センター神経研究所

O1P-J11
16:30
**海馬歯状回特異的な NMDA 受容体変異マウスの場所細胞特性**
**Impaired pattern separation of hippocampal place representation in the dentate gyrus-specific NMDA receptor mutant mice**
林　勇一郎[1](Yuichiro Hayashi)，鍋島　曜子[2](Yoko Nabeshima)，鍋島　陽一[2](Yo-ichi Nabeshima)，
宮川　　剛[1](Tsuyoshi Miyakawa)，船曳　和雄[1](Kazuo Funabiki)
[1]京都大院・医・先端領域，[2]京都大院・医・腫瘍生物

O1P-J12
16:45
**CADPS2/CAPS2 遺伝子と自閉症との関連－ノックアウトマウスの解析と自閉症患者におけるスプライシング異常**
**Autistic-like phenotypes in CADPS2/CAPS2 knockout mice and aberrant CADPS2 splicing in autistic patients**
定方　哲史[1](Tetsushi Sadakata)，鷲田　美和[1](Miwa Washida)，岩山　佳美[2](Yoshimi Iwayama)，
大倉　勇史[3](Takeshi Ohkura)，仙波りつ子[1](Ritsuko Katoh-Semba)，田中　三佳[4](Mika Tanaka)，

森　則夫[5](Norio Mori)，市川　宏伸[3](Hironobu Ichikawa)，糸原　重美[6](Shigeyoshi Itohara)，
吉川　武男[2](Takeo Yoshikawa)，古市　貞一[1](Teiichi Furuichi)
[1]理研・脳センター・分子神経形成，[2]理研・脳センター・分子精神，[3]東京都立梅ヶ丘病院，[4]理研・脳センター・リサーチリソースセンター，[5]浜松医科大学精神科，[6]理研・脳センター・行動遺伝

O1P-J13
17:00
**高温ストレス耐性ラットの血中のリン脂質およびグルコース値**
**Serum phospholipids and glucose level in rats with tolerance to heat stress**
古山富士弥[1](Fujiya Furuyama)，池本　敦[2](Atsushi Ikemoto)，宮澤　大介[3](Daisuke Miyazawa)，
山本　晶子[1](Akiko Yamamoto)，西野　仁雄[1](Hitoo Nishino)
[1]名古屋市立大院・医・脳神経生理，[2]秋田大学教育文化学部生活者科学講座，[3]金城学院大学薬学部

O1P-J14
17:15
**PPARαリガンドによる概日時計の制御とリズム障害治療薬としての可能性**
**PPARα is a potential therapeutic drug target of circadian rhythm sleep disorders**
大石　勝隆[1](Katsutaka Oishi)，白井　秀徳[1,2](Hidenori Shirai)，工藤　崇[3](Takashi Kudo)，
柴田　重信[3](Shigenobu Shibata)，石田直理雄[1,2](Norio Ishida)
[1]産総研・生物機能・生物時計，[2]筑波大・生命環境科学，[3]早稲田大・理工・薬理

## Oral Session 1P-K　15:00-16:00　Room K

## 情報伝達 1
## Signal Transduction 1

Chairperson　山内　淳司 (Junji Yamauchi) (国立成育医療センター研究所薬剤治療)

O1P-K01
15:00
**神経栄養因子-3によるシュワン細胞の遊走はRas-Tiam1-Rac1経路を必要とする**
**Ras activation of Rac1 exchange factor Tiam1 mediates neurotrophin-3-induced Schwann cell migration**
山内　淳司[1](Junji Yamauchi)，宮本　幸[1](Yuki Miyamoto)，三部　篤[1](Atsushi Sanbe)，Jonah R. Chan[2]，
Eric M. Shooter[2]，田上　昭人[1](Akito Tanoue)
[1]国立成育医療センター研究所・薬剤治療，[2]スタンフォード大学・医・神経生物

O1P-K02
15:15
**BDNFによるcorticalニューロンの突起伸張はTiam1のチロシンリン酸化を必要とする**
**Tyrosine-phosphorylation of Tiam1 mediates BDNF-induced neurite outgrowth in cortical neurons**
宮本　幸[1](Yuki Miyamoto)，山内　淳司[1](Junji Yamauchi)，三部　篤[1](Atsushi Sanbe)，
William C. Mobley[2]，田上　昭人[1](Akito Tanoue)
[1]国立成育医療センター研究所・薬剤治療，[2]スタンフォード大学・医・神経学

O1P-K03
15:30
**細胞間シグナルシステムCD47-SHPS-1系による神経突起形成の制御**
**Regulation by CD47-SHPS-1 system of neurite development**
村田　考啓[1,2](Takaaki Murata)，大西　浩史[1](Hiroshi Ohnishi)，草苅　伸也[1](Shinya Kusakari)，
村田　陽二[1](Yoji Murata)，岡澤　秀樹[1](Hideki Okazawa)，古屋　信彦[2](Nobuhiko Furuya)，
的崎　尚[1](Takashi Matozaki)
[1]群馬大・生体調節研究所・バイオシグナル分野，[2]群馬大院・医・聴平衡覚外科学

O1P-K04
15:45
**PACAPによるPC12細胞の神経突起伸展におけるSTAT3の活性化**
**Activation of STAT3 by pituitary adenylate cyclase-activating polypeptide(PACAP)during PACAP-promoted neuritogenesis of PC12 cells**
石堂　正美(Masami Ishido)
国立環境研・環境リスク研究プログラム

**Oral Session 1P-K** 16:00-17:00 Room K

## 情報伝達 2
## Signal Transduction 2

Chairperson 鈴木 龍雄 (Tatsuo Suzuki) (信州大学大学院医学研究科神経可塑性学)

O1P-K05
16:00
**シナプス脱ユビキチン化酵素よる p65/RelA のスタビリティーの制御**
**Synaptic ubiquitin-specific protease (synUSP) is transported to nucleus and regulates p65/RelA stability**
田 慶宝(Qing-Bao Tian), 鈴木 龍雄(Tatsuo Suzuki)
信州大院・医・神経可塑

O1P-K06
16:15
**Involvement of ERK cascades in the anterior cingulate cortex in pain-related negative emotion in rat**
Hong Cao
Institute of Neurobiology, Fudan University, Shanghai, China

O1P-K07
16:30
**Fyn チロシンキナーゼ活性化を介する抗精神病薬作用機序の解析**
**Fyn protein tyrosine kinase as a target for antipsychotic drugs**
服部功太郎[1](Kotaro Hattori), 内野 茂夫[2](Shigeo Uchino), 伊早坂智子[1,3](Tomoko Isosaka),
高坂 新一[2](Shinichi Kohsaka), 八木 健[3](Takeshi Yagi), 湯浅 茂樹[1](Shigeki Yuasa)
[1]国立精神神経センター・神経研・微細構造, [2]国立精神神経センター・神経研・代謝, [3]阪大・生命機能・心生物学

O1P-K08
16:45
**Transcriptomics of unilateral irradiated and contralateral rat striata after gamma knife surgery**
Randeep Rakwal[1], Junko Shibato[1], Misato Hirano[1], Nobuo Kouyama[2], Yoko Katayama[2], Motohiro Hayashi[3], Hitoshi Iwahashi[4], Yoshinori Masuo[1]
[1]Human Stress Signal Research Center (HSS), AIST, Tsukuba, Japan, [2]Department of Physiology, School of Medicine, Tokyo Women`s Medical University, Tokyo, Japan, [3]Department of Neurosurgery, School of Medicine, Tokyo Women`s Medical University, Tokyo, Japan, [4]HSS, AIST, Ikeda, Japan

**Oral Session 1P-K** 17:00-17:45 Room K

## 虚血
## Ischemia

Chairperson 加藤 天美 (Amami Kato) (近畿大学脳神経外科)

O1P-K09
17:00
**海馬興奮毒性における二相性プロスタノイド産生 (脂質の網羅的質量分析)**
**Dual phase prostanoid production during excitotoxic hippocampal injury: A comprehensive lipidomics analysis**
吉川 圭介[1](Keisuke Yoshikawa), 北 芳博[2](Yoshihiro Kita), 島田 厚良[1](Atsuyoshi Shimada),
細川 昌則[1](Masanori Hosokawa), 清水 孝雄[2](Takao Shimizu)
[1]愛知県心身障害者コロニー 発達障害研究所 病理学部, [2]東京大学医学系研究科分子細胞生物学

O1P-K10
17:15
**虚血再灌流障害におけるプロスタグランジン $E_2$ EP3 受容体の関与**
**Involvement of prostaglandin $E_2$ EP3 receptors in stroke-reperfusion injury**
丹治 勇人[1](Hayato Tanji), 松尾 由理[1](Yuri Ikeda-Matsuo), 太田 梓[1](Azusa Ota),
植松 智[2](Satoshi Uematsu), 審良 静男[2](Shizuo Akira), 佐々木泰治[1](Yasuharu Sasaki)
[1]北里大・薬・薬理, [2]大阪大・微生物病研・自然免疫

O1P-K11
17:30
**脳主幹動脈閉塞性疾患患者における運動関連磁界の変調**
**Subclinical alteration of motor related magnetic field in patients with cerebral vascular occlusive disease.**
押野 悟(Satoru Oshino), 加藤 天美(Amami Kato), 平田 雅之(Masayuki Hirata),
齋藤 洋一(Youichi Saitoh), 貴島 晴彦(Haruhiko Kishima), 藤中 俊之(Toshiyuki Fujinaka),
吉峰 俊樹(Toshiki Yoshimine)
大阪大院・医・脳神経外科

## Oral Session 2P-A 16:00-16:45 Room A

### 回路網形成 5
### Network Formation 5

Chairperson 村上富士夫 (Fujio Murakami) (大阪大学大学院生命機能研究科)

O2P-A01
16:00
**FGF 8によって誘導されるSemaphorin3Fは中脳ドーパミン作動性ニューロン軸索の伸長方向を規定する**
**FGF8-induced semaphorin 3F regulates growth polarity of midbrain dopaminergic axons**
山内 健太[1](Kenta Yamauchi), 水嶋 茂樹[1](Shigeki Mizushima), 玉田 篤史[2](Atsushi Tamada),
山本 亘彦[1](Nobuhiko Yamamoto), 高島 成二[3](Seiji Takashima), 村上富士夫[1,2](Fujio Murakami)
[1]大阪大院・生命機能, [2]基生研・行動制御, [3]大阪大・保健センター

O2P-A02
16:15
**皮質脊髄投射のU字型発達**
**U-shaped development of corticospinal innervation**
上山 勉(Tsutomu Kamiyama), 前田 仁士(Hitoshi Maeda), 桜井 正樹(Masaki Sakurai)
帝京大・医・生理

O2P-A03
16:30
**マウス皮質脊髄路におけるGluR ε 2(2B)依存的シナプス除去**
**GluR ε 2(2B) dependent synapse elimination of corticospinal tract in mice**
大野 孝恵[1](Takae Ohno), 吉岡 昇[1](Noboru Yoshioka), 前田 仁士[1](Hitoshi Maeda),
三品 晶美[2](Masayoshi Mishina), 桜井 正樹[1](Masaki Sakurai)
[1]帝京大学・医・生理, [2]東京大院・薬理・分子神経生物

## Oral Session 2P-A 17:00-18:00 Room A

### 回路網形成 6
### Network Formation 6

Chairperson 小川 正晴 (Masaharu Ogawa) (理化学研究所脳科学総合研究センター細胞培養技術開発)

O2P-A04
17:00
**Reelinシグナルにおける転写因子としてのDisabled-1の役割**
**A role of Disable-1 as a transcription factor in Reelin signaling**
守村 敏史[1](Toshifumi Morimura), 久恒 智博[2,3](Chihiro Hisatsune), 服部 光治[4](Mitsuharu Hattori),
御子柴克彦[2,3,5](Katsuhiko Mikoshiba), 小川 正晴[1](Masaharu Ogawa)
[1]理研・脳センター・細胞培養, [2]東大・医科研・脳神経発生分化, [3]理研・脳センター・発生神経, [4]名古屋市立大・薬・病態生化, [5]科学技術振興機構・国際共同研究・カルシウム振動プロジェクト

O2P-A05
17:15
**膜タンパク質DNERの樹状突起局在はトランスサイトーシスに依存する**
**The somatodendritic targeting of DNER requires transcytosis**
福田 徹子(Tetsuko Fukuda), 横山 斉輔(Seisuke Yokoyama), 見学美根子(Mineko Kengaku)
理研・BSI・神経細胞極性

O2P-A06
17:30
**小脳顆粒細胞の移動における微小管骨格の動態**
**Microtubule dynamics regulating migration of cerebellar granule cells**
梅嶋 宏樹[1](Hiroki Umeshima), 平野 丈夫[2](Tomoo Hirano), 見学美根子[1](Mineko Kengaku)
[1]理研・BSI・神経細胞極性, [2]京都大院・理・生物物理

O2P-A07
17:45
**マウス脳内におけるMahya遺伝子の機能解析**
**Functional analysis of mouse Mahya genes**
市川 直哉(Naoya Ichikawa), 門脇 辰彦(Tatsuhiko Kadowaki)
名古屋大院・生命農・生命技術科学

**Oral Session 2P-B** 14:00-15:00 Room B

## シナプス伝達 (J)
## Synaptic Transmission (J)

This session is held in Japanese

Chairperson 山口（白石）陽子 (Yoko Shiraisi-Yamaguchi)（日本学術振興会, 長崎大学医学部）

O2P-B01
14:00
**リン酸化チロシンアダプター分子 Shc を介したスパイン形成制御に関わるシグナル伝達経路**
**The signaling pathways involved in dendritic spine formation via Shc, phospho-tyrosine adaptor molecule**
山口（白石）陽子[1,2](Yoko Shiraisi-Yamaguchi), 森 望[2](Nozomu Mori)
[1]日本学術振興会, [2]長崎大学大学院・医歯薬学総合研究科

O2P-B02
14:15
**内因性カンナビノイドシグナル制御におけるモノアシルグリセロールリパーゼの役割**
**Roles of monoacylglycerol lipase in the regulation of endocannabinoid signaling**
橋本谷祐輝[1](Yuki Hashimotodani), 少作 隆子[2](Takako Ohno-Shosaku), 狩野 方伸[3](Masanobu Kano)
[1]大阪大院・医・神経生理, [2]金沢大院・医・機能障害学, [3]大阪大院・医・細胞神経科学

O2P-B03
14:30
**発達期小脳の神経回路形成過程における登上線維シナプス除去への GABA 作動性シナプス伝達の関与**
**Strength of GABAergic transmission influences climbing fiber synapse elimination during cerebellar development**
中山 寿子[1](Hisako Nakayama), 橋本 浩一[1,2](Kouichi Hashimoto), 宮崎 太輔[3](Taisuke Miyazaki), 柳川右千夫[4](Yuchio Yanagawa), 小幡 邦彦[5](Kunihiko Obata), 渡辺 雅彦[3](Masahiko Watanabe), 狩野 方伸[1](Masanobu Kano)
[1]大阪大学院・医・細胞神経科学, [2]CREST-JST, [3]北海道大学院・医・第二解剖, [4]群馬大学院・医・遺伝発達行動学, [5]理研・脳科学総合研究センター・神経回路メカニズム研究グループ・小幡ユニット

O2P-B04
14:45
**蝸牛除去マウス脳幹ニューロンの GAD2 発現の増強**
**Increase in GAD2 expression in the periolivary region of the mouse following cochlear ablation**
宝谷 剛志(Takeshi Houtani), 濱田 聡子(Satoko Hamada), Stefan Trifonov, 加瀬 政彦(Masahiko Kase), 杉本 哲夫(Tetsuo Sugimoto)
関西医科大学・医・脳構築学

**Oral Session 2P-B** 15:00-16:00 Room B

## シナプス可塑性 1
## Synaptic Plasticity 1

Chairperson 平野 丈夫 (Tomoo Hirano)（京都大学理学研究科）

O2P-B05
15:00
**小脳長期抑圧を制御するデルタ2型グルタミン酸受容体はイオンチャネルとして機能していない**
**The δ2 glutamate receptor does not function as an ion channel to regulate cerebellar long-term depression**
掛川 渉(Wataru Kakegawa), 幸田 和久(Kazuhisa Kohda), 柚崎 通介(Michisuke Yuzaki)
慶應義塾大・医・生理

O2P-B06
15:15
**Ultrastructural analysis of Purkinje cell spines reveals a highly asymmetrical organization of the PSD and ER, having profound influence on molecular signal processing**
Thomas Launey[1], Tsutomu Hashikawa[2], Rieko Sakai[1], Tsuyako Hayashida[2], Yumiko Motoyama[1], Mariko Dacher[1], Masao Ito[1]
[1]Lab. Memory & Learning, RIKEN BSI, Wako-Shi, Japan, [2]Lab. for Neural Architecture, ibid.

O2P-B07
15:30
**小脳プルキンエ細胞 AMPA 受容体活動依存性トラフィッキングにおけるアクチン制御の関与**
**Regulation of actin cytoskeleton is involved in activity-dependent traffickings of AMPA-receptors in cerebellar Purkinje cell**
山口 和彦[1](Kazuhiko Yamaguchi), 立川 哲也[2](Tetsuya Tatsukawa), 戸高 宏[2](Hiroshi Todaka), Dharmaraj Elakkat[1], 永雄 総一[2](Soichi Nagao), 伊藤 正男[1](Masao Ito)
[1]理研・脳センター・記憶学習, [2]理研・脳センター・運動学習制御

O2P-B08 小脳抑制性シナプスにおける長期増強発現の分子メカニズム
15:45 Molecular mechanism of inhibitory synaptic plasticity in the cerebellum
川口 真也[1,2](Shin-ya Kawaguchi), 平野 丈夫[1,2](Tomoo Hirano)
[1]京都大院・理・生物物理, [2]戦略的創造研究推進事業 科学技術振興機構

**Oral Session 2P-B** 16:00-17:00 Room B

## シナプス可塑性 2
## Synaptic Plasticity 2

Chairperson 関口 正幸 (Masayuki Sekiguchi) (国立精神・神経センター神経研)

O2P-B09 LTP と長期可塑性現象 RISE のさらなる相違点
16:00 Further evidence for the distinction between LTP and RISE, a long-lasting synaptic plasticity accompanied by synaptogenesis.
冨永ー吉野恵子[1](Keiko Tominaga-Yoshino), 浦久保知佳[1](Tomoyoshi Urakubo),
岡田 誠剛[2](Masayoshi Okada), 松田 博子[2](Hiroko Matsuda), 小倉 明彦[1](Akihiko Ogura)
[1]大阪大院・生命機能, [2]関西医大・生理

O2P-B10 海馬 CA3 領域における TEA 誘導性シナプス可塑性
16:15 The characteristics of TEA-induced plasticity at hippocampal CA3 synapses
鈴木江津子(Etsuko Suzuki), 岡田 隆(Takashi Okada)
専修大院・文・心理

O2P-B11 扁桃体外側基底核におけるドパミン $D_2$ 受容体と 1 型ニューロテンシン受容体によるシナプス長期増強制御機構
16:30 The LTP regulation system by $D_2$ receptor and neurotensin receptor type-1 in the basolateral amygdala
天野 大樹[1,2](Taiju Amano), 和田恵津子[1,2](Etsuko Wada), 野田 百美[3](Mami Noda),
和田 圭司[1,2](Keiji Wada), 関口 正幸[1,2](Masayuki Sekiguchi)
[1]国立精神神経セ・四部, [2]CREST, [3]九大・院・薬・病態生理

O2P-B12 ラット大脳皮質の長期抑圧に対する Homer1a/Vesl-1S の影響は層によって異なる
16:45 Laminar variation of Homer1a/Vesl-1S effects on long-term depression in the rat visual cortex
植田 禎史[1,2](Yoshifumi Ueta), 山本 亮[1](Ryo Yamamoto), 杉浦 重樹[3](Shigeki Sugiura),
井ノ口 馨[4](Kaoru Inokuchi), 加藤 伸郎[1](Nobuo Kato)
[1]金沢医科大学・生理機能制御学, [2]京都大院・医・認知行動脳科学, [3]奈良医大, [4]三菱化学生命科学研究所

**Oral Session 2P-B** 17:00-18:00 Room B

## シナプス可塑性 3
## Synaptic Plasticity 3

Chairperson 酒井 裕 (Yutaka Sakai) (玉川大学脳科学研究所)

O2P-B13 スパイクタイミング依存可塑性が非同期性の細胞間に働いて生み出される局所的同期発火
17:00 Local synchrony formed by spike-timing-dependent plasticity working on asynchrony-favoring neurons
加藤 英之(Hideyuki Cateau), 深井 朋樹(Tomoki Fukai)
理研BSI

O2P-B14 2つの同期検出器をもつモデルは様々な条件でのシナプス可塑性を再現する
17:15 Two coincidence detectors model can reproduce synaptic plasticity induced by various protocols
倉重 宏樹[1](Hiroki Kurashige), 酒井 裕[2](Yutaka Sakai)
[1]玉川大院・工, [2]玉川大・工

O2P-B15 Actin-based metaplasticity in cocaine addiction
17:30
Shigenobu Toda, Hao-wei Shen, Ashley Bouknight, Peter W Kalivas
Dept. Neurosciences, Medical Univ. South Carlina, Charleston, SC, USA

O2P-B16 皮質線条体シナプス可塑性の分子機構の動力学モデル
17:45 A kinetic model for molecular mechanisms of cortico-striatal synaptic plasticity
中野 高志[1,2](Takashi Nakano), 土居 智和[3](Tomokazu Doi), 吉本潤一郎[1,2](Junichiro Yoshimoto),
銅谷 賢治[1,2](Kenji Doya)
[1]奈良先端科学技術大学院大学・情報, [2]沖縄大学院大学先行研究, [3]大阪バイオサイエンス研究所

**Oral Session 2P-C** 14:00-15:00 Room C

## 視覚 4
## Visual System 4

Chairperson 樫森与志喜 (Yoshiki Kashimori) (電気通信大学量子・物質工学科)

O2P-C01 視覚カテゴリー化タスクに必要な物体の特徴を抽出するための神経メカニズム
14:00 Neural mechanism for extracting object features critical for visual categorization task
曽我 充哉[1](Mitsuya Soga), 藤田 一寿[1](Kazuhisa Fujita), 樫森与志喜[1,2](Yoshiki Kashimori)
[1]電気通信大院・情報システム学研究科・情報ネットワーク学専攻, [2]電気通信大学量子・物質工学科

O2P-C02 機能的モデルによる初期視覚野細胞特性の新解析法
14:15 Functional model characterization for neurons in the early visual cortex
佐々木耕太[1](Kota Sasaki), 大澤 五住[1,2](Izumi Ohzawa)
[1]大阪大院・基礎工・機能創成, [2]大阪大院・生命機能

O2P-C03 Effective resolution of visual filters applied to natural images
14:30
Michel J. Vidal-Naquet, Manabu Tanifuji
Riken Brain Science Institute, Wako-Shi, Saitama, Japan

O2P-C04 ヒトレチノトピック視覚野における意識的および無意識的視覚処理の相対的重み
14:45 Relative weights on conscious and non-conscious visual processing in human retinotopic areas
山城 博幸[1,2](Hiroyuki Yamashiro), 山本 洋紀[1](Hiroki Yamamoto), 齋木 潤[1](Jun Saiki),
眞野 博彰[3](Hiroaki Mano), 梅田 雅宏[3](Masahiro Umeda), 田中 忠蔵[3,4](Chuzo Tanaka)
[1]京都大学大学院人間・環境学研究科, [2]日本学術振興会, [3]明治鍼灸大学脳神経外科, [4]明治鍼灸大学医療情報学研究室

**Oral Session 2P-C** 15:00-16:00 Room C

## 視覚 5
## Visual System 5

Chairperson 伊藤 浩之 (Hiroyuki Ito) (京都産業大学工学部)

O2P-C05 Effectiveness of dithering to destroy spike coincidences
15:00
Sonja Gruen[1,2], Antonio Pazienti[1], Markus Diesmann[1]
[1]Gruen Research Unit, RIKEN Brain Science Inst, Wako-Shi, Japan, [2]BCCN Berlin, Germany

O2P-C06 Contribution of the GABAergic pathway to the correlated activities of chicken retinal ganglion cells
15:15
Xue Liu, Yi Zhou, Pei-Ji Liang
Department of Biomedical Engineering, Shanghai Jiao Tong University, Shanghai, China

O2P-C07 15:30
Precisely timed inhibition can modulate the internal structure of bursts in salamander retina
Xin Jin[1], Toshihiko Hosoya[2]
[1]Department of Biomedical Engieering, Shanghai Jiao Tong University, Shanghai, China, [2]RIKEN Brain Science Institute, Wako-shi, Saitama, Japan

O2P-C08 15:45
外側膝状体における同期振動発火の非定常ダイナミクス - ブートストラップ法による有意性検定 -
Non-stationary dynamics of synchronous oscillatory spike events in the LGN - Bootstrap significance test -
伊藤 浩之(Hiroyuki Ito)
京都産業大学・工・情報通信工

**Oral Session 2P-C** 16:00-17:00 Room C

## 視覚 6
## Visual System 6

Chairperson 神谷 之康 (Yukiyasu Kamitani) (ATR脳情報研究所)

O2P-C09 16:00
ネコ一次視覚野ニューロンの受容野周囲刺激による反応抑制は受容野刺激応答強度の時間変化に比例して変化する
Strength of surround suppression is proportional to the time course of response magnitude in cat V1
七五三木聡(Satoshi Shimegi)
大阪大院・医・認知行動科学

O2P-C10 16:15
大きさ恒常性の神経基盤と成り得る、V4 野神経細胞の視差・大きさ選択性
Disparity- and size-selective neurons in macaque area V4 as a neural basis for size constancy
田中 慎吾(Shingo Tanaka), 藤田 一郎(Ichiro Fujita)
大阪大院・生命機能

O2P-C11 16:30
ヒトの視覚皮質における階層的群化表現に基づく競合バイアス化
Biased competition based on hierarchical grouping representations in the human visual cortex
河西 哲子(Tetsuko Kasai), 守谷 大樹(Hiroki Moriya)
北海道大学 大学院教育学研究科

O2P-C12 16:45
多重解像度局所画像複合器による fMRI 信号からの視覚像再構成
Reconstruction of visual images from fMRI signals by combination of multi-resolution local image decoders
宮脇 陽一[1,2](Yoichi Miyawaki), 内田 肇[2,3](Hajime Uchida), 山下 宙人[2](Okito Yamashita),
佐藤 雅昭[2](Masa-aki Sato), 田邊 宏樹[4](Hiroki C. Tanabe), 定藤 規弘[4](Norihiro Sadato),
神谷 之康[4](Yukiyasu Kamitani)
[1]（独）情報通信研究機構, [2]（株）国際電気通信基礎技術研究所・脳情報研究所, [3]奈良先端科学技術大学院大学, [4]生理学研究所

**Oral Session 2P-C** 17:00-18:00 Room C

## 痛覚 (J)
## Pain (J)

This session is held in Japanese

Chairperson 野口 光一 (Koichi Noguchi) (兵庫医科大学解剖学第2講座)

O2P-C13 17:00
脳卒中後疼痛患者における tractography と rTMS の治療効果
The diffusion tensor image and tractography of the patients with post-stroke pain
後藤 哲(Tetsu Goto), 齋藤 洋一(Youichi Saitoh), 貴島 晴彦(Haruhiko Kishima),
平田 雅之(Masayuki Hirata), 押野 悟(Satoru Oshino), 谷 直樹(Naoki Tani),
橋本 直哉(Naoya Hashimoto), 加藤 天美(Amami Kato), 吉峰 俊樹(Toshiki Yoshimine)
大阪大院・医・脳神経外科

O2P-C14 グリシントランスポーター阻害による神経因性疼痛制御
17:15 Blockade of glycine transporters ameliorated mechanical allodynia developed in neuropathic pain model in mice
森田　克也(Katsuya Morita)，本山　直世(Naoyo Motoyama)，北山　友也(Tomoya Kitayama)，
森岡　徳光(Norimitsu Morioka)，土肥　敏博(Toshihiro Dohi)
広島大院・医歯薬学総合・歯科薬理学

O2P-C15 痒みの脳内メカニズムに関するヒトfMRI研究
17:30 Neural correlates associated with perceptual difference between itching and pain: A human fMRI study
望月　秀紀[1,2](Hideki Mochizuki)，定藤　規弘[2](Norihiro Sadato)，谷内　一彦[3](Kazuhiko Yanai)
[1]国立精神・神経センター，[2]自然科学研究機構生理学研究所，[3]東北大学大学院医学系研究科

O2P-C16 ミノサイクリンはインターフェロンγによる脊髄ミクログリアの活性化および異痛症を抑制する
17:45 Minocycline attenuates spinal microglia activation and tactile allodynia caused by interferon-γ
増田　隆博(Takahiro Masuda)，津田　誠(Makoto Tsuda)，井上　和秀(Kazuhide Inoue)
九州大院・薬・薬理

## Oral Session 2P-D　14:00-15:00　Room D

## 幹細胞 3
## Stem Cells 3

Chairperson　田賀　哲也 (Tetsuya Taga)（熊本大学発生医学研究センター）

O2P-D01 Wnt3aシグナリングによる海馬前駆細胞の細胞周期調節
14:00 Wnt3a promotes mouse hippcampal progenitor cell proliferation by shortening cell cycle
鹿川　哲史[1](Tetsushi Kagawa)，吉永　豊[1](Yutaka Yoshinaga)，清水　健史[1](Takeshi Shimizu)，
井上　俊洋[1](Toshihiro Inoue)，高田　慎治[2](Shinji Takada)，田賀　哲也[1](Tetsuya Taga)
[1]熊本大・発生研・転写制御，[2]統合バイオ

O2P-D02 Wntシグナルは成体哺乳類の網膜再生を促進する
14:15 Wnt signaling promotes retinal regeneration in adult mammals
小坂田文隆[1,2](Fumitaka Osakada)，大音壮太郎[1](Sotaro Ooto)，赤木　忠道[1](Tadamichi Akagi)，
万代　道子[1](Michiko Mandai)，赤池　昭紀[2](Akinori Akaike)，高橋　政代[1](Masayo Takahashi)
[1]理化学研究所　発生・再生科学総合研究センター　網膜再生医療研究チーム，[2]京都大学大学院　薬学研究科　薬品作用解析学

O2P-D03 TsukushiはWntシグナルを阻害して網膜幹細胞の増殖を抑制する
14:30 Tsukushi inhibits proliferation of retinal stem cells by Wnt signalling inhibition
太田　訓正[1,3](Kunimasa Ohta)，伊藤　綾子[1](Ayako Ito)，栗山　正[1,2](Sei Kuriyama)，
大沼　信一[4](Shin-ichi Ohnuma)，小阪美津子[5](Mitsuko Kosaka)，中川　真一[6](Shin-ichi Nakagawa)，
田中　英明[1,2](Hideaki Tanaka)
[1]熊本大院・医薬・神経分化学，[2]熊本大・COE，[3]科技構，[4]ケンブリッジ大学，[5]理研CDB，[6]理研中川主幹研究ユニット

O2P-D04 Hes5を発現する蝸牛支持細胞は高い増殖能力を持つ
14:45 Hes5-expressing supporting cells in the mammalian cochlea possess a high proliferative potential
小島　憲[1](Ken Kojima)，小野　和也[1](Kazuya Ono)，竹林　慎治[1](Shinji Takebayashi)，
大塚　俊之[2](Toshiyuki Ohtsuka)，伊藤　壽一[1](Juichi Ito)
[1]京大院・医・耳鼻科，[2]京大・ウイルス研

**Oral Session 2P-D** 15:00-15:45 Room D

## 幹細胞 4
## Stem Cells 4

Chairperson 新郷 哲郎 (Tetsuro Shingo) (岡山大学大学院医歯薬学総合研究科脳神経外科)

O2P-D05
15:00
**持続的脳深部刺激による慢性期脳虚血モデルに対する神経再生効果**
**Continuous deep brain stimulation has a regenrative effect on the ischemic rat model in the chronic stage**
森本 尊雅(Takamasa Morimoto), 新郷 哲郎(Teturo Shingo), 蔵本 智士(Satoshi Kuramoto),
田尻 直輝(Naoki Tajiri), 門田 知倫(Tomohito Kadota), 安原 隆雄(Takao Yasuhara),
三好 康之(Yasuyuki Miyoshi), 伊達 勲(Isao Date)
岡山大院・医歯薬・神経病態外科 (脳神経外科)

O2P-D06
15:15
**Effects of the monomeric, oligomeric and fibrillar Aβ42 peptideson the proliferation and differentiation of adult neural stemcells from SVZ**
Hee Soon Choi, ChaeJung Heo, Keun-A Jang
Department of Pharmacology, College of Medicine, Seoul National University, Seoul, Korea

O2P-D07
15:30
**Enhanced survival of neural progenitor cells derived TH positive neurons under surveillance of olfactory ensheating cells on exposure to 6-hydoxydopamine**
Kavita Seth, Nishi Srivastava, Ashok K Agrawal
DEV. TOX. DIV., I T RC, LUCKNOW, INDIA

**Oral Session 2P-D** 16:00-17:00 Room D

## 神経新生
## Neurogenesis

Chairperson 山口 正洋 (Masahiro Yamaguchi) (東京大学大学院医学系研究科細胞分子生理学)

O2P-D08
16:00
**虚血サル海馬のニューロン新生と DSCAM**
**DSCAM in the hippocampal neurogenesis of ischemic monkeys**
山嶋 哲盛[1](Tetsumori Yamashima), Boryana Popivanova[2], Jianzhong Guo[1],
向田 直史[2](Naofumi Mukaida)
[1]金沢大院・医・再生脳外科, [2]金沢大・がん研

O2P-D09
16:15
**不飽和脂肪酸は海馬神経前駆細胞の増殖を促進する**
**Polyunsaturated fatty acids promote proliferation of neural progenitor cells in the hippocampal dentate gyrus**
前川 素子[1,2](M Maekawa), 松股 美穂[2,3](M Matsumata), 大和田祐二[4](Y Owada), 紺谷 昌仙[5](M Kontani),
原 芳信[2,3](Y Hara), 河島 洋[5](H Kawashima), 木曽 茂樹[5](Y Kiso), 湯浅 茂樹[1](S Yuasa),
大隅 典子[2,3](N Osumi)
[1]国立精神・神経セ, [2]東北大学・院・医, [3]CREST, JST, [4]山口大学・院・医, [5]サントリー・健康科学

O2P-D10
16:30
**マウス成体脳における神経新生の機能的意義**
**Functional significance of adult neurogenesis in the mouse brain**
今吉 格[1](Itaru Imayoshi), 糸原 重美[2](Shigeyoshi Itohara), 池田 敏男[3](Toshio Ikeda),
影山龍一郎[1](Ryoichiro Kageyama)
[1]京都大学・ウイルス研究所, [2]理化学研究所脳科学総合研究センター, [3]国立長寿医療センター研究所

O2P-D11
16:45
**食餌時間に同調したマウス嗅球新生顆粒細胞の生死決定臨界期の存在**
**Feeding-related time window of fate decision of newborn granule cells in the adult mouse olfactory bulb**
横山 健(Takeshi Yokoyama), 森 憲作(Kensaku Mori), 山口 正洋(Masahiro Yamaguchi)
東京大院・医・細胞分子生理

## Oral Session 2P-D　17:00-18:00　Room D

### 栄養因子
### Trophic Factors

Chairperson　船越　洋（Hiroshi Funakoshi）（大阪大学大学院医学系研究科分子再生医学分野）

O2P-D12
17:00
CNTFによる後根神経節ニューロンの生存・神経突起伸長促進機構の解析
Molecular mechanisms for the neuropoietic actions of CNTF on cultured adult rat dorsal root ganglion neurons
三五　一憲(Kazunori Sango)，柳澤比呂子(Hiroko Yanagisawa)
東京都神経研・発生形態

O2P-D13
17:15
ツメガエル外胚葉に存在する自己分泌型FGFシグナリングの神経誘導・形態形成における関与
Autocrine FGF signaling in ectoderm is involved in neural induction and patterning in *Xenopus* development
岡本　治正(Harumasa Okamoto)，本郷　育子(Ikuko Hongo)
産業技術総合研究所・脳神経情報

O2P-D14
17:30
肝細胞増殖因子 (HGF) はラット生後発生過程のオリゴデンドロサイト前駆細胞 (OPC) に栄養因子として機能する
Hepatocyte growth factor (HGF) as a trophic factor for oligodendrocyte progenitor cells (OPCs) during postnatal development in the rat
大谷　若菜(Wakana Ohya)，船越　洋(Hiroshi Funakoshi)，中村　敏一(Toshikazu Nakamura)
大阪大院・医・分子再生医学

O2P-D15
17:45
中脳ドーパミン性神経細胞におけるEGFの栄養因子的作用
Neurotrophic effects of EGF / ErbB1 signals on developing midbrain dopaminergic neurons
岩倉百合子(Yuriko Iwakura)，鄭　英君(Ying Zheng)，朴　英善(Ying-Shan Piao)，
横幕　大作(Daisaku Yokomaku)，青木　弘之(Hiroyuki Aoki)，武井　延之(Nobuyuki Takei)，
那波　宏之(Hiroyuki Nawa)
新潟大・脳研・分子神経生物学

## Oral Session 2P-E　14:00-15:00　Room E

### パーキンソン病 1
### Parkinson's Disease 1

Chairperson　橋本　款（Makoto Hashimoto）（東京都神経科学総合研究所）

O2P-E01
14:00
前シナプス装置のスカフォールドおよびレビー小体の構成成分であるSept4はα-シヌクレインの神経毒性を抑制する
Sept4, a component of presynaptic scaffold and Lewy bodies,is required for the suppression of α-synuclein neurotoxicity
木下　専[1](Makoto Kinoshita)，猪原　匡史[1,2](Masafumi Ihara)，萩原　明[1](Akari Hagiwara)，
服部　信孝[3](Nobutaka Hattori)，宮川　剛[4](Tsuyoshi Miyakawa)
[1]京都大院・医，[2]京都大院・医・神経内科，[3]順天堂大・脳神経内科，[4]藤田保健衛生大・総医研

O2P-E02
14:15
パーキンソン病関連神経毒1BnTIQはtubulinに結合する
1BnTIQ, a Parkinson Disease-related neurotoxin binds to tubulin
幸田　龍紀[1](Ryuki Kohta)，古武弥一郎[1](Yaichiro Kotake)，泉　俊輔[2](Syunsuke Izumi)，
杉原　数美[1](Kazumi Sugihara)，細谷　孝充[3,4](Takamitsu Hosoya)，平松　俊行[3,4](Toshiyuki Hiramatsu)，
大坪　裕子[4](Yuko Otsubo)，古山　浩子[4](Hiroko Koyama)，鈴木　正昭[4,5](Masaaki Suzuki)，
太田　茂[1](Shigeru Ohta)
[1]広島大院・医歯薬，[2]広島大院・理，[3]東工大院・生命理工，[4]岐阜大院・医，[5]理研フロンティア

O2P-E03 14:30
変異型βシヌクレインによる神経変性機序の解析
Molecular mechanism of neurodegeneration caused by familial mutaions (P123H and V70M) of β-synuclein
橋本　款(Makoto Hashimoto)，魏　建設(Jianshe Wei)，中井　雅晶(Masaaki Nakai)，
藤田　雅代(Masayo Fujita)
都神経研・病態代謝化学

O2P-E04 14:45
パーキンソン病モデルサルにおけるドパミン合成系酵素遺伝子導入の長期効果
Long-term recovery in a primate model of Parkinson disease after gene transfer of dopamine-synthesizing enzymes
Shin-ichi Muramatsu[1]，小野　文子[3](Fumiko Ono)，滝野　直美[1](Naomi Takino)，
西田　紘子[1](Hiroko Nishida)，浅利さやか[1](Sayaka Asari)，池口　邦彦[1](Kunihiko Ikeguchi)，
藤本　健一[1](Ken-ichi Fujimoto)，塚田　秀夫[4](Hideo Tsukada)，寺尾　恵治[3](Keiji Terao)，
小澤　敬也[2](Keiya Ozawa)，中野　今治[1](Imaharu Nakano)
[1]自治医大・神経内科，[2]自治医大　遺伝子治療，[3]筑波霊長類医科学センター，[4]浜松ホトニクス

**Oral Session 2P-E** 15:00-15:45 Room E

## パーキンソン病 2 (J)
## Parkinson's Disease 2 (J)
This session is held in Japanese

Chairperson　東海林幹夫 (Mikio Shoji) (弘前大学大学院医学研究科)

O2P-E05 15:00
L-DOPA 誘発ジスキネジアモデルラットの淡蒼球内節では L-DOPA 投与により GABA が過剰に遊離される
L-DOPA persuades enhanced GABA release in the medial globus pallidus in a rat model of L-DOPA-induced dyskinesia
冨山　誠彦[1](Masahiko Tomiyama)，新井　陽[1](Akira Arai)，朱　剛[2](Gang Zhu)，
木村　珠喜[1](Tamaki Kimura)，東海林幹夫[1](Mikio Shoji)
[1]弘前大学大学院医学研究科脳研脳神経内科，[2]弘前大学大学院神経精神科

O2P-E06 15:15
グルタミン酸誘発ドパミンニューロン死における内在性ドパミンの関与
Involvement of endogenous dopamine in dopaminergic neuronal death induced by glutamate
泉　安彦[1](Yasuhiko Izumi)，澤田　秀幸[2](Hideyuki Sawada)，山本　憲幸[1](Noriyuki Yamamoto)，
久米　利明[1](Yoshiaki Kume)，香月　博志[1](Hiroshi Katsuki)，赤池　昭紀[1](Akinori Akaike)
[1]京都大学大学院薬学研究科薬品作用解析学分野，[2]国立病院機構宇多野病院

O2P-E07 15:30
ゾニサミドの実験的振戦に対する効果
Effects of zonisamide on experimental tremors in rats
三輪　英人(Hideto Miwa)，浜　喜和(Kiwa Hama)，梶本　賀義(Yoshinori Kajimoto)，
中西　一郎(Ichiro Nakanishi)，近藤　智善(Tomoyoshi Kondo)
和歌山県医大・医・神経内科

**Oral Session 2P-E** 16:00-17:30 Room E

## 大脳基底核
## Basal Ganglia

Chairpersons　高田　昌彦 (Masahiko Takada) (東京都神経科学総合研究所)
藤山　文乃 (Fumino Fujiyama) (京都大学医学部医学研究科高次脳形態学教室)

O2P-E08 16:00
ラット線条体パッチ・マトリックスからの投射経路を再検討する
A reevaluation of the network via patch and matrix compartments of rat neostriatum
藤山　文乃[1](Fumino Fujiyama)，孫　在隣[1](Jaerin Sohn)，雲財　知[1](Tomo Unzai)，
金子　武嗣[1,2](Takeshi Kaneko)
[1]京都大院・医・高次脳形態学，[2]CREST

O2P-E09 16:15
Molecular basis for impulsive and self-controlled behaviors in delay-discounting task
Seiji Hayashizaki, Yumi Ito, Masahiko Takada
Tokyo Metropolitan Institute for Neuroscience, Tokyo, Japan

O2P-E10 16:30
視床下核—淡蒼球神経回路モデルの神経活動モードの解析
Dynamical modes in a computational model of the subthalamo-pallidal circuit
北野　勝則[1](Katsunori Kitano), 深井　朋樹[2](Tomoki Fukai)
[1]立命館大・情報理工・知能情報, [2]理化学研究所・脳総研

O2P-E11 16:45
Role of the primate lateral habenula in negative motivational control of behavior
Masayuki Matsumoto, Okihide Hikosaka
Laboratory of Sensorimotor Research, NEI, NIH, USA

O2P-E12 17:00
ドーパミン誘導性運動に対する前頭前皮質 - 視床下核経路の機能
The medial prefrontal cortex controls dopamine-induced motor behavior via the subthalamic nucleus activation
八十島安伸(Yasunobu Yasoshima), 小林　和人(Kazuto Kobayashi)
福島医大・医・生体機能

O2P-E13 17:15
パーキンソン病モデルサルの大脳基底核ニューロンに見られる異常発振
Abnormal oscillatory discharges of basal ganglia neurons in parkinsonian monkeys
橘　　吉寿[1,2](Yoshihisa Tachibana), 岩室　宏一[1,3](Hirokazu Iwamuro), 高田　昌彦[4](Masahiko Takada),
南部　　篤[1,2](Atsushi Nambu)
[1]生理研・生体システム, [2]総研大, [3]東京大・脳神経外科, [4]東京都神経研・統合生理

## Oral Session 2P-F　14:00-15:00　Room F

## 認知 1
## Cognition 1

Chairperson　花川　　隆 (Takashi Hanakawa) (国立精神・神経センター神経研究所)

O2P-F01 14:00
視知覚時のシータ、ガンマ同期によるネットワーク形成
Network formation by theta and gamma-band synchronization in visual perception
北城　圭一(Keiichi Kitajo), 尾崎　　隆(Takashi Ozaki), 山口　陽子(Yoko Yamaguchi)
理化学研究所・脳科学総合研究センター・創発知能ダイナミクス研究チーム

O2P-F02 14:15
注意の瞬き中の脳波位相同期現象
EEG phase synchronizaion during Attentional Blink
中谷　智恵(Chie Nakatani), Cees van Leeuwen
理研脳科学総合研究センター・認知動力学研究チーム

O2P-F03 14:30
視覚におけるノイズとパターンの相互作用
Interaction between noise and pattern in vision
脇坂　崇平(Sohei Wakisaka)
神戸大院・理

O2P-F04 14:45
手足の心内回転課題に伴う身体部位局在性脳活動
Mental rotation of hands and feet involves somatotopically organized brain regions
花川　　隆(Takashi Hanakawa), 細田　千尋(Chihiro Hosoda), 進藤　誠吾(Seigo Shindo),
本田　　学(Manabu Honda)
国立精神・神経センター・神経研・疾病研究第七部

## Oral Session 2P-F　15:00-16:00　Room F

### 認知 2
### Cognition 2

Chairperson　入來　篤史（Atsushi Iriki）（理化学研究所脳科学総合研究センター象徴概念発達研究チーム）

O2P-F05
15:00
**Effects of prefrontal and anterior cingulate lesions in monkeys on a Wisconsin Card Sorting Task (WCST) analogue**
Mark J. Buckley[1], Farshad A. Mansouri[2], Phillip G.F. Browning[1], Hassan Hoda[2], Sze C. Kwok[1], Majid Mahboubi[2], Adam Phillips[2], Keiji Tanaka[2]
[1]Oxford University, [2]RIKEN Brain Science Institution

O2P-F06
15:15
**Mnemonic function of lateral prefrontal cortex in conflict-induced behavioral adjustment**
Farshad A. Mansouri[1], Mark J. Buckley[2], Keiji Tanaka[1]
[1]RIKEN Brain Science Institute, Japan, [2]Oxford University, UK.

O2P-F07
15:30
**非論理的推論を可能にする人間の認知バイアスの脳活動**
**Neural correlates of human cognitive bias resulting in illogical inference**
小川　昭利[1](Akitoshi Ogawa), 山崎由美子[1](Yumiko Yamazaki), 上野　賢一[3](Kenichi Ueno), Kang Cheng[2,3], 入來　篤史[1](Atsushi Iriki)
[1]理研・脳センター・象徴概念発達研究チーム, [2]理研・脳センター・認知機能表現研究チーム, [3]理研・脳センター・RRC

O2P-F08
15:45
**副腎皮質刺激ホルモン放出因子は注意機能を上昇させる**
**Corticotropin releasing factor increases attentional function in rats**
大村　優(Yu Ohmura), 山口　拓(Taku Yamaguchi), 泉　剛(Takeshi Izumi), 松本真知子(Machiko Matsumoto), 吉岡　充弘(Mitsuhiro Yoshioka)
北海道大院・医・神経薬理

## Oral Session 2P-F　16:00-17:00　Room F

### アルツハイマー病 1 (J)
### Alzheimer's Disease 1 (J)

This session is held in Japanese

Chairperson　高島　明彦（Akihiko Takashima）（理化学研究所脳科学総合研究センター）

O2P-F09
16:00
**認知症患者血漿におけるアネキシン A5 濃度上昇（分子マーカー）**
**Elevation of circulating level of annexin A5 in dementia patients (molecular marker)**
相馬　仁[1](Hitoshi Sohma), 誉田　大仁[3](Hirohito Honda), 水江　由佳[4](Yuka Mizue), 山口　真美[1](Mami Yamaguchi), 村上　新治[2](Shinji Murakami), 前田　哲[5](Tetsu Maeda), 小海　康夫[1](Yasuo Kokai)
[1]札幌医大・医・分子機能, [2]札幌医大・保健医療・作業療法, [3]三洋化成工業(株), [4]札幌IDL, [5]釧路北病院

O2P-F10
16:15
**帰脾湯による記憶障害改善作用のメカニズム**
**The mechanism of memory improvement by Kihi-to**
城屋敷枝里(Eri Joyashiki)
富山大・和漢研・薬効解析部

O2P-F11
16:30
**RB1CC1 発現低下は mTOR 経路を介して神経細胞萎縮を起こし、アルツハイマー病に関与する**
**RB1CC1 insufficiency causes neuronal atrophy through mTOR signaling alteration and involved in pathology of Alzheimer's diseases**
茶野　徳宏(Tokuhiro Chano), 岡部　英俊(Hidetoshi Okabe)
滋賀医科大学・医・臨床検査医学

O2P-F12 16:45
エストロジェンはムスカリン性アセチルコリン受容体 M5 欠損による脳血管収縮に対して代償的に作用する
Estrogen prevents cerebrovascular deficits by the lack of M5 muscarinic acetylcholine receptor through MAPK activation
新家　瑠奈[1](Runa Araya), 北村　尚士[1](Naohito Kitamura), 木村　哲也[2](Tetsuya Kimura), Jurgen Wess[3], 山田　真久[1](Masahisa Yamada)
[1]理研・脳センター・山田研究ユニット, [2]理研・脳センター・アルツハイマー病, [3]Bioorganic Chemistry, NIDDKD, USA

**Oral Session 2P-F** 17:00-18:00 Room F

## アルツハイマー病 2
## Alzheimer's Disease 2

Chairperson　樋口　真人 (Makoto Higuchi) (放射線医学総合研究所分子イメージング研究センター)

O2P-F13 17:00
脳虚血に伴う過剰の eNOS 発現はβ－アミロイドの蓄積に関与する
Aberrant expression of eNOS is associated with accumulation of beta-amyloid in brain ischemia-induced microvascular injury
韓　　峰[1](Feng Han), 福永　浩司[1,2](Kohji Fukunaga)
[1]東北大院・薬・薬理, [2]東北大学・ 21世紀COEプログラム

O2P-F14 17:15
安定同位体利用 NMR 法による GM1 ミセル結合型アミロイドβの構造解析
Conformation and intermolecular interaction of amyloid β-peptide in association with GM1 micelles as revealed by NMR spectroscopy
内海　真穂[1,2](Maho Utsumi), 山口　芳樹[1](Yoshiki Yamaguchi), 山本　直樹[3](Naoki Yamamoto), 柳澤　勝彦[3](Katsuhiko Yanagisawa), 加藤　晃一[1,2](Koichi Kato)
[1]名市大・院薬, [2]分子科学研究所, [3]長寿医療センター研究所

O2P-F15 17:30
Human chorionic gonadotropin increases exploratory behaviour and impairs episodic-like memory in PS1M146V mice
Anna M. Barron[1,2,3], Kevin Taddei[1,2,3], Giuseppe Verdile[1,2,3], Ralph N. Martins[1,2,3]
[1]School of Psychiatry & Clinical Neurosciences, University of Westen Australia, Australia, [2]Centre of Excellence in Alzheimer's Disease Research and Care, Edith Cowan University, [3]Sir James McCusker Alzheimer's Disease Research Unit, Hollywood Private Hospital

O2P-F16 17:45
P301S 変異タウトランスジェニックマウスにおいてはマイクログリアの活性化がタウ病理、神経変性に先行する
Microglial activation precedes tau pathology and neurodegeneration in a P301S mutant tau transgenic mouse
吉山　容正[1,2](Yasumasa Yoshiyama), 樋口　真人[2,3](Makoto Higuchi), John_Q. Trojanowski[2], Virginia_M.-Y. Lee[2]
[1]国立病院機構　千葉東病院　神経内科, [2]ペンシルバニア大学, [3]放射線医学研究所

**Oral Session 2P-G** 14:00-15:00 Room G

## 脳イメージング
## Brain Imaging

Chairperson　谷内　一彦 (Kazuhiko Yanai) (東北大学大学院医学系研究科)

O2P-G01 14:00
The contribution of the dorsolateral prefrontal cortex to executive function
Ji Hyun Ko[1], Oury Monchi[2], Alain Ptito[1], Michael Petrides[1], Antonio_P Strafella[3,4]
[1]Montreal Neurological Institute, McGill Univ., Canada, [2]Geriatric Institute, Univ. of Montreal, Canada, [3]Toronto Western Research Institute and Hospital, Univ. of Toronto, Canada, [4]Centre for Addiction and Mental Health, Univ. of Toronto, Canada

O2P-G02 興奮映像観察時のヒトの fMRI 脳活動と自律神経反応の相関
14:15 Correlation between human brain activities and the autonomic nervous system elicited by emotional video movies

田中 靖人[1](Yasuto Tanaka), 光吉 俊二[2](Shunji Mitsuyoshi), 柴崎 晃一[3](Koichi Shibasaki),
加藤 誠[4](Makoto Kato), 南 哲人[4](Tetsuto Minami), 村田 勉[4](Tsutomu Murata)
[1]情報通信研究機構 バイオICTグループ, [2]エージーアイ株式会社, [3]日本SGI, [4]情報通信研究機構

O2P-G03 知っている人と知らない人での他者の意思決定の推測についての脳活動
14:30 Brain activities related to inferring familiar and unfamiliar persons` decision-makings: An fMRI study

立花 良之[1](Yoshiyuki Tachibana), 佐々 裕子[1,2](Yuko Sassa), 横山 悟[1](Satoru Yokoyama),
杉浦 元亮[1,3](Motoaki Sugiura), 川島 隆太[1,2](Ryuta Kawashima)
[1]東北大学加齢医学研究所脳機能開発研究分野, [2]科学技術振興機構・社会技術研究開発事業, [3]生理学研究所大脳皮質機能研究系

O2P-G04 Abnormalities in white matter structure in autism spectrum disorders detected by diffusion
14:45 tensor imaging

Roger J. Jou[1,2], Sarah J. Paterson[2], Xenophon Papademetris[3], Lawrence H. Staib[3],
Robert T. Schultz[2,3]
[1]Dept of Psychiatry, Yale University, New Haven, USA, [2]Child Study Center, Yale University, New Haven, CT, USA, [3]Dept of Diagnostic Radiology, Yale University, New Haven, CT, USA

**Oral Session 2P-G** 15:00-16:00 Room G

## 細胞イメージング Cellular Imaging

Chairperson 寺川 進 (Susumu Terakawa) (浜松医科大学光量子医学研究センター細胞イメージング)

O2P-G05 カエル脳幹部呼吸神経活動の可視化
15:00 Respiratory neuronal activities of the frog brainstem visualized *in vitro*

越久 仁敬[1](Yoshitaka Oku), 木村 直史[2](Naofumi Kimura), 増宮 晴子[1](Haruko Masumiya),
岡田 泰昌[3](Yasumasa Okada)
[1]兵庫医大・生理1, [2]慈恵医大・薬理2, [3]慶應大・医・月が瀬リハセンター・内科

O2P-G06 二光子励起機能的カルシウムイメージング法によって明らかになったマウス大脳皮質視覚野の興奮性細胞と抑制性細胞における方位選択性の違い
15:15 Difference in response selectivity between inhibitory and excitatory neurons in visual cortex, as revealed by *in vivo* two-photon functional $Ca^{2+}$ imaging

惣谷 和広[1](Kazuhiro Sohya), 亀山 克朗[1](Katsuro Kameyama), 柳川右千夫[2](Yuchio Yanagawa),
津本 忠治[1](Tadaharu Tsumoto)
[1]理化学研究所・脳科学総合研究センター, [2]群馬大院・医・遺伝発達行動学分野

O2P-G07 消去・再生可能な蛍光蛋白をもちいたマウス神経回路網の可視化
15:30 Optical dissection and reconstruction of neural networks in the mouse cortex using a photo-reversible fluorescent protein

八田 公平[1,2](Kohei Hatta), 大村 朋美[2](Tomomi Omura)
[1]兵庫県立大院・生命理学・生体情報学 I, [2]理研CDB

O2P-G08 生体内蛍光イメージングの落とし穴 —虚血による影響—
15:45 Pitfall in the study of intravital fluorescence imaging during ischemia

山本 清二(S Yamamoto), 王 勇(Y Wang), 宮川 厚夫(A Miyakawa), 櫻井 孝司(T Sakurai),
寺川 進(S Terakawa)
浜松医科大学・光量子医学研究センター

**Oral Session 2P-G** 16:00-17:00 Room G

## 社会的行動 1
## Social Behavior 1

Chairperson 澤本 伸克 (Nobukatsu Sawamoto) (京都大学大学院医学研究科附属高次脳機能総合研究センター)

O2P-G09 16:00
**視線と頭の方向に対するサル扁桃体顔ニューロンの応答性**
**Activity of monkey amygdalar face neurons sensitive to both gaze and head directions**
田積 徹[1,2,3](Tooru Tazumi), 堀 悦郎[2,3](Etsuro Hori), 小野 武年[2,3](Taketoshi Ono),
西条 寿夫[2,3](Hisao Nishijo)
[1]聖泉大学・人間学部・人間心理, [2]富山大学大学院 医学薬学研究部, [3]科学技術振興機構

O2P-G10 16:15
**パーソナリティに依存した他人の心的状況を理解するための皮質回路**
**Personality-dependent cortical network during understanding other persons' mental states**
地村 弘二(Koji Jimura), 小西 清貴(Seiki Konishi), 浅利 知輝(Tomoki Asari),
近添 淳一(Junichi Chikazoe), 宮下 保司(Yasushi Miyashita)
東京大学・医・統合生理

O2P-G11 16:30
**親子間における顔表情認知の神経基盤**
**Neural basis of emotional facial recognition between parents and children**
篠崎 淳[1](Jun Shinozaki), 澤本 伸克[1](Nobukatsu Sawamoto), 村井 俊哉[2](Toshiya Murai),
花川 隆[1](Takashi Hanakawa), 福山 秀直[1](Hidenao Fukuyama)
[1]京都大学大学院医学研究科附属高次脳機能総合研究センター, [2]京都大学大学院医学研究科精神医学教室

O2P-G12 16:45
**結婚相手と友人選び：視覚情報と社会的文脈が意思決定大脳メカニズムに与える影響**
**Who is to marry or to be a friend?: an fMRI study of social decision making**
杉浦 元亮[1,2,3](Motoaki Sugiura), 舟山 梨沙[3](Risa Funayama), 佐々 祐子[2,4](Yuko Sassa),
Hyeonjeong Jeong[2], 涌澤 圭介[2,6](Keisuke Wakusawa), 立花 良之[2,6](Yoshiyuki Tachibana),
堀江 薫[5,7](Kaoru Horie), 佐藤 滋[5,7](Shigeru Sato), 川島 隆太[2,4](Ryuta Kawashima)
[1]生理研・大脳皮質機能, [2]東北大・加齢研, [3]宮教大, [4]RISTEX, JST, [5]東北大院・国際文化, [6]東北大院・医,
[7]東北大・言語認知研究拠点

**Oral Session 2P-G** 17:00-18:00 Room G

## 社会的行動 2
## Social Behavior 2

Chairperson 佐久間康夫 (Yasuo Sakuma) (日本医科大学大学院システム生理学)

O2P-G13 17:00
**幼少期における豊かな環境飼育はオープンフィールドでの雄ラット性行動を減弱する**
**Rearing in enriched environment calms down male rat copulatory activities in open field**
浦川 将[1](Susumu Urakawa), 近藤 保彦[1](Yasuhiko Kondo), 美津島 大[2](Dai Mitsushima),
西野 仁雄[3](Hitoo Nishino), 佐久間康夫[1](Yasuo Sakuma)
[1]日本医科大学・医・生理学第一, [2]横浜市立大学・医・神経内分泌学, [3]名古屋市立大学・医・脳神経生理

O2P-G14 17:15
**Altered Drosophila aggression, locomotion, and neuromuscular excitability by isolated rearing.**
Atsushi Ueda, Chun-Fang Wu
Dpt of Biological Sciences, University of Iowa, Iowa City, USA

O2P-G15 17:30
**Motopsin/PRSS12 欠損マウスにおける社会行動の異常**
**Mice lacking motopsin/PRSS12 gene showed abnormal social behavior.**
三井 真一[1](Shinichi Mitsui), 大迫 洋治[1](Yoji Osako), 由利 和也[1](Kazunari Yuri),
横井 史章[2](Fumiaki Yokoi), D. Deng Mai[2], Li Yuqing[2], 山口 希[3](Nozomi Yamaguchi)
[1]高知大・医・解剖, [2]アラバマ大・医・神経, [3]同志社大・工・環境システム

O2P-G16
17:45
自閉症スペクトラム障害における社会的文脈に応じた動的な意思決定過程の調整メカニズムの障害
Dysfunction of dynamical modulation of decision making process depending on social context in autism spectrum disorder
高橋　英之[1](Hideyuki Takahashi)，大森　隆司[2](Takashi Omori)，室橋　春光[3](Harumitu Murohashi)，
神尾　陽子[4,5](Yoko Kamio)
[1]北海道大院・情報・複合情報，[2]玉川大・学術研究所，[3]北海道大院・教育，[4]国立精神神経センター，[5]科学技術振興機構・社会技術研究開発センター

**Oral Session 2P-H** 14:00-15:00 Room H

## 精神障害 (J)
## Psychiatric Disorders (J)

This session is held in Japanese

Chairperson 橋本　亮太 (Ryota Hashimoto)（大阪大学大学院医学系研究科附属子どものこころの分子統御機構研究センター）

O2P-H01
14:00
ＰＡＣＡＰ遺伝子は統合失調症と関連する
Pituitary adenylate cyclase-activating polypeptide is associated with schizophrenia
橋本　亮太[1,2](Ryota Hashimoto)，功刀　　浩[2](Hiroshi Kunugi)，馬場　明道[3](Akemichi Baba)
[1]大阪大院・医・子どものこころ，[2]国立精神セ・神経研究所・疾病三部，[3]大阪大院・薬・神経薬理学

O2P-H02
14:15
IL-1 受容体アンタゴニストノックアウトマウスにおいて誘導される不安様行動の解析
IL-1 receptor antagonist (IL-1Ra) KO mice show anxiety-like behavior
若林　千里[1,2](Chisato Wakabayashi)，岩倉洋一郎[1](Yoichiro Iwakura)
[1]東大・医科研・細胞機能，[2]（独）日本学術振興会

O2P-H03
14:30
うつ病発症関連因子としての TRAP1 の同定および機能解析
Identification and functional characterization of TRAP1 as a candidate gene for major depression
窪田　杏子[1](Kyoko Kubota)，井上　　浄[1](Kiyoshi Inoue)，橋本　亮太[2,3,4](Ryota Hashimoto)，
功刀　　浩[4](Hiroshi Kunugi)，斉藤　　治[5](Osamu Saito)，小菅　麻子[6](Asako Kosuga)，
巽　　雅彦[7](Masahiko Tatsumi)，上島　国利[6](Kunitoshi Ueshima)，熊本奈都子[1](Natsuko Kumamoto)，
遠山　正彌[1](Masaya Tohyama)
[1]大阪大院・医・神経機能形態学，[2]大阪大院・医・付属子どものこころの分子統御機構研究センター，[3]大阪大院・医・精神医学，[4]国立精神・神経センター神経研究所疾病研究第三部，[5]国立精神・神経センター武蔵病院精神科，[6]昭和大・医・精神医学，[7]横浜診療クリニック

O2P-H04
14:45
縫線核含有中脳切片培養系におけるメタンフェタミン、MDMA および 5-MeO-DiPT 長期処置によるセロトニン神経への影響
Chronic effects of methamphetamine, MDMA and 5-MeO-DiPT on raphe serotonergic neurons in rat organotypic mesencephalic slice culture
樋口　　萌(Megumi Higuchi)，鈴木　祐一(Yuichi Suzuki)，中川　貴之(Takayuki Nakagawa)，
金子　周司(Shuji Kaneko)
京大院・薬・生体機能解析

**Oral Session 2P-H** 15:00-15:45 Room H

## 統合失調症
## Schizophrenia

Chairperson 宮川　　剛 (Tsuyoshi Miyakawa)（藤田保健衛生大学総合医学研究所）

O2P-H05
15:00
ERK による DISC1 のリン酸化とその生理機能の解析
Regulation of DISC1 through phosphorylation by ERK
田谷真一郎(Shinichiro Taya)，朝来　淳子(Junko Uraguchi-Asaki)，黒田　啓介(Keisuke Kuroda)，
貝淵　弘三(Kozo Kaibuchi)
名古屋大院・医・神経情報薬理

O2P-H06
15:15
神経新生は海馬 Akt の活性化を介して感覚運動系制御に関与している
Neurogenesis correlates sensorimotor gating via activation of hippocampal Akt
松崎　秀夫[1,2](Hideo Matsuzaki)，和久田智靖[2](Tomoyasu Wakuda)，関　規寛[2](Norihiro Seki)，
岩田　泰秀[2](Yasuhide Iwata)，鈴木　勝昭[2](Katsuaki Suzuki)，武井　教使[2,3](Nori Takei)，
遠山　正彌[1](Masaya Tohyama)，森　則夫[2,3](Norio Mori)
[1]大阪大・子どものこころの分子統御機構研究センター，[2]浜松医大・精神神経医学講座，[3]浜松医大・子どものこころの発達研究センター

O2P-H07
15:30
新規統合失調症脆弱性因子としての 14-3-3 ε の同定
Identification of *YWHAE*, a gene encoding 14-3-3ε, as a novel susceptibility gene for schizophrenia
匹田　貴夫[1](Takao Hikita)，池田　匡志[2](Masashi Ikeda)，田谷真一郎[1](Shinichiro Taya)，
宮川　剛[3](Takeshi Miyakawa)，貝淵　弘三[1](Kozo Kaibuchi)，尾崎　紀夫[4](Norio Ozaki)，
岩田　仲生[2](Nakao Iwata)
[1]名古屋大院・医・神経情報薬理，[2]藤田保健衛生大・医・精神，[3]京大院・医・生体遺伝子機能，[4]名古屋大院・医・精神

**Oral Session 2P-H**　16:00-17:00　Room H

## 依存・乱用
## Addiction and Abuse

Chairperson　西川　徹（Toru Nishikawa）（東京医科歯科大学大学院精神行動医科学分野）

O2P-H08
16:00
ラット脳 methamphetamine 応答性 *mrt3* および *as-mrt3* 遺伝子の構造と発現
Expression and genomic organization of methamphetamine-induced transcript *mrt3* and *as-mrt3* in the rat central nervous system
山本　直樹(Naoki Yamamoto)，村岡新一郎(Shinichiro Muraoka)，海野　麻未(Asami Umino)，
西川　徹(Toru Nishikawa)
東京医科歯科大院・医歯・精神行動医科学

O2P-H09
16:15
Chronic administration of nicotine retards the development of morphine dependency and tolerance in mice
Abbas Haghparast, Nima Naderi, Abbas Khani, Amir Alizadeh, Fereshteh Motamedi
Neuroscience Research Center, Shaheed Beheshti Medical University, Tehran, Iran

O2P-H10
16:30
Nicotine and morphine interactions; new protocol for morphine dependency in mice
Abbas Khani, Abbas Haghparast, Nima Naderi, Amir-Mohammad Alizadeh, Fereshteh Motamedi
Neuroscience Research Center, Shaheed Beheshti Medical University, Tehran, Iran

O2P-H11
16:45
キュー依存性の薬物欲求における前頭前野の役割－機能的 MRI・TMS の組み合わせ研究
A role of dorsolateral prefrontal cortex in cue-induced craving
林　拓也[1,2](Takuya Hayashi)，Ji Hyun Ko[2]，Antonio Strafella[2]，Alain Dagher[2]
[1]国循研・放医，[2]モントリオール神経研究所

**Oral Session 2P-H**　17:00-18:00　Room H

## 神経内分泌
## Neuroendocrine System

Chairperson　東田　陽博（Haruhiro Higashida）（金沢大学21世紀COE革新脳科学プログラム）

O2P-H12
17:00
CD38 は細胞内のカルシウムの濃度の調節を通じてオキシトシンの放出に影響する
CD38 can influence oxytocin release by regulating intracellular calcium concentration.
金　鐸(Duo Jin)，劉　鴻翔(Hong Xiang Liu)，橋井美奈子(Minako Hashii)，平井　宏和(Hirokazu Hirai)，
東田　陽博(Haruhiro Higashida)
金沢大・21世紀COE革新脳科学プログラム

O2P-H13 GnRH ニューロンにみられる gabazin 抵抗性 $GABA_A$ 受容体電流とニューロステロイドによる修飾
17:15 Presence of gabazine-resistant $GABA_A$ receptor currents and their modulation by neurosteroids in rat GnRH (LHRH) neurons
尹　成珠(Chengzhu Yin), 田中　伸幸(Nobuyuki Tanaka), 石井　寛高(Hirotaka Ishii),
加藤　昌克(Masakatsu Kato), 佐久間康夫(Yasuo Sakuma)
日本医科大学・生理学第一

O2P-H14 ゼブラフィッシュ糖質コルチコイド受容体遺伝子突然変異体の解析
17:30 Role of glucocorticoid receptor in vision and behavior
武藤　彩[1,2](Akira Muto), Limor Ziv[2], Michael Taylor[2], Miyuki Suzawa[2], Herwig Baier[2]
[1]遺伝研・初期発生, [2]カリフォルニア大学サンフランシスコ校生理学部門

O2P-H15 摂食調節と体重調節における内因性ノシセプチンの生理的役割
17:45 A physiological role for endogenous nociceptin in the regulation of food intake and weight gain
小泉美和子[1,2](Miwako Koizumi), Barbara Cagniard[2], 廣中　直行[1](Naoyuki Hironaka), Niall P. Murphy[2]
[1]JST・ERATO・下條潜在脳機能プロジェクト, [2]理研・脳センター・神経回路メカニズム

## Oral Session 2P-J　14:00-15:00　Room J

### 遺伝子制御 Gene Regulation

Chairperson　中島　欽一 (Kin-ichi Nakajima) (奈良先端大学)

O2P-J01 Epigenetic regulation of critical period plasticity in visual cortex
14:00
Judy CG Sng[1], Andreas Lennartsson[2], Michela Fagiolini[1,3], Piero Carninci[2], Takao Hensch[1,3]
[1]RIKEN Brain Science Institute, Laboratory for Neuronal Circuit Development, [2]RIKEN Genome Science Laboratory, [3]Children's Hospital, Harvard Medical School, Boston MA 02115

O2P-J02 GSK 遺伝子の転写に対するカリウムの効果
14:15 Effect of potassium on the transcription of GSK3$\alpha$ and GSK3$\beta$
村山　洋(Ohoshi Murayama), 山口　瞳(Akira Yamaguchi), 中西　滋之(Shigeyuki Nakanishi),
前田　康紀(Yasunori Maeda), 上野　瞳(Hitomi Ueno), 小山　香織(Kaori Koyama),
松田　基夫(Motoo Matsuda)
麻布大院・環境保健学研究科

O2P-J03 突起形態と転写制御におけるアクチン結合モチーフを有する転写活性化因子 MKL1/2 の機能解析
14:30 Molecular characterization of MKL1/2, coactivators for SRF in morphology and transcription of neurons
田渕　明子[1](Akiko Tabuchi), 石川　充[1](Mitsuru Ishikawa), 阪上　洋行[2](Hiroyuki Sakagami),
西嶋　直紀[1](Naoki Nishijima), 塩田　惇[1](Jun Shiota), 津田　正明[1](Masaaki Tsuda)
[1]富山大院・医薬・分子神経生物, [2]東北大院・医・細胞生物

O2P-J04 Imprinting of the cerebral cytochrome P450s and developmental neurotoxicity of lindane
14:45
Ashu Johri[1], Alok Dhawan[1], Ram L. Singh[2], Devendra Parmar[1]
[1]Developmental Toxicology Division, ITRC, Lucknow, India, [2]Department of Biochemistry, Dr. R.M.L. Awadh University, Faizabad

## Oral Session 2P-J　15:00-16:00　Room J

### 化学感覚 1 Chemical Senses 1

Chairperson　坂野　仁 (Hitoshi Sakano) (東京大学大学院理学系研究科生物化学)

O2P-J05 後脳における味情報の時空間コーディング
15:00 Spatiotemporal coding of gustatory information in hindbrain
齋藤　勇輝[1](Yuuki Saito), 藤田　一寿[1](Kazuhisa Fujita), 樫森与志喜[2](Yoshiki Kashimori)
[1]電気通信大学大学院・情報システム学研究科・情報ネットワーク学専攻, [2]電気通信大学　量子・物質工学科

O2P-J06
15:15
線虫 C. elegans における G タンパクを介した温度受容メカニズム
G protein-coupled novel temperature sensing mechanism
久原　篤[1](Atsushi Kuhara)，奥村　将年[1](Masatoshi Okumura)，木村幸太郎[1,2](Koutarou D Kimura)，
松本　邦弘[1](Kunihiro Matsumoto)，森　郁恵[1](Ikue Mori)
[1]名大　院理　生命，[2]名古屋大・理・生命，[3]現所属：国立遺伝学研究所

O2P-J07
15:30
嗅覚受容体遺伝子の単一発現を保障する分子機構
An LCR *H* for the mouse odorant receptor (OR) genes acts in *cis*, and not in *trans*
西住　裕文(Hirofumi Nishizumi)，熊坂　耕平(Kohei Kumasaka)，坂野　仁(Hitoshi Sakano)
東京大学・大学院理学系研究科・生物化学専攻

O2P-J08
15:45
ショウジョウバエの求愛行動に関わる新しいフェロモン受容体候補
A new candidate pheromone receptor contributing to the male courtship behavior of Drosophila
小金澤雅之(Masayuki Koganezawa)，山元　大輔(Daisuke Yamamoto)
東北大学大学院生命科学研究科

**Oral Session 2P-J**　16:00-17:00　Room J

## 化学感覚 2
## Chemical Senses 2

Chairperson　森　憲作 (Kensaku Mori)（東京大学大学院医学系研究科細胞分子生理）

O2P-J09
16:00
匂いの質感を読み解くメカニズムの解明
Decoding an innate aversive quality from an odor map in the mouse olfactory bulb
小早川　高[1](Ko Kobayakawa)，小早川令子[1](Reiko Kobayakawa)，森　憲作[2](Kensaku Mori)，
坂野　仁[1](Hitoshi Sakano)
[1]東京大学理学系研究科生物化学専攻，[2]東京大学医学系研究科機能生物学専攻細胞分子生理学教室

O2P-J10
16:15
ラット嗅球における顆粒細胞から僧帽細胞への抑制の覚醒状態依存的変化
Behavioral state-dependent change of granule-to-mitral inhibition in the rat olfactory bulb
津野　祐輔(Yusuke Tsuno)，柏谷　英樹(Hideki Kashiwadani)，森　憲作(Kensaku Mori)
東京大院・医・細胞分子生理

O2P-J11
16:30
マウスの嗅覚系のニューラルネットモデル：匂い地図を考慮したアテンションのシミュレーション
A neural network model of the olfactory system of mice:Simulated attention behavior taking the odorant map into account
曽　智[1](Zu Soh)，辻　敏夫[1](Toshio Tsuji)，滝口　昇[2](Noboru Takiguchi)，
大竹　久夫[3](Hisao Ohtake)
[1]広島大学大学院工学研究科　複雑システム工学専攻，[2]広島大学大学院先端物質科学研究科 分子生命機能科学専攻，[3]大阪大学大学院先工学研究科　生命先端工学専攻

O2P-J12
16:45
味覚嫌悪学習獲得後の条件刺激再呈示によってラット腹側淡蒼球での細胞外 GABA 遊離が増加する
The re-presentation of conditioned stimulus after acquisition of conditioned taste aversion increase ventral pallidum GABA release in rats.
乾　賢[1](Tadashi Inui)，志村　剛[1](Tsuyoshi Shimura)，山本　隆[2](Takashi Yamamoto)
[1]大阪大学・院人間科学・行動生理，[2]大阪大学・院歯学研究科

## Oral Session 2P-J　17:00-18:00　Room J

### 受容体 Receptors

Chairperson　久保　義弘 (Yoshihiro Kubo) (生理学研究所神経機能素子研究部門)

O2P-J13
17:00
**アダプター蛋白質を介した高親和性コリントランスポーターの細胞内輸送**
**Adaptor protein-dependent trafficking of high-affinity choline transporter**
三澤日出巳[1](Hidemi Misawa), 藤ヶ谷浩文[1](Hirofumi Fujigaya), 森脇　康博[1](Yasuhiro Moriwaki),
奥田　隆志[1](Takashi Okuda), 中津　史[2](Fubito Nakatsu), 大野　博司[2](Hiroshi Ohno)
[1]共立薬大・薬理, [2]理研免疫アレルギー・免疫系構築

O2P-J14
17:15
**シナプスから核へのシグナル伝達**
**Signal transport from synapse to the nucleus**
西　真弓(Mayumi Nishi), 細川　康二(Koji Hosokawa), 藤川　和世(Kazuyo Fujikawa),
河田　光博(Mitsuhiro Kawata)
京都府立医大院・医・生体構造科学

O2P-J15
17:30
**C 末端による代謝型グルタミン酸受容体と G 蛋白質との結合制御**
**Coupling profile of the metabotropic glutamate receptor1 is regulated by the C-terminal tail**
立山　充博[1,2](Michihiro Tateyama), 久保　義弘[1,2,3](Yoshihiro Kubo)
[1]生理学研究所・分子生理・神経機能素子, [2]SORST, [3]医科歯科大学

O2P-J16
17:45
**NMDA 受容体チャネルの開口を介した 5´AMP キナーゼによる $GABA_B$ 受容体のリン酸化**
**Phosphorylation of $GABA_B$ receptor R2 subunit by 5´AMP-activated protein kinase through opening NMDA receptor channel.**
倉本　展行[1](Nobuyuki Kuramoto), 山下　智美[1](Tomomi Yamashita), 折田　将成[1](Masanari Orita),
杉山　千絵[1](Chie Sugiyama), Stephen Moss[2], 荻田喜代一[1](Kiyokazu Ogita)
[1]摂南大・薬・薬理, [2]ペンシルバニア大学

## Oral Session 2P-K　14:00-15:00　Room K

### 方法 Methodology

Chairperson　平井　宏和 (Hirokazu Hirai) (群馬大学大学院医学系研究科神経生理学分野)

O2P-K01
14:00
**8型 AAV ベクターによる乏突起細胞へ高効率遺伝子導入**
**Efficient transduction of oligodendrocytes with AAV8 vectors**
浅利さやか[1](Sayaka Asari), 西田　紘子[1](Hiroko Nishida), 奈良　優子[1](Yuko Nara),
滝野　直美[1](Naomi Takino), 古寺　美加[1](Mika Kodera), Wei-Zhong Xiao[1,2], 佐々木康郎[3,4](Yasuo Sasaki),
菊地　哲[3,4](Satoru Kikuchi), 松下　卓[2](Takashi Matsushita), 岡田　尚巳[2](Takashi Okada),
星　美奈子[3,4](Minako Hoshi), 小澤　敬也[2](Keiya Ozawa), 中野　今治[1](Imaharu Nakano),
村松　慎一[1](Shin-ichi Muramatsu)
[1]自治医科大学, [2]自治医大遺伝子治療部, [3]三菱生命科学研究所, [4]東京工業大学

O2P-K02
14:15
**レンチウイルスベクターを用いた遺伝子レスキュー　－ CD38 遺伝子の脳部位特異的発現による機能回復－**
**Lentiviral-vector-mediated gene rescue -Functional recovery by region-specific expression of CD38 gene-**
平井　宏和[1,2,3](Hirokazu Hirai), 寅嶋　崇[1,3,5](Takashi Torashima), Duo Jin[3,4], Hong-Xiang Liu[3,4],
Olga Lopatina[3,4], 野田　百美[6](Mami Noda), 東田　博陽[3,4](Haruhiro Higashida)
[1]群馬大院・医・神経生理, [2]科学技術振興機構　SORST, [3]金沢大２１世紀COE・革新脳科学, [4]金沢大院・医・脳細胞遺伝子, [5]金沢大院・医・シナプス発達機能学, [6]九州大院・薬・生命薬学

O2P-K03 14:30
レンチウィルスを用いた、樹状突起膜特異的標識法の開発
Development of dendritic membrane-targeting signals using lentiviral vectors
亀田 浩司[1](Hiroshi Kameda), 日置 寛之[1](Hiroyuki Hioki), 古田 貴寛[1](Takahiro Furuta),
大平 耕司[1](Koji Ohira), 松田 和郎[1](Wakoto Matsuda), 中村 公一[1,2](Kouichi Nakamura),
金子 武嗣[1,2](Takeshi Kaneko)
[1]京都大院・医・高次脳形態, [2]科学技術振興機構 戦略的創造研究推進事業

O2P-K04 14:45
神経細胞活動性履歴の標識と反屈束の機能的な非対称性
Labeling history of neuronal activity and functional asymmetry of fasciculus retroflexus
一條 裕之[1](Hiroyuki Ichijo), 永井 健治[2](Takeharu Nagai), 濱田 理人[1](Michito Hamada),
小林麻己人[3](Makoto Kobayashi), 高橋 智[1](Satoru Takahashi)
[1]筑波大学・大学院人間総合科学研究科・基礎医学系・解剖発生, [2]北海道大学電子科学研究所, [3]筑波大学先端学際領域研究センター

**Oral Session 2P-K** 15:00-16:00 Room K

## 感覚運動系の可塑性
## Sensori-Motor Plasticity

Chairperson 岡ノ谷一夫 (Kazuo Okanoya) (理化学研究所生物言語研究チーム)

O2P-K05 15:00
ジュウシマツ cDNA マイクロアレイを用いた歌制御神経核特異的な遺伝子の同定
Isolation of Song nuclei specific genes by using cDNA microarray in Bengalese finch
加藤 真樹(Masaki Kato), 岡ノ谷一夫(Kazuo Okanoya)
理研・BSI・生物言語

O2P-K06 15:15
ジュウシマツの視床皮質ループを構成する歌制御神経核間における選択的聴覚応答の発火時期の相関
Temporal relations of auditory selectivity among song-related thalamo-cortical brain loop nuclei in Bengalese finches
関 義正[1,2](Yoshimasa Seki), 岡ノ谷一夫[2](Kazuo Okanoya)
[1]千葉大院・自然科学, [2]理化学研究所 脳センター 生物言語研究チーム

O2P-K07 15:30
Spatial temporal credit assignment neural network model
Adam P.D. Ponzi
DEI Lab, RIKEN BSI, Saitama, Japan.

O2P-K08 15:45
Can theta burst transcranial magnetic stimulation affect cortico-muscular synchronization in humans?
Murat Saglam[1], Hayato Yanagida[1], Kaoru Matsunaga[2], Nobuki Murayama[1], Yuki Hayashida[1], Ryoji Nakanishi[2]
[1]Dept of Graduate School of Science and Technology, Kumamoto University, Japan, [2]Dept. of Neurology, Kumamoto Kinoh Hospital,Japan

**Oral Session 2P-K** 16:00-17:00 Room K

## ブレインマシン / コンピュータ・インターフェイス
## BMI/BCI

Chairperson 柴田 智広 (Tomohiro Shibata) (奈良先端科学技術大学院大学情報科学研究科)

O2P-K09 16:00
Dynamic online target control of SSVEP-based brain-computer interface with multiple commands
Hovagim Bakardjian, Pablo Martinez, Andrzej Cichocki
Laboratory for Advanced Brain Signal Processing, BSI, RIKEN

O2P-K10 16:15
単一試行活動による意思決定の予測 ～即時および遅延 go/no-go 課題を用いて～
Single-trial based neural prediction of immediate and delayed go/no-go decisions.
長谷川良平[1,2](Ryohei_P Hasegawa), 長谷川由香子[1,2](Yukako_T Hasegawa), Mark_A Segraves[2]
[1]産業技術総合研究所・脳神経情報研究部門, [2]ノースウェスタン大学・神経生物学生理学部門

O2P-K11 ユーザーの筋電信号を利用するロボットとの動的・協調的インタラクション
16:30 Virtual force/tactile sensos for dynamic and cooperative interaction with a robot using user's biological signals
為井 智也(Tomoya Tamei), 石井 信(Shin Ishii), 柴田 智広(Tomohiro Shibata)
奈良先端科学技術大学院大学・情報科学研究科

O2P-K12 海馬位相コードに基づく認知地図生成：実空間神経ロボティクスによる考察
16:45 A neuro-robotics study in the real world: Formation of the cognitive map by using theta phase coding
我妻 広明(Hiroaki Wagatsuma), 池田 達也(Tatsuya Ikeda), 山口 陽子(Yoko Yamaguchi)
理研・脳センター・創発知能ダイナミクス

**Oral Session 2P-K** 17:00-17:45 Room K

**学際領域 (J)**
**Interdisciplinary Areas (J)** This session is held in Japanese

Chairperson 篠本 滋 (Shigeru Shinomoto) (京都大学理学研究科物理学教室)

O2P-K13 クロマフィン細胞における分泌顆粒動態の４次元解析
17:00 4-D imaging of secretory granule movements in chromaffin cells
林 光紀[1](Mitsunori Hayashi), 関口 瑞季[1,2](Mizuki Sekiguchi), 松田 冬香[1](Fuyuka Matsuda),
笹川 展幸[1](Nobuyuki Sasakawa), 熊倉鴻之助[1](Konosuke Kumakura)
[1]上智大・生命研, [2]東京大・医科研

O2P-K14 線虫*C. elegans* の food-NaCl 連合学習の神経応答シミュレーション
17:15 Simulation study: Changes in the neural network of *C. elegans* induced by food-NaCl associative learning
鈴木 芳代[1,2](Michiyo Suzuki), 坂下 哲哉[1](Tetsuya Sakashita), 小林 泰彦[1](Yasuhiko Kobayashi),
辻 敏夫[3](Toshio Tsuji)
[1]日本原子力研究開発機構・量子ビーム応用・バイオ応用技術, [2]日本学術振興会, [3]広島大学・院工・複雑システム工学

O2P-K15 演題取下げ
Withdrawn

O2P-K16 神経発火における不規則性の時間変化についてのベイズ解析
17:30 A Bayesian analysis of the time dependent irregularity of neuronal firing
下川 丈明(Takeaki Shimokawa), 篠本 滋(Shigeru Shinomoto)
京都大院・理

# ポスター発表 Poster Session 大会第1日目 9月10日(月) Day 1 September 10 (Mon)

Core Time Odd numbers : 11:00 ～ 12:00
Even numbers : 13:00 ～ 14:00

## シナプス 1
## Synapse 1

P1-a01 **Effects univibrissae rearing on response properties of barrel cortical neurons in C-Fiber-depleted rats**
s. Azizollahi[1], A. Shamsizade[1,2], V. Shey Bani[1], S. Arabzadeh[1], M. Afarinesh[1]
[1]Neuroscience Research Center, Kerman Univ of Med Sci, Iran, [2]Dept of Physi, Rafsanjan Univ of Med Sci, Iran

P1-a02 **Low concentration of nicotine triggers a novel regulatory pathway in neuromuscular synapses**
Vasiliy V. Fedorin, Olga P. Balezina
Department of Physiology, Moscow State University, Moscow, Russia

P1-a03 **活動電位による calyx of Held シナプス前末端 Ca2+ 上昇の性質**
**Single action potential-evoked $Ca^{2+}$ transients at the calyx of Held presynaptic terminal**
中村 行宏[1](Yukihiro Nakamura), David DiGregorio[2], 高橋 智幸[1,3](Tomoyuki Takahashi)
[1]同志社大・生命医科学, [2]パリ第5大学, [3]沖縄科学技術大学院大学

P1-a04 **Regulation of the differentiation of excitatory synapses by the SALM family of cell adhesion-like molecules**
Jaewon Ko[1], Seho Kim[1], Hye Sun Chung[2], Hyun Kim[2], Eunjoon Kim[1]
[1]Department of Biological Sciences, KAIST, Daejeon, South Korea, [2]Department of Anatomy, College of Medicine, Korea University, 126-1, 5-Ka, Seoul 136-705, Korea

P1-a05 **シナプス可塑性関連プロテアーゼニューロプシンによる 120kDa タンパク質のプロセシング**
**A 120 kDa protein localized in the synaptic membrane is cleaved by plasticity-related protease neuropsin**
松井 信樹[1](Nobuki Matsui), 田村 英紀[1](Hideki Tamura), 伊藤 康一[2](Kouichi Itoh),
塩坂 貞夫[1](Sadao Shiosaka)
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P1-a06 **カドヘリン8欠損による脊髄膠様質への侵害性機械および冷覚シナプス入力の減弱**
**Impairment of afferent synaptic inputs for mechanical and cold sensation to substantia gelatinosa neurons of the rat spinal dorsal horn through cadherin-8 expressing synaptic glomeruli**
古江 秀昌[1](Hidemasa Furue), 鈴木 祥宏[2](Sachihiro_SC Suzuki), 古賀 浩平[1](Kohei Koga),
能見 光雄[3](Mitsuo Nohmi), 竹市 雅俊[2](Masatoshi Takeichi), 吉村 恵[1](Megumu Yoshimura)
[1]九州大院・医・統合生理, [2]理研CDB, [3]佐大・総合分析

P1-a07 **単一シナプスにおけるグルタミン酸放出の可視化解析**
**Optical imaging of glutamate release at individual hippocampal synapses**
坂本 寛和(Hirokazu Sakamoto), 並木 繁行(Shigeyuki Namiki), 飯沼 将(Sho Iinuma),
廣瀬 謙造(Kenzo Hirose)
名古屋大院・医・細胞生理

P1-a08 **Cbln ファミリー分子の生化学的解析**
**Characterization of a transneuronal cytokine family Cbln**
飯島 崇利[1](Takatoshi Iijima), 三浦会理子[2](Eriko Miura), 松田 恵子[1](Keiko Matsuda),
亀川 雄一[1](Yuichi Kamekawa), 渡辺 雅彦[2](Masahiko Watanabe), 柚崎 通介[1](Michisuke Yuzaki)
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P1-a09 ラット海馬スライスおける興奮性シナプス伝達は灌流速度により変化する
Change in superfusion rate alters excitatory synaptic transmission in rat hippocampal slice
木村 眞吾(Shingo Kimura), 川崎 敏(Satoshi Kawasaki), 渡辺 修二(Shuji Watanabe),
藤田 玲子(Reiko Fujita), 佐々木和彦(Kazuhiko Sasaki)
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P1-a10 ホスホリパーゼ $A_2$ 活性化によるラット脊髄膠様質ニューロンのグリシンと GABA 作動性抑制性シナプス伝達の促進
Enhancement by phospholipase $A_2$ activation of glycinergic and GABAergic inhibitory transmission in rat substantia gelatinosa neurons
柳 涛(Tao Liu), 藤田 亜美(Tsugumi Fujita), 岳 海源(Hai-Yuan Yue), 水田恒太郎(Kotaro Mizuta),
朴 蓮花(Lian-Hua Piao), 中塚 映政(Terumasa Nakatsuka), 熊本 栄一(Eiichi Kumamoto)
佐賀大・医・生体構造機能学

P1-a11 Cbln1 はプルキンエ細胞への興奮性シナプス前終末形成を誘導する
Cbln1 directly induces excitatory presynaptic assembly in cerebellar Purkinje cells
石田 綾[1](Aya Ishida), 松田 恵子[1](Keiko Matsuda), 近藤 哲郎[1,2](Tetsuro Kondo),
飯島 崇利[1](Takatoshi Iijima), 柚崎 通介[1](Michisuke Yuzaki)
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P1-a12 シナプス前末端内の $Ca^{2+}$ 除去機構：ミトコンドリアへの $Ca^{2+}$ 取り込みは末端内 $Ca^{2+}$ イオン濃度がある臨界レベル以上で生ずる
$Ca^{2+}$ clearance mechanisms in presynaptic terminals : $Ca^{2+}$ uptake to mitochondria started when $[Ca^{2+}]_i$ increased to a critical level
鈴木 直哉(Naoya Suzuki), 茂藤 優司(Yuji Motou)
名大院・理・物理

P1-a13 ラット大脳皮質における2層・5層錐体細胞へのパルバルブミン陽性抑制性入力の分布
Differential distribution of parvalbumin positive boutons for two populations of pyramidal cells
松下 敦子(A. Matsushita), 一戸 紀孝(N. Ichinohe), A. Knight, K. S. Rockland
理化学研究所・脳科学総合研究センター

P1-a14 Proteinase-activated receptor-1 活性化による成熟ラット脊髄膠様質のグルタミン酸作動性興奮性シナプス伝達促進
Enhancement by PAR-1 activation of glutamatergic excitatory transmission in adult rat substantia gelatinosa neurons
藤田 亜美(Tsugumi Fujita), 柳 涛(Tao Liu), 中塚 映政(Terumasa Nakatsuka),
熊本 栄一(Eiichi Kumamoto)
佐賀大・医・生体構造機能学

P1-a15 大脳皮質非錐体細胞の樹状突起特性の解析
Dendritic dimensions of cortical nonpyramidal cells
窪田 芳之[1](Yoshiyuki Kubota), 苅部 冬紀[1](Fuyuki Karube), 野村 真樹[3](Masaki Nomura),
青柳富誌生[3](Toshio Aoyagi), 望月 敦史[2](Atsushi Mochizuki), 川口 泰雄[1](Yasuo Kawaguchi)
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P1-a16 大脳皮質バレル野及び島皮質におけるカラム間コミュニケーションへの $GABA_A$ IPSP の関与
Differential involvement of $GABA_A$ IPSPs in inter-columnar communication in the barrel and insular cortices
齋藤 充(Mitsuru Saito), 佐藤 元(Hajime Sato), 豊田 博紀(Hiroki Toyoda),
姜 英男(Youngnam Kang)
大阪大院・歯・高次脳口腔機能

P1-b01 大脳皮質回路における発火パタンに依存した結合特異性
Firing pattern dependent specificity of cortical feed-forward subnetworks
大塚 岳(Takeshi Otsuka), 森島美絵子(Mieko Morishima), 川口 泰雄(Yasuo Kawaguchi)
生理学研究所大脳神経回路論部門

P1-b02 黒質網様部から上丘中間層 GABA 作動性ニューロンへの抑制性入力
Evidence of nigral inhibitory inputs to GABAergic neurons in mouse superior colliculus
伊佐かおる[1,2](Kaoru Isa)，金田　勝幸[1,3](Katsuyuki Kaneda)，柳川右千夫[4](Yuchio Yanagawa)，
小幡　邦彦[5](Kunihiko Obata)，伊佐　　正[1,2,3](Tadashi Isa)
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P1-b03 $\alpha_2$-アドレナリン受容体を介する小脳 GABA 作動性シナプス伝達抑制の生後発達
$\alpha_2$-Adrenoceptor activation reduces GABAergic synaptic transmission in the developing mouse cerebellar cortex
廣野　守俊(Moritoshi Hirono)，松永　　渉(Wataru Matsunaga)，小幡　邦彦(Kunihiko Obata)
理化学研究所・脳センター・神経回路メカニズム研究グループ

P1-b04 シナプス形成過程における神経支配依存的な遺伝子発現解析
Analysis of innervation-dependent gene expression during synaptogenesis
福井　　愛[1](Ai Fukui)，稲木美紀子[1](Mikiko Inaki)，油谷　浩幸[3](Hiroyuki Aburatani)，
能瀬　聡直[1,2](Akinao Nose)
[1]東京大院・理・物理，[2]東京大院・新領域・複雑理工，[3]東京大・先端研

P1-b05 SAD キナーゼによるアクティブゾーン特異蛋白質 Bassoon および Piccolo のリン酸化。
SAD kinase directly phosphorylates the active zone proteins Bassoon and Piccolo.
Susumu Higa，所　　崇(Takashi Tokoro)，北島　　勲(Isao Kitajima)，大塚　稔久(Toshihisa Ohtsuka)
富山大院・医

P1-b06 ComplexinII と Parkin との相互作用
Interaction of complexin II with parkin
兼田　　悠[1](Hisashi Kaneda)，高橋　正身[2](Masami Takahashi)，片岡　正和[1](Masakazu Kataoka)
[1]信州大院・工・環境機能，[2]北里大・医

P1-b07 大脳皮質非錐体細胞への興奮性・抑制性シナプス入力比
An excitatory and inhibitory synapse ratio on cortical nonpyramidal cell subtypes of the rat
関川　明生(Akio Sekigawa)，窪田　芳之(Yoshiyuki Kubota)，畑田さゆり(Sayuri Hatada)，
川口　泰雄(Yasuo Kawaguchi)
自然科学研究機構・生理研・大脳神経回路論

P1-b08 培養海馬神経細胞の個々のシナプスにおける神経伝達物質放出確率と GluR2 の発現量の解析
Presynaptic release probability and GluR2 abundance at individual synapses in cultured hippocampal neurons
徳岡　宏文[1](Hirofumi Tokuoka)，合田裕紀子[1,2](Yukiko Goda)
[1]英国医学研究所・細胞生物学部門，[2]ユニバーシティ・カレッジ・ロンドン

## 神経伝達物質、修飾物質 1
## Neurotransmitters and Modulators 1

P1-b09 Age-related changes in ionized calcium-binding adapter molecule 1 in the gerbil hippocampal CA1 region
Jung Hoon Choi[1], Choong Hyun Lee[1], Dae Won Chung[1], In Ku Hwang[1], Moo Ho Won[2], Yeo Sung Yoon[1], Heungshik S Lee[1], In Se Lee[1]
[1]Department of Anatomy and Cell Biology, College of Veterinary Medicine, Seoul National University, [2]Department of Anatomy and Neurobiology, College of Medicine, Hallym University

P1-b10 Choline acetyltransferase of a peripheral type (pChAT) synthesizes acetylcholine in adult rat dorsal root ganglion
Jean-Pierre Bellier, Hiroshi Kimura
Molecular Neuroscience Research Center, Shiga University of Medical Science, Otsu, Japan

P1-b11 Nanoparticle induced neurotoxicity in the mice olfactory bulb: analysis of neurotransmitter and proinflammatory cytokine mRNA levels

Tin-Tin Win-Shwe[1], Dai Mitsushima[2], Shoji Yamamoto[1], Atsushi Fukushima[1], Hidekazu Fujimaki[1]
[1]Research Center for Environmental Risk, National Institute for Environmental Studies, Tsukuba, Japan, [2]Department of Physiology, Yokohama City University Graduate School of Medicine, 3-9 Fukuura, Kanazawa, Yokohama

P1-b12 ラット海馬ニューロステロイド合成に対するレチノイン酸の影響
Effect of retinoic acid on neurosteroidogenesis in cultured rat hippocampus

宗綱　栄二[1](Eiji Munetsuna), 川戸　佳[2](Suguru Kawato), 小南　思郎[1](Shiro Kominami),
山崎　岳[1](Takeshi Yamazaki)
[1]広島大院・総合科学研究科, [2]東京大院・総合文化・広域科学

P1-b13 DOPS 芳香族誘導体は DOPA 応答を惹起しうる
Aromatic derivatives of DOPS could induce DOPAergic responses

室田　裕大[1](Yasuhiro Murota), 矢上　達郎[1,3](Taturou Yagami), 青木　令奈[1](Reina Aoki),
梶原　康弘[2](Yasuhiro Kajihara), 山本　直毅[2](Naoki Yamamoto), 五嶋　良郎[1](Yoshio Goshima)
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P1-b14 発達期小脳からのグルタミン酸および GABA の放出を連続観察する
Spatiotemporal observation of glutamate and GABA release from developing cerebellar cortex

平野翔一郎[1](Shoichiro Hirano), 大河内健吾[1](Kengo Okochi), 井上　尚久[1](Naohisa Inoue),
穂積　直裕[2](Naohiro Hozumi), 吉田　祥子[1](Sachiko Yoshida)
[1]豊橋技術科学大学・物質工学系, [2]愛知工業大学

P1-b15 線条体における２－アラキドノイルグリセロールを介した逆行性シグナル伝達に関与する分子の細胞内配置
Subcellular arrangement of molecules for 2-arachidonoyl-glycerol-mediated retrograde signaling in the striatum

内ヶ島基政[1](Motokazu Uchigashima), 鳴島　円[2](Madoka Narushima), 深谷　昌弘[1](Masahiro Fukaya),
Istvan Katona[3], 狩野　方伸[2](Masanobu Kano), 渡辺　雅彦[1](Masahiko Watanabe)
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P1-b16 酵素担持と微弱光励起による新しいグルタミン酸測定デバイスの開発
New system for released glutamate detection using the covalent immobilization of glutamate dehydrogenase and evanescent light

井上　尚久[1](Naohisa Inoue), 大河内健吾[1](Kengo Okochi), 平野翔一郎[1](Shoichiro Hirano),
穂積　直裕[2](Naohiro Hozumi), 吉田　祥子[1](Sachiko Yoshida)
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P1-b17 ラット培養神経細胞におけるマグネシウムの影響
Role of magnesium in neural process of rat cultured neurons

古川由里子[1,2](Yuriko Furukawa), 島田　明佳[1,2](Akiyoshi Shimada), 河西奈保子[1,2](Nahoko Kasai),
鳥光　慶一[1,2](Keiichi Torimitsu)
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P1-b18 小脳外顆粒層での GABA 放出と一過性の VGAT 分布
Non-neuronal GABA release in external granular layer and transient VGAT distribution

大河内健吾[1](Kengo Okochi), 田中真理子[1](Mariko Tanaka), 平野翔一郎[1](Shoichiro Hirano),
森島　寿貴[1](Toshitaka Morishima), 川口　泰雄[2](Yasuo Kawaguchi), 柳川右千夫[3](Yuchio Yanagawa),
穂積　直裕[4](Naohiro Hozumi), 吉田　祥子[1](Sachiko Yoshida)
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P1-b19 海馬神経細胞シナプスにおけるステロイドの合成と局所分泌
Steroid synthesis and synaptocrinology in the hippocampal synapses
北條　泰嗣[1,2](Yasushi Hojo)，肥後　心平[1,2](Shimpei Higo)，向井　秀夫[1,2](Hideo Mukai)，John H. Morrison[3]，William G. M Janssen[3]，小南　思郎[4](Shiro Kominami)，原田　信広[5](Nobuhiro Harada)，
木本　哲也[1,2](Tetsuya Kimoto)，川戸　　佳[1,2](Suguru Kawato)
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P1-b20 ラット中脳縫線核におけるグルタミン酸作動性ニューロンの解析
Characterization of glutamatergic neurons in the rat midbrain raphe nuclei
日置　寛之[1](Hiroyuki Hioki)，中村　　悠[1](Hisashi Nakamura)，馬　　雲飛[1](Yun-Fei Ma)，
中村　公一[1,2](Kouichi Nakamura)，早川　　隆[1](Takashi Hayakawa)，金子　武嗣[1,2](Takeshi Kaneko)
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P1-b21 GPR7(NPBW1) 受容体選択的な新規 Neuropeptide B (NPB) ペプチドアナログの解析
Development of a potent and selective GPR7 agonist: a systematic structure-activity study of neuropeptide B
金坂　真生(Maki Kanesaka)，松田　匡雄(Masao Matsuda)，平野　　篤(Atsushi Hirano)，
田中　健一(Kenichi Tanaka)，金谷　章生(Akio Kanatani)，鴇田　　滋(Shigeru Tokita)
万有製薬つくば研究所代謝性疾患領域研究部

P1-b22 Relaxin3 遺伝子の発現調節解析
Regulatory mechanism of relaxin3 gene expression
渡邊　義久[1](Yoshihisa Watanabe)，吉本　寛司[2](Kanji Yoshimoto)，田中　雅樹[1](Masaki Tanaka)
[1]京都府立医科大・附属脳・血管系老化研究センター・細胞生物学，[2]京都府立医科大・医・法医学

P1-b23 セリンラセマーゼノックアウトマウスを用いた脳内 D- セリンの機能解析
Study of D-serine function in vivo by establishing and analysis of serine racemase knockout mouse
趙　英らん[1](Ying-Luan Zhao)，高田　義美[1](Yoshimi Takata)，橋本　謙二[2](Kenji Hashimoto)，
崎村　建司[3](Kenji Sakimura)，森　　寿[1](Hisashi Mori)
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P1-b24 発達期大脳皮質における細胞外タウリンの $GABA_A$ 受容体を介する役割
The role of $GABA_A$ receptor-mediated actions of taurine in developing cerebral cortex
古川　智範[1](T Furukawa)，山田　順子[3](J Yamada)，井上　浩一[1](K Inoue)，松島　芳隆[2](Y Matsushima)，
柳川右千夫[4](Y Yanagawa)，福田　敦夫[1,3](A Fukuda)
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P1-b25 マウス脳での SNAP-25 family タンパク質の局在の解析
Differential expression of SNAP-25 family proteins in mouse brain
山森　早織(Saori Yamamori)，板倉　　誠(Makoto Itakura)，菅谷　大地(Daichi Sugaya)，
高橋　正身(Masami Takahashi)
北里大・医・生化学

P1-b26 アストロサイトにおけるアシル -CoA 合成酵素 lipidosin の発現と髄鞘再生時の発現増大
Expression of an acyl-CoA synthetase, lipidosin, in astrocytes and its up-regulation during experimental remyelination.
宋　　時栄[1](しょん そん)，加藤千恵子[1](Chieko Kato)，安達栄治郎[2](Eijiro Adachi)，
橋本　有弘[3](Naohiro Hashimoto)
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## 受容体、輸送体 1
## Receptors and Transporters 1

P1-b27 **X 線照射による胎生期マウス大脳皮質におけるグルタミン酸作動性ニューロンの異常**
**Aberration of glutamatergic neurons in X-ray irradiated mouse embryonic cortex**
呉　てぃん[1](Ting Wu)，伊奈　鮎香[1](Ayuka Ina)，吉田さちね[1](Sachine Yoshida)，今野　仁子[1](Jinko Konno)，
首藤　文洋[1](Fumihiro Shutoh)，孫　　学智[2](Xue-Zhi Sun)，久野　節二[1](Setsuji Hisano)
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P1-b28 **ラット脳におけるシグマ -1 受容体発現の生後成長変化**
**Sigma-1 receptor expression in rat brain during postnatal development**
榎屋　友幸[1](Tomoyuki Enokiya)，山本　秀子[2](Hideko Yamamoto)，山本　敏文[1,2](Toshifumi Yamamoto)
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P1-b29 **マウス皮質発生期におけるカハール・レチウス細胞での小胞性グルタミン酸輸送体２の発現**
**Cajal-Retzius cells express vesicular glutamate transporter 2 (VGLUT2) during the mouse corticogenesis**
伊奈　鮎香(Ayuka Ina)，今野　仁子(Jinko Konno)，吉田さちね(Sachine Yoshida)，
首藤　文洋(Fumihiro Shutoh)，久野　節二(Setsuji Hisano)
筑波大学院・人間総合科学・感性認知脳科学・システム脳科学

P1-b30 **神経系に発現するオーファン G タンパク質共役型受容体の解析**
**Localization and characterization of an orphan G protein-coupled receptor expressed in the nervous system**
植田　高史(Takashi Ueda)，鵜川　眞也(Shinya Ugawa)，石田　雄介(Yusuke Ishida)，
島田　昌一(Shoichi Shimada)
名古屋市大院・医・分子形態

P1-b31 **$M_1$ ムスカリン性アセチルコリン受容体は終脳の投射ニューロンに豊富に発現している**
**$M_1$ subtype of muscarinic acetylcholine receptor is preferentially expressed in the principal neurons of the mouse telencephalon**
山崎美和子(Miwako Yamasaki)，飯塚　　尭(Yutaka Iizuka)，渡辺　雅彦(Masahiko Watanabe)
北海道大院・医・解剖発生学

P1-b32 **細胞内 pH を調整する IP3 受容体結合タンパクの新規機能解明**
**IP3 receptor binding protein, IRBIT, specifically binds to and activates sodium bicarbonate cotransporter 1**
藤本　一朗[1](Ichiro Fujimoto)，白壁　恭子[1](Kyoko Shirakabe)，Giuseppina Priori[1]，
山田　秀臣[2](Hideomi Yamada)，安東　英明[3](Hideaki Ando)，水谷　顕洋[3](Akihiro Mizutani)，
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P1-b33 **摂食受容体 MCH1R における膜貫通部位プロリンの解析**
**The critical role of transmembrane prolines in melanin-concentrating hormone receptor**
斎藤祐見子[1](Yumiko Saito)，津留崎幸子[1](Sachiko Tsurusaki)，相崎　良美[2,3](Yoshimi Aizaki)，
中野　満恵[3](Mitsue Nakano)，松原真由美[1](Mayumi Matsubara)
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P1-b34 **マウス大脳皮質体性感覚野におけるＫＣＣ２局在と GABA 回路網の生後発達変化**
**Postnatal development of the KCC2-localization and GABA network in the mouse somatosensory cortex**
高山　千利(Chitoshi Takayama)
北海道大学・医・分子解剖

P1-b35 脊髄ミクログリアにおける $P2X_7$ 受容体活性化を介したグルタミン酸トランスポーター機能の制御機構
Involvement of $P2X_7$ receptor in the regulation of glutamate transporters in cultured spinal microglia
森岡　徳光[1](Norimitsu Morioka)，北山　友也[1](Tomoya Kitayama)，森田　克也[1](Katsuya Morita)，
仲田　義啓[2](Yoshihiro Nakata)，土肥　敏博[1](Toshihiro Dohi)
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P1-b36 選択的セロトニン再取り込み阻害薬の小胞性モノアミントランスポーターに対する阻害作用
Inhibition of selective serotonin reuptake inhibitors on vesicular monoamine transporter 2
田村　浩平[1,3](Kohei Tamura)，巽　　国子[1,2](Kuniko Tatsumi)，安本　　智[1,2](Satoshi Yasumoto)，
山本　敏文[1,2](Toshifumi Yamamoto)，池田　和隆[1](Kazutaka Ikeda)，山本　秀子[1](Hideko Yamamoto)
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P1-b37 グルタミン酸受容体デルター2とその足場タンパク質デルフィリンの結合の PKA による調節
PKA may regulate the binding of GluR$\delta$2 to its scaffolding protein, Delphilin
園田　智子[1](Tomoko Sonoda)，望月千恵子[1](Chieko Mochizuki)，山下　哲司[1](Tetsuji Yamashita)，
兼子　佳子[1](Keiko Kaneko)，宮城　洋平[2](Yohei Miyagi)，茂里　　康[3](Yasushi Shigeri)，
矢間　　太[4](Futoshi Yazama)，奥田　研爾[1](Kenji Okuda)，川本　　進[1,5](Susumu Kawamoto)
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P1-b38 哺乳類松果体細胞における神経性制御の基盤：ニコチン性 ACh 受容体の発達と特性
Molecular basis of the autonomic regulation of mammalian pinealocytes with focus on nicotinic receptors
河　　和善(Kazuyoshi Kawa)
東北大院・医・生体情報学

P1-b39 脈絡叢上皮細胞における Notch2 の微絨毛表面への非対称性局在
Asymmetric localization of Notch2 on the microvillous surface in choroid plexus epithelial cells
田中　正彦[1,2](Masahiko Tanaka)，小久保正博[3](Masahiro Kokubo)，丸野内　棣[2](Tohru Marunouchi)
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P1-b40 神経シナプスに存在する PDZ ドメインを含むタンパク質、Delphilin はモノカルボン酸トランスポーター MCT2 と結合する
Delphilin, the PDZ domain-containing protein, interacts with the monocarboxylate transporter 2
兼子　佳子[1](Keiko Kaneko)，園田　智子[1](Tomoko Sonoda)，宮城　洋平[2](Yohei Miyagi)，
山下　哲司[1](Tetsuji Yamashita)，奥田　研爾[1](Kenji Okuda)，川本　　進[1,3](Susumu Kawamoto)
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P1-b41 無刺激状態マウス培養アストロサイトにおけるアラキドン酸及びその代謝物を介した P2X7 受容体活性制御
Regulatory mechanism for P2X7 receptor activation by arachidonic acid and/or it's metabolites in mouse cultured astrocytes under resting conditions
宮木　　純(Jun Miyaki)，長澤　一樹(Kazuki Nagasawa)，藤本　貞毅(Sadaki Fujimoto)
京都薬大・衛生化学

## グリア、グリア-ニューロン相互作用 1
## Glia and Glia-Neuron Interaction 1

P1-b42 アストロサイト上のリガンドは神経細胞の PKC の活性化を通じて興奮性シナプスの形成を促進する
A cell surface ligand of astrocytes promotes excitatory synapse-formation through PKC-activation in neurons
濱　　裕(Hiroshi Hama)，安藤　亮子(Ryoko Ando)，宮脇　敦史(Atsushi Miyawaki)
独立行政法人理科学研究所・脳科学総合研究センター・細胞機能探索技術開発チーム

P1-b43 ラットアストロサイトにおけるコリン取り込みの機能的特徴
Functional characterization of choline transport in rat astrocyte
田島　裕久(Hirohisa Tajima)，稲津　正人(Masato Inazu)，山田　朋子(Tomoko Yamada)，
松宮　輝彦(Teruhiko Matsumiya)
東京医大・医・薬理学

P1-b44 脳弓下器官のグリア細胞に発現する $Na_x$ チャンネルは、体液ナトリウムイオンレベルの上昇を感知し、乳酸を介して神経細胞への情報伝達を制御する。
Glial $Na_x$ channels control lactate signaling to neurons for brain [$Na^+$] sensing
清水　秀忠[1](Hidetada Shimizu)，檜山　武史[1,3](Takeshi Y. Hiyama)，長倉　彩乃[1,3](Ayano Nagakura)，
藤川　顕寛[1,3](Akihiro Fujikawa)，渡辺　英治[2,3](Eiji Watanabe)，野田　昌晴[1,3](Masaharu Noda)
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P1-b45 初代培養アストログリア細胞のグルタミン合成酵素に与える Bisphenol A の影響
Effect of bisphenol A on the protein level of glutamine synthetase in primary-cultured astroglial cells
金松　知幸[1](Tomoyuki Kanamatsu)，中嶋　一行[2](Kazuyuki Nakajima)，小山　晶子[1](Akiko Koyama)，
川戸　雪絵[1](Yukie Kawato)
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P1-b46 リゾレシチン誘導坐骨神経脱髄モデルマウスにおける AX2 の役割
Role of Annexin2 in lysolecithin-induced demyelination of the peripheral nervous system
片伯部哲也(Tetsuya Katakabe)，林　　明子(Akiko Hayashi)，馬場　広子(Hiroko Baba)
東京薬科大学・薬・機能形態

P1-b47 *Caspr1* 遺伝子変異マウスの有髄神経線維におけるパラノード構成タンパクの局在
Distribution of paranodal proteins in myelinated nerves from Shambling mice with a *Caspr1* mutation
高岸　芳子[1](Yoshiko Takagishi)，千島　由子[2](Yuko Chishima)，孫　　暁陽[1](Xiaoyang Sun)，
織田　銑一[2](Sen-ichi Oda)，村田　善晴[1](Yoshiharu Murata)
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P1-b48 逆行性神経回路標識によるマウス脳弓下器官の神経細胞の解析
Characterization of neurons in the mouse subfornical organ by retrograde labeling
長倉　彩乃[1,3](Ayano Nagakura)，檜山　武史[1,3](Takeshi Y. Hiyama)，渡辺　英治[2,3](Eiji Watanabe)，
野田　昌晴[1,3](Masaharu Noda)
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P1-c01 Amphotericin B によるミクログリアの細胞機能変化
Effect of Amphotericin B on cellular functions of cultured microglia
本吉　晃子[1](Akiko Motoyoshi)，中嶋　秀満[2](Hidemitsu Nakajima)，中村　洋一[1](Yoichi Nakamura)
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P1-c02 C6 グリオーマではホルボールエステルによって開口放出が抑制される
Phorbol 12-myristate 13-acetate suppresses $Ca^{2+}$-dependent peptide hormone release from clonal rat glioma C6 cells
安田 圭一(Keiichi Yasuda), 板倉 誠(Makoto Itakura), 菅谷津貴子(Tsukiko Sugaya),
山森 早織(Saori Yamamori), 高橋 正身(Masami Takahashi)
北里大学・医学部・生化学

P1-c03 麻酔下個体脳における単一アストロサイト ラベリング法
Intracellular labeling of single cortical astrocytes in vivo
三嶋 恒子(Tsuneko Mishima), 酒谷 誠一(Seiichi Sakatani), 平瀬 肇(Hajime Hirase)
理化学研究所BSI・平瀬ユニット

P1-c04 マクロファージ及びミクログリアによるサイトカイン産生における Iba1 の役割
Role of Iba1 in cytokine production in macrophages and microglia
前田詠里子[1](Eriko Maeda), 原田 佳奈[1](Kana Harada), 大澤 圭子[2](Keiko Ohsawa),
高坂 新一[2](Shinichi Kohsaka), 秀 和泉[1](Izumi Hide), 仲田 義啓[1](Yoshihiro Nakata)
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P1-c05 アストロサイトのセルアセンブリ
Astroglial cell assemblies
長谷川彩子(Ayako Hasegawa), 松木 則夫(Norio Matsuki), 池谷 裕二(Yuji Ikegaya)
東京大院・薬・薬品作用学

P1-c06 ZNRF1 によるユビキチン・プロテアソーム系を介した、グルタミン合成酵素の発現制御機構
ZNRF1 regulates glutamine synthetase protein expression in Schwann cells via a ubiquitin-proteasome pathway
齋藤 文典(Fuminori Saitoh), 荒木 敏之(Toshiyuki Araki)
国立 精神・神経センター 神経研究所

P1-c07 培養アストロサイトに発現するグルタミントランスポーターに対する Forskolin の増強作用
Forskolin up-regulates expression of mRNA for a glutamine transporter in cultured rat neocortical astrocytes
大西 知絵(Chie Ohnishi), 小椋 正人(Masato Ogura), 谷浦 秀夫(Hideo Taniura),
中道 範隆(Noritaka Nakamichi), 米田 幸雄(Yukio Yoneda)
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P1-c08 培養アストロサイトに発現するグルタミントランスポーターの cAMP シグナルによる機能増強
Promoted function by cAMP signals of a glutamine transporter expressed in cultured rat neocortical astrocytes
小椋 正人(Masato Ogura), 谷浦 秀夫(Hideo Taniura), 大西 知絵(Chie Ohnishi),
中道 範隆(Noritaka Nakamichi), 米田 幸雄(Yukio Yoneda)
金沢大院・自然科学(薬)・薬物学

P1-c09 細胞外 S100B によるカイニン酸誘導ガンマ振動の振幅上昇
Extracellular S100B increases the amplitude of kainate-induced gamma oscillation *in vivo*
酒谷 誠一[1](Seiichi Sakatani), 大島 章子[2](Akiko Seto-Ohshima), 糸原 重美[2](Shigeyoshi Itohara),
平瀬 肇[1](Hajime Hirase)
[1]理研・脳センター・平瀬ユニット, [2]理研・脳センター・行動遺伝

P1-c10 生後発達期ラット孤束核における神経活動依存的なアストロサイトの被覆
Activity-dependent glial coverage in the rat nucleus of tractus solitarius during postnatal development
太城 康良(Yasura Tashiro), 河合 良訓(Yoshinori Kawai)
東京慈恵会医科大・医・解剖第一

P1-c11 硫化水素のアストロサイトにおける感受性の、分化および reactive 化に伴う変化
The sensitivity of differentiated astrocytes to hydrogen sulfide is diminished in the reactive astrocytes
津金麻実子[1,2](Mamiko Tsugane)，永井　康雄[3](Yasuo Nagai)，木村　由佳[1](Yuka Kimura)，
岡　淳一郎[2](Jun-Ichiro Oka)，木村　英雄[1](Hideo Kimura)
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P1-c12 ApoE は LDL receptor-related protein 4 (LRP4) のリガンドである
Identification of apoE as a ligand for LDL receptor-related protein 4 (LRP4)
鈴木　龍雄(Tatsuo Suzuki)，ルー　永浩(Yonghao Lu)，田　　慶宝(Qing-Bao Tian)
信州大・院・医・神経可塑性

## 血液脳関門 Blood-Brain Barrier

P1-c13 血液脳関門における claudin subtype の mRNA 発現プロファイル
mRNA exparession profile of claudin subtypes in mouse blood-brain barrier
大槻　純男[1,2](Sumio Ohtsuki)，山口　啓史[1](Hirofumi Yamaguchi)，淺島　朋子[1,2](Tomoko Asashima)，
寺崎　哲也[1,2](Tetsuya Terasaki)
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P1-c14 BBB キット ( 血液脳関門 in vitro 再構成モデル )
BBB kit (Blood-Brain Barrier in vitro Reconstruction Model)
中川　慎介[1,5](Shinsuke Nakagawa)，Maria Deli[2,5]，中尾しのぶ[1](Shinobu Nakao-Nishioku)，
林　健太郎[3](Kentaro Hayashi)，中桶　了太[1](Ryota Nakoke)，片岡　泰文[4,5](Yasufumi Kataoka)，
丹羽　正美[1,5](Masami Niwa)
[1]長崎大院・医歯薬・神経薬理，[2]ハンガリーサイエンスアカデミー，[3]長崎大学病院・脳神経外科，[4]福岡大学・薬・薬学疾患管理学，[5]ファーマコセル株式会社

P1-c15 5-HTP 投与によって引き起こされる血中セロトニンの増加に対する脳内セロトニン増加の寄与
Contribution of increase in brain serotonin (5-HT) upon increase in blood 5-HT induced by 5-HTP administration in rat
中谷　康司(Yasushi Nakatani)，関　　由成(Yoshinari Seki)，菊地　宏美(Hiromi Kikuchi)，
上林　英里(Eri Kambayashi)，麓　　正樹(Masaki Fumoto)，鈴木　郁子(Ikuko Sato-Suzuki)，
有田　秀穂(*Hideho Arita*)
東邦大・医・統合生理

## 創薬 Drug Development

P1-c16 *in silico* ドラッグスクリーニングによる新規 UCH-L1 活性促進化合物の同定
Identification of novel UCH-L1-potentiating compounds by *in silico* drug screening
平山　和徳[1,2](Kazunori Hirayama)，青木　俊介[1](Shunsuke Aoki)，西川　香里[1](Kaori Nishikawa)，
松本　　隆[2](Takashi Matsumoto)，和田　圭司[1](Keiji Wada)
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P1-c17 人参養栄湯薬効成分，陳皮による老齢脳での脱髄回復について
A Japanese herbal medicine Ninjinyoueito ingredients, Aurantii nobilis pericarpium, rescue from aging-induced demyelination
佐藤菜名子[1](Nanako Sato)，清和　千佳[1](Chika Seiwa)，安川　明男[2](Akio Yasukawa)，
川喜田卓也[4](Takuya Kawakita)，工藤　千秋[5](Chiaki Kudo)，高尾　正克[6](Masakatsu Takao)，
小松　靖弘[3](Yasuhiro Komatsu)，阿相　皓晃[1](Hiroaki Asou)
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P1-c18 タランチュラから得られたカルシムチャネル毒の解析と応用
Calcium Channel Blocker Peptides Isolated from Tarantula Venom : identification, characterization and utilization
小野　世吾(Seigo Ono)，木村　忠史(Tadashi Kimura)，久保　泰(Tai Kubo)
産業技術総合研究所

P1-c19 Effects of N-(2-propylpentanoyl) urea on impairment of learning and memory induced by common carotid artery occlusion in mice
Oraphan Wanakhachornkrai, Boonyong Tantisira, Mayuree Tantisura H.
Interdepartment of Physiology, Chulalongkorn University

P1-c20 Minocycline attenuates neuronal cell death and improves cognitive impairment in Alzheimer's disease models
minji Kim, Yoori Choi, Hye-Sun Kim, Ki Young Shin, Eun-Mee Kim, Yoo-Hun Suh
Department pharmacology,College of Medicine, Seoul National University, Seoul, Korea

## 神経誘導、パターン形成、進化
## Neural Induction, Pattern Formation, Evolution

P1-c21 Critical role of SDF-1/CXCR4 in the development of boundary cap cells and CNS/PNS segregation
Yan Zhu[1], Tomoko Matsumoto[1], Sakae Mikami[2], Takashi Nagasawa[2], Fujio Murakami[1]
[1]Grad Sch of Frontier Biosci, Osaka Univ, [2]Inst for Frontier Med Sci, Kyoto Univ

P1-c22 中脳前後軸形成における beta2 Chimaerin の解析
Analysis of beta2 Chimaerin in rostrocaudal polarity formation of the optic tectum
鈴木－平野明日香[1](Asuka Suzuki-Hirano)，木村　淳[2](Jun Kimura)，仲村　春和[1](Harukazu Nakamura)
[1]東北大学院・生命・脳構築，[2]Cincinnati Children's Hospital Medical Center

P1-c23 フェレット外側膝状体のスライス培養を用いた神経入力非依存的な分化過程の解析
Developmental processes in the ferret LGN independent of input from retina and visual cortex
岩井　玲奈(Lena Iwai)，河崎　洋志(Hiroshi Kawasaki)
東京大院・医・神経機能解明ユニット

P1-c24 ゼブラフィッシュ胚発生における *Sufatase FP3* 遺伝子の機能解析
Functional analysis of the *Sufatase FP3* gene during zebrafish embryonic development
塩見　健輔(Kensuke Shiomi)，桑野　剛英(Takahide Kuwano)，桝　和子(Kazuko Keino-Masu)，
桝　正幸(Masayuki Masu)
筑波大・人間総合科学・分子神経

P1-c25 ゼブラフィッシュ中枢神経系発生における Strawberry notch ファミリー遺伝子の役割
The role of Strawberry notch family genes in the central nervous system development of zebrafish
高野　愛[1](Ai Takano)，蔵地　理代[1](Riyo Zochi)，日比　正彦[2](Masahiko Hibi)，
寺島　俊雄[1](Toshio Terashima)，勝山　裕[1](Yu Katsuyama)
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P1-c26 ゼブラフィッシュプルリン遺伝子のクローニングと発生初期網膜における発現
The cloning of zebrafish purpurin gene and its expression in early developing retina
永島　幹子[1](Mikiko Nagashima)，田中　聖之[2](Masayuki Tanaka)，村山　大育[2](Daisuke Murayama)，
馬渡　一浩[1](Kazuhiro Mawatari)，加藤　聖[2](Satoru Kato)
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P1-c27 大脳皮質形成における Sonic hedgehog シグナルの役割
The role of sonic hedgehog signaling in the developing cerebral cortex
駒田 致和(Munekazu Komada), 才津 浩智(Hirotomo Saitsu), 塩田 浩平(Kohei Shiota),
石橋 誠(Makoto Ishibashi)
京都大院・医・生体構造医学・形態形成機構学

P1-c28 人脳における前頭極から中心溝までの前頭葉の長さ
Length of the frontal lobe from the anterior pole of the frontal lobe to the central sulcus in human brain
北村 泰子(Taiko Kitamura), 山田 仁三(Jinzo Yamada)
東京医大・組織神経解剖学

P1-c29 マウス発生過程における Tsukushi の発現パターン
Expression pattern of Tsukushi during mouse development
伊藤 綾子[1](Ayako Ito), 新明 洋平[1,2](Youhei Shinmyou), 中山 りか[4](Rika Nakayama),
大島なおこ[4](Naoko Oshima), 田中 英明[1,2](Hideaki Tanaka), 太田 訓正[1,3](Kunimasa Ohta)
[1]熊本大院・医薬・神経分化, [2]熊本大・COE, [3]科技構, [4]理研CDB

P1-c30 キノコ体発生における Wnt シグナリングの役割
Role of Wnt signaling in the mushroom body development
清水 一道(Kazumichi Shimizu), Makoto Sato, Shoko Yoshida, Tetsuya Tabata
東大・分生研・形態形成

P1-c31 DMAP1 は神経細胞の移動と分化に関与する
Involvement of DMAP1 in the neural migration and differentiation in zebrafish
田中 英臣[1,2](Hideomi Tanaka), 天羽龍之介[1,3](Ryunosuke Amou), 白木 利幸[1](Toshiyuki Shiraki),
小林 恵美[1](Megumi Kobayashi), 中山 涼子[1](Ryoko Nakayama), 岡本 仁[1,2](Hitoshi Okamoto)
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P1-c32 ケモカイン SDF-1 α は嗅内皮質から海馬歯状回への軸索伸長と誘導を調節する
The chemokine SDF-1 α regulates axonal elongation and pathfinding from entorhinal cortex to dentate gyrus in hippocampus
大島 洋一[1](Yoichi Ohshima), 久保 武一[1](Takekazu Kubo), 山下 俊英[1](Toshihide Yamashita),
小山 隆太[2](Ryuta Koyama), 中川 正法[3](Masanori Nakagawa)
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P1-c33 マウス大脳皮質構築過程の神経細胞層形成に対するニューロトロフィン -3 の影響
Neurotrophin-3 influences cortical laminar formation during development of mouse cerebral cortex
大塚 正成(Masanari Ohtsuka), 福光 秀文(Hidefumi Fukumitsu), 古川 昭栄(Shoei Furukawa)
岐阜薬科大学 分子生物学教室

P1-c34 発生期マウス大脳皮質に強く発現する転写抑制因子 RP58 が制御する下流遺伝子の解析
Analysis of the downstream effectors for a transcriptional repressor RP58 in the embryonic cerebral cortex
丸山 千秋[1](Chiaki Ohtaka-Maruyama), 三輪 昭子[1](Akiko Miwa), 葛西 正孝[2](Masataka Kasai),
岡戸 晴生[1](Haruo Okado)
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P1-c35 RP58 が新皮質と海馬の領域化に関与する可能性について
RP58 may be involved in regionalization of neocortex and hippocampus
岡戸 晴生[1](Haruo Okado), 丸山 千秋[1](Chiaki Ohtaka-Maruyama), 三輪 昭子[1](Akiko Miwa),
川野 仁[2](Hitoshi Kawano), 葛西 正孝[3](Masataka Kasai)
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## 神経幹・前駆細胞と細胞分化，移植 1
## Neural Stem/Progenitor Cells and Cellular Differentiation, Transplantation 1

P1-c36 **神経幹細胞に対する気分安定薬の効果のマイクロアレイ解析**
**Microarray analysis of neurospheres treated with mood stabilizers.**
松下　雄一[1,2](Yuichi Matsushita)，東　　幹人[2](Mikito Higashi)，等　　誠司[2](Seiji Hitoshi)，
池中　一裕[1,2](Kazuhiro Ikenaka)
[1]総合研究大学院大学，[2]生理学研究所

P1-c37 **ショウジョウバエ RNAi スクリーニングによって得られた神経発生必須遺伝子のマウスホモローグ / ショウジョウバエでの遺伝子発現解析**
**Profiling of gene expression required for mouse/*Drosophila* neurogenesis, which are identified by *Drosophila* RNAi Screening**
Mohammad Saharul Islam，小泉　恵太(Keita Koizumi)，Sarwat Amina，東田　陽博(Haruhiro Higashida)
金沢大・21世紀COE革新脳科学プログラム

P1-c38 **ADモデルマウス脳における OLig2 遺伝子の発現変化とその意味**
**Decreased expression of Olig2 in response to β-amyloid accelerates the death of neural stem/progenitor cells.**
内田　洋子(Yoko Uchida)，五味不二也(Fujiya Gomi)
東京都老人総合研究所・老年病ゲノムチーム

P1-c39 **ラット嗅上皮剥離後の嗅球における Olig2 の発現変化について**
**Expression pattern of Olig2 in the rat olfactory bulb following olfactory epithelium lesion**
平田　和穂[1](Kazuho Hirata)，金丸　孝昭[2](Takaaki Kanemaru)，三野原元澄[3](Motozumi Minohara)，
吉良　潤一[3](Jun-ichi Kira)
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P1-c40 **ニューロン分化における C2HC 型 Zn フィンガー転写因子 dNZF の解析**
**Analysis of *Drosophila* C2HC-type Zn finger factor dNZF in neuronal differentiation**
大迫　俊二[1](Shunji Ohsako)，飯島　　樹[1,2](Itsuki Iijima)，橋爪　　卓[1,2](Masaru Hashizume)，
山田　隆文[1](Takafumi Yamada)
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P1-c41 **BDNF による神経幹細胞から分化した神経前駆細胞への生存保護効果**
**BDNF supports the survival of neuronal progenitors differentiated from neural stem cells in vitro**
久戸瀬哲也(Tetsuya Kudose)，福光　秀文(Hidefumi Fukumitsu)，古川　昭栄(Shoei Furukawa)
岐阜薬科大院・薬

P1-c42 **髄腔内成長因子投与による変異 SOD1 遺伝子導入ラット NG2 陽性神経前駆細胞の活性化**
**Intrathecal infusion of growth factors stimulates NG2-expressing progenitors in mutant *SOD1*-transgenic rats**
割田　　仁(Hitoshi Warita)，青木　正志(Masashi Aoki)，水野　秀紀(Hideki Mizuno)，
糸山　泰人(Yasuto Itoyama)
東北大院・医・神経内科

P1-c43 **細胞周期抑制剤による神経幹細胞からの神経分化促進における TGFβ1 の発現変化**
**Enhanced expression of TGFβ1 in cell cycle blocker-induced neuronal differentiation of neurosphere**
三角　吉代(Sachiyo Misumi)，飛田　秀樹(Hideki Hida)，金　　へ亨(Hye-Jung Kim)，
西野　仁雄(Hitoo Nishino)
名古屋市立大院・医・脳神経生理

P1-c44 hGFAP プロモーター下流に DsRed をもつレンチウィルスを用いたアストロサイト特異的標識法の確立
Astrocyte-specific labeling with a recombinant lentiviral vector carrying DsRed protein driven by a human glial fibrillary acidic protein promoter
佐藤　薫[1](Kaoru Sato), Rachel_E Ventura[2], James_E Goldman[2], 中澤　憲一[1](Ken Nakazawa)
[1]国立医薬品食品衛生研究所・薬理部, [2]コロンビア大学病理部

P1-c45 遺伝子マーカーによる reeler 大脳皮質の形成異常の解析
Characterization of the reeler Cerebral Cortex by expression of molecular markers
出來本秀行(Hideyuki Dekimoto), 寺島　俊雄(Toshio Terashima), 勝山　裕(Yu Katsuyama)
神戸大院・医・神経発生

P1-c46 Notch シグナルはマウス発生期小脳に存在する EGF 応答性細胞のグリア細胞への分化を抑制する
Notch signaling inhibits glial differentiation of the EGF-responsive cells in developing mouse cerebellum
関本　研一[1](Kenichi Sekimoto), 岡野 - 内田孝幸[1](Takayuki Okano-Uchida), 倉知　正[1](Masashi Kurachi),
磯　達也[2](Tatsuya Iso), 倉林　正彦[2](Masahiko Kurabayashi), 齋藤　繁[3](Shigeru Saito),
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P1-c47 Xrcc4 発現抑制による神経発生時の神経細胞死
Massive apoptosis during neurogenesis induced by XRCC4 inhibition
川端　昌弘[1](Masahiro Kawabata), 川端　晃幸[2](Teruyuki Kawabata), 荻野　哲也[3](Tetsuya Ogino)
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P1-c48 神経特異的ファミリータンパク質 BRINP のタンパク質相互作用解析
Analysis of BRINP-interacting proteins
一色　真明[1](Masaaki Isshiki), 石井美奈子[2](Minako Ishii), 本宮　真[3](Makoto Motomiya),
小林美和子[2](Miwako Kobayashi), 松岡　一郎[1,2](Ichiro Matsuoka)
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P1-d01 神経幹細胞の発生における glial cells missing 遺伝子の役割
glial cells missing (gcm) genes in the induction of neural stem cells
等　誠司[1](Seiji Hitoshi), 田中　謙二[1](Kenji Tanaka), 細谷　俊彦[2](Toshihiko Hosoya),
堀田　凱樹[3](Yoshiki Hotta), 池中　一裕[1](Kazuhiro Ikenaka)
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P1-d02 内包出血モデルにおける内因性神経幹細胞の活性化
Activation of endogenous neural stem cells in intracerebral hemorrhage model rat
増田　匡(Tadashi Masuda), 熊崎　路子(Michiko Kumasaki), 櫻井　輝美(Terumi Sakurai),
飛田　秀樹(Hideki Hida)
名古屋市立大学大学院・医学研究科・脳神経生理

P1-d03 小脳顆粒細胞の分化におけるホメオボックス遺伝子 *Mbh1* の機能
*Mbh1* is involved in cerebellar granule cell differentiation downstream of a proneural gene, *Math1*
川内　大輔(Daisuke Kawauchi), 斎藤哲一郎(Tetsuichiro Saito)
千葉大院・医・発生生物

P1-d04 NSS 法による胚性幹細胞の神経分化における遺伝子発現及びタンパク質発現の変化
Changes in expression of genes and proteins during neural differentiation from embryonic stem cells by Neural Stem Sphere method
大津　昌弘[1](Masahiro Otsu), 龍野　良佑[2](Ryosuke Tatsuno), 堀越　朋恵[2](Tomoe Horikoshi),
赤間　邦子[2](Kuniko Akama), 村上　拡治[1](Koji Murakami), 中山　孝[3](Takashi Nakayama),
中村　愛[4](Megumi Nakamura), 戸田　年総[4](Tosifusa Toda), 笠井　久隆[5](Hisataka Kasai),
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P1-d05 カニクイサル胚性幹細胞を用いた超選択的神経分化誘導の評価と応用
Super selective neuronal differentiation and application of cynomolgus embryonic stem cells
遠藤　乙音[1](Otone Endo)，水野　正明[3](Masaaki Mizuno)，梶田　泰一[2](Yasukazu Kajita)，
吉田　純[2](Jun Yoshida)
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P1-d06 脳外傷後の損傷周囲大脳皮質からの神経幹細胞の単離
Isolation of neural stem cells from damaged rat cerebral cortex after traumatic brain injury
伊藤　龍生[1](Tatsuki Itoh)，佐藤　隆夫[1,2](Takao Satou)，橋本　重夫[3](Shigeo Hashimoto)，
伊藤　浩行[1](Hiroyuki Ito)
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P1-d07 NG2 プロテオグリカンと免疫細胞マーカーを発現するラットおよびヒトグリオーマ細胞
Rat and human glioma cells express NG2 proteoglycan and immune cell markers
Anna Smirkin[1]，松本　洋明[2](Hiroaki Matsumoto)，大西　丘倫[2](Takanori Ohnishi)，
今井　嘉紀[1](Yoshinori Imai)，高橋　寿明[1](Hisaaki Takahashi)
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## 細胞移動、突起伸展、回路網形成 1
## Migration, Axonal and Dendritic Outgrowth, Network Formation 1

P1-d08 コンドロイチン硫酸のガイダンス効果を検討する培養実験系の確立
New method for investigating guidance effects of chondroitin sulfates *in vitro*
安藤　覚[1](Satoru Ando)，杉浦　信夫[2](Nobuo Sugiura)，木全　弘治[2](Koji Kimata)，
高橋　智[1](Satoru Takahasi)，一條　裕之[1](Hiroyuki Ichijo)
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P1-d09 小脳に投射する三叉神経脊髄路核神経細胞の起源と発生
The origin and development of the precerebellar neurons in the spinal trigeminal nucleus
岡田　拓也(Takuya Okada)，桝　和子(Kazuko Keino-Masu)，桝　正幸(Masayuki Masu)
筑波大院・人間総合科学・分子神経生物

P1-d10 移動及び核形成初期における小脳前核神経細胞群の動態観察
Real-time imaging of precerebellar neurons during migration and early steps of nucleogenesis
渡辺　治康(Haruyasu Watanabe)，村上富士夫(Fujio Murakami)
大阪大院　生命機能　脳神経工学

P1-d11 GABA、グルタミン酸による海馬新生顆粒細胞の分化
GABA and glutamate regulate the differentiation of newborn hippocampal granule cells
市川　淳也(Junya Ichikawa)，池谷　裕二(Yuji Ikegaya)，松木　則夫(Norio Matsuki)，
小山　隆太(Ryuta Koyama)
東京大院・薬・薬品作用学

P1-d12 一次体性感覚野臨界期の終了時期決定におけるオリゴデンドロサイトの役割
Termination of lesion-induced barrel plasticity is independent of oligodendrocyte differentiation in the mouse somatosensory cortex
戸田　智久[1](Tomohisa Toda)，早川　格[1](Itaru Hayakawa)，松林　完[1](Yutaka Matsubayashi)，
田中　謙二[2](Kenji Tanaka)，池中　一裕[2](Kazuhiro Ikenaka)，Richard_Q Lu[3]，河崎　洋志[1](Hiroshi Kawasaki)
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P1-d13 スリングショットによる海馬神経細胞樹状突起形成の制御
Slingshot regulates primary dendrite formation in rat hippocampal neurons
赤塚　淳一(Junichi Akatsuka)，木内　泰(Tai Kiuchi)，遠藤　光晴(Mitsuharu Endo)，
大橋　一正(Kazumasa Ohashi)，水野　健作(Kensaku Mizuno)
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P1-d14 ラット膨大後皮質生後発達における層特異的遺伝子発現解析
Layer-specific gene expression in developing rat retrosplenial cortex
宮下 俊雄[1](Toshio Miyashita), Marie Wintzer[1], 小西 智一[2](Tomokazu Konishi),
一戸 紀孝[1](Noritaka Ichinohe), Kathleen Rockland[1]
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P1-d15 プロトカドヘリンαの細胞内領域は、セロトニン神経の正常な投射の形成に必須である
Cytoplasmic domain of Protocadhrin-α is essential for establishment of normal serotonergic projection
香取 将太[1](Shota Katori), 濱田 俊[2](Shun Hamada), 福田 絵美[1](Emi Fukuda),
江角 重行[3](Shigeyuki Esumi), 野口由紀子[1](Yukiko Noguchi), 山本 秀子[4](Hideko Yamamoto),
山本 敏文[5](Toshifumi Yamamoto), 八木 健[1](Takeshi Yagi)
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P1-d16 UNC-51 は、ショウジョウバエ脳において軸索・樹上突起パターニングを制御している。
UNC-51 regulates axonal and dendritic patterning in the Drosophila brain
望月 洋明[1](Hiroaki Mochizuki), 戸田 浩史[1,2](Hirofumi Toda), 友田 利文[2](Toshifumi Tomoda),
古久保 - 徳永克男[1](Katsuo Furukubo-Tokunaga)
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P1-d17 LECT2 は神経突起の伸長と分岐を調節する
Leukocyte cell-derived chemotaxin 2 (LECT2) regulates neuritic extension and branching
輿水 洋平(Yohei Koshimizu), 大富美智子(Michiko Ohtomi)
東邦大学・理・生物分子科学科

P1-d18 ドレブリンによる軸索・樹状突起伸展の促進
Enhancement of axon and dendrite outgrowth by drebrin expression at early developmental stages in hippocampal neurons
水井 利幸(Toshiyuki Mizui), 片山 理人(Michihito Katayama), 白尾 智明(Tomoaki Shirao)
群馬大院・医・高次細胞機能

P1-d19 PTPζによる DNER の局在制御機構の解析
Receptor-type protein tyrosine phosphataseζ(PTPζ) associates with DNER and regulates its subcellular localization
深澤 伸名[1](Nobuna Fukazawa), 横山 斉輔[2](Seisuke Yokoyama), 見学美根子[2](Mineko Kengaku),
前田 信明[1](Nobuaki Maeda)
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P1-d20 発達期マウス小脳におけるコンドロイチン硫酸転移酵素の発現解析
Expression of chondroitin sulfate sulfotransferases in the developing mouse cerebellum
石井 万幾(Maki Ishii), 前田 信明(Nobuaki Maeda)
東京都神経研・分子発生

P1-d21 樹状突起スパイン形成におけるソニックヘッジホッグの役割
The role of sonic hedgehog in dendritic spine formation
佐々木信成(Nobunari Sasaki), 栗栖 純子(Junko Kurisu), 見学美根子(Mineko Kengaku)
独立行政法人理化学研究所脳科学総合研究センター

P1-d22 大脳皮質および海馬形成における Rac1 の機能解析
Essential role of Rac1 in development of cerebral cortex and hippocampus
葛西 秀俊[1](Hidetoshi Kassai), 寺島 俊雄[2](Toshio Terashima), 深谷 昌弘[3](Masahiro Fukaya),
三浦会里子[3](Eriko Miura), 渡辺 雅彦[3](Masahiko Watanabe), 饗場 篤[1](Atsu Aiba)
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P1-d23 神経前駆細胞の移動における活性酸素種の解析
Role of reactive oxygen species on cell migration in neural progenitor cells
諫田 泰成(Yasunari Kanda), 渡辺 康裕(Yasuhiro Watanabe)
防衛医大・薬理

P1-d24 マウス大脳皮質視覚野の脳梁軸索投射形成におけるシナプス前・後細胞の神経活動の役割
Roles of pre- and post-synaptic neuron activity in interhemispheric connection development in the mouse visual cortex
水野　秀信[1,2](Hidenobu Mizuno)，平野　丈夫[1,2](Tomoo Hirano)，田川　義晃[1,2](Yoshiaki Tagawa)
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P1-d25 ARD1-NAT1 アセチルトランスフェラーゼ複合体による tubulin アセチル化を介した樹状突起発達制御
ARD1-NAT1 complex regulates neuronal dendritic arborization through α-tubulin acetylation
大川　宜昭[1](Noriaki Ohkawa)，杉崎俊一郎[1,2](Shunichiro Sugisaki)，徳永　絵理[1](Eri Tokunaga)，
藤谷　和子[1](Kazuko Fujitani)，瀬藤　光利[1](Mitsutoshi Setou)，井ノ口　馨[1,2,3](Kaoru Inokuchi)
[1]三菱化学生命科学研究所，[2]横浜国立大学，[3]CREST，JST

P1-d26 BIG-2 は標的糸球への嗅細胞軸索投射を制御する
Involvement of BIG-2 in convergence of olfactory axons to target glomeruli
後藤（金子）智美[1](Tomomi Kaneko-Goto)，吉原　誠一[1](Sei-ichi Yoshihara)，
吉原　良浩[1,2](Yoshihiro Yoshihara)
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P1-d27 大脳皮質上層ニューロンの内側投射のメカニズム
Molecular mechanism that regulates medially oriented axonal growth of upper layer neurons in cortical development
趙　　虹(Hong Zhao)，丸山　拓郎(Takuro Maruyama)，白崎　竜一(Ryuichi Shirasaki)，
山本　亘彦(Nobuhiko Yamamoto)
大阪大院　　生命機能

P1-d28 ネトリン-1 による海馬苔状線維の順行性投射機構の維持
A possible involvement of Netrin-1 as a guidance cue for hippocampal mossy fibers
村松里衣子(Rieko Muramatsu)，池谷　裕二(Yuji Ikegaya)，松木　則夫(Norio Matsuki)，
小山　隆太(Ryuta Koyama)
東京大院・薬・薬品作用

P1-d29 ヒストン脱アセチル化酵素6の脳における役割
Possible role of histone deacetylase 6 in brain
川口　禎晴(Yoshiharu Kawaguchi)，時田　義人(Yoshito Tokita)，深田　斉秀(Masahide Fukada)，
青木　英子(Eiko Aoki)，正木　茂夫(Shigeo Masaki)，中山　敦雄(Atsuo Nakayama)
愛知県コロニー・発達障害研究所・発生障害学部

P1-d30 視床皮質軸索の枝分かれ形成における視床と大脳皮質細胞の神経活動の役割
The role of neural activities of thalamic and cortical neurons in thalamocortical axon branching
山田　成人[1](Akito Yamada)，上阪　直史[2](Naofumi Uesaka)，早野　泰史[1](Yasufumi Hayano)，
田端　俊英[2](Toshihide Tabata)，狩野　方伸[2](Masanobu Kano)，山本　亘彦[1](Nobuhiko Yamamoto)
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## 再生
## Regeneration

P1-d31 末梢神経損傷時のシンデカン - 1の発現変動
Nerve injury induces the expression of syndecan-1 heparan sulfate proteoglycan
村上　公一(Koichi Murakami)，吉田　成孝(Shigetaka Yoshida)
旭川医大・解

P1-d32 嗅球グリア細胞の移植による黒質線条体ドーパミン神経路の再生には繊維性瘢痕の形成抑制が伴う
Regeneration of nigrostiatal dopaminergic axons by transplantation of olfactory ensheathing cells is accompanied by elimination of the fibrotic scar formation in the lesion site
川野　仁(Hitoshi Kawano)，李　洪鵬(Hong Peng Li)，滕　錫川(Xi Chun Teng)，
川村　光毅(Koki Kawamura)
東京都神経研・発生形態

P1-d33 ゼブラフィッシュ視神経軸索の再生
Regeneration of optic nerve axons in zebrafish
杉木　隼人[1](Hayato Sugiki)，堀江　秀典[2](Hidenori Horie)，井上　宏子[1](Hiroko Inoue)
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P1-d34 魚類視神経再生過程に発現するガレクチンの変動とその役割
Upregulation of galectins in retina and its possible role in fish optic nerve regeneration
杉谷　加代[1](Kayo Sugitani)，胡桃沢智子[1](Tomoko Kurumizawa)，馬渡　一浩[1](Kazuhiro Mawatari)，
金田　学[2](Manabu Kaneda)，加藤　聖[2](Satoru Kato)
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P1-d35 Purpurin による損傷後網膜神経節細胞の生存および軸索再生
Cell survival and axonal regeneration of injured retinal ganglion cells by purpurin
郡山　恵樹[1](Yoshiki Koriyama)，桜井　裕之[2](Hirokyuki Sakurai)，松川　通[1](Toru Matsukawa)，
馬渡　一浩[2](Kazuhiro Mawatari)，加藤　聖[1](Satoru Kato)
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P1-d36 イヌの脊髄損傷モデルについて
New canie spinal cord injury model free from laminectory
井上　勝也[1,2](Katsuya Inoue)，遠藤　克昭[1,2](Katsuaki Endo)，藤川　孝満[1,2](Takamitsu Fujikawa)，
福田　正順[2](Seijyun Fukuda)，中村　達雄[2](Tatsuo Nakamura)
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P1-d37 Neuroprotective effects of transplanted olfactory ensheathing cells on axotimized retinal ganglion cells in adult rats
Mingmei Wu[1], Bing Hou[1], Hao Yang[1], Na Luo[1], Xi-Ying Jiao[1], Kwok-Fai So[2], Gong Ju[1], Si-Wei You[1]
[1]Institute of Neurosciences, The Fourth Military Medical University, Xi' an, PR China, [2]Department of Anatomy, Faculty of Medicine, The University of Hong Kong, Hong Kong, China

P1-d38 OFF 神経節細胞と ON 神経節細胞の生存および軸索再生能
Difference in regeneration and survival between OFF-center ganglion cells and ON-center cells in cat retina
渡部　眞三(Masami Watanabe)，時田　義人(Yoshito Tokita)
愛知県心身障害者コロニー発達障害研究所周生期学部

## 神経修復，リハビリテーション
## Neural Repair and Rehabilitation

P1-d39 ニューロンとアストロサイトは線維芽細胞に比べゲノム全体に働くヌクレオチド除去修復活性が低い
Neurons and astrocytes exhibit lower activities of global genome nucleotide excision repair than do fibroblasts
山本　亜弥[1](Aya Yamamoto)，伊藤　康一[2](Kouichi Itoh)，新野　秀人[1](Hideto Shinno)，
中村　祐[1](Yu Nakamura)
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P1-d40 脳卒中後の麻痺手運動時における一次感覚運動野の活動 − NIRS による横断的検討−
Primary sensorimotor activation during affected hand grasping after hemiparetic stroke: A cross-sectional NIRS study
武田湖太郎[1,2](Kotaro Takeda), 今井　樹[2,3](Itsuki Imai), 前田　眞治[4](Masaharu Maeda),
加藤　宏之[1,2](Hiroyuki Kato)
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P1-d41 後根切断後の脊髄におけるオリゴデンドロサイト前駆細胞の増殖及び分化
Responses of oligodendrocyte progenitors in the spinal cord following dorsal rhizotomy in the adult rat
近藤　照義[1](Teruyoshi Kondo), 中村桂一郎[2](Kei-Ichiro Nakamura), 高村　徳人[3](Norito Takamura),
徳永　仁[3](Jin Tokumasa), 山本　隆一[3](Ryuichi Yamamoto)
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P1-d42 発達初期片側除皮質ラットにおける皮質脊髄路の大規模変化
Large-scale change in the corticofugal projections in rats with neonatal hemi-decortication
高橋　雅人[1,5](Masahito Takahashi), Anusara Vattanajun[1,4], 梅田　達也[1](Tatsuya Umeda),
伊佐かおる[1,2](Kaoru Isa), 伊佐　正[1,2,3](Tadashi Isa)
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P1-d43 適度な運動はパーキンソン病モデルに対して神経保護効果を有する
Exercise has a neuroprotective effect in parkinsonian rat model
田尻　直輝(Naoki Tajiri), 新郷　哲郎(Tetsuro Shingo), 元　文姫(Wenji Yuan),
森本　尊雅(Takamasa Morimoto), 門田　知倫(Tomohito Kadota), 安原　隆雄(Takao Yasuhara),
三好　康之(Yasuyuki Miyoshi), 伊達　勲(Isao Date)
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## 体性運動 1
## Somatomotor System 1

P1-d44 Developmental changes in mouse striatal neurons differ to those seen in rat
Hyunchul Lee, Atomu Sawatari
Department of Physiology and Bosch Institute, University of Sydney, Australia

P1-d45 Sensory basis of motor program selection in the cricket behavior
Tetsuya Matsuura[1], Masamichi Kanou[2], Shinsuke Morita[2], Tsuneo Yamaguchi[3]
[1]Dept. Welfare Eng., Iwate Univ., Morioka, Japan, [2]Grad. Sch. Sci. and Eng., Ehime Univ., Matsuyama, Japan, [3]Kawasaki Coll. Allied Health Profession, Kurashiki, Japan

P1-d46 三叉神経運動核および周辺のニューロンにおける GAD 67 とグリシントランスポーター 2 mRNA の発現
Neurons expressing GAD 67 and glycine transporter 2 mRNA in and around the trigeminal motor nucleus of the rat
河田　亮(Akira Kawata), 林　弘之(Hiroyuki Hayashi), 都築　英子(Hideko Tsuzuki),
大栗　重彦(Shigehiko Oguri), 杉山　朋久(Tomohisa Sugiyama), 赤城　忠臣(Tadaomi Akagi),
東　一善(Kazuyoshi Higashi), 高橋　理(Osamu Takahashi)
神奈川歯科大学人体構造学講座組織学分野

P1-d47 手首運動を利用した定量的運動機能検査システムの構築
Development of a quantitative evaluation system for motor function using wrist movement
李　鍾昊[1](Jongho Lee), 鏡原　康裕[2](Yasuhiro Kagamihara), 筧　慎治[1](Shinji Kakei)
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P1-d48 シングルニューロントレースによる線条体投射系の解析
The striatofugal system from patch and matrix compartments in rat neostriatum
孫　在隣[1](Jaerin Sohn)，藤山　文乃[1](Fumino Fujiyama)，金子　武嗣[1,2](Takeshi Kaneko)
[1]京都大院・医・高次脳形態，[2]CREST

P1-e01 マウスナー細胞と形態学的に相同な後脳網様体脊髄路ニューロン間の機能的結合
Functional connection from Mauthner cell to homologous reticulospinal neurons in the goldfish hindbrain
根木　大輔[1](Daisuke Neki)，中山　寿子[2](Hisako Nakayama)，藤井　高志[2](Takashi Fujii)，
小田　洋一[1](Yoichi Oda)
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P1-e02 腕運動時における体性感覚反射ゲインのタイムコーディング
Time-domain coding of somatosensory reflex gain during arm movements
木村　聡貴[1](Toshitaka Kimura)，五味　裕章[1,2](Hiroaki Gomi)
[1]NTT・NTTコミュニケーション科学基礎研究所，[2]JST・ERATO下條潜在脳機能プロジェクト

P1-e03 視覚 - 運動情報の不一致と自己主体感の関係
The relationship between the sense of self-agency and visuo-motor incongruency
浅井　智久(Tomohisa Asai)，丹野　義彦(Yoshihiko Tanno)
東京大院・総合文化研究科

P1-e04 指の体性感覚情報が左右指運動に与える影響
Influence of finger somatosensory information on the coordination of bimanual finger movements
櫻田　武[1,2](Takeshi Sakurada)，五味　裕章[2](Hiroaki Gomi)，井澤　淳[2,3](Jun Izawa)，
伊藤　宏司[1](Koji Ito)
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P1-e05 手操作運動課題におけるサル AIP 野ニューロン応答の情報量解析
Information analysis of Monkey AIP neurons during hand manipulation task
清水　崇司[1](Takashi Shimizu)，石田　文彦[1](Fumihiko Ishida)，村田　哲[2](Akira Murata)，
阪口　豊[1](Yutaka Sakaguchi)
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P1-e06 無動化除脳ウサギの中脳刺激により誘発された後肢歩行パターンの特徴
Hindlimb locomotor pattern evoked by the midbrain stimulation in decerebrate, paralyzed rabbits
松山　清治[1](Kiyoji Matsuyama)，石黒　雅敬[1](Masanori Ishiguro)，青木　藩[2](Mamoru Aoki)
[1]札幌医大・医・第二生理，[2]北海道文教大・人間科学・理学療法

P1-e07 線条体投射ニューロンに対する大脳皮質および視床からの入力の定量的解析
Quantitative analysis of inputs to projection neurons from the neocortex or the thalamus in the rat neostriatum
薗村　貴弘[1](Takahiro Sonomura)，古田　貴寛[2](Takahiro Furuta)，古田　敬子[2](Keiko-O Furuta)，
雲財　知[2](Tomo Unzai)，岩井　治樹[1](Haruki Iwai)，植村　正憲[1](Masanori Uemura)，
金子　武嗣[2,3](Takeshi Kaneko)
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P1-e08 課題達成要求がタイミング一致課題の脳活動に及ぼす影響
Effects of task requirements on the brain activity in the coincident timing task
大築　立志[1](Tatsuyuki Ohtsuki)，加賀城聡子[1](Akiko Kagajo)，門田　宏[2](Hiroshi Kadota)，
中島八十一[2](Yashoichi Nakajima)，中澤　公孝[2](Kimitaka Nakazawa)，工藤　和俊[1](Kazutoshi Kudo)
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P1-e09 ヒトの倒立振子バランス制御におけるムダ時間の影響
Balance control of the virtual inverted pendulum: effects of experimentally introduced time delays
澤畑　博人(Hirohito Sawahata)，都築　仁志(Hitoshi Tsuzuki)，落合　亮平(Ryohei Ochiai)，
山口　峻司(Takashi Yamaguchi)
山形大院・理工・生体センシング機能工学

P1-e10 小脳プルキンエ細胞複雑スパイクは運動指令を内因的座標系でコードする
Cerebellar complex spikes encode movement in intrinsic coordinate frames
角田　吉昭[1](Yoshiaki Tsunoda)，松井　航[1](Wataru Matsui)，田桑　弘之[3](Hiroyuki Takuwa)，
Peter Strick[2]，Donna Hoffman[2]，筧　慎治[1](Shinji Kakei)
[1]東京都神経研　認知行動，[2]ピッツバーグ大学，[3]愛媛大学

P1-e11 サルにおけるリズム学習
Rhythm learning in the monkey
鴻池　菜保(Naho Konoike)，宮地　重弘(Shigehiro Miyachi)，三上　章允(Akichika Mikami)
京都大学霊長類研究所・行動発現分野

P1-e12 タイミング一致課題における情報のフローによるヒト補足運動野活動の減少
Continuous information flow decreases the activity of continuous information flow decreases the activity of supplementary motor area (SMA) in coincident timing task
工藤　和俊[1](Kazutoshi Kudo)，門田　宏[2](Hiroshi Kadota)，中島八十一[2](Yashoichi Nakajima)，
中澤　公孝[2](Kimitaka Nakazawa)，大築　立志[1](Tatsuyuki Ohtsuki)
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## 眼球運動
## Oculomotor System

P1-e13 Activity of pursuit neurons in the caudal part of the frontal eye fields (FEF) during static roll-tilt
Sergey Kurkin[1], Teppei Akao[1], Junko Fukushima[2], Kikuro Fukushima[1]
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P1-e14 舌下神経前位核における抑制性ニューロンの膜特性　-VGAT-Venus transgenic rat を用いた解析 -
Membrane properties of prepositus inhibitory neurons in VGAT-Venus transgenic rats
紫野　正人[1](Masato Shino)，柳川右千夫[2](Yuchio Yanagawa)，川口　泰雄[3](Yasuo Kawaguchi)，
小澤　瀞司[1](Seiji Ozawa)，齋藤　康彦[1](Yasuhiko Saito)
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P1-e15 サッカード適応の加速と異方向転移が示唆する小脳の可塑性
Cerebellar plasticity suggested by the facilitation and transfer of saccadic adaptation
藤田　昌彦(Masahiko Fujita)
法政大・工・経営工

P1-e16 Square-wave jerk を再現する固視微動モデル
A model of small involunatay eye movements to reproduce squre-wave jerks
小濱　剛[1](Takeshi Kohama)，神山　斉己[2](Yoshimi Kamiyama)
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P1-e17 サッカード中の視覚の安定性維持における MST の能動的関与
An active role of MST in maintaining visual stability during saccades in the natural environment
寒　重之[1](Shigeyuki Kan)，三崎　将也[2,4](Masaya Misaki)，岩田　一樹[2,3](Kazuki Iwata)，
小池　耕彦[2,3](Takahiko Koike)，宮内　哲[1,2,3](Satoru Miyauchi)
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P1-e18 サル小脳半球第７小葉のサッケードとパーシュート眼球運動応答の特徴
Responsiveness of primate cerebellar hemispheric lobule VII to saccade and smooth pursuit eye movements
北澤　宏理[1](Hiromasa Kitazawa)，大木　雅文[2](Masafumi Ohki)，永雄　総一[1,3](Soihi Nagao)
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P1-e19 サッケード眼球運動とその手がかり刺激に対する小脳半球第７小葉の神経細胞の応答
Visual responses of monkey cerebellar hemispheric lobule VII during saccade eye movement preparation
茂木有加利[1](Eucaly Mogi)，蛯名　悠希[2](Yuki Ebina)，永雄　総一[1](Soichi Nagao)
[1]理化学研究所・BSI・運動学習制御，[2]長岡技大・電気電子情報工学

P1-e20 早発輻輳眼球運動の特性
Properties of the anticipatory convergence eye movement
戸田　春男(Haruo Toda)，飯島　淳彦(Atsuhiko Iijima)，板東　武彦(Takehiko Bando)
新潟大院・医歯学・統合生理

P1-e21 一次視覚野除去後の損傷視野内における視覚運動変換過程への上丘の寄与
Involvement of superior colliculus in visual-oculomotor processing after unilateral V1 lesion
高浦　加奈[1](Kana Takaura)，吉田　正俊[1,2,3](Masatoshi Yoshida)，伊佐　正[1,2,3](Tadashi Isa)
[1]総合研究大学院大学・生命科学，[2]生理学研究所・認知行動発達，[3]科学技術振興機構・CREST

P1-e22 前頭眼野追跡眼球運動ニューロンに対する頸部固有受容器からの入力
Neck proprioceptive inputs to pursuit neurons in the caudal frontal eye fields (FEF)
齊藤　展士[1,2](Hiroshi Saito)，赤尾　鉄平[1](Teppei Akao)，Sergei Kurkin[1]，福島　菊郎[1](Kikuro Fukushima)
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P1-e23 前頭眼野滑動性眼球運動ニューロンの前庭応答潜時
Latency of vestibular responses of pursuit neurons in the caudal frontal eye fields (FEF) to whole body rotation
赤尾　鉄平[1](Teppei Akao)，齊藤　展士[1](Hiroshi Saito)，福島　順子[2](Junko Fukushima)，Sergei Kurkin[1]，
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P1-e24 追跡眼球運動発現におけるトップダウン的注意の効果
The effect of a symbolic cue on human smooth pursuit initiation
三浦健一郎(Kenichiro Miura)，青木　佑紀(Yuki Aoki)，田端　宏充(Hiromitsu Tabata)，
河野　憲二(Kenji Kawano)
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## 視覚 1
## Visual System 1

P1-e25 Amphiphysin Ir は ARPE-19 細胞のレセプター介在性エンドサイトーシスに関与する
Amphiphysin Ir participates in receptor-mediated endocytosis in ARPE-19
細谷　修(Osamu Hosoya)，筒井　研(Ken Tsutsui)，筒井　公子(Kimiko Tsutsui)
岡山大・院・医歯薬

P1-e26 注意にもとづく視覚グルーピングに選択的なサル頭頂間溝皮質ニューロン活動
Selective responses of neurons in the intraparietal sulcus of the monkey during a visual grouping task guided by attention
横井　功[1,2](Isao Yokoi)，小松　英彦[1,2](Hidehiko Komatsu)
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P1-e27 サル下側頭皮質における色関連領野の機能的 MRI 計測
Discrete color-related subregions in the monkey inferior temporal cortex
原田 卓弥[1,2](Takuya Harada), 郷田 直一[1,2](Naokazu Goda), 小川 正[1,2](Tadashi Ogawa),
伊藤 南[1,2](Minami Ito), 豊田 浩士[1](Hiroshi Toyoda), 定藤 規弘[1,2](Norihiro Sadato),
小松 英彦[1,2](Hidehiko Komatsu)
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P1-e28 ポップアウト刺激によるサル視覚皮質活動：fMRI 研究
Influence of visual saliency in monkey visual cortex
郷田 直一[1,2](Naokazu Goda), 原田 卓弥[1,2](Takuya Harada), 小川 正[1,2](Tadashi Ogawa),
伊藤 南[1,2](Minami Ito), 豊田 浩士[1](Hiroshi Toyoda), 定藤 規弘[1,2](Norihiro Sadato),
小松 英彦[1,2](Hidehiko Komatsu)
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P1-e29 サル一次視覚野における 5-HT1B および 5-HT2A 受容体の修飾効果
Functional effects of 5-HT1B and 5-HT2A receptors in macaque V1
定金 理[1](Osamu Sadakane), 渡我部昭哉[1](Akiya Watakabe), 小松 勇介[1](Yusuke Komatsu),
七五三木聡[2](Satoshi Shimegi), 内藤 智之[2](Tomoyuki Naito), 尾崎 弘展[2](Hironobu Osaki),
佐藤 宏道[2](Hiromichi Sato), 山森 哲雄[1](Tetsuo Yamamori)
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P1-e30 ケイジドグルタミン酸の光分解により惹起された視覚野内興奮性信号伝播の特徴
Signal propagation properties in the visual cortex evoked by flush photolysis of a caged glutamate
小山内 実(Makoto Osanai), 上垣 英聖(Hidesato Uegaki), 八木 哲也(Tetsuya Yagi)
大阪大院・工・電気電子情報

P1-e31 追跡眼球運動中のサル MT/MST 野ニューロンの活動特性
Characteristics of neuronal responses in cortical areas MT/MST of monkey during pursuit eye movements
稲場 直子(Naoko Inaba), 河野 憲二(Kenji Kawano)
京都大院・医・認知行動脳科学

P1-e32 方位マッチング課題はオプティック・フローに対する MST ニューロン応答に影響する
Match to heading tasks influence MST neuronal responses to optic flow
佐藤 暢哉[1,2,3](Nobuya Sato), Charles J. Duffy[3]
[1]科学技術振興機構, [2]日本大学医学研究科応用システム神経科学, [3]ロチェスター大学メディカルセンター神経学部門

P1-e33 注視解除による視覚探索への影響
Express visual search
加藤利佳子[1,2](Rikako Kato), 伊佐 正[1,2,3](Tadashi Isa)
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P1-e34 網膜 AII アマクリン細胞の発火特性におけるギャップ結合の生理的役割
A Physiological role of gap junctions in firing properties of retinal AII amacrine cells
田丸 文信(Fuminobu Tamalu), 渡辺 修一(Shu-Ichi Watanabe)
埼玉医大・医・生理

P1-e35 外側膝状体における代謝型グルタミン酸受容体の活動依存的調節の年齢依存性
Developmental change of activity-dependent regulation of mGluR1$\alpha$ in the lateral geniculate nucleus
吉田 三穂[1](Miho Yoshida), 佐藤 武正[2](Takemasa Satoh), 中村 公一[3](Kouichi Nakamura),
金子 武嗣[3](Takeshi Kaneko), 畠 義郎[1](Yoshio Hata)
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P1-e36 バイオロジカルモーション知覚処理の神経機序：脳磁図による検討
Neural mechanism of biological motion processing: An MEG study
平井　真洋[1,2](Masahiro Hirai)，金桶　吉起[1](Yoshiki Kaneoke)，中田　大貴[1](Hiroki Nakata)，
柿木　隆介[1](Ryusuke Kakigi)
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P1-e37 発達期視覚野においてBDNFタンパク質量はERK、CaMK、PI3K、p38MAPK、Cdk5阻害では変化しないが、PKA阻害により増加する
BDNF protein level is increased by inhibition of PKA, but not ERK, CaMK, PI3K, p38MAPK or Cdk5 in developing visual cortex
一坂　吏志[1](Satoshi Ichisaka)，仙波りつ子[2](Ritsuko Katoh-Semba)，高村　明孝[3](Hironori Takamura)，
都田　博子[4](Hiroko Miyakoda)，難波　栄二[4](Eiji Nanba)，小島　一宏[3](Kazuhiro Kojima)，
津本　忠治[5,6](Tadaharu Tsumoto)，畠　　義郎[3](Yoshio Hata)
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P1-e38 ラット一次視覚野における層特異的脳梁結合
Laminar specificity of callosal connections in the rat striate cortex
佐藤　武正[1](Takemasa Satoh)，畠　　義郎[2](Yosio Hata)
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P1-e39 タライラッハ空間におけるヒト視覚野位置の不一致性と不確定性
Inconsistency and uncertainty in the locations of human visual areas in Talairach space
山本　洋紀[1](Hiroki Yamamoto)，福永　雅喜[2](Masaki Fukunaga)，田中　忠蔵[3](Chuzo Tanaka)，
梅田　雅宏[2](Masahiro Umeda)，江島　義道[4](Yoshimichi Ejima)
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P1-e40 細胞外電気刺激による網膜神経節細胞の興奮に関して - 計算論的および実験的解析 -
On the direct activation of retinal ganglion cells by extracellular electrical stimulation - computational and experimental analyses -
小佐井貴雄(Takao Kosai)，本村　珠美(Tamami Motomura)，林田　祐樹(Yuki Hayashida)，
村山　伸樹(Nobuki Murayama)
熊本大院・自然科学研究科・情報電気電子工学

P1-e41 自由行動下におけるラット外側膝状体神経活動の慢性記録
Chronic recording of neural activity of LGN in behaving rat.
赤崎　孝文(Takafumi Akasaki)，畠　　義郎(Yoshio Hata)
鳥取大院・医・生体高次

P1-e42 ノイズの付加がパターン弁別に及ぼす効果
Effect of noise on pattern discrimination
佐々木　仁[1](Hitoshi Sasaki)，坂根　貞嗣[1](Sadatsugu Sakane)，石田　卓也[1](Takuya Ishida)，
轟原　正義[2](Masayoshi Todorokihara)，北村　多平[3](Tahei Kitamura)，青木　亮三[1](Ryozo Aoki)
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P1-e43 新規網膜神経細胞試料における自発性膜電位変化
Spontaneous membrane voltage changes in retinal neurons dissociated by a new method
本村　珠美(Tamami Motomura)，林田　祐樹(Yuki Hayashida)，村山　伸樹(Nobuki Murayama)
熊本大学大学院自然科学研究科

P1-e44 ラット網膜における電位依存性ナトリウムチャネルサブタイプの発現パターン
Expression pattern of voltage-gated sodium channel subtypes in the rat retina
金子　優子(Yuko Kaneko)，渡辺　修一(Shu-Ichi Watanabe)
埼玉医科大学・医・生理

P1-e45 眼優位性可塑性における抑制回路の動的役割：発達過程における視覚皮質の回路モデル
Dynamic role of inhibitory circuits in ocular dominance plasticity: A network model of the developing visual cortex
深井 朋樹[1](Tomoki Fukai), 姜 時友[1](Siu Kang), 矢崎 - 杉山陽子[2](Yoko Yazaki-Sugiyama),
加藤 英之[1](Hideyuki Cateau), ヘンシュ貴雄[2,3](Takao K Hensch)
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## 聴覚、前庭感覚 1
## Auditory and Vestibular Systems 1

P1-e46 Modifications in perineuronal nets of rat vestibular nuclear neurons during postnatal development and in injury
Chun Wai Ma[1], Chun Hong Lai[1], Daisy Kwok Yan Shum[2], Ying Shing Chan[1]
[1]Department of Physiology, The University of Hong Kong, [2]Department of Biochemistry, The University of Hong Kong

P1-e47 Responsiveness of thalamic neurons to rotational and translational motions in the horizontal plane
K.P. Ng, C.H. Lai, Y.C. Tse, Y.S. Chan
Department of Physiology, The University of Hong Kong, Hong Kong, China

P1-e48 Developmental changes in excitatory and inhibitory transmission at central vestibular synapses of rats
Y.S. Chan[1], YC Tse[1], SK Lai[1], C.H. Lai[1], WH Yung[2]
[1]Dept of Physiology, The University of Hong Kong, Hong Kong, [2]Dept of Physiology, The Chinese University of Hong Kong.

P1-f01 定常聴覚刺激提示時における聴覚磁場応答
Effect of implicit response in Auditory MEG measurement
冨田 教幸[1](Noriyuki Tomita), 田森 佳秀[2](Yoshi Tamori)
[1]金沢工業大学大学院・工学研究科・システム設計工学専攻, [2]人間情報システム研究所

P1-f02 ピッチ調整の聴覚フィードバック及びフィードフォワード制御に関わる神経基盤
Neural substrates involved in auditory feedback and feed-forward controls of pitch modulation
豊村 暁[1,2](Akira Toyomura), 小山 幸子[1,2](Sachiko Koyama), 宮本 環[1](Tamaki Miyamoto),
寺尾 敦[1](Atsushi Terao), 大森 隆司[3](Takashi Omori), 室橋 春光[1](Harumitsu Murohashi),
栗城 眞也[1](Shinya Kuriki)
[1]北海道大学, [2]科学技術振興機構, [3]玉川大学

P1-f03 純音に挿入された時間的 gap に対する聴覚誘発反応：脳波と脳磁図の同時計測
Neural Responses to Gap Embedded in Continuous Sound: Simultaneous Recordings of MEG and EEG.
豊巻 敦人[1](Atsuhito Toyomaki), 小山 幸子[1,2](Sachiko Koyama), 豊澤 悠子[2](Yuko Toyosawa),
竹内 文也[1](Fumiya Takeuchi), 竹市 博臣[3](Hiroshige Takeichi), 栗城 慎也[1](Shinya Kuriki)
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P1-f04 コウモリの聴覚系における周波数処理の皮質下降性変調の神経機構
Neural mechanism of corticofugal modulation of frequency processing in bat auditory system
樫森与志喜[1,2](Yoshiki Kashimori), 廣岡 成一[2](Seiichi Hirooka), 藤田 一寿[2](Kazuhisa Fujita)
[1]電気通信大・量子物質工学科, [2]電気通信大院・情報システム研究科

P1-f05 スナネズミの聴覚皮質における神経活動に関する音刺激の立ち上がり部分の振幅変化の効果
Effect of a change in amplitude at sound onset on activities of auditory cortical neurons in the Mongolian gerbils
辻　将也(Masaya Tsuji)
同志社大院・工・知識専攻

P1-f06 アブラコウモリのエコーロケーション音に対する下丘の神経反応
Neural responses of inferior colliculus to echolocation sounds in Japanese house bat (*Pipistrellus abramus*).
後藤　和廣[1](Kazuhiro Goto)，飛龍志津子[2](Shizuko Hiryu)，力丸　裕[1](Hiroshi Riquimaroux)
[1]同志社大院・工・知識工学，[2]同志社大学　研究開発推進機構

P1-f07 メスキンカチョウ聴覚回路における最初期遺伝子 Arc の発現パターン解析
*Arc* mRNA expression pattern in female zebra finch auditory pathway
藤村　彬(Akira Fujimura)，堀田　耕司(Koji Hotta)，岡　浩太郎(Kotaro Oka)
慶應義塾大院・基礎理工・生命システム情報

P1-f08 下丘に発現する P2X1
P2X1 in the inferior colliculus
瀧　公介(Kousuke Taki)，工藤　基(Motoi Kudo)
滋賀医大・解剖

P1-f09 音の特徴の情報処理に音楽経験が与える影響
Effects of musical experience on the automatic auditory feature processing
小野健太郎[1](Kentaro Ono)，中村　昭範[1](Akinori Nakamura)，吉山　顕次[2](Kenji Yoshiyama)，
綿古里武志[1](Takeshi Kinkori)，文堂　昌彦[3](Masahiko Bundo)，伊藤　健吾[1](Kengo Ito)
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P1-f10 言語音認識機構のラットモデル：聴覚野に依存する合成言語音の弁別学習
A rat model for investigating speech sound recognition in humans: discrimination learning of synthetic speech sounds dependent on the presence of the auditory cortex
工藤　雅治(Masaharu Kudoh)，羽深　将人(Masato Habuka)，菱田　竜一(Ryuichi Hishida)，
澁木　克栄(Katsuei Shibuki)
新潟大・脳研・システム脳生理

P1-f11 光学的計測によるモルモット聴覚皮質コア領域とベルト領域の機能的接続
Functional connections between the core and belt fields of the guinea pig auditory cortex observed by optical imaging
堀川　順生(Junsei Horikawa)，沼田　亮太(Ryota Numata)，内山　大輔(Daisuke Uchiyama)，
杉本　俊二(Shunji Sugimoto)
豊橋技科大・知識情報

P1-f12 聴覚属性の背景的および自発的皮質表現
Ongoing and spontaneous cortical representations of auditory attributes
吉田　崇将[1](Takamasa Yoshida)，桂　卓成[1,2](Takusige Katura)，山崎　享子[2](Kyoko Yamazaki)，
田中　繁[3](Shigeru Tanaka)，岩本　光正[1](Mitsumasa Iwamoto)，田中　尚樹[1,2](Naoki Tanaka)
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## 嗅覚、味覚、化学感覚 1
## Olfaction, Taste, Chemical Senses 1

P1-f13 舌の侵害性刺激が及ぼす味覚への影響の検討
A study of the influences on tastes by oral noxious stimuli
石田　雄介(Yusuke Ishida)，鵜川　眞也(Shinya Ugawa)，植田　高史(Takashi Ueda)，
島田　昌一(Shoichi Shimada)
名古屋市立大学院・医・分子形態学

P1-f14 ツメガエル V1R 遺伝子群は主嗅覚系で発現する
Amphibian V1R gene family is expressed in the *Xenopus* main olfactory system
伊藤 敦子[1,2](Atsuko Date-Ito), 市川 眞澄[3](Masumi Ichikawa), 森 裕司[2](Yuji Mori),
山岸 公子[1](Kimiko Hagino-Yamagishi)
[1]都臨床研・一分子, [2]東大院・農学生命科学, [3]都神経研・基盤

P1-f15 鋤鼻ニューロンの投射がマウス副嗅球の糸球体形成に果たす役割
Vomeronasal axons play a key role in the glomerular formation of the accessory olfactory bulb.
横須賀 誠[1](Makoto Yokosuka), 佐原 資謹[2,3](Yoshinori Sahara), 小牟田 縁[2](Yukari Komuta),
斎藤 徹[1](Toru Saito), 市川 眞澄[4](Masumi Ichikawa), 中村 俊[2](Shun Nakamura)
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P1-f16 誘導性 Cre/loxP システムを用いた嗅球 mitral/tufted cell の単一細胞レベルのラベリング
Transgenic mice expressing tamoxifen-inducible Cre recombinase in mitral/tufted cells of the olfactory bulb
米倉淳一郎(Junichiro Yonekura), 横井 峰人(Mineto Yokoi)
京大院・医

P1-f17 ラット前嗅核における左右匂い入力に対する緻密な調整について
Close co-ordination of binasal inputs in the rat anterior olfactory nucleus
菊田 周[1,2](Shu Kikuta), 柏谷 英樹[1](Hideki Kashiwadani), 加我 君孝[2](Kimitaka Kaga),
森 憲作[1](Kensaku Mori)
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P1-f18 マウスにおける揮発性化学物質の嗅覚検知閾値
Olfactory detection threshold of ordinary volatile chemicals in mice
黒河 佳香(Yoshika Kurokawa), 北條理恵子(Rieko Hojo), 中島 大介(Daisuke Nakajima),
藤巻 秀和(Hidekazu Fujimaki)
独立行政法人国立環境研究所

P1-f19 嗅球における c-Fos 陽性ニューロンの分布に対する遠心性入力の影響
Effects of centrifugal inputs on distribution of c-Fos immunoreactive neurons in the olfactory bulb
松谷 伸二(Shinji Matsutani)
北里大・看護・機能形態

P1-f20 刺激依存的に嗅覚2次神経の可塑性を司る分子の探求
Activity-dependent expression of semapholin 7A in the mouse olfactory bulb
井上 展子(Nobuko Inoue), 芹沢 尚(Shou Serizawa), 今井 猛(Takeshi Imai),
坂野 仁(Hitoshi Sakano)
東京大学理学系研究科生物化学専攻

P1-f21 一対の鏡像異性体分子に応答するマウス嗅覚受容体の最大多数タイプはヒトが高感度となる異性体に高感度を示す
The largest subtype of murine odorant receptors responsive to a pair of enantiomers is more sensitive to a more potent partner of the enantiomers in human perception
廣野 順三[1](Junzo Hirono), 浜名 洋[1](Hiroshi Hamana), 江村 誠[2](Makoto Emura),
佐藤 孝明[1](Takaaki Sato)
[1]産業技術総合研究所・セルエンジニアリング, [2]高砂香料工業・研究開発本部

P1-f22 Mitral cell layer 形成における sema6A の機能解析
Possible roles of *sema6A* in the mitral cell layer formation
井ノ口 霞(Kasumi Inokuchi), 竹内 春樹(Haruki Takeuchi), 芹沢 尚(Shou Serizawa),
坂野 仁(Hitoshi Sakano)
東京大学大学院・理・生物化学

P1-f23 細胞認識分子の組み合わせによる嗅神経細胞の軸索分離
Combinatorial code of cell- recognition molecules for the axonal segregation of olfactory sensory neurons.
中嶋　藍(Ai Nakashima)，竹内　春樹(Haruki Takeuchi)，芹沢　尚(Shou Serizawa)，
坂野　仁(Hitoshi Sakano)
東京大院・理・生化

P1-f24 *Pax6* 変異ヘテロ接合マウスの嗅球における介在神経細胞の減少
Decreased interneurons in the *Pax6* mutant mouse olfactory bulb
羽場　芙弥[1](Hasumi Haba)，野村　真[1](Tadashi Nomura)，大隅　典子[1,2](Noriko Osumi)
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P1-f25 マウス培養鋤鼻細胞中のネスチン陽性細胞増殖に対する主要尿タンパク質の効果
Major urinary proteins increase the number of nestin-positive cells in mouse vomeronasal cell cultures
権　蓉丹[1](Rongdan Quan)，村本　和世[1](Kazuyo Muramoto)，椛　秀人[1,2](Hideto Kaba)
[1]高知大・医・生理，[2]生理研・環境適応機能発達

P1-f26 オキシトシンは副嗅球における長期増強の誘導を促進する
Oxytocin facilitates the induction of long-term potentiation in the accessory olfactory bulb
方　龍雲[1](Long-Yun Fang)，椛　秀人[1,2](Hideto Kaba)
[1]高知大・医・生理，[2]生理研・環境適応機能発達

P1-f27 げっ歯類鋤鼻系における ESP ペプチドの受容
The reception of ESP peptides in rodent vomeronasal neurons
垉紗智子(Sachiko Haga)，木本　裕子(Hiroko Kimoto)，柳川　太一(Taichi Yanagawa)，佐藤　幸治(Koji Sato)，
東原　和成(Kazushige Touhara)
東京大院・新領域・先端生命科学

P1-f28 神経活動の時空間パターンを用いた濃度不変な匂い表現の計算論
Olfactory computation using spatiotemporal pattern of network activity for odor representation
牧野　悌也(Yoshinari Makino)，安池　誠(Makoto Yasuike)，中　雄樹(Yuki Naka)，
三浦　治己(Haruki Miura)，矢野　雅文(Masafumi Yano)
東北大学・電気通信研究所

P1-f29 ヒスタミンによる匂い応答の修飾
Histamine modulates olfactory response in the olfactory epithelium
大熊　真人[1](Mahito Ohkuma)，三村　英也[2](Hideya Mimura)，今田　英己[1](Hideki Imada)，
河合　房夫[1](Fusao Kawai)，内藤　健晴[2](Kensei Naito)，宮地　栄一[1](Ei-ichi Miyachi)
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## 感覚運動系の可塑性
## Sensori-Motor Plasticity

P1-f30 Plasticity for functional restoration of forelimb movements following neonatal unilateral decortication.
A. Vattanajun[1], M. Takahashi[2,3], K. Isa[3], T. Isa[3,4]
[1]Dept. Physiol., Phramongkutklao Col. Med., Bangkok, Thailand, [2]Dept. Orthopaedic Surgery, Kyorin Univ., Mitaka, Japan, [3]Dept. Dev. Physiol., Natl. Inst. Physiol. Sci., Okazaki, Japan, [4]Grad. Univ. Advanced Studies, Hayama, Japan

P1-f31 投擲運動のプリズム適応における視覚情報の効果：投擲物の視覚情報が適応に与える影響
Role of visual information on prism adaptation of throwing movements: The effect of visual information of the thrown ball
石川　拓海(Takumi Ishikawa)，阪口　豊(Yutaka Sakaguchi)
電気通信大学大学院　情報システム学研究科

P1-f32 熟練ピアニストによる打鍵動作時の主働筋・拮抗筋間のシナジーの利用
Expert pianists exploit an efficient agonist-antagonist muscular synergy when striking the keys on the piano.
古屋 晋一(Shinichi Furuya), 木下 博(Hiroshi Kinoshita)
大阪大院・医・予防環境医学

P1-f33 ゼブラフィッシュの逃避運動を誘発する感覚入力の発達に伴った再編成
Remodeling of sensory nerve projection onto Mauthner cell during development in zebrafish larva
谷本 昌志(M. Tanimoto), 小橋 常彦(T. Kohashi), 小田 洋一(Y. Oda)
名古屋大院・理・生命理

P1-f34 皮質脊髄路損傷後に大脳皮質活動とは独立した EMG-EMG コヒーレンスは増大する
Increase of intermuscular coherence independent of cortical activity during recovery from corticospinal tract lesion in monkeys
西村 幸男[1,2](Yukio Nishimura), 伊佐 正[1,2](Tadashi Isa)
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P1-f35 外側皮質脊髄路損傷後の回復過程における神経成長関連タンパク GAP-43 の脳内発現
Gene expression of GAP-43 in the monkey motor cortex after lesion of the lateral corticospinal tract
肥後 範行[1,2](Noriyuki Higo), 西村 幸男[2,3](Yukio Nishimura), 村田 弓[1,2,4](Yumi Murata),
大石 高生[1,2,5](Takao Oishi), 齋藤紀美香[3](Kimika Saito), 高橋 雅人[3,6](Masahito Takahashi),
坪井 史治[3,7](Fumiharu Tsuboi), 伊佐 正[2,3](Tadashi Isa)
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P1-f36 サル皮質脊髄路：一次運動野における手指制御領域から脊髄への軸索投射の定量的解析
Quantitative analysis of corticospinal projection from digit area of motor cortex of rhesus monkey
齋藤紀美香[1](Kimika Saito), 西村 幸男[1,2](Yukio Nishimura), 大石 高生[2,3](Takao Oishi),
肥後 範行[2,4](Noriyuki Higo), 村田 弓[2,4](Yumi Murata), 伊佐 正[1,2](Tadashi Isa)
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P1-f37 求心路遮断性疼痛に対する鏡療法の有効性
Willed visuomotor imagery alleviates deafferentation pain: Possible implications of mirror visual feedback treatment
住谷 昌彦[1](Masahiko Sumitani), 宮内 哲[2](Satoru Miyauchi), 齋藤 洋一[3](Youichi Saitoh),
柴田 政彦[4](Masahiko Shibata), 眞下 節[1](Takashi Mashimo)
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（＊P1-f37は発表者の都合により，9月12日にP3-k15として発表されます。）

## 自律神経系 1
## Autonomic Nervous System 1

P1-f38 自然発症性高血圧ラットの延髄孤束核における炎症性分子の遺伝子発現プロファイル
Gene expression profile of inflammatory molecules in the nucleus tractus solitarii of the spontaneously hypertensive rat (SHR)
和気 秀文[1](Hidefumi Waki), Sabine Gouraud[1], 前田 正信[1](Masanobu Maeda), Julian F.R. Paton[2]
[1]和歌山県立医科大学・医・第二生理, [2]英国ブリストール大学生理学講座

P1-f39 感情制御方略が予期不安時の自律神経活動に与える影響
Influence of emotion regulation on autonomic activity during anticipatory anxiety
村上 裕樹(Hiroki Murakami), 大平 英樹(Hideki Ohira), 松永 昌宏(Masahiro Matsunaga)
名古屋大院・環境学・心理

P1-f40 アペリン-12の腹腔内投与後脳内ニューロンにおけるc-Fos蛋白の発現
Neuronal expression of c-Fos protein in the brain after intraperitoneal injection of apelin-12 in Wistar rats
高山 清茂(Kiyoshige Takayama), 岩崎 裕美(Hiromi Iwazaki)
群馬大・医・保健学科

P1-f41 演題取下げ
Withdrawn

P1-f42 体液量増加による正中視索前核のノルアドレナリン放出調節へのGABA受容体機構の関与
Involvement of GABA receptor mechanisms in the control of noradrenaline release in the median preoptic nucleus in response to volume expansion in rats
宮久保浩子[1](Hiroko Miyakubo), 田中 淳一[1](Junichi Tanaka), 林 泰資[2](Yasushi Hayashi),
野村 正彦[3](Masahiko Nomura)
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P1-f43 カプシエイトは末梢交感神経系を局所的に活性化する
Capsiate topically activates peripheral sympathetic nervous system
小野 郁(Kaori Ono), 原 佳子(Yoshiko Hara), 安居 昌子(Masako Yasui),
高橋 迪雄(Michio Takahashi)
味の素株式会社・健康基盤研究所

P1-f44 カプシエイトは褐色脂肪組織の交感神経活動を亢進する
Capsiate enhances sympathetic nerve activity of brown adipose tissue
安居 昌子[1](Masako Yasui), 小野 郁[1](Kaori Ono), 原 佳子[1](Yoshiko Hara),
永島 計[2](Kei Nagashima), 高橋 迪雄[1](Michio Takahashi)
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P1-f45 カメの脊髄神経節ー交感神経節複合体におけるシナプス関連タンパク質の免疫組織化学的検索
Chelonian spinal nerve-sympathetic ganglial complex: A synapsin I and synaptic vesicle protein immunohistochemical study.
門田 哲夫(Tetsuo Kadota), 中野 真人(Masato Nakano), 跡部 好敏(Yoshitoshi Atobe), R.C. Goris,
船越 健悟(Kengo Funakoshi)
横浜市大・医・神経解剖

P1-f46 サルの尾側延髄における呼吸性ニューロン
Respiratory neurons in the caudal VRG of the monkey
佐々木秀一[1](Shuichi Sasaki), 小国 英一[2](Eiichi Oguni), 佐々木誠一[3](Seiichi Sasaki)
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P1-f47 体表面の局所加温が冷え性日本人女性の生理的機能に及ぼす影響
Effects of local heating of body surface on physiological functions in Japanese women sensitive to the cold
安井 宏[1,2](Hiroshi Yasui), 梅野 克身[1,2](Katsumi Umeno), 堀 悦郎[1,2](Etsuro Hori),
永嶋 義直[3](Yoshinao Nagashima), 矢田 幸博[3](Yukihiro Yada), 鈴木 敏幸[3](Toshiyuki Suzuki),
小野 武年[1,2](Taketoshi Ono), 西条 寿夫[1,2](Hisao Nishijo)
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P1-f48 ヒスタミンH1受容体欠損マウスにおける換気量の日内変動
Circadian profile of ventilation in histamine H1 receptors lacking mice
岩瀬みち子(Michiko Iwase), 石黒 卓(Takashi Ishiguro), 金丸みつ子(Mitsuko Kanamaru),
泉崎 雅彦(Masahiko Izumizaki), 大嶋 康義(Yasuyosi Ohshima), 本間 生夫(Ikuo Homma)
昭和大学 医 第二生理

P1-g01 視床下部室傍核および青斑核の下行性自律神経線維におけるCOX発現は脳室内投与CRFにより活性化する
Activation of COX isozymes in spinally projecting neurons in the PVN and LC by centrally administered CRF in rats
山口奈緒子[1](Naoko Yamaguchi), 岡田尚志郎[1](Shoshiro Okada), 清水 孝洋[1](Takahiro Shimizu), 臼井 大介[1,2](Daisuke Usui), 横谷 邦彦[1](Kunihiko Yokotani)
[1]高知大・医・薬理, [2]高知大・医・小児思春期医学

P1-g02 発達小脳に現れる呼吸性神経活動
Neonatal cerebellum broadly displays respiratory activities
荒田 晶子(Akiko Arata), 藤井 盛光(Morimitsu Fujii), 伊藤 正男(Masao Ito)
理化学研究所・脳科学センター・記憶学習チーム

## 神経免疫 Neuroimmunology

P1-g03 プロテオーム解析による末梢神経炎の新規自己抗原候補解析
Identification of new antigenic candidates in autoimmune neuropathies by serological proteome analysis
遠藤 真澄(Masumi Endoh)
国立感染症研究所ハンセン病研究センター

P1-g04 実験的自己免疫性脳脊髄炎（EAE）におけるCD4及びCD8陽性細胞T細胞サブセットの時系列的変化
Chronological changes of $CD4^+$ and $CD8^+$ T cell subsets in the periphery and CNS during the course of EAE
薗部 佳史(Yoshifumi Sonobe), 金 世杰(Shijie Jin), 王 金岩(Jinyan Wang),
川ノ口 潤(Jun Kawanokuchi), 竹内 英之(Hideyuki Takeuchi), 水野 哲也(Tetsuya Mizuno),
錫村 明生(Akio Suzumura)
名古屋大・環医研・神経免疫

P1-g05 The effects of viscero-somatic interactions on thalamic mast cell recruitment in rat developing cystitis
David Dubayle[1], Jacques Serviere[2], Daniel Menetrey[1]
[1]Laboratory of Neurophysics and Physiology - CNRS UMR 8119, [2]INRA INA-PG

## 本能と情動行動 Instinct and Emotional Behavior

P1-g06 トリプトファン2,3,ジオキシゲナーゼ（TDO）遺伝子欠損マウスは、全身／脳内トリプトファン代謝と高次脳機能が修飾される
Alteration of systemic/brain tryptophan metabolism and modulation of anxiety-related behavior in tryptophan 2,3-dioxygenase (TDO)-deficient mice
金井 将昭(Masaaki Kanai), 船越 洋(Hiroshi Funakoshi), 高橋 寿明(Hisaaki Takahashi),
中村 敏一(Toshikazu Nakamura)
大阪大院・医学系研究科・生化学・分子生物学・分子再生医学分野

P1-g07 5HTTLPR s/s遺伝子型を持つ人々はロマンティック？
Healthy carriers of the 5HTTLPR s/s genotype are romantic?
松永 昌宏[1,2](Masahiro Matsunaga), 磯和 勅子[1,3](Tokiko Isowa), 木村 健太[1](Kenta Kimura),
宮腰 誠[1](Makoto Miyakoshi), 金山 範明[1](Noriaki Kanayama), 村上 裕樹[1](Hiroki Murakami),
小長谷敏浩[2](Toshihiro Konagaya), 福山 誠介[4,5](Seisuke Fukuyama), 篠田 淳[4](Jun Shinoda),
山田 實紘[4](Jitsuhiro Yamada), 大平 英樹[1](Hideki Ohira)
[1]名古屋大院・環境学・心理, [2]愛知医科大・消化器内科, [3]三重県立看護大・老年看護, [4]木沢記念病院・中部療護センター, [5]神奈川歯科大・生理

P1-g08 同種他個体による恐怖条件反応の緩和作用
Two types of social buffering differentially attenuates conditioned fear responses in male rats
清川　泰志[1,2](Yasushi Kiyokawa)，菊水　健史[1](Takefumi Kikusui)，武内ゆかり[1](Yukari Takeuchi)，
森　裕司[1](Yuji Mori)
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P1-g09 危険予測能力における X11L の役割
A possible role for an adaptor protein, X11L/mint2, in risk assessment
佐野　良威[1](Yoshitake Sano)，Chad Boulay[1]，鈴木　利治[2](Toshiharu Suzuki)，Niall P. Murphy[1]，
糸原　重美[1](Shigeyoshi Itohara)
[1]理研・脳センター，[2]北大院・薬

P1-g10 ASK1 ノックアウトマウスの行動的特徴
Behavioral characteristics of ASK1 KO mice
熊倉かれん[1](Karen Kumakura)，野村　洋[1](Hiroshi Nomura)，野口　拓也[2](Takuya Noguchi)，
一條　秀憲[2](Hidenori Ichijo)，松木　則夫[1](Norio Matsuki)
[1]東京大院・薬・薬品作用学，[2]東京大院・薬・細胞情報学

P1-g11 前脳特異的αCaMKII 過剰発現マウスにおける不安障害発現のメカニズムの解析
Mechanisms of increase in anxiety by overexpressing of CaMKII in forebrain
古市　隆大[1](Takahiro Furuichi)，遠藤　健吾[1](Kengo Endo)，吉田　太郎[1](Taro Yoshida)，
佐度　恵[1](Megumi Sado)，梶井　靖[2](Yasushi Kajii)，喜田　聡[1](Satoshi Kida)
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P1-g12 ラット扁桃体中心核から中脳中心灰白質を経て疑核後核に至る神経路の化学的性質
Chemical property of the neural pathways from the central amygdaloid nucleus to the nucleus retroambiguus via the midbrain periaqueductal gray in the rat
岡　達郎(Tatsuro Oka)，津森登志子(Toshiko Tsumori)，横田　茂文(Shigefumi Yokota)，
安井　幸彦(Yukihiko Yasui)
島根大学・医・神経形態学

P1-g13 内側膝状体周辺領域の構成
Organization of the regions surrounding the medial geniculate nucleus
本村　啓介[1](Keisuke Motomura)，小坂　俊夫[2](Toshio Kosaka)
[1]九州大院・医・精神病態，[2]九州大院・医・神経形態

P1-g14 報酬予測脳活動に対する知覚的曖昧性の影響
Brain activity for reward prediction on ambiguous perception
山本　愛実[1](Manami Yamamoto)，奥田　次郎[2](Jiro Okuda)，鮫島　和行[2](Kazuyuki Samejima)，
Schultz Wolfram[3]，坂上　雅道[2](Masamichi Sakagami)
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P1-g15 横臥時におけるブタ海馬の局所集合電位の解析
Analysis of local field potentials in the hippocampus of piglets during rest
齋藤　敏之[1](Toshiyuki Saito)，藤原　清悦[2](Sei-etsu Fujiwara)，今野兼次郎[3](Kenjiro Konno)，
山口　峻司[4](Takashi Yamaguchi)，伊崎　義憲[2](Yoshinori Izaki)，明間　立雄[2](Tateo Akema)
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P1-g16 温熱的感覚の部位差
The regional difference in temperature related sensations
中村真由美[1](Mayumi Nakamura)，依田　珠江[2](Tamae Yoda)，安原　祥[3](Saki Yasuhara)，
斉藤　恭世[3](Yasuyo Saito)，春日　桃子[1](Momoko Kasuga)，永島　計[3](Kei Nagashima)，
Larry I. Crawshaw[4]，彼末　一之[1,3](Kazuyuki Kanosue)
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P1-g17 アロマターゼノックアウトマウスにおける回転車活動量の分析
Analyses of running wheel activity (RWA) in aromatase-knockout (ArKO) mice
渡井 浩太[1](Kota Watai), 津田夢芽子[1](Mumeko Tsuda), 仲田真理子[1](Mariko Nakata),
戸田 勝巳[2](Katsumi Toda), 小川 園子[1](Sonoko Ogawa)
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P1-g18 視床下部の防御反応部位から小脳の限局した微小帯域のプルキンエ細胞への興奮性作用はオレキシン作動性線維が介する。
Orexinergic neurons mediate excitatory signals to Purkinje cells in a descrete microzone of the cerebellum
西丸 直子[1](Naoko Nisimaru), 白井 良徳[1](Yoshinori Shirai), 藤井 盛光[1](Morimitsu Fujii),
桜井 武[2](Takeshi Sakurai), 山本 三幸[2](Miyuki Yamamoto), 伊藤 正男[1](Masao Ito)
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P1-g19 雌マウス性行動の調節に果たすエストロゲンレセプター・ベータ (ER-$\beta$) の役割
Role of estrogen receptor b (ERb) and sexual experience in female reproductive behavior behavior in mice
肖 凱(Kai Xiao), 津田夢芽子(Mumeko Tsuda), 小川 園子(Sonoko Ogawa)
筑波大院・人間総合科学

P1-g20 離乳後の母ラットにおけるエストロゲン抗不安および抗うつ作用
Anxiolytic and antidepressant effects of estradiol in postpartum rats after weaning pup
古田 都[1](Miyako Furuta), 二宮 碧[2](Midori Ninomiya), 功刀 浩[1](Hiroshi Kunugi)
[1]国立精神神経センター・神経研究所・疾病研究第3部, [2]早稲田大学 理工学部

P1-g21 側坐核におけるドーパミン代謝に対する腹側被蓋野への CART 投与効果
Dopamine metabolism in the nucleus accumbens induced by injection of CART peptide into the ventral tegmental area in rtas
佐々木和男(Kazuo Sasaki), 村上 由紘(Yoshihiro Murakami), 望月 崇裕(Takahiro Mochizuki),
金 主賢(Juhyon Kim), 中島 一樹(Kazuki Nakajima)
富山大院・理工学研究部

P1-g22 コモンマーモセットの視覚探索行動の強さを調べるための新しいテスト
A new test to measure the strength of visual exploration in common marmoset
山中 創[1](Hajime Yamanaka), 石橋 英俊[1](Hidetoshi Ishibashi), 中村 克樹[1,2](Katsuki Nakamura)
[1]国立精神・神経センター 神経研究所, [2]科学技術振興機構 戦略的創造研究推進事業

## 学習、記憶 1
## Learning and Memory 1

P1-g23 注意の瞬き発生時において新規な光景をエンコードするために増加するシータ脳波パワー
Theta EEG power enhancement for encoding novel scene at attentional blink period
水原 啓暁[1](Hiroaki Mizuhara), 山口 陽子[2](Yoko Yamaguchi)
[1]京都大院・情報学・知能情報学, [2]理化学研究所・脳科学総合研究センター・創発知能ダイナミクス研究チーム

P1-g24 前脳特異的な CREB 活性化による記憶固定化能力向上のメカニズムの解析
Enhancement of memory formation and LTP by up-regulation of CREB activity
鈴木 章円[1](Akinobu Suzuki), 福島 穂高[1](Hotaka Fukushima), Min Zhuo[2], 喜田 聡[1](Satoshi Kida)
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P1-g25 海馬体における長期増強の誘導・維持に関する動物種差
Comparison of hippocampal LTP induction and maintenance between monkeys and rodents
田村 了以[1](Ryoi Tamura), 永福 智志[1](Satoshi Eifuku), 北村 貴志[1](Takashi Kitamura),
上野 照子[1](Teruko Uwano), 小野 武年[2](Taketoshi Ono)
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P1-g26 ショウジョウバエ細胞接着因子 Klingon は長期記憶形成に必要であり、Notch シグナル活性により制御されている
The Drosophila cell-adhesion molecule Klingon is required for long-term memory and is regulated by Notch signaling activity
松野　元美[1](Motomi Matsuno), Tim Tully[2], 齋藤　実[1,3](Minoru Saitoe)
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P1-g27 海馬 CA3 神経回路の連想的神経活動は活動依存的に組織化される
Activity-dependent organization of associative neuronal dynamics of the hippocampal CA3 network in vitro
菊池　修(Osamu Kikuchi), 片山　統裕(Norihiro Katayama), 辛島　彰洋(Akihiro Karashima),
中尾　光之(Mitsuyuki Nakao)
東北大院・情報科学・バイオモデリング論

P1-g28 ワモンゴキブリの長期記憶形成における NO の役割
The role of NO in the formation of long-term memory in cockroaches
倉持　友和(Tomokazu Kuramochi), 渡邉　英博(Hidehiro Watanabe), 水波　誠(Makoto Mizunami)
東北大・院生命科学

P1-g29 ERK は恐怖記憶の形成に必要である。
ERK is required for fear memory formation
原田　武志[1](Takeshi Harada), 城山　優治[2](Yuji Kiyama), 中尾　和貴[3](Kazuki Nakao),
真鍋　俊也[2](Toshiya Manabe), 饗場　篤[1](Atsu Aiba)
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P1-g30 受動的回避反応課題を用いた記憶再固定化と記憶強化の解析
Mechanisms of memory reconsolidation associated with memory enhancement in passive avoidance task
福島　穂高(Hotaka Fukushima), 鈴木　章円(Akinobu Suzuki), 喜田　聡(Satoshi Kida)
東農大院・農・バイオ

P1-g31 物体認知記憶固定化・再固定化に対する遺伝子発現制御の役割の解析
Roles of gene expression regulation in consolidation and reconsolidation of object recognition memory
岡野絵美子(Emiko Okano), 鈴木　章円(Akinobu Suzuki), 遠藤　健悟(Kengo Endoh),
喜田　聡(Satoshi Kida)
東農大院・農・バイオ

P1-g32 ラット海馬 CA1 錐体細胞における異シナプス連合性 LTP の樹状突起膜電位の光計測
Optical imaging of dendritic propagating potential in heterosynaptic associative LTP of rat hippocampal CA1 pyramidal neurons
米山　誠[1](Makoto Yoneyama), 福島　康弘[2](Yasuhiro Fukushima), 山崎　吉之[2](Yoshiyuki Yamazaki),
塚田　稔[2](Minoru Tsukada)
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P1-g33 転写因子 BMAL1 は記憶形成に重要な役割を担う
Transcription factor BMAL1 plays critical roles in memory formation
長谷川俊介(Shunsuke Hasegawa), 細田　浩司(Hiroshi Hosoda), 喜田　聡(Satoshi Kida)
東農大・応生科・バイオサイエンス

P1-g34 レチノイン酸受容体の記憶・学習に対する役割の解析
Roles of retinoic acid receptors in learning and memory
榎本　初音(Hatune Enomoto), 崔　泰樹(Tesu Choi), 内田　周作(Shusaku Uchida),
宮尾　貴久(Takahisa Miyao), 喜田　聡(Satoshi Kida)
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P1-g35 空間記憶の再固定化・消去のメカニズムの解析
Mechanism of reconsolidation and extinction of spatial memory
金　亮(Ryang Kim)，吉田有理紗(Yurisa Yoshida)，鈴木　章円(Akinobu Suzuki)，
喜田　聡(Satoshi Kida)
東京農大院・農・バイオサイエンス

P1-g36 恐怖記憶再固定化・消去の相互作用メカニズムの解析
Mechanisms of reconsolidation and extinction of contextual fear memory
間宮　則(Nori Mamiya)，鈴木　章円(Akinobu Suzuki)，喜田　聡(Satoshi Kida)
東農大・応生科・バイオ

P1-g37 イントロンプローブを用いた in situ hybridization 法による最初期遺伝子の発現解析とその利用
Use of intron and exon probes to characterize the expression of immediate early genes
井澤　傑[1](Takeshi Izawa)，雲出　佑[1](Yu Kumode)，山森　哲雄[2](Tetsuo Yamamori)，
八木　健[1](Takeshi Yagi)，木津川尚史[1](Takashi Kitsukawa)
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P1-g38 条件性連合学習におけるラットの海馬と新皮質ニューロン集団活動のアトラクタ変動性
Attractor variability of neuronal population activity in hippocampus and neocortex of rats during conditional association learning
原　康治郎[1,2](Kojiro Hara)，高橋　晋[2](Susumu Takahashi)，櫻井　芳雄[1,2](Yoshio Sakurai)
[1]京都大学院・文・心理，[2]科学技術振興機構，戦略的創造推進事業

P1-g39 RhoGAP タンパク質 RICS の神経系における機能解析
Functional analysis of the Rho GTPase-activating protein RICS in the nervous system
坂上　史佳(Fumika Sakaue)，林　寛敦(Tomoatsu Hayashi)，那須 - 西村教子(Yukiko Nasu-Nishimura)，
中村　勉(Tsutomu Nakamura)，秋山　徹(Tetsu Akiyama)
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P1-g40 ニューロン活動とシナプス可塑性が協同的に作用する系における動的ネットワーク構造の形成
A cooperative dynamics of network of neural oscillators between the neuronal activity and the synatptic weight.
青木　高明[1,2](Takaaki Aoki)，青柳富誌生[1](Toshio Aoyagi)
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P1-g41 ショウジョウバエ *Calcineurin B2* 遺伝子は記憶形成に関与する
Involvement of the *Calcineurin B2* gene in *Drosophila* courtship memory
中井　康弘(Yasuhiro Nakai)，相垣　敏郎(Toshiro Aigaki)，坂井　貴臣(Takaomi Sakai)
首都大院・理工・生命科学

P1-g42 ソングバード幼鳥の大脳基底核におけるさえずり関連活動
Singing-related activity in basal ganglia of juvenile songbird
柳原　真(Shin Yanagihara)，Neal Hessler
理研・脳

P1-g43 意志決定課題における行動モデルと大脳基底核の神経活動
Neuronal activity of the nucleus accumbens and the globus pallidus in a decision making task
伊藤　真[1](Makoto Ito)，銅谷　賢治[1,2](Kenji Doya)
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P1-g44 麻酔下ラットにおける、前頭前野と海馬神経活動の関係
Relationship between prefrontal and hippocampal neuronal spike activities in anesthetized rats
藤原　清悦(Sei-etsu Fujiwara)，伊崎　義憲(Yoshinori Izaki)，明間　立雄(Tatsuo Akema)
聖マリアンナ医大・医・生理

P1-g45 *nitric oxide synthase* dsRNA が長期記憶の形成を抑制する
*Nitric oxide synthase* dsRNA interfere with long-term memory formation in an insect
高橋　俊文[1](Toshifumi Takahashi)，國遠　尚代[2](Hisayo Kunitou)，浜田明日香[2](Asuka Hamada)，
松本　幸久[1](Yukihisa Matsumoto)，三戸　太郎[2](Taro Mito)，野地　澄晴[2](Sumihare Noji)，
水波　　誠[1](Makoto Mizunami)
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P1-g46 麻酔下ラットにおける神経連絡に関連した海馬と前頭前野の集合電位パワー変化の関係
Relationship between hippocampal and prefrontal local field potential power changes related to neuronal connectivity in anesthetized rats
伊崎　義憲(Yoshinori Izaki)，藤原　清悦(Sei-etsu Fujiwara)，明間　立雄(Tatsuo Akema)
聖マリアンナ医科大学・生理

P1-g47 ショウジョウバエ *chico* 変異体による学習不全と加齢性記憶障害の抑制
Olfactory learning deficits and suppression of age-related memory impairment by a mutation in *chico*,a *Drosophila* insulin receptor substrate
長野慎太郎[1,2](Shintaro Naganos)，齊藤　　実[2](Minoru Saitoe)
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P1-g48 胎生期 MAM 投与ラットにおける空間認知障害と海馬異所性ニューロンとの関連性
The relation of the spatial cognition deficit and the hippocampal heterotopic neurons in the prenatal MAM-treated rats
今井　英明(Hideaki Imai)，杉岡　幸三(Kouzo Sugioka)，寺島　俊雄(Toshio Terashima)
神戸大院・医・脳科学・神経発生

## 認知 1
## Cognition 1

P1-h01 時間認知におけるサル前頭前野の役割
Role of monkey prefrontal cortex in temporal processing
湯本　直杉[1](Naosugi Yumoto)，陸　　暁峰[2](Xiaofeng Lu)，宮地　重弘[3](Shigehiro Miyachi)，
南部　　篤[4](Atsushi Nambu)，深井　朋樹[5](Tomoki Fukai)，高田　昌彦[1](Masahiko Takada)
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P1-h02 社会的注意に対するアイコンタクトの効果とその神経機構 -fNIRS と EEG による
Effects of eye contact on social attention and their neural basis investigated by fNIRS and EEGs
小林　恒之[1,2](Tsuneyuki Kobayashi)，竹内　幹伸[1,3](Mikinobu Takeuchi)，高本　孝一[1](Koichi Takamoto)，
堀　　悦郎[1,2](Etsuro Hori)，松井　三枝[2,4](Mie Matsui)，石川　亮宏[5](Akihiro Ishikawa)，
河野　　理[5,6](Satoru Kohno)，小野　武年[1](Taketoshi Ono)，西条　寿夫[1,2](Hisao Nishijo)
[1]富山大院・医学薬学研究部・システム情動科学，[2]科学技術振興機構，[3]富山大・医・脳外科学，[4]富山大・医・心理学，[5]島津製作所，医療機器事業部，技術部，[6]京都大・医・高次脳機能総合研究センター

P1-h03 サルを用いたメタ記憶の神経生理学的研究のための行動課題
Behavioral paradigm for neurophysiological studies of metamemory in monkeys
田中　暁生(Akio Tanaka)，船橋新太郎(Shintaro Funahashi)
京都大学大学院人間・環境学研究科

P1-h04 複雑な報酬予告刺激に対するドーパミン細胞の応答
Dopamine responses to complex reward-predicting stimuli
野元　謙作[1,2](Kensaku Nomoto)，Takeo Watanabe[3]，坂上　雅道[1](Masamichi Sakagami)
[1]玉川大・脳研，[2]東京大院・医・精神医学，[3]ボストン大・心理

P1-h05 複数ステップに渡る行動選択過程において、線条体がコードする強化因子
Neuronal coding of reinforcers during multi-step choice task in the striatum
山田　　洋(Hiroshi Yamada)，井之川　仁(Hitoshi Inokawa)，木村　　實(Minoru Kimura)
京都府立医大・神経生理

P1-h06 ヒヨコのリスク感受性選択
Risk-sensitive choice in chicks
松島 俊也(Toshiya Matsushima), 川森 愛(Ai Kawamori)
北海道大院・理学・行動知能

P1-h07 サル運動性視床における Antisaccade に関連した神経活動
Neural correlates of antisaccades in the primate motor thalamus
國松 淳[1](Jun Kunimatsu), 田中 真樹[1,2](Masaki Tanaka)
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P1-h08 リズム知覚によるタッピング運動への影響
Influence of rhythm perception on finger tapping
嶋田 育代[1](Ikuyo Shimada), 石山 敦士[1](Atsushi Ishiyama), 中村 克樹[1,2,3](Katsuki Nakamura),
泉 明宏[1,2](Akihiro Izumi)
[1]早稲田大学大学院、先進理工学研究科、電気・情報生命工専攻, [2]国立精神・神経センター神経研究所,
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P1-h09 ドーパミン D1 受容体阻害剤の線条体注入による、試行錯誤学習課題に及ぼす影響
Selective impairment of reward-based adaptive choice of actions by intra-striatal injection of dopamine D1 receptor antagonist
上田 康雅[1](Y. Ueda), 鮫島 和行[2](K. Samejima), 銅谷 賢治[3](K. Doya), 木村 實[1](M. Kimura)
[1]京都府立医大・神経生理, [2]玉川大学 脳科学研究所 脳科学研究施設, [3]沖縄科学技術研究基盤整備機構 神経計算ユニット

P1-h10 ニホンザルにおける数に基づく操作課題遂行中の行動解析
Behavioral analysis of Japanese monkeys during a numerosity-based operation task
奥山 澄人[1](Sumito Okuyama), 虫明 元[1](Hajime Mushiake), 丹治 順[3](Jun Tanji),
乾 敏郎[2](Toshio Inui)
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P1-h11 視線のポップアウト
Pop-out of gaze
永福 智志(Satoshi Eifuku), 大湊 絢(Jun Ominato), 小野 武年(Taketoshi Ono),
田村 了以(Ryoi Tamura)
富山大学大学院・医学薬学研究部・統合神経科学

P1-h12 サル前頭前野における錐体細胞・介在細胞のゴール表現の遷移
Transition of goal representation of putative pyramidal neurons and interneurons in the primate prefrontal cortex
坂本 一寛[1](Kazuhiro Sakamoto), 吉田 隼[2](Shun Yoshida), 虫明 元[2](Hajime Mushiake),
合原 一幸[3](Kazuyuki Aihara), 丹治 順[4](Jun Tanji)
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P1-h13 自己肯定に関する脳機能イメージング研究
Cortical mechanism of positive self-concept : an fMRI study
福島 愛[1,2](Ai Fukushima), 杉浦 元亮[1,2](Motoaki Sugiura), 三浦 直樹[1,3](Naoki Miura),
内田 信也[1,4](Shinya Uchida), 関口 敦[1](Atsushi Sekiguchi), 堀江 薫[5,6](Kaoru Horie),
佐藤 滋[5,6](Shigeru Satoh), 川島 隆太[1,6](Ryuta Kawashima)
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P1-h14 頭頂葉の視覚 - 触覚バイモーダルニューロンによる自己と他者身体部位の表現
Self and other's body part representation in parietal visuo-tactile bimodal areas
石田 裕昭(Hiroaki Ishida), 村田 哲(Akira Murata)
近畿大・医・第一生理

P1-h15 数値選択課題が頭頂葉脳活動に及ぼす影響：近赤外線スペクトロスコピー (NIRS) による研究
Choosing a smaller number or a larger number in the number comparison task elicited different responses in the parietal region -A NIRSstudy-
洞口　貴弘(Takahiro Horaguchi)，緒方　洋介(Yosuke Ogata)，山本　三幸(Miyuki Yamamoto)
筑波大院・人間総合科学・感性認知脳科学

P1-h16 行動ルールの同定に関与する神経機構の fMRI による検討
Neural activity in the human brain involved in strategic rule identification
橘　香織[1,2](Kaori Tachibana)，虫明　元[3](Hajime Mushiake)，鈴木　匡子[1](Kyoko Suzuki)，
森　悦朗[1](Etsuro Mori)，丹治　順[4](Jun Tanji)
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P1-h17 時間弁別課題の刺激呈示期間におけるサル前頭前野ニューロンの活動
Cue-period activity of monkey prefrontal neurons during a duration-discrimination task
生塩　研一(Ken-ichi Oshio)，千葉　惇(Atsushi Chiba)，稲瀬　正彦(Masahiko Inase)
近畿大・医・第一生理

P1-h18 BCAA 投与が精神疲労と神経認知に及ぼす影響
Effect on neurocognition and mental fatigue after BCAA administrations
山本　隆宣(Takanobu Yamamoto)
帝塚山大学、心理福祉、心理

P1-h19 電気緊張距離を用いた視覚再構成のニューラルネットワークモデル
Neural network model for visual reconstruction using the morphoelectrotonic transform theory
渡邊　紀文(Norifumi Watanabe)，石崎　俊(Shun Ishizaki)
慶應義塾大学大学院政策・メディア研究科

P1-h20 サル前頭葉における情報量尺度の表現
Neural representation of information measures in the monkey frontal cortex
中村　清彦(Kiyohiko Nakamura)，高橋　大樹(Hiroki Takahashi)，小松三佐子(Misako Komatsu)
東工大院・総合理工・知能システム科学

P1-h21 ブローカ野における語結合に関する初期の階層的統語処理
Early hierarchical syntactic processing in Broca's area for merging words
飯島　和樹[1,2](Kazuki Iijima)，福井　直樹[3](Naoki Fukui)，酒井　邦嘉[1,2](Kuniyoshi L. Sakai)
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P1-h22 Chronic treatment of methylphenidate injures behavioral/cognitive functions by degeneration of dopaminergic neuron in rats
Sara Wardhani[1]，Norifumi Shioda[1]，Kohji Fukunaga[1,2]
[1]Department of Molecular Pharmacology, Tohoku University, Sendai, Japan, [2]Tohoku University 21st Century COE Program `CRESCENDO`

P1-h23 乳児の聴覚情報処理における大脳左右半球間の機能分化
Hemispheric differences in the activation patterns in the infant brain during auditory stream processing
保前　文高[1,2](Fumitaka Homae)，渡辺　はま[1,2](Hama Watanabe)，中野　珠実[2](Tamami Nakano)，
多賀厳太郎[1,2](Gentaro Taga)
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## アルツハイマー病、他の認知症、老化 1
## Alzheimer Disease, Other Dementia, Aging 1

P1-h24 血液脳関門を介したamyloid-β peptide(1-40)の排出輸送はactivated α2-macroglobulinとの複合体形成によって減少する
Cerebral clearance of human amyloid-β peptide (1-40) across the blood-brain barrier is reduced by complex formation of activated α2-macroglobulin
伊藤 慎悟[1,2](Shingo Ito), 大槻 純男[1,2](Sumio Ohtsuki), 上家 潤一[1,2](Junichi Kamiie),
祢津 泰子[1](Yasuko Nezu), 寺崎 哲也[1,2](Tetsuya Terasaki)
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P1-h25 APP代謝におけるX11およびX11Lの役割
The roles of X11 and X11L in APP metabolism
齋藤 有紀[1](Yuhki Saito), 佐野 良威[2](Yoshitake Sano), 中矢 正[1](Tadashi Nakaya),
糸原 重美[2](Shigeyoshi Itohara), 鈴木 利治[1](Toshiharu Suzuki)
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P1-h26 γ部位で切断されたAPP細胞内断片の同定
Novel intracellular domain fragment of Alzheimer amyloid precursor protein (AICD) via γ-secretase γ-site cleavage
亀谷富由樹[1](Fuyuki Kametani), 久米 秀明[1,2](Hideaki Kume)
[1]財団法人東京都医学研究機構・東京都精神医学総合研究所, [2]国立精神神経センター疾病第6部

P1-h27 α7ニコチン受容体やphosphatidylinositol 3-kinase経路を介したドネペジル慢性処置の作用
Effects of chronic treatment with donepezil via α7-nicotinic receptor and phosphatidylinositol 3-kinase cascade
高鳥 悠記[1](Yuki Takada-Takatori), 久米 利明[2](Toshiaki Kume), 大木 雄太[2](Yuta Ohgi),
新留 徹広[3](Tetsuhiro Niidome), 藤井 健志[1](Takeshi Fujii), 岡部 進[1](Susumu Okabe),
杉本 八郎[3](Hachiro Sugimoto), 赤池 昭紀[2](Akinori Akaike)
[1]同志社女大薬・薬・薬理, [2]京都大院・薬・薬品作用解析, [3]京都大院・薬・創薬神経科学

P1-h28 ゲルろ過HPLC法によるアルツハイマー病患者髄液を中心とした蛋白質およびリポ蛋白質解析
Analysis of lipoprotein in cerebral spinal fluid (CSF)of Alzheimer's disease(AD)patients using HPLC
赤津 裕康[1](Hiroyasu Akatsu), 岡崎 三代[2](Mitsuyo Okazaki), 伊藤 仁一[3](Jinichi Itoh),
山本 孝之[1](Takayuki Yamamoto), 道川 誠[4](Makoto Michikawa), 横山 信治[3](Shinji Yokoyama)
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P1-h29 N-カドヘリンはアミロイドβ蛋白の放出を促進する
N-cadherin enhances extracellular secretion of Aβ
植村 健吾[1](Kengo Uemura), 葛谷 聡[1](Akira Kuzuya), 青柳 信寿[1](Nobuhisa Aoyagi),
安藤 功一[1](Koichi Ando), 高橋 良輔[1](Ryosuke Takahashi), 下濱 俊[2](Shun Shimohama),
木下 彩栄[3](Ayae Kinoshita)
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P1-h30 インビボクロスリンク法を用いたガンマセクレターゼ新規構成蛋白質の探索
A screening for novel components of gamma-secretase complex using by in vivo crosslink method
片山 泰一[1](Taiichi Katayama), 三條 伸夫[2](Nobuo Sanjo), Paul Fraser[3], Peter St.George-Hyslop[3],
佐藤 康二[1](Kohji Sato)
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P1-h31 神経変性モデル SAMP10 マウスにおけるミクログリア退行変性の早期出現
Early onset of microglial dystrophy in SAMP10 mice
石井さなえ(Sanae Ishii)，島田　厚良(Atsuyoshi Shimada)，吉川　圭介(Keisuke Yoshikawa)，
千葉　陽一(Youichi Chiba)，河村　則子(Noriko Kawamura)，熊谷　直子(Naoko Kamagai)，
古川　絢子(Ayako Furukawa)，細川　昌則(Masanori Hosokawa)
愛知県心身障害者コロニー・発達障害研究所・病理学

P1-h32 エンドサイトーシス経路異常による GM1 ガングリオシドの細胞外放出の促進
Accelerated release of exosome-associated GM1 ganglioside by endocytic pathway abnormality
湯山　耕平[1](Kohei Yuyama)，山本　直樹[1,2](Naoki Yamamoto)，柳澤　勝彦[1](Katsuhiko Yanagisawa)
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P1-h33 βアミロイド前駆体蛋白質の細胞内蓄積とβアミロイド分泌に及ぼすシンタキシン 5 アイソフォームの効果
Effects of Syntaxin5 isoforms on the accumulation of intracellular βAPP and the secretion of Aβ peptides
須賀　圭[1](Kei Suga)，齋藤　綾子[1](Ayako Saito)，富山　貴美[2](Takami Tomiyama)，
森　啓[2](Hiroshi Mori)，赤川　公朗[1](Kimio Akagawa)
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P1-h34 SAMP10 マウス大脳皮質におけるプロテオグリカン関連エピトープ 6B4 の発現低下
Reduced expression of the 6B4-epitope in perineuronal nets of the cerebral cortex of SAMP10 mice
松井ふみ子[1](Fumiko Matsui)，斉藤　優子[1,2](Yuko Saito)，河村　則子[1](Noriko Kawamura)，
慶野　裕美[1](Hiromi Keino)，千葉　陽一[1](Yoichi Chiba)，熊谷　直子[1](Naoko Kumagai)，
石井さなえ[1](Sanae Ishii)，吉川　圭介[1](Keisuke Yoshikawa)，島田　厚良[1](Atsuyoshi Shimada)，
前田　信明[3](Nobuaki Maeda)，大平　敦彦[1](Atsuhiko Oohira)，細川　昌則[1](Masanori Hosokawa)
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P1-h35 アミロイドβタンパク注入マウスに認められる認知記憶障害におけるマトリックスメタロプロテアーゼの役割
Implication of matrix metalloprotease-9 in cognitive impairment induced by i.c.v. injection of Aβ in mice
溝口　博之[1](H Mizoguchi)，田熊　一敞[1](K Takuma)，福崎　笑子[1](E Fukuzaki)，
衣斐　大祐[1](D Ibi)，秋田　史生[1](F Akita)，染谷　栄一[1](E Someya)，T Alkam[2]，恒川　浩子[2](H Tsunekawa)，
毛利　彰宏[2](A Mouri)，野田　幸裕[3](Y Noda)，鍋島　俊隆[2](T Nabeshima)，山田　清文[1](K Yamada)
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P1-h36 老齢マウス脳シナプス膜中の特異な GM1 ganglioside 集積ドメイン
Unique GM1-ganglioside-enriched microdomain in synaptosomes prepared from aged mouse brain
山本　直樹[1,2](Naoki Yamamoto)，湯山　耕平[1](Kohei Yuyama)，柳澤　勝彦[1](Katsuhiko Yanagisawa)
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P1-h37 老齢マカクサルとツパイ前頭皮質におけるソマトスタチン陽性変成構造
Somatostatin-immunoreactive degenerating structures in prefrontal cortex of aged tree shrews (Tupaia) and macaque monkeys
山下　晶子[1](Akiko Yamashita)，清水　慶子[2](Keiko Shimizu)，Eberhard Fuchs[3]，
林　基治[2](Motoharu Hayashi)，泰羅　雅人[1](Masato Taira)
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P1-h38 [$^{11}$C]BF-227 を用いたアルツハイマー病におけるアミロイド斑の分子イメージング
In vivo detection of amyloid deposits in Alzheimer disease patients using [$^{11}$C]BF-227
谷内　一彦[1](Kazuhiko Yanai)，岡村　信行[1](Nobuyuki Okamura)，工藤　幸司[2](Yukitsuka Kudo)，
荒井　啓行[3](Hiroyuki Arai)
[1]東北大院・医・機能薬理，[2]東北大・先進医工学，[3]東北大・院・老年医学

P1-h39 プロテアソーム活性阻害とユビキチン化封入体形成がSAMP10マウス由来培養神経細胞に与える影響
Impact of proteasomal inhibition and formation of ubiquitinated inclusions on cultured neurons from SAMP10 mice
千葉 陽一(Yoichi Chiba), 島田 厚良(Atsuyoshi Shimada), 熊谷 直子(Naoko Kumagai),
河村 則子(Noriko Kawamura), 吉川 圭介(Keisuke Yoshikawa), 石井さなえ(Sanae Ishii),
古川 絢子(Ayako Furukawa), 細川 昌則(Masanori Hosokawa)
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P1-h40 家族性アルツハイマー病におけるアミロイド前駆体タンパク質のN型糖鎖の変化
Change of N-glycan structures of amyloid precursor protein in familial Alzheimer's disease
遠藤 玉夫(Tamao Endo), 赤阪(萬谷)啓子(Keiko Akasaka-Manya), 櫻井 洋子(Yoko Sakurai),
萬谷 博(Hiroshi Manya)
都老人研・老化ゲノム機能

P1-h41 神経変性モデルSAMP10マウスの海馬の興奮毒性に対する脆弱性とミクログリアの応答異常
Vulnerability to excitotoxic insults and defective microglial response in SAMP10 mouse
島田 厚良(Atsuyoshi Shimada), 石井さなえ(Sanae Ishii), 吉川 圭介(Keisuke Yoshikawa),
千葉 陽一(Yoichi Chiba), 河村 則子(Noriko Kawamura), 熊谷 直子(Naoko Kumagai),
古川 絢子(Ayako Furukawa), 細川 昌則(Masanori Hosokawa)
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P1-h42 軟性食餌飼育がドパミン遊離量に及ぼす影響
Effect of Semi-fluid diet breeding on the hippocampal dopamine release
串田 祥生[1](Sachio Kushida), 堀 紀雄[1](Norio Hori), 木本 克彦[1,3](Katsuhiko Kimoto),
豊田 實[1](Minoru Toyoda), 新田 淳美[4](Atsumi Nitta), 鍋島 俊隆[4](Toshitaka Nabeshima),
小野塚 実[2,3](Minoru Onozuka)
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## ポリグルタミン病、ALS、その他の神経変性疾患
## Polyglutamine Diseases, ALS, Other Neurodegenerative Disorders

P1-h43 DNA損傷3'-末断端に対するaprataxinの修復機能
Aprataxin, the causative gene product for EAOH/AOA1, repairs damaged 3'-ends of DNA single strand breaks
他田 正義[1](Masayoshi Tada), 小山 哲秀[1](Akihide Koyama), 五十嵐修一[1](Shuichi Igarashi),
横関 明男[1](Akio Yokoseki), 高橋 哲哉[1](Tetsuya Takahashi), 志賀 篤[1](Atsushi Shiga),
西澤 正豊[1](Masatoyo Nishizawa), 小野寺 理[2](Osamu Onodera)
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P1-h44 新たな精神遅滞モデルショウジョウバエ、PQBP-1変異体の解析
A drosophila PQBP-1 mutant is a novel model for mental retardation
田村 拓也(Takuya Tamura), 堀内 大輔(Daisuke Horiuchi), 岡澤 均(Hitoshi Okazawa)
東京医科歯科大学・難研・神経病理

P1-h45 脊髄小脳失調症14型(SCA14)で見い出された変異γPKCが初代培養プルキンエ細胞に及ぼす影響
Effects of mutant γPKC found in SCA14 on the nature of primary cultured Purkinje cells
関 貴弘[1](Takahiro Seki), 島原 隆行[1](Takayuki Shimahara), 安部 奈々[1](Nana Abe),
山本 和央[1](Kazuhiro Yamamoto), 足立 直子[2](Naoko Adachi), 柏木香保里[2](Kaori Kashiwagi),
斎藤 尚亮[2](Naoaki Saito), 酒井 規雄[1](Norio Sakai)
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P1-h46 カテプシンD欠損に伴うリソソーム蓄積症の病態形成について
Possible roles of LC3, ubiquitin, and p62 in the accumulation of lysosomes in neurons deficient in cathepsin D
小池 正人[1](Masato Koike), 柴田 昌宏[1](Masahiro Shibata), 石井 哲郎[2](Tetsuro Ishii),
内山 安男[1](Yasuo Uchiyama)
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P1-h47 RNA 編集異常による孤発性 ALS モデルマウスの開発
Development of a mouse model of sporadic ALS by deficient RNA editing
郭　伸[1](Shin Kwak)，日出山拓人[1](Takuto Hideyama)，西本　祥仁[2](Yoshinori Nishimoto)，
山下　雄也[1](Takenari Yamashita)，辻　省次[1](Shoji Tsuji)，高橋　良輔[3](Ryosuke Takahashi)，
三澤日出巳[4](Hidemi Misawa)，鈴木　岳之[5](Takeshi Suzuki)
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P1-h48 DNA マイクロアレイによる変異 GFAP 導入グリア細胞応答に関わる遺伝子発現異常の解析
Global analysis of gene expression in glial cell transfected with mutant *GFAP*
吉田　誠克[1](Tomokatsu Yoshida)，中川　正法[1](Masanori Nakagawa)，水島かつら[2](Katsura Mizushima)，
内藤　裕二[2](Yuji Naito)
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P1-j01 Tet-On システムを利用した RNA 編集活性測定株の樹立
Establishment of Hela cell lines suitable for the measurement of GluR2 RNA editing efficiency using Tet-on system
山下　雄也[1](Takenari Yamashita)，只見智恵子[1,2](Chieko Tadami)，西本　祥仁[1,3](Yoshinori Nishimoto)，
木村　大輔[1,2](Daisuke Kimura)，鈴木　岳之[2](Takeyuki Suzuki)，郭　伸[1](Shin Kwak)
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P1-j02 Cdk5/p35 による変異型ハンチンチンの凝集阻害
Inhibition of mutant huntingtin aggregate formation by Cdk5/p35
上之薗紗有子[1](Sayuko Kaminosono)，小山　文隆[2](Fumitaka Oyama)，斎藤　太郎[1](Taro Saito)，
浅田　明子[1](Akiko Asada)，永井　義隆[3](Yoshitaka Nagai)，貫名　信行[2](Nobuyuki Nukina)，
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P1-j03 ハンチントン病モデルで酸化の障害
Oxidative damage in nucleic acids in Huntington's disease model
盛　子敬(Zijing Sheng)，山田　英孝(Hidetaka Yamada)，中別府雄作(Yusaku Nakabeppu)
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P1-j04 BACE1 によるナトリウムチャネル β4 サブユニットの神経突起伸長活性の制御
BACE1 cleavage mediates neurite morphology induced by sodium channel β4 subunit
宮崎　晴子[1,2](Haruko Miyazaki)，小山　文隆[1](Fumitaka Oyama)，Hong Kit Wong[1]，
金子　貢巳[1](Kumi Kaneko)，櫻井　隆[1](Takashi Sakurai)，玉岡　晃[2](Akira Tamaoka)，
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P1-j05 ハンチントン病モデルマウスにおけるナトリウムチャネル β4 サブユニットの発現抑制
Dysregulation of β4 gene transcription in the striatum of Huntington Disease transgenic mice
小山　文隆[1](Fumitaka Oyama)，宮崎　晴子[1,2](Haruko Miyazaki)，黒沢　大[1](Masaru Kurosawa)，
玉岡　晃[2](Akira Tamaoka)，金子　武嗣[3](Takeshi Kaneko)，貫名　信行[1](Nobuyuki Nukina)
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P1-j06 ヒト Cu/Zn- スーパーオキシドジスムターゼの酸化ダメージにおける Cys111 の役割
Role of Cys111 in an oxidative damage of human Cu/Zn-superoxide dismutase
藤原　範子[1](Noriko Fujiwara)，中堅三弥子[2](Miyako Nakano)，吉原　大作[1](Daisaku Yoshihara)，
大河原知水[1](Tomomi Ookawara)，江口　裕伸[1](Hironobu Eguchi)，谷口　直之[3](Naoyuki Taniguchi)，
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P1-j07 コレイン欠失は DARPP-32 タンパク質発現増強を導く
Chorein deficiency leads to increase of DARPP-32 protein exprssion.
松田三恵子(Mieko Matsuda), 中村 雅之(Masayuki Nakamura), 倉野 裕(Yutaka Kurano),
水野恵三子(Emiko Mizuno), 市場 美緒(Mio Ichiba), 加藤真依子(Maiko Kato), 揚村明日美(Asumi Agemura),
佐野 輝(Akira Sano)
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P1-j08 ニーマンピック病 C 型マウス脳におけるユビキチンおよびユビキチン化タンパク質の性状解析
Characterization of ubiquitin and polyubiquitin conjugates in brains of Niemann-Pick C disease mouse
嶋田 洋太[1](Yohta Shimada), 高田 耕司[1](Koji Takada), 福田 隆浩[2](Takahiro Fukuda),
大川 清[1](Kiyoshi Ohkawa)
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P1-j09 CMT 変異体の NF-L 繊維形成異常
Effect of Charcot-Marie-Tooth mutations on filament assembly of neurofilament-L protein
安木 淳[1](Jun Yasuki), 後藤 隆洋[2](Takahiro Gotow), 塩崎 元子[2,3](Motoko Shiozaki),
佐々木考寛[4](Takahiro Sasaki), 内山 安男[3](Yasuo Uchiyama), 久永 眞市[1](Shin-ichi Hisanaga)
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P1-j10 HGF による ALS マウスの治療効果は c-Met/HGF 受容体のチロシンおよびセリン残基の相反的リン酸化により調節されている
Reciprocal phosphorylation of tyrosine and serine residues of c-Met/HGF receptor in motoneurons implicates HGF-dependent disease retardation of a transgenic mouse model of ALS.
角山 圭一(Keiichi Kadoyama), 船越 洋(Hiroshi Funakoshi), 中村 敏一(Toshikazu Nakamura)
大阪大院・医・分子再生医学

P1-j11 球脊髄性筋萎縮症モデルにおける CHIP 高発現の効果
CHIP over-expression reduces mutant AR protein and ameliorates phenotypes of the SBMA transgenic mouse model
足立 弘明(Hiroaki Adachi), 和座 雅浩(Masahiro Waza), 徳井 啓介(Keisuke Tokui),
勝野 雅央(Masahisa Katsuno), 南山 誠(Makoto Minamiyama), 田中 章景(Fumiaki Tanaka),
祖父江 元(Gen Sobue)
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P1-j12 歯状核赤核淡蒼球ルイ体萎縮症 (DRPLA) トランスジェニックマウス脳の発現プロファイリングの包括的 cross-platform 解析
Comprehensive cross-platform gene expression profiling analysis of dentatorubral-pallidoluysian atrophy (DRPLA) transgenic mice
鈴木 一詩[1](Kazushi Suzuki), 周 家毅[1](Jiayi Zhou), 佐藤 俊哉[2](Toshiya Sato),
小宅 睦郎[3](Mutsuo Oyake), 山田 光則[4](Mitsunori Yamada), 高橋 均[4](Hitoshi Takahashi),
高橋 祐二[1](Yuji Takahashi), 後藤 順[1](Jun Goto), 辻 省次[1](Shoji Tsuji)
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P1-j13 SUMO-1 modification increases human SOD1 stability and aggregation
Erkang Fei, Nali Jia, Ming Yan, Zheng Ying, Qiang Sun, Hongfeng Wang, Tao Zhang,
Xiaochuan Ma, Husheng Ding, Xuebiao Yao, Yunyu Shi, Guanghui Wang
School of Life Sciences, USTC, Hefei,China

P1-j14 Role of satellite III repeats in stress response and neurodegeneration
Sonali Sengupta, Ganesh Subramaniam
Biological Sciences and Bioengineering Department, Indian Institute of Technology Kanpur, India

P1-j15 **p45 targets the polyglutamine disease protein ataxin-3 to the proteasome**

Hongfeng Wang[1], Nali Jia[1], Erkang Fei[1], Zhiming Wang[1], Chao Liu[1], Tao Zhang[1], Jun Fan[1], Mian Wu[1], Lin Chen[1], Nobuyuki Nukina[2], Jiangning Zhou[1], Guanghui Wang[1]
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P1-j16 **骨髄由来マイクログリアは網膜遺伝性神経血管変性を制御する**
**Bone marrow-derived microglia control inherited neurovascular degeneration of the retina**

佐々原　学(Manabu Sasahara), 西田　佳代(Kayo Nishida), 大石　明生(Akio Oishi), 淀井　有子(Yuko Yodoi), 亀田　隆範(Takanori Kameda), 吉村　長久(Nagahisa Yoshimura), 大谷　篤史(Atsushi Otani)
京都大院・医・眼科

P1-j17 **子宮内エレクトロポレーション法を用いたXPAの機能解析**
**Functional analysis of XPA using in utero electroporation method**

竹内　聖二(Seiji Takeuchi), 謝　敏カク(Minjue Xie), 八木　秀司(Hideshi Yagi), 佐藤　真(Makoto Sato)
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## 統合失調症 Schizophrenia

P1-j18 **統合失調症関連因子 Dysbindin の解析**
**Molecular mechanism of Dysbindin:a Schizophrenia-related factor**

桑原　隆亮[1](Ryusuke Kuwahara), 松崎　伸介[1](Shinsuke Matsuzaki), 奥田　洋明[1](Hiroaki Okuda), 河本　圭介[1](Keisuke Kawamoto), 橋本　亮太[2](Ryota Hashimoto), 遠山　正彌[1](Masaya Tohyama)
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P1-j19 **統合失調症における前頭前野の低ドーパミン不安定性のシステム・ダイナミクス理論**
**A system dynamics theory of the hypodopaminergic instability of the prefrontal cortex in schizophrenia**

田中　昌司(Shoji Tanaka)
上智大学・理工・電気電子

P1-j20 **シャンデリア細胞が前頭前野の高ドーパミン性の過活動を制御する**
**Chandelier neurons control prefrontal cortical hyperactivation with hyperdopaminergic transmission**

坂田　和優(Kazumasa Sakata), 海老　紘彰(Hiroaki Ebi), 田中　昌司(Shoji Tanaka)
上智大院・理工・電気電子

P1-j21 **バックグラウンドアクティビティが作動記憶に携わる前頭前野のニューロンの持続発火に影響する—計算機実験—**
**Background activity affects sustained firing of prefrontal neurons engaged in working memory - a computational study -**

野村　健介[1,2](Kensuke Nomura), 田中　昌司[2](Shoji Tanaka), 加藤元一郎[1](Motoichiro Kato), 鹿島　晴雄[1](Haruo Kashima)
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P1-j22 **無麻酔ラットにおける扁桃体ニューロン活動に対するフェンサイクリジン全身投与の影響**
**Effects of systemic Phencyclidine on firing activity of amygdala neurons in freely moving rats**

片山　規央(Tadahiro Katayama), 浄土　英一(Eiichi Jodo), 鈴木　喜明(Yoshiaki Suzuki), 星野　研洋(Ken-Yo Hoshino), 竹内　賢(Satoshi Takeuchi), 香山　雪彦(Yukihiko Kayama)
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P1-j23 メタンフェタミン及び MK-801 によるプレパルスインヒビションの障害には淡蒼球 - 下橋網様核神経が関与する
Implication of the pallidotegmental neurons in methamphetamine- and MK-801-induced impairment of prepulse inhibition of the startle reflex in mice
新井佐和子(Sawako Arai)，溝口　博之(H Mizoguchi)，永井　　拓(T Nagai)，高橋　健次(K Takahashi)，
亀井　浩行(H Kamei)，田熊　一敞(K Takuma)，山田　清文(K Yamada)
金沢大院・自然科学研究科・薬物治療学

P1-j24 脳内でのアクチビン阻害は、統合失調症様行動を引き起こす
Inhibition of activin signaling in the brain causes a schizophrenia-like behavior
上田　洋司[1](H. Ageta)，高嶋　記子[1](N. Takashima)，井ノ口　馨[1,2](K. Inokuchi)
[1]三菱化学生命科学研究所，[2]CREST, JST

P1-j25 幼若期ラット腹側海馬特異的低ミエリン化による行動異常
Neonatal hypomyelination in the ventral hippocampus induces behavioral changes in adulthood
牧之段　学[1](Manabu Makinodan)，辰巳　晃子[2](Kouko Tatsumi)，山内　崇平[1](Takahira Yamauchi)，
下田　重郎[1](Shigero Shimoda)，松吉ひろ子[2](Hiroko Matsuyoshi)，岸本　年史[1](Toshifumi Kishimoto)，
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P1-j26 統合失調症モデルの認知機能障害に対するエモジンの効果
Effect of emodin for cognitive impairment in model of schizophrenia
川村　宏樹[1](Hiroki Kawamura)，水野　　誠[1,2](Makoto Mizuno)，那波　宏之[1](Hiroyuki Nawa)
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P1-j27 フェンサイクリジンによる培養大脳皮質ニューロンからの急速なグルタミン酸放出の増加と BDNF の発現上昇
Phencyclidine rapidly induced glutamate release and increased BDNF expression in cultured cortical neurons
安達　直樹[1](Naoki Adachi)，沼川　忠広[1](Tadahiro Numakawa)，熊丸　絵美[1](Emi Kumamaru)，
泉　　愛子[1,2](Aiko Izumi)，功刀　　浩[1](Hiroshi Kunugi)
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P1-j28 神経系における DISC1-Kendrin 相互作用の役割
The role of the interaction between DISC1 and Kendrin in the nervous system
清水　尚子[1](Shoko Shimizu)，松崎　伸介[1,2](Shinsuke Matsuzaki)，服部　剛志[1,2](Tsuyoshi Hattori)，
三好　　耕[3](Ko Miyoshi)，遠山　正彌[1,2](Masaya Tohyama)
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P1-j29 統合失調症者の探索眼球運動および fMRI を用いた情動関連認知機能の特徴：健常者との比較
Characteristics of emotional related cognitive function in schizophrenia evaluating by exploratory eye movements, and fMRI: comparison with healthy subjects.
松岡　稔昌[1](Toshimasa Matsuoka)，森田喜一郎[2](Kiichiro Morita)，小路　純央[1](Yoshihisa Shoji)，
山本　寛子[1](Hiroko Yamamoto)
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P1-j30 覚せい剤行動感受性亢進と内側前頭前野における形態学的変化の相関
Methamphetamine sensitization parallels decrease in dopamine axon density and length reduaction of the GABA axon terminal in the medial prefrontal cortex
門田　朋子(Tomoko Kadota)，門田　　健(Ken Kadota)
千葉大院・医・環境生命医学

P1-j31 統合失調症関連遺伝子 DISC1 によるショウジョウバエ連合学習と睡眠覚醒の阻害
Overexpression of DISC1 impairs associative learning and arousal in *Drosophila*
丸山　泰[1](Y Maruyama)，安藤　徹也[1](T Ando)，本庄　賢[1](K Honjo)，霜田　政美[2](M Shimoda)，
望月　洋明[1](H Mochizuki)，戸田　浩史[1,3](H Toda)，神谷　篤[4](A Kamiya)，石田直理雄[5](N Ishida)，
友田　利文[3](T Tomoda)，澤　明[4](A Sawa)，古久保 - 徳永克男[1](K Furukubo-Tokunaga)
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P1-j32 DISC1 相互作用分子としてのメッセンジャーリボヌクレオプロテインの同定
Identification of novel ribonucleoproteins as DISC1-interacting molecules
坪井　大輔[1,2](Daisuke Tsuboi)，田谷真一郎[1](Shinichiro Taya)，貝淵　弘三[1](Kozo Kaibuchi)
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P1-j33 統合失調症関連因子 DISC1 と Dysbindin の機能解析
The functional analysis of DISC1 and Dysbindin
服部　剛志[1,2](Tsuyoshi Hattori)，松崎　伸介[1](Shinsuke Matsuzaki)，熊本奈都子[2](Natsuko Kumamoto)，
清水　尚子[2](Shoko Shimizu)，山田　浩平[2](Kouhei Yamada)，遠山　正彌[1](Masaya Tohyama)
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P1-j34 Olanzapine 投与ラット海馬における Bax, Bcl-2 mRNA 発現量の変化
Alterations in Bax and Bcl-2 mRNA expression levels in hippocampus of olanzapine administrated rats.
菊山　裕貴[1,2](Hiroki Kikuyama)，吉田　祥[1](Yasushi Yoshida)，花岡　忠人[1](Tadahito Hanaoka)，
水野　貴史[1,2](Takafumi Mizuno)，金澤　徹文[1](Tetsufumi Kanazawa)，米田　博[1](Hiroshi Yoneda)
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P1-j35 統合失調症関連遺伝子 Dysbindin の機能解析
Functional analysis of Dysbindin, one of schizophrenia susceptibility genes
熊本奈都子[1](Natsuko Kumamoto)，松崎　伸介[1,2,3](Shinsuke Matsuzaki)，服部　剛志[1,2](Tsuyoshi Hattori)，
清水　尚子[1](Shoko Shimizu)，山田　浩平[1](Kohe Yamada)，桑原　隆亮[1](Ryusuke Kuwahara)，
行岡　太志[1](Futoshi Yukioka)，窪田　杏子[1](Kyoko Kubota)，遠山　正彌[1,2,3](Masaya Thoyama)
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P1-j36 アリピプラゾールの前頭前野ドパミン遊離に対する２つの作用：脳内微小透析法による研究
Dual effects of aripiprazole on prefrontal dopamine release
黒木　俊秀[1](Toshihide Kuroki)，中原　辰雄[2](Tatsuo Nakahara)，長尾奈穂子[1](Naoko Nagao)，
本村　啓介[1](Keisuke Motomura)，橋本喜次郎[2](Kijiro Hashimoto)，神庭　重信[1](Shigenobu Kanba)
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P1-j37 統合失調症者の情動惹起機能の特徴：P300 と RORETA 解析
Characteristics of emotional charged cognitive function in patients with schizophrenia: P300 and LORETA analyses
森田喜一郎[1](Kiichiro Morita)，小路　純央[1,2](Yoshihisa Shoji)，松岡　稔昌[2](Toshimasa Matsuoka)，
森　圭一郎[2](Keiichiro Mori)，平井　聡[1](Satoshi Hirai)
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P1-j38 活性化ミクログリアに対する向精神薬の影響
The effect of psychotropic drugs on microglial activation induced by interferon-$\gamma$
門司　晃(Akira Monji)，加藤　隆弘(Takahiro Kato)，堀川　英喜(Hideki Horikawa)，
溝口　義人(Yoshito Mizoguchi)，Qian Bian，橋岡　禎征(Sadayuki Hashioka)，神庭　重信(Shigenobu Kanba)
九州大学大学院医学研究院精神病態医学

## 気分障害
## Mood Disorders

P1-j39 Pericentrin 変異マウスを用いた神経細胞 1 次繊毛の機能解析
Functional analysis of neuronal primary cilia using pericentrin mutant mice
三好　耕(Ko Miyoshi)，宮崎　育子(Ikuko Miyazaki)，浅沼　幹人(Masato Asanuma)
岡山大院・医歯薬・脳神経制御・神経情報

P1-j40 気分障害患者における神経栄養因子群発現異常の検討
Expression analysis of neurotrophic factors in patients with mood disorders.
大脇　孝治[1](Koji Otsuki)，内田　周作[1](Shusaku Uchida)，松原　敏郎[2](Toshio Matsubara)，
綿貫　俊夫[1](Toshio Watanuki)，若林　祐介[1](Yusuke Wakabayashi)，渡辺　義文[1](Yoshifumi Watanabe)
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P1-j41 気分障害患者末梢白血球における選択的スプライシング調節機構異常の検討
Expression analysis of adhesion molecule CD44 isoform mRNA in the patients with mood disorders
綿貫　俊夫(Toshio Watanuki)，内田　周作(Shusaku Uchida)，船戸　弘正(Hiromasa Funato)，
大脇　孝治(Koji Otsuki)，若林　祐介(Yusuke Wakabayashi)，渡辺　義文(Yoshifumi Watanabe)
山口大院・医・高次脳機能病態学

P1-j42 気分障害患者における血中シグマ 1 受容体発現に関する検討
Altered levels of whole blood sigma-1 receptor protein in patients with mood disorders
竹林　実[1,2](Minoru Takebayashi)，久岡　一恵[2](Kazue Hisaoka)，前田奈津子[2](Natsuko Maeda)，
土岡　麻美[2](Mami Tsuchioka)
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臨床研究部

P1-j43 抗うつ薬によるグリア細胞株由来神経栄養因子 (GDNF) 産生における FGF 受容体の関与
Antidepressants-induced GDNF production in rat C6 glioma cells involves activation of FGF receptors.
久岡　一恵[1](Kazue Hisaoka)，前田奈津子[1](Natsuko Maeda)，土岡　麻美[1,3](Mami Tsuchioka)，
竹林　実[1,2](Minoru Takebayashi)
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P1-j44 双極性障害治療薬バルプロ酸は *C. elegans* の $IP_3$ および DAG に依存する行動に作用する
Valproic acid, a drug for bipolar disorder, acts on both $IP_3$- and DAG- regulated behaviours in *C. elegans*
徳岡　涼美(Suzumi Tokuoka)，Saiardi Adolfo，Nurrish Stephen
英国医学研究所

P1-j45 IL-1β 脳室内慢性投与によるモノアミン線維密度と行動の変化
The effects of IL1-β on behavioral responses and the density of monoamine axons
木田　裕之(H Kida)，中村　彰治(S Nakamura)
山口大学・医・高次神経科学

P1-j46 不定愁訴の歯科治療による改善効果
Ameliorative effect of dental treatments on unexplained symptoms
大塚　剛郎[1,6](Takero Otsuka)，笹栗　健一[1,6](Kenichi Sasaguri)，岡田　成生[2](Naruo Okada)，
藤田　晃史[3](Akifumi Fujita)，渡邊　和子[4,6](Kazuko Watanabe)，佐藤　貞雄[1,6](Sadao Sato)，
小野塚　実[5,6](Minoru Onozuka)
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P1-j47 選択的セロトニン再取り込み阻害薬がヒト注意機構に及ぼす影響の検討：fMRI 研究
Serotonergic modulation on the human attentional system: an fMRI study
八幡　憲明[1](Noriaki Yahata)，高橋　英彦[2](Hidehiko Takahashi)，鈴木　秀典[1](Hidenori Suzuki)，
大久保善朗[3](Yoshiro Okubo)
[1]日医大・薬理，[2]放医研，[3]日医大・精神

P1-j48 The effect of subconvulsive electrical stimulation of the nucleus accumbens on depressive behavior
E. Toth, R. Gersner, M. Shabat-Simon, A. Zangen
Neurobiology Department, Weizmann Institute of Sciences, Rehovot, Israel

## 痛覚 1
## Pain 1

P1-k01 グリア由来 Interleukin- 1 β により活性化された侵害受容性三叉神経節ニューロンの炎症性痛覚過敏に対する関与について
Activation of interleukin receptor of nociceptive tigeminal ganglion neurons via satellite glial interleukin-1 β contributes to the hyperalgesia following peripheral inflammation
武田　守(Mamoru Takeda)，高橋　誠之(Masayuki Takahashi)，松本　茂二(Shigeji Matsumoto)
日本歯科大学　生命歯学部　生理学講座

P1-k02 ブラジキニンによるブラジキニン B2 受容体の細胞内取込みはプロスタグランジン受容体 EP3 の活性化により阻害される
Activation of prostaglandin receptor EP3 inhibits the bradykinin-induced B2 receptor sequestration
小崎　康子(Yasuko Kozaki)，神部　福司(Fukushi Kambe)，片野坂公明(Kimiaki Katanosaka)，
妹尾　久雄(Hisao Seo)，水村　和枝(Kazue Mizumura)
名古屋大・環医研

P1-k03 下り坂走後の骨格筋における発痛物質の検討
Investigation of pain-producing substances in skeletal muscles after downhill running.
浦井　久子(Hisako Urai)，宇田　宗弘(Munehiro Uda)，上　勝也(Katsuya Kami)
大阪体育大学大学院・スポーツ科学研究科

P1-k04 神経損傷に伴う侵害受容線維末梢端グルタミン酸受容体の感受性変化
Changes in sensitivity of glutamate receptors at the peripheral endings of the nociceptive fivers following nerve injury
八巻　史子[1](Fumiko Yamaki)，古山　昭[2](Akira Furuyama)，竹村　元秀[3](Motohide Takemura)，
小池　勇一[1](Yuichi Koike)，米原　典史[1](Norifumi Yonehara)
[1]奥羽大・薬・薬理，[2]奥羽大・歯・口腔生理，[3]大阪大院・歯・高次脳口腔機能学

P1-k05 関節炎ラット脊髄後角における神経興奮のグリア細胞依存性増強
Glia-mediated facilitation of neuronal excitation in the spinal dorsal horn in the arthritic rat
池田　弘(Hiroshi Ikeda)，西　良介(Ryosuke Nishi)，村瀬　一之(Kazuyuki Murase)
福井大院・工・知能システム

P1-k06 痛覚過敏における NOX1/NADPH oxidase の役割
The role of NOX1/NADPH oxidase on hyperalgesia
衣斐　督和(Masakazu Ibi)，松野　邦晴(Kuniharu Matsuno)，矢部　千尋(Chihiro Yabe)
京都府立医科大学・医・病態分子薬理学

P1-k07 神経因性疼痛における脊髄 HCN チャネルの寄与
Spinal HCN channels contribute to neuropathic pain
田辺　光男(Mitsuo Tanabe)，高須　景子(Keiko Takasu)，小野　秀樹(Hideki Ono)
名古屋市立大学・院・薬学研究科

P1-k08 ガムチューイングによるセロトニン性鎮痛効果と前頭前野の賦活
An activation of cerebral oxygenation in prefrontal cortex and analgesic effect during gum chewing
神谷　清(Kiyoshi Kamiya)，麓　正樹(Masaki Fumoto)，
菊地　宏美(Hiromi Kikuchi)，中谷　康司(Yasushi Nakatani)，関　由成(Yoshinari Seki)，
中里　茜(Akane Nakasato)，于　新軍(Xinjun Yu)，関山タマミ(Tamami Sekiyama)，
上林　英里(Eri Kambayashi)，鈴木　郁子(Ikuko Sato-Suzuki)，有田　秀穂(Hideho Arita)
東邦大学・医・統合生理

P1-k09 扁桃体高頻度刺激による鎮痛作用
High frequency stimulation delivered to the amygdala produced analgesia.
井沢　優美(Yumi Izawa)，川上　順子(Yoriko Kawakami)
東京女子医科大学・第一生理学

P1-k10 入力損傷のある三叉神経核におけるクロライド恒常性変化関連遺伝子
Changes in the chloride homeostasis-related genes in trigeminal nuclei of the adult rats with nerve injury
魏　兵[1](B Wei)，古川　智範[1](T Furukawa)，熊田　竜郎[1](T Kumada)，岡部　明仁[3](A Okabe)，
大野　浩司[2](Koji Ohno)，佐藤　康二[2](K Sato)，福田　敦夫[1](A Fukuda)
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P1-k11 海馬 Sub 領域における痛み刺激の修飾作用
High frequency stimulation delivered to the hippocampal subiculum modified nociceptive responses recorded in the prefrontal cortex.
中村　弘之(Hiroyuki Nakamura)，川上　順子(Yoriko Kawakami)
東京女子医科大学　第一生理学教室

P1-k12 ビンクリスチン誘発アロディニアにおけるマクロファージ関与の可能性
Possible involvement of macrophage on vincristine-induced allodynia in mice
木口　倫一(Norikazu Kiguchi)，前田　武彦(Takehiko Maeda)，雑賀　史浩(Fumihiro Saika)，
岩井　茂樹(Shigeki Iwai)，岸岡　史郎(Shiroh Kishioka)
和歌山医大・薬理

P1-k13 光計測法による扁桃体中心核の神経興奮伝播の計測
Propagation of neuronal excitation in the rat central nucleus of amygdala revealed by the optical imaging
切通　考貴(Takaki Kiritoshi)，池田　弘(Hiroshi Ikeda)，村瀬　一之(Kazuyuki Murase)
福井大院・工・知能システム

## 行動薬理
## Behavioral Pharmacology

P1-k14 緑の香りがもつ抗うつ薬様効果
Antidepressant-like effect of green odor in mice
中富　康仁[1](Yasuhito Nakatomi)，横山ちひろ[1,2](Chihiro Yokoyama)，木下清二朗[1](Seijiro Kinoshita)，
正木　大貴[1](Daiki Masaki)，土田　英人[1](Hideto Tsuchida)，尾上　浩隆[2](Hirotaka Onoe)，
吉本　寛司[1](Kanji Yoshimoto)，福居　顯二[1](Kenji Fukui)
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P1-k15 Monoamionergic activity enhancer の in vivo における効果
In vivo effects of a monoamionergic activity enhancer
石橋恵利子(Eriko Ishibashi)，恒川　浩子(Hiroko Tsunekawa)，楠本　治子(Haruko Kusumoto)，
南　亜紀子(Akiko Minami)，高畑　和恵(Kazue Takahata)，米田　文郎(Fumio Yoneda)
エフピー株式会社

P1-k16 アンフェタミンによるマウスの多動と前頭前皮質神経の活性化に対するセロトニン 1A 作動薬の抑制作用
Serotonin 1A (5-$HT_{1A}$) agonist decreases locomotor activity and prefrontal neuronal activation in amphetamine treated mice
土田 理恵[1](Rie Tsuchida), 久保 勝裕[1](Masahiro Kubo), 黒田真里子[1](Mariko Kuroda),
角田 享也[2](Michiya Kakuda), 新谷 紀人[1](Norihito Shintani), 吾郷由希夫[3](Yukio Ago),
松田 敏夫[2,3](Toshio Matsuda), 橋本 均[1,2](Hitoshi Hashimoto), 馬場 明道[1](Akemichi Baba)
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P1-k17 HPC-1/syntaxin1A ノックアウトマウスにおける自閉症様行動異常
Deletion of HPC-1/syntaxin1A gene causes autistic behavior in mice
藤原 智徳[1](Tomonori Fujiwara), 小藤 剛史[2](Takefumi Kofuji), 三嶋 竜弥[1](Tatsuya Mishima),
吉川 武男[3](Takeo Yoshikawa), 赤川 公朗[1](Kimio Akagawa)
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P1-k18 ラット線条体に対するガンマナイフ照射により引き起こされる組織学的変化の解析
Cytological changes underlying the behavioral alterations after the Gamma Knife irradiation on the rat striatum
神山 暢夫[1](Nobuo Kouyama), 片山 洋子[1](Yoko Katayama), 林 基弘[2](Motohiro Hayashi),
川上 順子[1](Yoriko Kawakami)
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P1-k19 ウィスターおよび LEC ラットの脳機能に対する銅代謝の影響
Effects of copper metabolism on neurologigal functions in Wistar and LEC rats
吉原 大作[1](Daisaku Yoshihara), 藤原 範子[1](Noriko Fujiwara), 磯 博行[2](Hiroyuki Iso),
北中 順恵[3](Nobue Kitanaka), 北中 純一[3](Junichi Kitanaka), 江口 裕伸[1](Hironobu Eguchi),
大河原知水[1](Tomomi Ookawara), 尾澤敬一郎[2](Keiichiro Ozawa), 下田 重朗[2](Shigero Shimoda),
竹村 基彦[3](Motohiko Takemura), 鈴木敬一郎[1](Keiichiro Suzuki)
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P1-k20 A study on impulsivity and choice between delayed reinforcers in T-maze
Hamed Yazdanpanah, Paria Khazaee, Azam Mesdaghinia
Department of Pharmacology, Kashan University of Medical Sciences, Kashan, Iran

## イメージング 1
## Imaging 1

P1-k21 運動の時間パターンを反映する BOLD 信号
BOLD signal reflects temporal patterns of movement
戸松 彩花[1](Saeka Tomatsu), 染谷 芳明[2](Yoshiaki Someya), 小川 誠二[2](Seiji Ogawa),
筧 慎治[1](Shinji Kakei)
[1]東京都神経科学総合研究所, [2]小川脳機能研究所

P1-k22 Web アプリケーションを用いた MRI 脳画像データベース
Web-based MRI Brain image database system.
松田 圭司[1](Keiji Matsuda), 大石 高生[1,2,3](Takao Oishi), 肥後 範行[1,3](Noriyuki Higo)
[1]産業技術総合研究所, [2]京都大・霊長研, [3]CREST・科学技術振興機構

P1-k23 近赤外分光計測画像法による手指筋出力の予測
Prediction for finger force from cortical activity measured by near-infrared spectroscopy(NIRS)
南部 功夫[1,3](Isao Nambu), 大須理英子[2,3](Rieko Osu), 安藤 創一[4](Soichi Ando), 内藤 栄一[2,3](Eiichi Naito),
川人 光男[3](Mitsuo Kawato)
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P1-k24 ヒト連合野における食品の好き嫌いと活動様式について
Taste preferences and activation in the association cortex in humans.
水野　潤造[1](Junzo Mizuno)，湯山　徳行[1](Noriyuki Yuyama)，久保　金弥[2](Kinya Kubo)，
丹羽　政美[3](Masami Niwa)，渡邊　和子[4](Kazuko Watanabe)，小野塚　実[1](Minoru Onozuka)
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P1-k25 かなひろいテスト時の前頭前野賦活：fMRIによる研究
Activation of prefrontal area by attention task (Kana Pick-out Test)
橘　　篤導[1](Atsumichi Tachibana)，平野　好幸[1,2,5](Yoshiyuki Hirano)，大塚　剛郎[2,3](Takero Otsuka)，
小野　弓絵[1,2](Yumie Ono)，金　　完哉[1,2](Wanjae Kim)，渡邊　和子[2,4](Kazuko Watanabe)，
小畠　隆行[5](Takayuki Obata)，池平　博夫[5](Hiroo Ikehira)，小野塚　実[1,2](Minoru Onozuka)
[1]神奈川歯科・生体機能・生理学，[2]神奈川歯科大・高次脳・口腔科学研究センター，[3]神奈川歯科・成長発達歯科・歯科矯正学，[4]星城大学・リハ・生理，[5]放医研・分子イメージング・先端生体計測

P1-k26 脳腫瘍患者の術前マッピング
Pre-surgical mapping of the brain with brain tumor
二橋　尚志[1](Takashi Nihashi)，竹林　成典[2,3](Shigenori Takebayashi)，文堂　昌彦[2](Masahiko Bundo)，
藤井　正純[3](Masazumi Fujii)，若林　俊彦[3](Toshihiko Wakabayashi)，吉田　　純[3](Jun Yoshida)，
藤澤　洋幸[1](Hiroyuki Fujisawa)，清水ひとみ[1](Hitomi Shimizu)，早坂　和正[1](Kazumasa Hayasaka)，
長縄　慎二[4](Shinji Naganawa)
[1]長寿医療セ放，[2]長寿医療セ脳，[3]名大医脳，[4]名大医放

P1-k27 Nonlinear local neurovascular coupling in the cerebral cortex
Jorge J Riera[1], Juan C Jimenez[2], Tohru Ozaki[3], Ryuta Kawashima[1], Xiaohong Wan[4]
[1]IDAC, Tohoku Univ, Sendai, [2]Inst of Cyber, Math and Phys, Havana, [3]The Inst of Stat Math, Tokyo, [4]RIKEN Brain Science Inst, Wako

P1-k28 ラットの高炭酸ガス負荷時における神経活動と血流反応の抑制
Suppression of neural activity and hemodynamic responses during hypercapnic challenges in rats
根本　正史(Masahito Nemoto)，星　　詳子(Yoko Hoshi)，佐藤　知絵(Chie Sato)
東京都精神医学総合研究所・脳機能解析研究チーム

P1-k29 マンガン増強MRIの神経薬理学的応用
The application of pharmacological MRI in manganese enhancement of brain
伊藤　康一[1](Kouichi Itoh)，藤井　博匡[2](Hirotada Fujii)
[1]徳島文理大・香川薬・分子薬理，[2]札幌医科大学・保健医療学部

Core Time Odd numbers : 11:00 ～ 12:00
Even numbers : 13:00 ～ 14:00

## シナプス 2
## Synapse 2

P2-a01 小脳プルキンエ細胞におけるアクチン重合依存的な Shank1 局在安定化
Actin-dependent regulation of Shank1 dynamics in Purkinje cell dendrites
布施 俊光(Toshimitsu Fuse), 尾藤 晴彦(Haruhiko Bito)
東京大院・医・神経生化学/21世紀COEプログラム

P2-a02 海馬歯状回における Cbln1 結合部位の存在
Existence of specific binding sites for Cbln1 in the hippocampus
近藤 哲朗[1,2](Tetsuro Kondo), 飯島 崇利[1](Takatoshi Iijima), 亀川 雄一[1](Yuichi Kamekawa),
松田 恵子[1](Keiko Matsuda), 石田 綾[1](Aya Ishida), 柚崎 通介[1](Michisuke Yuzaki)
[1]慶応大・医・生理, [2]産総研・脳神経情報

P2-a03 ラット小脳顆粒細胞 - 介在ニューロン間興奮性シナプス伝達の性質
Characterization of the excitatory synaptic transmission between granule cells and interneurons in rat cerebellar cortex
佐竹伸一郎[1,2](Shin' Ichiro Satake), 井本 敬二[1,2](Keiji Imoto)
[1]生理研・生体情報, [2]総研大・生理科学

P2-a04 海馬局所神経回路における CNQX/AP5 抵抗性の同期的振動活動
CNQX/AP5-resistant seizure-related oscillations in rat hippocampal slices
塚元 葉子[1](Yoko Tsukamoto), 礒村 宜和[2](Yoshikazu Isomura), 今西美知子[1](Michiko Imanishi),
深井 朋樹[2](Tomoki Fukai), 高田 昌彦[1](Masahiko Takada)
[1]東京都神経研・統合生理, [2]理研BSI・脳回路機能理論

P2-a05 ラット海馬 CA3 における樹状突起、シナプス構築
Dendro- and synapto-architecture of the rat hippocampus CA3
松田 正司[1](Seiji Matsuda), 小林 靖[2](Yasushi Kobayashi), 石塚 典生[3](Norio Ishizuka)
[1]愛媛大学大学院・医・解剖発生, [2]防衛医科大学校解剖学講座, [3]東京都神経科学総合研究所脳構造研究部門

P2-a06 高 $K^+$ による神経終末部の脱分極は細胞外 $Ca^{2+}$ に依存せずに神経伝達物質放出を増強する
High potassium-induced facilitation of transmitter release in the absence of extracellular calcium
石橋 仁(Hitoshi Ishibashi), 張 一成(Il-Sung Jang), 鍋倉 淳一(Junichi Nabekura)
生理研・発達生理・生体恒常

P2-a07 樹状突起フィロポディア形成を促進するテレンセファリンとＥＲＭタンパク質との相互作用
Dendritic filopodia formation is mediated by the interaction between telencephalin and ERM proteins
古谷 裕[1,2](Yutaka Furutani), 松野 仁美[1,2](Hitomi Matsuno), 川崎 美和[1](Miwa Kawasaki),
佐々木雄彦[3](Takehiko Sasaki), 森 憲作[4](Kensaku Mori), 吉原 良浩[1,2](Yoshihiro Yoshihara)
[1]理研・脳センター・シナプス分子機構, [2]科学技術振興機構, [3]秋田大・医, [4]東大・医

P2-a08 lobule 10 のマウスプルキンエ細胞において高頻度平行線維刺激により観察される slow な外向き電流の特性
Characterization of slow outward current observed in mouse cerebellar Purkinje neurons of lobule 10 evoked by tetanic parallel fibers stimulation in slice
石井 裕[1,2](Hiroshi Ishii), 久保 義弘[1,2,3](Yoshihiro Kubo)
[1]総合研究大学院大学・生命科学・生理科学, [2]生理学研究所・神経機能素子, [3]科学技術振興機構

P2-a09 線虫 *C. elegans* の RIA ニューロンをモデル系としたシナプス細胞内局在の解析
Analysis of subcellular localization of synapses in *C. elgans* RIA interneuron
木全　翼(Tsubasa Kimata)，久原　篤(Atsushi Kuhara)，谷澤　欣則(Yoshinori Tanizawa)，
森　郁恵(Ikue Mori)
名古屋大院・理・生命

P2-a10 マウス上丘での $GABA_B$ 受容体によるバースト発火の制御機構
Mechanisms of $GABA_B$ receptor-mediated inhibition of burst activities in mouse superior colliculus
金田　勝幸[1,2](Katsuyuki Kaneda)，Penphimon Phongphanphanee[1,2]，柳川右千夫[3](Yuchio Yanagawa)，
小幡　邦彦[4](Kunihiko Obata)，伊佐　正[1,2](Tadashi Isa)
[1]生理研・認知行動発達，[2]総研大，[3]群馬大学・医・遺伝発達，[4]理研・脳科学総合研究センター

P2-a11 シナプス伝達の強い活性化に伴い発現誘導されるタンパク質 Homer1a の機能解析
Involvement of Homer1a in the regulation of distribution of postsynaptic proteins
井上由理子[1](Yuriko Inoue)，有働　洋[1](Hiroshi Udo)，井ノ口　馨[2](Kaoru Inokuchi)，
杉山　博之[1](Hiroyuki Sugiyama)
[1]九州大学院・システム生命科学府，[2]三菱生命科学研究所

P2-a12 ショウジョウバエ神経筋シナプスにおける活動依存的グルタミン酸受容体局在調節機構の解明
Activity-dependent regulation of glutamate receptor localization in *Drosophila* neuromuscular synapses
森本－谷藤高子(Takako Morimoto-Tanifuji)，渋谷　謙吾(Kengo Shibuya)，宮川　博義(Hiroyoshi Miyakawa)
東薬大・生命科学・脳神経

P2-a13 ラット胚中枢神経系における自発興奮波の時空間パターンと origin の同定：光学的イメージングによる解析
Origins of the spontaneous depolarization wave in the embryonic rat CNS revealed by optical imaging with a voltage-sensitive dye
佐藤　容子[1,2](Yoko Momose-Sato)，佐藤　勝重[1](Katsushige Sato)，木下　雅恵[1](Masae Kinoshita)
[1]東京医歯大院・医・細胞生理，[2]関東学院大・人間環境・健康栄養

P2-a14 F-BAR/EFC ドメイン蛋白質 Rapostlin による樹状突起スパインの制御
An F-BAR/EFC protein Rapostlin regulates dendritic spine development
柿本　哲宏(Tetsuhiro Kakimoto)，加藤　裕教(Hironori Katoh)，根岸　学(Manabu Negishi)
京都大院・生命科学・生体システム学

P2-a15 SAD キナーゼのマウス神経筋接合部における局在
SAD kinase is a presynaptic component of mouse neuromuscular junctions.
所　崇[1](Takashi Tokoro)，吉田　淑子[2](Shoko Yoshida)，比嘉　進[1](Susumu Higa)，
北島　勲[1,3](Isao Kitajima)，大塚　稔久[1,3](Toshihisa Ohtsuka)
[1]富山大院・医・臨床分子病態検査，[2]富山大院・医・再生医学，[3]CREST

P2-a16 GLAST 分子欠損マウスにおけるバーグマングリアの形態分化異常および形態学的登上線維多重支配
GLAST is essential for cytological differentiation of Bergmann glia and climbing fiber monoinnervation by suppressing local ectopic innervation
宮崎　太輔[1](Taisuke Miyazaki)，田中　光一[2](Kouichi Tanaka)，渡辺　雅彦[1](Masahiko Watanabe)
[1]北海道大院・医・解剖発生，[2]東京医科歯科大・医・分子神経科学

P2-b01 視床－大脳皮質神経回路における二種類の同期タイミング
Two types of synchronous timing in a somatosensory thalamocortical circuit
井上　剛(Tsuyoshi Inoue)，井本　敬二(Keiji Imoto)
生理学研究所・神経シグナル研究部門

P2-b02 CaMKII との相互作用が不能な変異 Syntaxin-1A マウスの作成と解析
Making the knock-in mice that lack the interaction between CaMKII and syntaxin-1A
渡邊　裕美[1,2](Yumi Watanabe)，崎村　建司[3](Kenji Sakimura)，五十嵐道弘[1,2](Michihiro Igarashi)
[1]新潟大院・医歯学総合・分子細胞機能学，[2]新潟大学・超域研究機構，[3]新潟大学・脳研究所・細胞神経生物学

P2-b03 Cec（CG1305遺伝子がencodeするカルシウム）チャネルはシナプスの活性部位のエンドサイトシスに関与し、低い小胞要求の時のシナプス伝達を維持する
CO1305 encoded calcium (Cec) channels participate to active zone endocytosis and maintain synaptic transmission at low demand of vesicles.
黒見　坦[1](Hiroshi Kuromi)，上野　耕平[1](Kohei Ueno)，城所　良明[2](Yoshiaki Kidokoro)
[1]群馬大学院・医・遺伝発達行動，[2]群馬大学生体調節研究所

P2-b04 グルタミン酸受容体δ2サブユニットは小脳顆粒細胞のシナプス前終末の分化を誘導する
Glutamate receptor δ2 subunit induces presynaptic differentiation of a cerebellar granule neuron
畔柳　智明[1,2](Tomoaki Kuroyanagi)，平野　丈夫[1,2](Tomoo Hirano)
[1]京都大院・理・生物物理，[2]科学技術振興機構　CREST

P2-b05 蛍光グルタミン酸指示分子を用いたシナプス間隙からのグルタミン酸漏出の可視化
Imaging glutamate spillover from synaptic clefts using the fluorescent glutamate indicator
大久保洋平[1](Yohei Okubo)，関谷　敬[1](Hiroshi Sekiya)，並木　繁行[2](Shigeyuki Namiki)，
坂本　寛和[2](Hirokazu Sakamoto)，飯沼　将[2](Sho Iinuma)，廣瀬　謙造[2](Kenzo Hirose)，
飯野　正光[1](Masamitsu Iino)
[1]東京大院・医・細胞分子薬理，[2]名古屋大院・医・細胞生理

P2-b06 鶏胚脳幹聴覚神経回路網の機能発達過程の光学的解析
Functional development of synaptic connections in the chick auditory system: Optical imaging with a voltage-sensitive dye
佐藤　勝重[1](Katsushige Sato)，Joel Glover[3]，佐藤　容子[1,2](Yoko Momose-Sato)
[1]東京医歯大院・医・細胞生理，[2]関東学院大・人間環境・健康栄養，[3]オスロ大・医・生理

## シナプス可塑性 1
## Synaptic Plasticity 1

P2-b07 Information content of synapses with realistic short-term dynamics
Jin Bao, Dmitri Bibichkov
Department of Membrane Biophysics, Max-Plank Institute for Biophysical Chemistry, Goettingen, Germany

P2-b08 GABAB receptor-mediated modulation of glycine release in rat substantia gelatinosa neurons
IS Choi[1], JH Cho[1], EJ Park[1], MG Lee[2], BJ Choi[1], IS Jang[1]
[1]Dept Dental Pharmacol, Kyungpook Natl Univ, Daegu, Republic of Korea, [2]Dept Medical Pharmacol, Kyungpook Natl Univ, Daegu, Republic of Korea

P2-b09 Requirement of tonic and phasic dopamine signals in long-term potentiation of prefrontal synapses
S Otani, B Kolomiets, A Marzo, J Caboche, P Vanhoutte
Neurobiology, University of Paris VI, Paris

P2-b10 ラット孤束複合体ニューロンの入力周波数依存的興奮パターンを決定するシナプス短期可塑性
Short-term plasticity defines postsynaptic excitation pattern in the solitary complex neurons of the rat
山本　清文[1](Kiyofumi Yamamoto)，井村　泰子[1](Taiko Imura)，野口　淳[2](Jun Noguchi)，
加藤　総夫[1](Fusao Kato)
[1]慈恵医大・神経生理，[2]東京ガス株式会社

P2-b11 バレル皮質内 GABA 細胞への視床投射の特異性と可塑性との関与
Maturation of thalamocortical transmission to GABAergic neurons and its possible relevance to plasticity in the barrel cortex
木村　文隆[1](Fumitaka Kimura)，伊丹　千晶[2](Chiaki Itami)，池添　貢司[3](Koji Ikezoe)，
田村　弘[3](Hiroshi Tamura)，藤田　一郎[3](Ichiro Fujita)，小幡　邦彦[4](Kunihiko Obata)，
柳川右千夫[5](Yuchio Yanagawa)，大島　稔[6](Minoru Ohshima)
[1]大阪大院・医・神経生理，[2]埼玉医大・生理学教室，[3]大阪大院・基礎工・認知脳科学・CREST，[4]理化学研究所・神経回路メカニズム，[5]群馬大院・医・遺伝発達行動学，[6]関西鍼灸大学・生理

P2-b12 放射線による樹状突起スパインへの影響
Effect of X-irradiation on dendritic spine morphology of hippocampal neurons
白井　克幸[1,2,3](Katsuyuki Shirai)，水井　利幸[1,3](Toshiyuki Mizui)，吉田由香里[2,3](Yukari Yoshida)，
岡本　雅彦[1,2,3](Masahiko Okamoto)，鈴木　義行[2,3](Yoshiyuki Suzuki)，花村　健次[1](Kenji Hanamura)，
中野　隆史[2,3](Takashi Nakano)，白尾　智明[1](Tomoaki Shirao)
[1]群馬大院・医・高次細胞機能学，[2]群馬大院・医・腫瘍放射線学，[3]COE

P2-b13 文脈的不安関連行動の消去過程における成熟ラット前頭前野シナプス応答におよぼす幼若期ストレスの影響
Early postnatal stress alters the prefrontal synaptic response during extinction of contextual fear-related behavior in adult rats
小関　裕代[1,2](Hiroyo Koseki)，松本真知子[1,2](Machiko Matsumoto)，山口　拓[2](Taku Yamaguchi)，
泉　剛[2](Takeshi Izumi)，富樫　広子[1](Hiroko Togashi)，吉岡　充弘[2](Mitsuhiro Yoshioka)
[1]北海道医療大、薬、総合薬学、病態生理，[2]北海道大院、医、神経薬理

P2-b14 電気感覚認識における短期シナプス可塑性による行動に関連した感覚情報抽出の神経メカニズム
Neural mechanism of extracting behaviorally-relevant features of stimulus by short-term synaptic plasticity in electrosensory perception
藤田　一寿(Kazuhisa Fujita)，樫森与志喜(Yoshiki Kashimori)
電気通信大学大学院情報システム学研究科情報ネットワーク学専攻

P2-b15 δ2 グルタミン酸受容体のリン酸化は小脳プルキンエ細胞における長期抑制の導入に必要ではない
Phosphorylation of the δ2 glutamate receptor is not required for induction of long-term depression in cerebellar Purkinje cells
仲神　龍一[1](Ryoichi Nakagami)，幸田　和久[1](Kazuhisa Kohda)，掛川　渉[1](Wataru Kakegawa)，
近藤　哲朗[1,2](Tetsuro Kondo)，柚崎　通介[1](Michisuke Yuzaki)
[1]慶応大院・医・生理学，[2]産業技術総合研究所脳神経情報

P2-b16 海馬樹状突起スパインにおける性ステロイドの急性作用
Rapid actions of sex steroids in hippocampal dendritic spines
川戸　佳(Suguru Kawato)，畑中　悠佑(Yusuke Hatanaka)，釣木澤朋和(Tomokazu Tsurugizawa)
東京大院・総文・広域

P2-b17 青斑核による海馬 CA3 野シナプス伝達の調節
Locus coeruleus modulates hippocampal CA3 synaptic transmission
張　寧(Ning Zhang)，松木　則夫(Norio Matsuki)
東京大院・薬・薬品作用学

P2-b18 Cbln1 欠損マウスを用いた小脳学習および海馬学習における Cbln1 の機能解析
Impaired delay, but normal trace classical eyeblink conditioning in the Cbln1 mutant mice
江見　恭一[1](Kyoichi Emi)，幸田　和久[1](Kazuhisa Kohda)，川原　茂敬[2](Shigenori Kawahara)，
柚崎　通介[1](Michisuke Yuzaki)
[1]慶応義塾大・医・生理，[2]東京大学・院薬・神経生物物理

P2-b19 テレンセファリンノックアウトマウスにおける視覚応答特性の早期成熟
Accelerated maturation of visual response properties in telencephalin knockout mice
岩井　陽一[1,2](Youichi Iwai)，Michela Fagiolini[2]，水口　陽子[2](Yoko Mizuguchi)，
吉原　良浩[1,3](Yoshihiro Yoshihara)，三品　昌美[4](Masayoshi Mishina)，ヘンシュ貴雄[1,2](Takao K Hensch)
[1]科学技術振興機構、CREST，[2]理化学研究所・脳科学総合研究センター・神経回路発達研究チーム，[3]理化学研究所・脳科学総合研究センター・シナプス分子機構研究チーム，[4]東京大院・医・分子神経生物

P2-b20 GLP-1 は海馬 CA1 野のシナプス伝達と長期抑圧を増強する
Glucagon-like pepetide-1 modulates synaptic transmission and long-term depression in the CA1 area of hippocampal slices
岩井　孝志(Takashi Iwai)，鈴木　　学(Manabu Suzuki)，岡　淳一郎(Jun-Ichiro Oka)
東京理科大・薬・薬理

P2-b21 繰り返し LTD 誘発後の長期シナプス減弱の発現に必要なタンパク合成の時間枠
Critical time window of protein synthesis required for LOSS, a long-lasting decrease in synaptic strength accompanied by synapse elimination.
江頭　良明(Yoshihiro Egashira)，田中　庸弘(Tsunehiro Tanaka)，上窪　裕二(Yuji Kamikubo)，
篠田　　陽(Yo Shinoda)，冨永（吉野）恵子(Keiko Tominaga-Yoshino)，小倉　明彦(Akihiko Ogura)
大阪大院・生命機能・脳神経工学

P2-b22 線虫による記憶を忘れるメカニズムの遺伝学的解析
Genetic analyses of mechanisms for memory erasing in *Caenorhabditis elegans*
井上　明俊(Akitoshi Inoue)，石原　　健(Takeshi Ishihara)
九州大学・理・生物

P2-b23 小脳プルキンエ細胞における抑制性シナプス可塑性を制御するシグナル経路のシミュレーション解析
Kinetic simulation analysis of signaling cascades regulating inhibitory synaptic plasticity in a cerebellar Purkinje neuron
北川　雄一[1,2](Yuichi Kitagawa)，川口　真也[1,2](Shin-ya Kawaguchi)，平野　丈夫[1,2](Tomoo Hirano)
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## 神経伝達物質、修飾物質 2
## Neurotransmitters and Modulators 2

P2-b24 GAD65 ノックアウトマウスに対するエタノール急性投与の影響
Acute ethanol effects on GAD65 KO mouse
松永　　渉(Wataru Matusnaga)，小幡　邦彦(Kunihiko Obata)
理化学研究所　脳科学総合研究センター

P2-b25 ラット動眼神経核ニューロン K チャネルのオピエートによる修飾
Effects of enkephaline on $K^+$ currents of oculomotor neurons in brainstem slices from rats
櫻庭　　陽[1,2](Hinata Sakuraba)，西村　嘉洋[1](Yoshihiro Nishimura)，山本　哲朗[1](Tetsuro Yamamoto)
[1]三重大学大学院医学系研究科システム神経科学，[2]鈴鹿医療科学大学　鍼灸学部

P2-b26 線虫の温度情報処理に関わる神経回路におけるグルタミン酸を介した頭部運動ニューロンの制御機構の解析
Analysis of Glutamate-mediated control of head motor neurons through thermotaxis neural circuit
大西　憲幸[1](Noriyuki Ohnishi)，久原　　篤[1](Atsushi Kuhara)，奥村　将年[1](Masatoshi Okumura)，
大河内善史[1](Yoshifumi Okochi)，稲田　　仁[1](Hitoshi Inada)，森　　郁恵[1,2,3](Ikue Mori)
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P2-b27 新生マウス海馬でのドパミンによる自発性 GABA 作動性性後シナプス電流の頻度の増加
Increase in the frequency of spontaneous $GABA_A$-PSC by dopamine in neonatal mouse hippocampus
芳野　浩樹[1](Hiroki Yoshino)，小川　陽一[2](Yoichi Ogawa)，中川　恵樹[1](Keiju Nakagawa)，
法山　良信[1](Yoshinobu Noriyama)，岸本　年史[1](Toshifumi Kishimoto)，山下　勝幸[2](Masayuki Yamashita)
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P2-b28 GABA 作動性シナプス後電流を修飾する PRIP 分子の役割
Roles of phosholipase C-related inactive protein (PRIP) in modulating $GABA_A$ IPSCs
豊田　博紀[1](Hiroki Toyoda)，小川　丈夫[1](Takeo Ogawa)，齋藤　　充[1](Mitsuru Saito)，
佐藤　　元[1](Hajime Sato)，小林　真之[3](Masayuki Kobayashi)，平田　雅人[2](Masato Hirata)，
姜　　英男[1](Youngnam Kang)
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P2-b29 内側前頭前皮質の第５層錐体細胞の Afterhyperpolalization に対する神経修飾因子の効果
The differential effects of neuromodulators on slow component of afterhyperpolalization of layer V pyramidal cells in the rat medial prefrontal cortex
佐竹　隆宏(takahiro satake)，兼子　幸一(Koichi Kaneko)，中込　和幸(Kazuyuki Nakagome)
鳥取大学医学部統合内科医学講座精神行動医学分野

P2-b30 チロシン水酸化酵素の N 端は本酵素の細胞内安定性を制御する
N-terminus of tyrosine hydroxylase regulates the intracellular stability of the enzyme
中島　昭[1](Akira Nakashima)，林　宣宏[2](Nobuhiro Hayashi)，金子　葉子[1](Yoko S. Kaneko)，
森　啓至[1](Keiji Mori)，永津　俊治[3](Toshiharu Nagatsu)，太田　明[1](Akira Ota)
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P2-b31 シナプトタグミン７による調節性分泌制御機構の可視化解析
Role for synaptotagmin VII in fusion pore dynamics revealed by total internal reflection fluorescence microscopy
坪井　貴司[1](Takashi Tsuboi)，福田　光則[2](Mitsunori Fukuda)
[1]理研・脳研・細胞機能探索，[2]東北大院・生命科学・膜輸送機構

P2-b32 新線条体パッチ領域におけるエンケファリン遺伝子の発現はマトリックス領域 に比べて著しく弱い：トランスジェニックマウスを用いた解析
Enkephalin gene expression is much weaker in the patch compartment than in the matrix compartment of the striatum: Analysis with transgenic mice
越水　義登[1](Yoshinori Koshimizu)，武　勝昔[2](Shengxi Wu)，薗村　貴弘[3](Takahiro Sonomura)，
雲財　知[1](To Unzai)，藤山　文乃[1](Fumino Fujiyama)，金子　武嗣[1,4](Takeshi Kaneko)
[1]京都大院・医・高次脳形態，[2]第四軍医大学　基礎部　解剖学教研室，[3]鹿児島大学　医歯学総合研究科　歯科機能形態学，[4]科学技術振興機構　戦略的創造研究推進事業

P2-b33 海馬 CA 1 投射性の5HT 1A 受容体発現正中縫線核細胞はセロトニンを含有する
CA1-projecting median raphe neurons expressing 5HT1A receptors are serotonergic
瀧川　慎也(Shinya Takigawa)，金城　雄太(Yuta Kaneshiro)，玉利　健悟(Kengo Tamari)，
西村　嘉洋(Yoshihiro Nishimura)，山本　哲朗(Tetsuro Yamamoto)
三重大院・医・システム神経科学

P2-b34 セリンラセマーゼ mRNA は主に神経細胞において発現する
The serine racemase mRNA locates predominantly in neurons of rat brain
吉川　正信[1](Masanobu Yoshikawa)，高安　直子[2](Naoko Takayasu)，野田　節子[3](Setsuko Noda)，
佐藤　雄一[4](Yuichi Sato)，小林　広幸[1](Hiroyuki Kobayashi)，橋本　篤司[1](Atsushi Hashimoto)
[1]東海大・医・臨床薬理，[2]東海大・医・分子科学，[3]東海大・健康科学・看護学，[4]北里大・医療衛生・遺伝子診断学

P2-b35 whole mount 免疫染色によるマウス小脳 CRF 陽性登上線維投射の解析
Stereoscopic study of corticotropin-releasing factor positive climbing fiber projection in mouse cerebellum by whole mount immunostaining
澤田　和彦[1](Kazuhiko Sawada)，Richard Hawkes[2]，福井　義浩[1](Yoshihiro Fukui)
[1]徳島大院・ヘルスバイオサイエンス・機能解剖，[2]カルガリー大・医・解剖

P2-b36 腹側体壁形成における抑制性神経伝達の役割
The role of inhibitory neurotransmission in ventral body wall formation
柿崎　利和[1,3](Toshikazu Kakizaki)，加家壁美樹子[1](Mikiko Kayakabe)，斉藤　憲史[1,2](Kenji Saito)，
柳川右千夫[1,3](Yuchio Yanagawa)
[1]群馬大院・医・遺伝発達行動，[2]総合研究大学院大学，[3]戦略的創造研究推進事業

P2-b37 マーモセット GABA ニューロンの免疫組織化学的解析
Immunohistochemical analysis of GABA neurons in the brain of the common marmoset
唐沢　延幸[1](Nobuyuki Karasawa)，林　基治[2](Motoharu Hayashi)，森　琢磨[2](Takuma Mori)，
清水　慶子[2](Keiko Shimizu)，山田　敬喜[3](Keiki Yamada)，小野塚　実[4](Minoru Onozuka)
[1]星城大学・リハビリテーション学部，[2]京都大学霊長類研究所，[3]藤田保健衛生大学衛生学部，[4]神奈川歯科大学口腔生理学

P2-b38 ラット大脳新皮質由来培養アストロサイト及び神経細胞における D- セリン局在の解明
Immunohistochemical and biochemical determination of D-serine in the cultured astrocytes and neurons from the rat neocortex
小柄　渚[1](Nagisa Kozuka), 海野　麻未[1](Asami Umino), 嶋津　奈[1](Dai Shimazu),
窪田　哲朗[1](Tetsuro Kubota), 仙波　禮治[2](Reiji Semba), 川添　僚也[3](Tomoya Kawazoe),
福井　清[3](Kiyoshi Fukui), 山本　直樹[1](Naoki Yamamoto), 西川　徹[1](Toru Nishikawa)
[1]東京医科歯科大院・医歯・精神行動医科学, [2]愛知県心身障害者コロニー発達障害研究所, [3]徳島大学分子酵素学研究センター

P2-b39 ナメクジ脳において発現する一酸化窒素合成酵素遺伝子群とそのゲノム構造の解析
The genomic structure and the variants of nitric oxide synthase gene in the terrestrial slug Limax valentianus
松尾　亮太(Ryota Matsuo), 伊藤　悦朗(Etsuro Ito)
徳島文理・香川薬・機能生物

P2-b40 黒質特異的なチロシン水酸化酵素遺伝子の破壊後、線条体におけるチロシン水酸化酵素量は遅れて減少する
Delayed reduction of the protein level in the striatum after genetic ablation of the tyrosine hydroxylase gene in the substantia nigra
坂根　寛明[1](Hiroaki Sakane), 一瀬（鷲見）千穂[2](Chiho Sumi-Ichinose), 小島　雅代[1](Masayo Kojima),
麻生　能功[1](Yoshinori Aso), 村松　慎一[3](Shin-ichi Muramatsu), 中野　今治[3](Imaharu Nakano),
小澤　敬也[4](Keiya Ozawa), 野村　隆英[2](Takahide Nomura), 永津　郁子[5](Ikuko Nagatsu),
永津　俊治[2](Toshiharu Nagatsu), Daniel Metzger[6], Pierre Chambon[6], 一瀬　宏[1](Hiroshi Ichinose)
[1]東工大院・生命理工, [2]藤田保健衛生大・医・薬理, [3]自治医大・神経内科, [4]自治医大・遺伝子治療研究部, [5]藤田保健衛生大・医・解剖, [6]IGBMC, Illkirch, France

## 情報伝達、変換、修飾 1
## Signal Transduction and Modulation 1

P2-b41 視床下部ニューロンでの GnRH による CaM キナーゼ II を介した MAP キナーゼの活性化反応
Activation of MAP kinase by GnRH via activation of CaM Kinase II in cultured hypothalamic neurons
山本　秀幸[1](Hideyuki Yamamoto), 山中　理菜[1](Ayana Yamanaka), 平上ゆかり[1](Yukari Hiragami),
前田　紀子[1](Noriko Maeda), 徳　誠吉[1](Seikichi Toku), 川原　正博[2](Masahiro Kawahara),
内藤　康仁[3](Yasuhito Naito)
[1]琉球大・医・生化学, [2]九州保健福祉大学　薬学部　薬学科, [3]北里大学　薬学部　薬理学教室

P2-b42 演題取下げ
Withdrawn

P2-b43 N-glycans govern the innate immune response in neurocysticercosis
V Prabhakaran, V Rajshekhar, A Oommen
Neurological Sciences, Christian Medical College, Vellore

P2-b44 Calcium-dependent mechanisms underlying propagation of γPKC translocation along the dendrites of cerebellar Purkinje cell
Hiroshi Tsubokawa[1], Kaori Kashiwagi[2], Naoaki Saito[2], Mitsuyuki Nakao[1], Norihiro Katayama[1]
[1]Grad Schl Inf Sci Tohoku Univ, Sendai, Japan, [2]Biosignal Res Ctr, Kobe Univ, Kobe, Japan

P2-b45 ニコチンによる神経新生抑制のメカニズム—HC2S2 細胞による研究
Mechanisms of nicotinic inhibition of neurogenesis. -Studies with use of HC2S2 cells.
新郷　明子[1](Akiko S. Shingo), 鬼頭　昭三[2](Shozo Kito)
[1]兵庫大・健康科学部・脳科研, [2]順心会幸生リハビリテーション病院

P2-b46 ヒポカルシンとクレアチンキナーゼBのカルシウム依存性結合
Identification of creatine kinase B as a $Ca^{2+}$-dependent hippocalcin-associated protein in rat brain
小林　正明(Masaaki Kobayashi)，渋谷沙也香(Sayaka Shibuya)，花上　まゆ(Mayu Hanaue)，
高松　　研(Ken Takamatsu)
東邦大・医・細胞生理

P2-b47 タンパク質アルギニンメチル化酵素の発現を抑制したPC12細胞に見られる形態的変化について
The characterization of protein arginine N-methyltransferases (PRMTs)-depleted PC12 cells.
森　　泰丈[1](Yasutake Mori)，宮田　信吾[1,2](Shingo Miyata)，種子田　艶[1](Tsuya Taneda)，
幸坂　　葵[1](Aoi Kousaka)，遠山　正彌[1,2](Masaya Tohyama)
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P2-b48 $Ca^{2+}$/カルモジュリン依存性プロテインキナーゼIIαノックアウトマウス脳と不活性型ノックインマウス脳の生化学的比較解析
Biochemical comparisons of brains from $Ca^{2+}$/calmodulin-dependent protein kinase IIα knock-out mice and inactivated knock-in mice
山肩　葉子[1,2](Yoko Yamagata)，柳川右千夫[3](Yuchio Yanagawa)，井本　敬二[1,2](Keiji Imoto)
[1]生理研，[2]総研大，[3]群馬大

P2-c01 脳切片の興奮伝搬に及ぼすサイアミラール抑制作用
Inhibitory effects of thiamylal on excitation propagation in brain slices
藤原　直士[1](Naoshi Fujiwara)，小川真有美[2](Mayumi Ogawa)，瀬尾　憲司[3](Kenji Seo)
[1]新潟大・医（保健）・検査技術，[2]新潟大学院・医歯学総合・麻酔，[3]新潟大学院・医歯学総合・歯科麻酔

P2-c02 ドーパミンで発生する$Na^+$電流応答に対するRhoとホスホリパーゼDによる促進作用
Facilitation of dopamine-induced $Na^+$ current response by Rho GTPase and phospholipase D in *Aplysia* neurons
川崎　　敏[1](Satoshi Kawasaki)，渡辺　修二[1](Shuji Watanabe)，木村　眞吾[1](Shingo Kimura)，
藤田　玲子[2](Reiko Fujita)，佐々木和彦[1](Kazuhiko Sasaki)
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P2-c03 培養神経細胞と神経膠細胞におけるAprataxinの発現
The appearance of aprataxin in rat cultured neuron and glia
二宮　孝文[1](Takafumi Ninomiya)，菊池　　真[1](Shin Kikuchi)，市川　量一[1](Ryoichi Ichikawa)，
小塚　直樹[2](Naoki Kozuka)，舘　　延忠[2](Nobutada Tachi)，辰巳　治之[1](Haruyuki Tatsumi)
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P2-c04 ウシガエル交換神経節細胞におけるチメロサールの影響
The effect of thimerosal on bullfrog sympathetic ganglion cells
島崎　由佳(Yuka Shimazaki)，能見　光雄(Mitsuo Nohmi)
佐賀大学・総合分析実験センター

P2-c05 RGMaシグナルにおけるPTENとFAKの関与
The involvement of PTEN and FAK in RGMa signaling
遠藤　光晴(Mitsuharu Endo)，山下　俊英(Toshihide Yamashita)
千葉大院・医・神経生物学

P2-c06 恐怖条件付けによって扁桃体で発現上昇するポロキナーゼSnkの脳内発現
Distribution of theSerum inducible kinase (Snk) mRNA in the rat nervous system; its up-regulation in lateral and basolateral amygdala after cued fear conditioning
赤澤　智宏[1](Chihiro Akazawa)，中村　泰子[2](Yasuko Nakamura)，前野　浩志[2](Hiroshi Maeno)，
和田　圭司[2](Keiji Wada)，高坂　新一[2](Shinichi Kohsaka)
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P2-c07 一側線条体へのガンマナイフ照射が両側線条体の蛋白質発現に及ぼす影響
Proteomics of unilateral irradiated and contralateral striata after gamma knife surgery
平野　美里[1](Misato Hirano), Randeep Rakwal[1], 神山　暢夫[2](Nobuo Kouyama),
片山　洋子[2](Yoko Katayama), 林　　基弘[3](Motohiro Hayashi), 小川　陽子[4](Yoko Ogawa),
吉田　康一[4](Yasukazu Yoshida), 岩橋　　均[4](Hitoshi Iwahashi), 増尾　好則[1](Yoshinori Masuo)
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P2-c08 生細胞内における$\alpha$CaMKIIの自己リン酸化と$Ca^{2+}$依存的活性制御機構の解析
Regulation of alpha CaMKII activation by its autophosphorylation and change in $Ca^{2+}$ level in living cells
加藤　健一(Kenichi Katou), 岩本　　拓(Taku Iwamoto), 喜田　　聡(Satoshi Kida)
東京農業大学・バイオサイエンス学科

P2-c09 TBCドメインをもつ成体ラット脳由来新規タンパク質に関する解析
The isolation of bdif-1 derived from the adult brain, and its function as Rab-GAP
伊藤　武司(Takeshi Ito), 古川　　弘(Hiromu Furukawa), 植木　孝俊(Takatoshi Ueki),
佐藤　康二(Kohji Sato)
浜松医科大学・医・解剖学講座

P2-c10 Map-based model of neural activity with chaotic spike-bursting oscillations
Lev V. Vdovin, Vladimir I Nekorkin
Nonlinear dynamics department, Institute of Applied Physics of the RAS, Nizhny Novgorod , Russia

## 軸索輸送、細胞骨格
## Axonal Transport and Cytoskeleton

P2-c11 MAP1BのN末とtubulinとの相互作用
Interaction between N-terminal 126 amino acids of MAP1B and tubulin
五味不二也(Fujiya Gomi), 内田　洋子(Yoko Uchida)
東京都老人総合研究所・老年病ゲノム研究チーム

P2-c12 ショウジョウバエの新規細胞内トラフィッキング制御因子の変異体は進行性変性症状を示す
Loss of a novel regulator for protein trafficking results in progressive degeneration in Drosophila
曽根　雅紀[1,2](Masaki Sone), 内田　敦子[2](Atsuko Uchida), 小松亜由美[2](Ayumi Komatsu),
鈴木えみ子[3](Emiko Suzuki), 岡澤　　均[1](Hitoshi Okazawa), 星野　幹雄[2](Mikio Hoshino),
鍋島　陽一[2](*Yo-ichi Nabeshima*)
[1]東京医歯・難治研, [2]京都大・医, [3]遺伝研

P2-c13 マウス脳内樹状突起スパインサイズとスパイン内ドレブリンA量の関係
Relation between dendritic spine size and its drebrin A level in adult mouse brain
小林　千穂[1](Chiho Kobayashi), Chiye Aoki[2], 白尾　智明[1](Tomoaki Shirao)
[1]群馬大院・医・高次細胞機能学, [2]ニューヨーク大学・神経科学センター

P2-c14 脳において生後発達期に増加するSept8とVAMP2の結合の解析
Identification of VAMP2 as a binding partner for a brain enriched septin, Sept8
伊東　秀記(Hidenori Ito), 岩本　郁子(Ikuko Iwamoto), 須藤　香織(Kaori Sudo), 森下　理香(Rika Morishita),
浅野　富子(Tomiko Asano), 永田　浩一(Koh-ichi Nagata)
愛知県コロニー・研・神経制御

P2-c15 神経活性によるチューブリンポリグルタミン酸付加を介した微小管修飾制御
Neural activity modifies microtubules by tubulin polyglutamylation
小西　慶幸[1](Yoshiyuki Konishi), 瀬藤　光利[1,2](Mitsutoshi Setou)
[1]三菱化学生命科学研究所・分子加齢医学, [2]自然科学研究機構・生理学研究所

P2-c16 CLP36 はアクチニンとの結合を通じて神経突起形成を制御する
CLP36 is a new member of growth cone-associated proteins that regulates neurite formation through the interaction with alpha-actinin
大野　浩司(Koji Ohno)，片山　泰一(Taiichi Katayama)，佐藤　康二(Kohji Sato)
浜松医科大学・医・解剖

P2-c17 微小管結合蛋白 doublecortin-like kinase1 の神経細胞内機能
Possible involvement of a microtubule-associated protein, doublecortin-like kinase 1, in postsynaptic function
申　義庚(Euikyung Shin)，岡部　繁男(Shigeo Okabe)
東京医科歯科大学

P2-c18 ドレブリン結合タンパク質 Spikar の核外での機能はスパイン形成に寄与している
Spikar distributed in the cytoplasm rescues impairment of spine formation induced by Spikar siRNA
山崎　博幸[1](Hiroyuki Yamazaki)，加藤　健一[1,2](Kenichi Kato)，関野　祐子[2](Yuko Sekino)，
白尾　智明[1](Tomoaki Shirao)
[1]群馬大・院・医・高次細胞機能，[2]CREST、科学技術振興機構

P2-c19 細胞骨格関連分子 Septin12 の生化学・細胞生物学的性状解析
Biochemical and cell biological analyses of Sept12 in the rat brain
永田　浩一(Koh-ichi Nagata)，伊東　秀記(Hidenori Ito)，須藤　香織(Kaori Sudo)，
岩本　郁子(Ikuko Iwamoto)，森下　理香(Rika Morishita)，浅野　富子(Tomiko Asano)
愛知県コロニー研・神経制御

## 遺伝子制御、エピジェネティクス、ゲノム情報学
## Gene Regulation, Epigenetics, Genome Informatics

P2-c20 Induction of stanniocalcin1 expression by histone deacetylase in mouse neuroblastoma cells
Yu Sheung Law, K Lai, C Wong
Department of Biology, Hong Kong Baptist University, Hong Kong

P2-c21 マウスてんかん誘導モデルにおける脳内遺伝子発現変化の解析
GeneChip analysis of the neocortical and hippocampal gene expression in kainate- and nicotine-injected mice
赤星　軌征[1](Noriyuki Akahoshi)，村島　善也[2](Yoshiya L Murashima)，氷見　敏行[3](Toshiyuki Himi)，
石崎　泰樹[1](Yasuki Ishizaki)，石井　　功[1](Isao Ishii)
[1]群馬大院・医・分子細胞機能，[2]東京都精神研，[3]武蔵野大・薬・安全性学

P2-c22 GSK-3beta 発現調節に関わるシス因子の検索
Deletion analysis of cis-elements regulating GSK-3β gene promoter
中西　滋之(Shigeyuki Nakanishi)，村山　　洋(Ohoshi Murayama)，小山　香織(Kaori Koyama)，
松田　基夫(Motoo Matsuda)
麻布大院・環境保健・遺伝子生物学

P2-c23 サル大脳新皮質の運動関連領野と前頭前野における網羅的遺伝子発現解析
Gene expression analysis of motor-related areas and prefrontal area of the monkey neocortex
佐藤　　明[1,7](Akira Sato)，西村　幸男[2,7](Yukio Nishimura)，大石　高生[3,7](Takao Oishi)，
肥後　範行[4,7](Noriyuki Higo)，村田　　弓[4,5,7](Yumi Murata)，尾上　浩隆[6,7](Hirotaka Onoe)，
伊佐　　正[2,7](Tadashi Isa)，小島　俊男[1,7](Toshio Kojima)
[1]理研・ゲノム・比較システム解析，[2]生理研・認知行動発達，[3]京大霊長研・器官調節，[4]産総研・脳神経情報，[5]筑波大院・人間総合科学，[6]理研フロンティア・分子プローブ機能評価，[7]CREST・JST

P2-c24 哺乳類に特有なミエリン構成タンパク質 Opalin 遺伝子の進化保存領域に見いだされたオリゴデンドロサイトエンハンサー
An oligodendrocyte enhancer in a phylogenetically conserved intron region of the mammalian myelin gene *Opalin*
有賀　純[1](Jun Aruga)，吉川　文生[2](Fumio Yoshikawa)，野崎　弥生[1](Yayoi Nozaki)，
榊　佳之[3](Yoshiyuki Sakaki)，豊田　敦[3](Atsushi Toyoda)，古市　貞一[2](Teiichi Furuichi)
[1]理研・脳センター・比較神経，[2]理研・脳センター・分子神経形成，[3]理研・ゲノム科学センター

P2-c25 BDNF 遺伝子プロモーター I の転写制御機構の解析と神経活動のクロマチン構造への組み込み
Integration of neuronal activity into chromatin on the BDNF gene promoter I against the repressive transcriptional activity of REST/NRSF in neuron
原　大智(Daichi Hara)，宮下　敏秀(Toshihide Miyashita)，田渕　明子(Akiko Tabuchi)，
津田　正明(Masaaki Tsuda)
富山大院・薬・分子神経生物学

P2-c26 プロトカドヘリンβクラスター遺伝子発現—片側アリルの差次的発現が生み出す神経細胞の多様性—
Monoallelic gene regulation and combinatorial expression of *Protocadherin-β* family genes confer neuronal diversity in the brain.
平野　敬三(Keizo Hirano)，金子　涼輔(Ryosuke Kaneko)，井澤　傑(Takeshi Izawa)，
木津川尚史(Takashi Kitsukawa)，八木　健(Takeshi Yagi)
大阪大学大学院院　生命機能研究科

P2-c27 神経系細胞株における ER-α変異体の発現とその調節
Estradiol regulates alternative splicing of ER-α mRNA in the differentiated NG108-15 neuronal cells
相澤　修(Shu Aizawa)，山室　裕(Yutaka Yamamuro)
日本大院・生物資源・生物資源生産科学

P2-c28 骨関連遺伝子の脳内における発現
Possible functional expression of bone-related genes in mouse brain
黒川晋太郎(Shintaro Kurokawa)，宝田　剛志(Takeshi Takarada)，米田　幸雄(Yukio Yoneda)
金沢大院・自然科学（薬）・薬物学

P2-c29 プロトカドヘリンα遺伝子クラスターのゲノム構造改変による発現パターン解析
Exchange of genome structure in *Protocadherin-α* gene cluster alters their neuronal expression pattern
野口由紀子[1,2](Yukiko Noguchi)，河村　佳見[2](Yoshimi Kawamura)，平林　敬浩[2](Takahiro Hirabayashi)，
内村　有邦[2](Arikuni Uchimura)，川口　将史[1,2](Masahumi Kawaguchi)，香取　将太[2](Shota Katori)，
平山　晃斉[2](Teruyoshi Hirayama)，八木　健[1,2](Takeshi Yagi)
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P2-c30 アクチン結合性転写活性化因子 MKL1/2 の組織・時期特異的分布
Distribution of MKL1/2, actin-binding coactivators for SRF, in developing rodent brain
石川　充[1](Mitsuru Ishikawa)，西嶋　直樹[1](Naoki Nishijima)，阪上　洋行[2](Hiroyuki Sakagami)，
津田　正明[1](Masaaki Tsuda)，田渕　明子[1](Akiko Tabuchi)
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P2-c31 アクチン結合モチーフを有する転写活性化因子 MKL1/2 の突起形態と遺伝子発現における機能解析
Functional analysis of MKL1/2, actin-binding coactivators for SRF, in neuronal morphology and gene expression
西嶋　直紀(Naoki Nishijima)，石川　充(Mitsuru Ishikawa)，堤下　寛之(Hiroyuki Tsutsumishita)，
塩田　惇(Jun Shiota)，津田　正明(Masaaki Tsuda)，田渕　明子(Akiko Tabuchi)
富山大院・薬・分子神経生物

P2-c32 電気的活動パターンにより制御される遺伝子群の解析
Molecules regulated by patterns of electrical activity
尾崎美和子[1,2,3](Miwako Ozaki)，澤村　直哉[1](Naoya Sawamura)，市川　道教[4](Michinori Ichikawa)
[1]早稲田大学・生命医療工学研究所，[2]早稲田オリンパスバイオサイエンス研究所，[3]早稲田大学・先端バイオ研究所，[4]ブレインビジョン株)

## 神経幹・前駆細胞と細胞分化，移植 2
## Neural Stem/Progenitor Cells and Cellular Differentiation, Transplantation 2

P2-c33 **Stem cell transplantation in rat model cognitive dysfunction: Neurobehavioral, Neurochemical and Immunohistochemical assessment**
Nishi Srivastava, Kavita Seth, Reyaz Ansari, Ashok K. Agrawal
Devlopmental Toxicology Division Industrial Toxicology Research Centre M.G. Marg Lucknow, India

P2-c34 **Colocalization of neuronal markers and cell-cycle markers in GABAergic neuron progenitors in the mouse spinal cord**
Jing Huang[1], Feng Feng[1], Nobuaki Tamamaki[2], Yuchio Yanagawa[3], Kunihiko Obata[4], Yun-Qing Li[1], Sheng-Xi Wu[1]
[1]Department of Anatomy, the Fourth Military Medical University, Xi' an, China, [2]Dept. Morphol. Neural Sci., Kumamoto Univ., Kumamoto, Japan, [3]Gunma Univ., Maebashi, Japan, [4]RIKEN, Wako, Japan

P2-c35 **Retardation of P19 neuronal differentiation by ifenprodil**
Danko Georgiev, Hideo Taniura, Yukio Yoneda
Lab Mol Pharmacol, Kanazawa University, Kanazawa, Japan

P2-c36 **Lysophosphatidic acid stimulates neuronal differentiation through $LPA_1$**
Shinsuke Ninose[1], Shinya Shano[1], Ryutaro Moriyama[1], Jerold Chun[2], Nobuyuki Fukushima[1]
[1]Department of Life Science, Kinki University, Japan, [2]The Scripps Research Institute, Ja Lolla, CA

P2-c37 **ショウジョウバエの嗅覚神経細胞を多様に分化させる分子機構の探索**
**Searching for mechanisms that diversify the olfactory receptor neurons in *Drosophila***
遠藤　啓太[1,2](Keita Endo)，伊藤　　啓[1](Kei Ito)
[1]東京大・分生研・高次構造，[2]JST・バイオインフォ

P2-c38 **IGFBPL-1 は発生期脳において新たに誕生した神経細胞の局在部位に強く発現する傾向を有する**
**IGFBPL-1 tends to strongly be expressed where newly born neurons are located in the developing brain**
権田　裕子[1](Yuko Gonda)，櫻井　　準[1](Hitoshi Sakurai)，田畑　秀典[1](Hidenori Tabata)，
平田　幸男[1](Yukio Hirata)，味岡　逸樹[1](Itsuki Ajioka)，仲嶋　一範[1,2](Kazunori Nakajima)
[1]慶應大・医・解剖，[2]慈恵大・DNA研・分子神経

P2-c39 **bHLH 転写因子による GDNF 受容体 (GFR$\alpha$ 1) の発現誘導**
**Induction of GDNF receptor, GFR$\alpha$ 1, expression by bHLH transcriptional factors**
本間　俊作(Shunsaku Homma)
福島県立医科大学・医学部・神経解剖発生学講座

P2-c40 **神経幹細胞のニューロンへの分化に対するドコサヘキサエン酸の影響**
**Effect of docosahexaenoic acid on the neurogenesis of neural stem cell**
片倉　賢紀(Masanori Katakura)，橋本　道男(Michio Hasimoto)，蒲生　修治(Shuji Gamoh)，
川北　映輔(Eisuke Kawakita)，奥井　俊之(Toshituki Okui)，紫藤　　治(Osamu Shido)
島根大学・医・環境生理

P2-c41 **培養神経幹 / 前駆細胞の神経分化における時計遺伝子の関与**
**Clock genes regulate neurogenic transcription factors and the neuronal differentiation of adult neural stem/progenitor cells**
君和田友美[1,2](Tomomi Kimiwada)，桜井省花子[1](Mikako Sakurai)，大橋　洋輝[1](Hiroki Ohashi)，
青木　俊介[1](Shunsuke Aoki)，冨永　悌二[2](Teiji Tominaga)，和田　圭司[1](Keiji Wada)
[1]国立精神・神経センター　神経研究所　疾病研究第四部，[2]東北大院　医　神経外科

P2-c42 Meis2 の大脳皮質形成における機能的役割
Meis2 activities in the control of three distinct developmental steps during the corticogenesis
田辺　康人[1,2](Yasuto Tanabe)
[1]阪大院・生命機能・脳神経工学，[2]三菱化学生命科学研究所

P2-c43 マウス脊髄における Foxp1 の発現パターンの発生学的解析
Developmental expression pattern of Foxp1 in the mouse spinal cord
久岡　朋子(Tomoko Hisaoka)，森川　吉博(Yoshihiro Morikawa)，仙波恵美子(Emiko Senba)
和歌山医大・医・第二解剖

P2-c44 NMDA 受容体拮抗薬によるダメージに対する神経幹細胞の神経保護効果のメカニズム
Direct/indirect mechanism of neuroprotection by neural stem cell against NMDA antagonist-induced damage
小野　貴文(Takafumi Ono)，鵜飼　渉(Wataru Ukai)，吉永　敏弘(Toshihiro Yoshinaga)，
石井　貴男(Takao Ishii)，渡邊　公彦(Kimihiko Watanabe)，館農　勝(Masaru Tateno)，
橋本　恵理(Eri Hashimoto)，齋藤　利和(Toshikazu Saito)
札幌医科大学医学部神経精神医学講座

P2-c45 老齢ラット大脳皮質における NG2 陽性細胞の領域特異的な変化について
Region-specific degeneration of NG2+ cells in the cerebral cortex of aged rats
田村　泰久[1](Yasuhisa Tamura)，崔　翼龍[1](Yilong Cui)，渡辺　恭良[1,2](Yasuyoshi Watanabe)，
片岡　洋祐[1,2](Yosky Kataoka)
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P2-c46 成熟脳海馬の神経系前駆細胞に発現する特定遺伝子
Differential expression of a particular gene responsible for astroglial differentiation in neural progenitor cells of adult mouse hippocampus
前田紗弥香(Sayaka Maeda)，渡邊　真未(Mami Watanabe)，玉置　啓祐(Keisuke Tamaki)，
中道　範隆(Noritaka Nakamichi)，米田　幸雄(Yukio Yoneda)
金沢大院・自然科学（薬）・薬物学

P2-c47 胎生期大脳壁の脳室面で誕生する娘細胞における転写因子発現のタイム テーブル
Time-table for transcription factor expression in daughter cells generated at the ventricular surface of the developing neocortex
宮田　卓樹[1,2,3](Takaki Miyata)，落合　和[1](Wataru Ochiai)，高原　大志[1](Taishi Takahara)，
小川　正晴[2](Masaharu Ogawa)
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P2-c48 胎生期神経前駆細胞の Ngn2 は Tbr2 の発現を誘導する
Ngn2 acts upstream of Tbr2 for neuron supply in the developing neocortex
落合　和[1](Wataru Ochiai)，高原　大志[1](Taishi Takahara)，宮田　卓樹[1,2](Takaki Miyata)
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P2-d01 胎生期大脳皮質の脳室帯において Rac1 は不均一に発現する
Non-uniform expression of Rac1 on the ventricular surface of developing neocortex
美濃部さやか[1,2](Sayaka Minobe)，落合　和[2](Wataru Ochiai)，宮田　卓樹[2,3](Takaki Miyata)
[1]三重大学・医・解剖，[2]名古屋大学・医，[3]科学技術振興機構、戦略的創造研究推進事業

## 細胞移動、突起伸展、回路網形成 2
## Migration, Axonal and Dendritic Outgrowth, Network Formation 2

P2-d02 ラット皮質 - 脊髄共培養系における単一軸索後退のタイムラプス観察
Axonal regressive events in rat corticospinal slice coculture studied with time-lapse confocal imaging of single axons
吉岡　昇(Noboru Yoshioka)，大野　孝恵(Takae Ohno)，桜井　正樹(Masaki Sakurai)
帝京大学医学部生理学講座

P2-d03 PC12D 細胞の突起伸展およびラッフル形成誘導のシグナル伝達
NGF-dependent formation of ruffles in PC12D cells required a different pathway from that for neurite outgrowth
神　絵里香(Erika Jin)，佐野　　護(Mamoru Sano)
京都府立医科大学大学院　生物学教室

P2-d04 Heparanase upregulation in astrocytes and macrophages recruited to the injured spinal cord
Yi Zhang[1], C.H. Chau[1], Y.S. Chan[2], D.K.Y. Shum[1]
[1]Department of Biochemistry, The University of Hong Kong, HK, China, [2]Departments of Physiology, The University of Hong Kong, HK, CHINA

P2-d05 Wnt5 signaling for glomerular patterning in the *Drosophila* olfactory system
Masao Sakurai[1,2], Tomoko Aoki[1], Shingo Yoshikawa[2], Kyoko Ishikawa[1], John B Thomas[2], Chihiro Hama[1]
[1]CDB, Riken, Japan, [2]Salk Institute for Biological Studies

P2-d06 Change in the microtubule-anchoring proteins at the centrosome during the differentiation of mouse neurons
Yusaku Ohama, Kensuke Hayashi
Life Science Inst. Sophia Univ. Tokyo, Japan

P2-d07 Function of draxin in the migration of neural crest cells
Su Yuhong, Iftekhar Bin Naser, MD Shahidul Islam, Yohei Shinmyo, Sanbing Zhang, Giasudin Ahmed, Sandy Chen, Hideaki Tanaka
Department of Developmental Neurobiology, University of Kumamoto, Kumamoto, Japan

P2-d08 Analysis of a novel guidance molecule, draxin, in chick embryonic tectobulbar axon projection.
Iftekhar Bin Naser, Yuhong Su, MD Shahidul Islam, Yohei Shinmyo, Sanbing Zhang, Giasuddin Ahmed, Sandy Chen, Hideaki Tanaka
Department of Developmental Neurobiology, University of Kumamoto, Kumamoto, Japan

P2-d09 細胞間接着による大脳皮質層形成機構の解析
Regulation of cortical laminar formation by cell-cell contacts
大石　康二[1](Koji Oishi)，刀川夏詩子[1](Kashiko Tachikawa)，佐々木慎二[1](Shinji Sasaki)，
仲嶋　一範[1,2](Kazunori Nakajima)
[1]慶應大・医・解剖，[2]慈恵医大・DNA研・分子神経

P2-d10 新皮質の形成において DISC1 が果たす役割の解析
The analyses of the role of DISC1 in the formation of the neocortex
久保健一郎[1](Ken-ichiro Kubo)，神谷　篤[2,3](Atsushi Kamiya)，澤　　明[2,3,4](Akira Sawa)，
仲嶋　一範[1,5](Kazunori Nakajima)
[1]慶應義塾大学医学部解剖学，[2]Department of Psychiatry, Johns Hopkins University School of Medicine, [3]Department of Neuroscience, Johns Hopkins University School of Medicine, [4]Graduate Program in Cellular Molecular Medicine, Johns Hopkins University School of Medicine, [5]慈恵医大・DNA研・分子神経

P2-d11 発生期大脳新皮質における多極性移動神経細胞の挙動を制御する分子の解析
Functional analyses of the molecules that control the behavior of multipolar migration neurons in the developing cerebral neocortex
佐々木慎二[1](Shinji Sasaki)，田畑　秀典[1](Hidenori Tabata)，刀川夏詩子[1](Kashiko Tachikawa)，
平田　幸男[1](Yukio Hirata)，仲嶋　一範[1,2](Kazunori Nakajima)
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P2-d12 Sema4D/PlexinB1 による神経突伸長促進作用機序の解析
Molecular mechanism of enhancement of neurite outgrowth by Sema4D
高原　瑞恵(Mizue Takahara)，立野　　翔(Sho Tachino)，稲垣　　忍(Shinobu Inagaki)
大阪大院・医・保健・神生研

P2-d13 **Shootin1 と神経突起伸長に関する定量的解析**
**Quantitative analysis of Shootin1 and neurite elongation.**
浅野　卓也[1](Takuya Asano)，鳥山　道則[2](Michinori Toriyama)，作村　諭一[1](Yuichi Sakumura)，
稲垣　直之[2](Naoyuki Inagaki)，石井　信[1](Shin Ishii)
[1]奈良先端大・情報科学，[2]奈良先端大・バイオサイエンス

P2-d14 **樹状突起・軸索投射を制御するホメオドメイン型転写因子 Homothorax の機能解析**
**A homeodomain transcriptional factor, Homothorax, controls dendritic and axonal targeting in the *Drosophila* brain**
安藤　舞(Mai Ando)，戸谷　洋子(Yoko Totani)，古久保ー徳永克男(Katsuo Furukubo-Tokunaga)
筑波大院・生命環境・生物科学

P2-d15 **細胞体による軸索伸長の遠隔制御**
**Axonal calcium sweep: somatic remote control of axon elongation**
池谷　裕二(Yuji Ikegaya)，山田　隆二(Ryuji_X Yamada)，佐々木拓哉(Takuya Sasaki)，
市川　淳也(Junya Ichikawa)，小山　隆太(Ryuta Koyama)，松木　則夫(Norio Matsuki)
東京大院・薬・薬品作用

P2-d16 **視床皮質投射における CRMP1 の役割**
**Analysis of thalamocortical projection in *crmp1* deficient mice**
冨田　祐介(Yusuke Tomita)，臼井　洋(Hiroshi Usui)，山下　直也(Naoya Yamashita)，
中村　史雄(Fumio Nakamura)，五嶋　良郎(Yoshio Goshima)
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P2-d17 **脚間核を介した手綱核の左右非対称な神経伝導路**
**The interpeduncular nucleus in zebrafish transmitted left-right information from the habenulae to the distinct targets in the hindbrain**
相澤　秀紀(Hidenori Aizawa)，後藤　翠(Midori Goto)，岡本　仁(Hitoshi Okamoto)
理研・脳センター・発生遺伝子制御

P2-d18 **Sema4D/Plexin-B1 は R-Ras GAP 活性を介して PTEN を活性化し、成長円錐崩壊を引き起こす**
**Sema4D/Plexin-B1 activates PTEN via R-Ras GAP activity,inducing growth cone collapse**
伊藤　由梨(Yuri Ito)，生沼　泉(Izumi Oinuma)，加藤　裕教(Hironori Katoh)，根岸　学(Manabu Negishi)
京都大学院・生命・生体システム学

P2-d19 **海馬苔状線維経路探索における脳由来神経栄養因子 (BDNF) の役割～レンチウイルスを用いた解析～**
**Involvement of BDNF in mossy fiber fasciculation**
田村　誠(Makoto Tamura)，田村　直寛(Naohiro Tamura)，小山　隆太(Ryuta Koyama)，
池谷　裕二(Yuji Ikegaya)，松木　則夫(Norio Matsuki)，山田　麻紀(Maki Yamada)
東京大院・薬・薬品作用

P2-d20 **フェムト秒レーザーにより切断された海馬神経細胞回路網における機能的再結合**
**Functional regeneration of hippocampal neurons on multielectrode array dish controlled by femtosecond laser processing**
細川　千絵[1,2](Chie Hosokawa)，清原　藍[1](Ai Kiyohara)，工藤　卓[1](Suguru N. Kudoh)，
田口　隆久[1](Takahisa Taguchi)
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P2-d21 **発達過程の小脳における神経認識分子 NB-3 の局在解析**
**Neural cell recognition molecule NB-3 in the developing cerebellum**
桜井　都衣[1](Kunie Sakurai)，鈴木　康仁[1](Yasuhito Suzuki)，豊島　学[1](Manabu Toyoshima)，
武田　泰生[2](Yasuo Takeda)，霜田　靖[1](Yasushi Shimoda)，Domna Karagogeos[3]，
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P2-d22 マウス嗅上皮における嗅覚受容体の領域特異的な発現に関与する分子の解析
Involvement of molecules in sub-areal expression of odorant receptor genes in the most ventro-lateral zone of the mouse olfactory epithelium
高橋　弘雄(Hiroo Takahashi)，吉原　誠一(Sei-ichi Yoshihara)，坪井　昭夫(Akio Tsuboi)
奈良医大・先端・生命システム

P2-d23 C.elegans における UNC-6 および UNC-5 局在異常を示す変異体の分離
Isolation of mutants that have abnormal localization of Netrin/UNC-6 or its receptor UNC-5 in C. elegans.
和賀　真子[1](Naoko Waga)，朝倉　太郎[1](Taro Asakura)，小倉　顕一[1](Ken-ichi Ogura)，
五嶋　良郎[1,2](Yoshio Goshima)
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P2-d24 嗅覚系の発生過程における転写因子 Arx 及び Fez の下流因子の解析
Downstream targets of Arx and Fez transcription factors regulating the development of mouse olfactory system
吉原　誠一[1](Sei-ichi Yoshihara)，日比　正彦[2](Masahiko Hibi)，北村　邦夫[3](Kunio Kitamura)，
吉原　良浩[4](Yoshihiro Yoshihara)，坪井　昭夫[1](Akio Tsuboi)
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P2-d25 netrin-G 分子群は神経回路特異的に樹状突起内の区画化を誘導する
Axonal netrin-Gs transneuronally determine lamina-specific subdendritic segments
西村 - 穐吉幸子(Sachiko Nishimura-Akiyoshi)，新美　君枝(Kimie Niimi)，仲柴　俊昭(Toshiaki Nakashiba)，
糸原　重美(Shigeyoshi Itohara)
理研・脳総研

P2-d26 大脳皮質神経細胞における HDAC9 の神経活動依存的な核・細胞質間移動による遺伝子発現と樹状突起形成の制御
Activity-dependent nucleocytoplasmic translocation of HDAC9 is required for gene expression and dendritic growth in cortical development
菅生　紀之[1](Noriyuki Sugo)，大城　洋明[1](Hiroaki Oshiro)，宋　　文杰[2](Wen-Jie Song)，
山本　亘彦[1](Nobuhiko Yamamoto)
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P2-d27 生後発達期における大脳皮質抑制性神経細胞移動の *in vivo* タイムラプスイメージング
*In vivo* time-lapse imaging of migration of cortical interneurons during postnatal development
稲田　浩之(Hiroyuki Inada)，和気　弘明(Hiroaki Wake)，鍋倉　淳一(Junichi Nabekura)
生理研・発達生理学・生体恒常

P2-d28 大脳皮質ニューロン細胞表面における DCC の分布はネトリン -1 により変化する
Netrin-1 regulates cell surface distribution of its receptor DCC in hamster primary cortical neurons
松本　英子(Hideko Matsumoto)，永島　雅文(Masabumi Nagashima)
埼玉医大・医・解剖

P2-d29 新規ゼブラフィッシュセマフォリンの同定
Identification of a novel zebrafish semaphorin
谷口　雅彦[1](Masahiko Taniguchi)，三上　義礼[2](Yoshinori Mikami)，増田　知之[3](Tomoyuki Masuda)，
木村　昌史[4](Masafumi Kimura)，吉田　知之[2](Tomoyuki Yoshida)，三品　昌美[2](Masayoshi Mishina)，
清水　孝雄[4](Takao Shimizu)
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## 栄養因子、サイトカイン
## Trophic Factors and Cytokines

P2-d30 **運動による脳内 TGF-beta の活性化機構**
**Mechanism of activation of transforming growth factor-beta in the brain by exercise**
藤川　哲兵(Teppei Fujikawa)，山田　裕之(Hiroyuki Yamada)，岩城　遥子(Yoko Iwaki)，
松村　成暢(Shigenobu Matsumura)，井上　和生(Kazuo Inoue)，伏木　　亨(Tohru Fushiki)
京大院・農・食品生物

P2-d31 **オンコスタチン M 受容体欠損マウスにおける後根神経節小型痛覚受容ニューロンの発生**
**The development of small nociceptive neurons in the dorsal root ganglia of oncostatin M receptor-deficient mice**
森川　吉博[1](Yoshihiro Morikawa)，小森　忠祐[1](Tadasuke Komori)，宮島　　篤[2](Atsushi Miyajima)，
仙波恵美子[1](Emiko Senba)
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P2-d32 **ニューレグリンの皮質抑制性シナプス発達促進作用**
**Neonatal heregulin / neuregulin signaling promotes development of cortical GABA neurons.**
阿部　佑一(Yuichi Abe)，難波　寿明(Hisaaki Namba)，那波　宏之(Hiroyuki Nawa)
新潟大学・脳研究所・分子神経生物

P2-d33 **Hypoxia inducible factor-1 α は ES 由来神経幹細胞からドーパミンニューロンへの分化作用に関与する**
**Hypoxia inducible factor 1-α participate in dopaminergic differentiation from ES-derived neural stem cells**
金　　泰善(Tae-Sun Kim)，飛田　秀樹(Hideki Hida)，増田　　匡(Tadashi Masuda)，
鄭　　且均(Cha-Gyun Jung)，西野　仁雄(Hitoo Nishino)
名市大院・医・脳神経生理

P2-d34 **幼若期上皮成長因子投与マウスの前頭皮質、海馬における GABA 抑制機能の低下**
**Neonatal EGF challenge attenuates GABAergic development in the neocortex and hippocampus**
難波　寿明(Hisaaki Namba)，阿部　佑一(Yuichi Abe)，那波　宏之(Hiroyuki Nawa)
新潟大・脳研・分子神経生物

P2-d35 **初代培養グリア細胞における LPS 誘導性 GDNF 遺伝子発現機構の解析**
**Analysis of regulatory mechanisms of LPS-induced GDNF gene expression in primary cultured glial cells**
田中　達英(Tatsuhide Tanaka)，大橋憲太郎(Kentaro Oh-hashi)，設楽　裕信(Hironobu Shitara)，
平田　洋子(Yoko Hirata)，木内　一壽(Kazutoshi Kiuchi)
岐阜大・工・生命工

P2-d36 **大脳皮質形成過程における幹細胞因子の役割**
**Roles of stem cell factor during mouse corticogenesis**
宗宮　仁美[1](Hitomi Soumiya)，福光　秀文[1](Hidefumi Fukumitsu)，国貞　隆弘[2](Takahiro Kunisada)，
古川　昭栄[1](Shoei Furukawa)
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P2-d37 **うつ病モデルマウスにおける神経栄養因子 mRNA 発現量と MAP キナーゼのリン酸化の解析**
**Decreases in the expression of neurotrophins and the phosphorylation of MAP kinase in an animal model of depression**
牧野　晃久(Akihisa Makino)，福光　秀文(Hidefumi Fukumitsu)，古川　昭栄(Shoei Furukawa)
岐阜薬大・薬・分子生物

P2-d38 BDNFで増強されるシナプス成熟をグルココルチコイドが抑制する
Glucocorticoid reduced the BDNF-mediated synaptic maturation in developing hippocampal neurons
熊丸 絵美[1](Emi Kumamaru), 沼川 忠広[1](Tadahiro Numakawa), 安達 直樹[1](Naoki Adachi), 矢ケ崎有希[1](Yuki Yagasaki), 泉 愛子[1,2](Aiko Izumi), 功刀 浩[1](Hiroshi Kunugi)
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P2-d39 グルココルチコイド暴露によるBDNF誘導グルタミン酸放出抑制のメカニズム
Glucocorticoid depresses the BDNF-induced glutamate release via the reduction of TrkB-GR interaction
沼川 忠広[1](Tadahiro Numakawa), 熊丸 絵美[1](Emi Kumamaru), 安達 直樹[1](Naoki Adachi), 矢ケ崎有希[1](Yuki Yagasaki), 泉 愛子[1,2](Aiko Izumi), 功刀 浩[1](Hiroshi Kunugi)
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P2-d40 ミクログリアからのATP誘発ケモカイン放出におけるNFATの関与
Involvement of NFAT in chemokine release from microglia induced by ATP
片岡 彩子(Ayako Kataoka), 津田 誠(Makoto Tsuda), 井上 和秀(Kazuhide Inoue)
九州大学・薬・薬理

P2-d41 Neuro2a細胞における小胞体ストレス障害に対するgenipinの保護効果
Protective effect of genipin on the typical ER stress-induced cell death
山崎眞津美[1](Matsumi Yamazaki), 千葉 賢三[1,2](Kenzo Chiba)
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P2-d42 BDNFとNT3の日内変動は幼若ラットの大脳皮質の機能発達に重要
Circadian changes of BDNF and NT-3 are important for cortical development of juvenile rats
浜武 通子[1](Michiko Hamatake), 松田 素子[1](Motoko Matsuda), 須藤 香織[1](Kaori Sudo), 永田 浩一[1](Koh-ichi Nagata), 一坂 吏志[2](Satoshi Ichisaka), 畠 義郎[2](Yoshio Hata), 仙波りつ子[1](Ritsuko Katoh-Semba)
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P2-d43 マウス神経系発達過程におけるTrkBスプライス変異型の発現比較
Distinct expression profiles of TrkB variants in the developing mouse nervous system
熊ノ郷晴子(Haruko Kumanogoh), 浅見 淳子(Junko Asami), 井上 高良(Takayoshi Inoue), 中村 俊(Shun Nakamura)
国立精神・神経センター・神経研・診断

## 神経細胞死、アポトーシス 1
## Neuronal Death and Apoptosis 1

P2-d44 神経細胞死におけるp25/Cdk5の核移行
The nuclear translocation of p25/Cdk5 in cultured neurons undergoing cell death
斎藤 太郎(Taro Saito), 遠藤 良(Ryo Endo), 浅田 明子(Akiko Asada), 久永 真市(Shin-ichi Hisanaga)
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P2-d45 The effect of TNF-$\alpha$ and IL-1$\beta$ against the A$\beta$25-35 induces neuronal cell death in mouse organotypic hippocampal slice cultures (OHSCs)
Huiling Jiang[1], HL Jiang[1], EC Suh[1], JY Kim[1], YA Kim[1], IH Cho[2], SJ Lee[2], KE Lee[1]
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P2-d46 無セルロプラスミン患者脳におけるミクログリアとアストロサイトによる神経細胞死の抑制
Microglia and astroglia prevent oxidative neuronal cell death in patient with aceruloplasminemia.
大城　聡[1](Satoru Oshiro)，曽根　敏男[2](Toshio Sone)，森岡　勝樹[3](Masaki Morioka)，
小林　新[4](Shin Kobayashi)，中嶋　一行[5](Kazuyuki Nakajima)
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P2-d47 ツニカマイシン添加後の小胞体ストレス誘導型アポトーシスにおける Bim の発現上昇の重要性
Importance of up-regulated Bim expression during tunicamycin-induced ER stress-mediated apoptosis in PC12 cells
下家　浩二(Koji Shimoke)，岸　宗一郎(Soichiro Kishi)，濱田　雅史(Masashi Hamada)，
笹谷　晴恵(Harue Sasaya)，藤田　裕子(Yuko Fujita)，池内　俊彦(Toshihiko Ikeuchi)
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P2-d48 小胞体ストレスによるアルツハイマー病発症機構においての Caspase-4 の役割
Signaling pathway of caspase-4 in endoplasmic reticulum stress-induced apoptosis of familial Alzheimer disease
行岡　太志[1](Futoshi Yukioka)，松崎　伸介[1,2](Shinsuke Matsuzaki)，河本　恵介[1](Keisuke Kawamoto)，
遠山　正彌[1,2](Masaya Tohyama)
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P2-e01 ラット運動ニューロン培養系における下垂体アデニル酸シクラーゼ活性化ポリペプチド（PACAP）の生存促進作用
Survival-promoting effect of pituitary adenylate cyclase-activating polypeptide (PACAP) on rat motoneurons in culture
富松　直子(Naoko Tomimatsu)，荒川　義弘(Yoshihiro Arakawa)
東京大学医学部附属病院・臨床試験部

P2-e02 カスパーゼ-4 活性化によるカスパーゼ-9, -3 を介した神経細胞死誘発機構
The mechanism of neuronal death through the activation of caspase-9,-3 via caspase-4 in human neuroblastoma SH-SY5Y cells
山室　晶子(Akiko Yamamuro)，大嶋　優(Yu Oshima)，吉岡　靖啓(Yasuhiro Yoshioka)，
笠井　淳司(Atsushi Kasai)，前田　定秋(Sadaaki Maeda)
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P2-e03 PC12 細胞におけるツニカマイシン、タプシガルギン、ポリグルタミン誘導 ER ストレスおよび細胞死に対するプロテアソーム阻害剤の影響
Effects of proteasome inhibitor on tunicamycin-, thapsigargin-, and polyglutamine-induced ER stress and cell death in PC12 cells
中山　均[1](Hitoshi Nakayama)，濱田　雅史[2](Masashi Hamada)，趙　晶[1](Jing Zhao)，
下家　浩二[2](Kouji Shimoke)，磯崎　稔[1](Minoru Isosaki)，佐藤　広康[1](Hiroyasu Satoh)，
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P2-e04 神経細胞死関連遺伝子のゲノムワイドスクリーニング
A genomic screen for novel genes with a cell-death-related function in neurons
米倉　真一(Shinichi Yonekura)，高澤　隆紀(Takanori Takazawa)，渡部　和彦(Kazuhiko Watabe)
東京都神経研・分子神経病理

P2-e05 グリセロアルデヒド3リン酸脱水素酵素のアミロイド様凝集と神経細胞死への関与
Amyloid-like aggregate of human GAPDH is involved in neuronal cell death
天野　渉(Wataru Amano)，中嶋　秀満(Hidemitsu Nakajima)，東　泰孝(Yasu-Taka Azuma)，
竹内　正吉(Tadayoshi Takeuchi)
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P2-e06 Cyclophosphamide による培養大脳皮質ニューロンの細胞死抑制効果
Survival effect of cyclophosphamide in cultured cortical neurons
北澤　裕美[1](Hiromi Kitazawa)，沼川　忠広[2](Tadahiro Numakawa)，安達　直樹[2](Naoki Adachi)，
熊丸　絵美[2](Emi Kumamaru)，Madinyet Niyaz[1]，泉　愛子[2](Aiko Izumi)，功刀　浩[2](Hiroshi Kunugi)，
工藤　玄恵[1](Motoshige Kudo)
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P2-e07 ER ストレス下のグリアにおけるコンドロイチン硫酸の機能
Function of chondoroitin sulfate in ER stress exposed glial cells
名取　貴光(Takamitsu Natori)，三宅　邦夫(Kunio Miyake)，長井　薫(Kaoru Nagai)
山梨大院・医・環境遺伝医学

P2-e08 AMPA は海馬神経細胞において早期に necrosis と DNA 断片化を誘導する
Alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) rapidly induces necrosis and DNA fragmentation in hippocampal neurons
茨木　京子(Kyoko Ibaraki)，山本　清二(Seiji Yamamoto)，宮川　厚夫(Atsuo Miyakawa)，
寺川　進(Susumu Terakawa)
浜松医科大学・光量子医学研究センター・細胞イメージング

P2-e09 細胞内遊離高マンノース型オリゴ糖による小胞体ストレス誘導細胞死の抑制効果
Free intracellular high-mannose oligosaccharides protect cell death induced by endoplasmic reticulum (ER) stress
三宅　邦夫(Kunio Miyake)，名取　貴光(Takamitsu Natori)，長井　薫(Kaoru Nagai)
山梨大院・医・環境遺伝

P2-e10 酸化ストレス誘導性細胞死に対する PDGF-B の神経細胞保護は Phosphoinositide 3-kinase/Akt シグナル経路を介する
Neuroprotection of PDGF-B against Oxidative Stress via the PI3K/Akt signal
鄭　蓮順(Lianshun Zheng)，石井　陽子(Yoko Ishii)，尾矢　剛志(Takeshi Oya)，
徳永　綾乃(Ayano Tokunaga)，濱島　丈(Takeru Hamashima)，石澤　伸(Shin Ishizawa)，
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P2-e11 Neuro2a 細胞の小胞体ストレス誘導細胞死における DNA 脱メチル化の影響
DNA demethylation is involved in ER stress induced cell death in mouse neuroblastoma Neuro2a cells
長井　薫(Kaoru Nagai)，名取　貴光(Takamitsu Natori)，三宅　邦夫(Kunio Miyake)
山梨大院・医・環境遺伝医学

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## Somatomotor System 2

P2-e12 覚醒ネコにおける随意運動時横隔膜の活動様式
Activity patterns of diaphragm during voluntary movements in awake cats
宇賀美奈子[1,2](Minako Uga)，丹羽　正利[3](Masatoshi Niwa)，落合　直之[1](Naoyuki Ochiai)，
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P2-e13 ニホンサルのトレッドミル歩行における一次運動野の神経細胞活動
Neuronal activity in primary motor cortex during treadmill locomotion of the Japanese monkey
中隋　克己[1](Katsumi Nakajima)，森　大志[2](Futoshi Mori)，村田　哲[1](Akira Murata)，
稲瀬　正彦[1](Masahiko Inase)
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P2-e14 前肢運動中の頭部固定ラットからの傍細胞・マルチユニット記録
Juxtacellular/multiunit recordings from head-restraint rats performing forelimb movement task
礒村　宜和[1,2](Yoshikazu Isomura)，春国　梨恵[1](Rie Harukuni)，田中さやか[1](Sayaka Tanaka)，
相澤　秀紀[3](Hidenori Aizawa)，深井　朋樹[1](Tomoki Fukai)
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P2-e15 第一中足骨骨頭下の足底への各種速度圧刺激と姿勢運動
Postural movements induced by pressure stimulus with various velocities to the plantar sole under first metatarsal head
藤原　勝夫(Katsuo Fujiwara)，澁谷久美子(Kumiko Shibuya)，中　　正美(Masami Naka)，
黒川　　望(Nozomi Kurokawa)
金沢大学・医学系研究科・運動生体管理学

P2-e16 全身性ジストニアモデルマウスにおける大脳基底核の活動異常
Abnormal activity of basal ganglia neurons in a DYT1 transgenic mouse model of early-onset generalized dystonia.
知見　聡美[1](Satomi Chiken)，Pullanipally Shashidharan[2]，南部　　篤[1](Atsushi Nambu)
[1]生理研・生体システム，[2]マウントサイナイ医科大学

P2-e17 サルの淡蒼球における大脳皮質運動領野からの視床下核を介した投射様式
Organization of motor cortical inputs in the globus pallidus via the subthalamic nucleus in the monkey
岩室　宏一[1,2](Hirokazu Iwamuro)，橘　　吉寿[1](*Yoshihisa Tachibana*)，斉藤　延人[2](Nobuhito Saito)，
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P2-e18 精密把握運動中に見られる脊髄 - 筋活動間のコヒーレンス
Spinomuscular coherence in monkey performing a precision grip task
武井　智彦[1,4](Tomohiko Takei)，関　　和彦[2,3](Kazuhiko Seki)
[1]京大院・人環，[2]生理学研究所・発達生理，[3]総研大院・生理科学，[4](独)　日本学術振興会特別研究員

P2-e19 ゼブラフィッシュの M-escape と Non-M escape は異なる感覚系によって駆動される
Different pathways for Mauthner and Non-Mauthner escapes in zebrafish
小橋　常彦(Tsunehiko Kohashi)，小田　洋一(Yoichi Oda)
名古屋大院・理・生命理学

P2-e20 運動野から入力を受けるサル淡蒼球ニューロンの運動課題遂行中の活動様式
Activity of globus pallidus neurons in behaving monkeys that receive inputs from cortical motor areas.
高良　沙幸[1,2](Sayuki Takara)，畑中　伸彦[1,2](Nobuhiko Hatanaka)，高田　昌彦[3](Masahiko Takada)，
南部　　篤[1,2](Atsushi Nambu)
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統合生理研究部門

P2-e21 運動課題遂行中のサルにおける淡蒼球ニューロン活動のグルタミン酸および GABA 作動性調節
Glutamatergic and GABAergic modulation of monkey pallidal neuron activity in relation to motor task.
畑中　伸彦[1,2](Nobuhiko Hatanaka)，高良　沙幸[1,2](Sayuki Takara)，橘　　吉寿[1,2](Yoshihisa Tachibana)，
高田　昌彦[3](Masahiko Takada)，南部　　篤[1,2](Atsushi Nambu)
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P2-e22 動揺床面上における除脳ラットの姿勢調節
Postural adjustment of decerebrate rats on the rotating platform
岩井　　史(Fumihito Iwai)，山口　峻司(Takashi Yamaguchi)
山形大院・理工・生体センシング機能工学

P2-e23 倒立振子バランス制御における眼球運動
Eye movements during balance control of the inverted pendulum
落合　亮平(Ryohei Ochiai)，澤畑　博人(Hirohito Sawahata)，山口　峻司(Takashi Yamaguchi)
山形大院・理工・生体センシング機能工学

P2-e24 線条体ストリオソーム・マトリックスにおけるコリン作動性介在ニューロンに対するミューオピオイド受容体の調節作用
Modulation of μ opioid receptors on cholinergic interneurons in the striosomes/matrix compartments of the striatum.
三浦　正巳[1](Masami Miura)，斎野・斉藤幸子[2](Sachiko Saino-Saito)，増田　正雄[1](Masao Masuda)，
小林　和人[3](Kazuto Kobayashi)，青崎　敏彦[1](Toshihiko Aosaki)
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P2-e25 前頭葉内側運動野の動作関連細胞活動における座標表現
Reference frame of action-related neuronal activity in the medial frontal motor areas
中島　　敏[1](Toshi Nakajima)，虫明　　元[1](Hajime Mushiake)，保坂　亮介[2](Ryosuke Hosaka)，
丹治　　順[3](Jun Tanji)
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P2-e26 サルの頸髄レベルでの皮質脊髄路切断後における手指巧緻性の回復；C2 での切断と C5 での切断の回復比較
Recovery of the finger dexterity after lesion of the corticospinal tract at cervical segments in monkey; Lesion at the C2 compared to the C5
坪井　史治[1,2](Fumiharu Tsuboi)，西村　幸男[2,3](Yukio Nishimura)，齋藤紀美香[2](Kimika Saito)，
高橋　雅人[2,4](Masahito Takahashi)，伊佐　　正[2,3](Tadashi Isa)
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P2-e27 脊髄前角の C- 終末の起始細胞は VII 層内側のコリン作動性ニューロンである
C-terminals in the spinal ventral horn originate in cholinergic neurons in the medial lamina VII
中田　隆博[1](Takahiro Nakata)，松田　正司[2](Seiji Matsuda)，松村　譲兒[3](George Matsumura)，
小林　　靖[1](Yasushi Kobayashi)
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## 視覚 2
## Visual System 2

P2-e28 初期視覚系の周辺文脈依存効果を基とした自然画像における図地分離メカニズム
Figure/ground segregation in natural images based on surround modulation in early vision
渡辺　哲次(Satoshi Watanabe)，西村　　悠(Haruka Nishimura)，酒井　　宏(Ko Sakai)
筑波大学・システム情報工学・コンピュータサイエンス

P2-e29 遮蔽された表面に対する V1/V2 応答の空間特性
Spatial specificity of V1/V2 responses to an occluded surface
番　　浩志[1,2](Hiroshi Ban)，山本　洋紀[1](Hiroki Yamamoto)，花川　　隆[3](Takashi Hanakawa)，
浦山　慎一[4](Shin-ichi Urayama)，麻生　俊彦[4](Toshihiko Aso)，福山　秀直[4](Hidenao Fukuyama)，
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P2-e30 サル TE 野ニューロン活動と色知覚の関係
Relationship between color perception and neural activity in area TE of the monkey
松茂良岳広[1](Takehiro Matsumora)，鯉田　孝和[2](Kowa Koida)，小松　英彦[1,2](Hidehiko Komatsu)
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P2-e31 Explicit representation of 3D object parts in monkey inferotemporal cortex

Yukako Yamane[1], Eric Carlson[1,2], Zhihong Wang[1], Katherine Bowman[1,3], Charles E Connor[1]
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P2-e32 ヒト両眼視システムによる、パターン変化の速い像と遅い像に対する奥行き弁別
Depth discrimination for transient and sustained images by the human stereoscopic system

高野　真希(Maki Takano), 土井　隆弘(Takahiro Doi), 藤田　一郎(Ichiro Fujita)
大阪大院・生命機能

P2-e33 初期視覚野細胞における3次元両眼受容野解析
An evaluation of 3-d binocular receptive fields in the early visual cortex

田渕　有香[1](Yuka Tabuchi), 佐々木耕太[2](Kota Sasaki), 大澤　五住[1,2](Izumi Ohzawa)
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P2-e34 マカクザル視覚皮質における第III層錐体細胞の電気生理学的性質の発達による変化
Developmental changes in electrophysiological properties of layer III pyramidal neurons in macaque visual cortices

丸山　敦子[1](Atsuko Maruyama), 石川　理子[2](Ayako Ishikawa), 細山　大輔[2](Daisuke Hosoyama),
吉村由美子[1,3](Yumiko Yoshimura), 田村　弘[1,2](Hiroshi Tamura), 佐藤　宏道[2](Hiromichi Sato),
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P2-e35 巨大画像より近似的に抽出された独立成分の解析
Approximate independent components of large natural scenes

松田　源立(Yoshitatsu Matsuda)
青山学院大・理工・情報テクノロジー

P2-e36 MEG/fMRI統合解析技術を用いた3次元物体知覚に関わる脳神経活動の可視化
Neural dynamics involved in the 3-D object perception as aseessed by MEG/fMRI integrated imaging technique

岩木　直[1](Sunao Iwaki), Giorgio Bonmassar[2], John W Belliveau[2]
[1]産総研, [2]マサチューセッツ総合病院

P2-e37 方位選択性を説明するフィードフォワードモデルとフィードバックモデルにおける符号化効率の違い
Difference in coding efficiency between feedforward and feedback models of orientation selectivity

大泉　匡史[1](Masafumi Oizumi), 三浦　佳二[1,2](Keiji Miura), 岡田　真人[1,2](Masato Okada)
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P2-e38 ネコ第一次視覚野における内因性光学的信号の経時的変化特性
Dynamics of intrinsic optical signals in cat primary visual cortex

鈴木　航(Wataru Suzuki), 永井　翠(Midori Nagai), 大塚　俊幸(Toshiyuki Ootsuka),
王　鋼(Gang Wang)
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P2-e39 A new approach to estimating receptive fields from responses to natural stimuli

Nicolas A. Lesica[1], Toshihiko Hosoya[2]
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P2-e40 The origin of spreading burst activities in the local circuit of the superior colliculus

P. Phongphanphanee[1,2], K. Kaneda[1,2], T. Isa[1,2,3]
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P2-e41 Visual cortical representation in cats reared under the deprivation of pattern vision

Toshiki Tani, Jerome Ribot, Shigeru Tanaka
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P2-e42 Critical contribution of slow-decay synaptic potential to complex-cell receptive field formation

Masanobu Miyashita[1,2], Shigeru Tanaka[1]
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P2-e43 神経特異的抗原 C38 は神経成熟を促進する
C38 enhances neuronal maturation of P19 embryonal carcinoma cells.

若林 毅俊[1](Taketoshi Wakabayashi), 森 徹自[1](Tetsuji Mori), 高森 康晴[1](Yasuharu Takamori),
小阪 淳[2](Jun Kosaka), 三木友香里[2](Yukari Miki), 山田 久夫[1](Hisao Yamada)
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P2-e44 視覚空間情報とヒゲ空間情報の不一致で生ずる斜視マウス視覚野応答の抑圧
Visual cortical depression induced by spatial sensory mismatching between visual and whisker inputs in strabismic mice

吉武 講平(Kouhei Yoshitake), 任海 学(Manavu Tohmi), 工藤 雅治(Masaharu Kudoh),
澁木 克栄(Katsuei Shibuki)
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P2-e45 2光子励起グルタミン酸活性化法によるラット視覚野錐体細胞に対するシナプス入力細胞のマッピング
Synaptic connections to layer 2/3 pyramidal neurons in rat visual cortex revealed by two-photon activation of caged glutamate

松崎 政紀[1,2](Masanori Matsuzaki), Graham C.R. Ellis-Davies[3], 河西 春郎[1](Haruo Kasai)
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P2-e46 NMDA 誘発マウス網膜障害モデルにおける外側外状体の経時的・部位的変化
Time-dependent and exofocal alterations in the lateral geniculate nucleus following retinal damage in mice

伊藤 保志[1](Yasushi Ito), 嶋澤 雅光[1](Masamitsu Shimazawa), 井口 勇太[1](Yuta Inokuchi),
福光 秀文[2](Hidefumi Fukumitsu), 古川 昭栄[2](Shoei Furukawa), 新家 眞[3](Makoto Araie),
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P2-e47 視覚情報伝達におけるコレシストキニン A 受容体の機能：体内時計の調節
Function of CCK-A receptors for the visual signal transductions: Regulation of the circadian clock system

池田 真行[1](Masayuki Ikeda), 島添 隆雄[2](Takao Shimazoe), 瀧口 総一[3](Souichi Takiguchi)
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P2-e48 遠方縮瞳誘導による近視の改善
A novel device improves myopic visual acuity through pupil constriction during far accommodation

堀江 秀典[1,2,3](Hidenori Horie), 湯田 兼次[4](Kenji Yuda), 大川 栄一[5](Eiichi Ookawa),
久原 聡[5](Satoshi Hisahara), 魚里 博[6](Hiroshi Uozato), 堀江 寛子[2](Hiroko Horie),
中嶋 聡美[2](Satomi Nakajima), 谷岡 健吉[7](Kenkichi Tanioka), 大川 裕司[7](Yuji Ohkawa),
松原 智紀[7](Tomoki Matubara), Wolfram Tetzlaff[8]
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## 聴覚、前庭感覚 2
## Auditory and Vestibular Systems 2

P2-f01 **聴覚皮質ニューロンのローパスフィルタ特性によって引き起こされる音脈分凝**
**Auditory stream segregation induced by low-pass filtering characteristics of auditory cortical neurons**
杉本　俊二(Shunji Sugimoto)，田中　宏幸(Hiroyuki Tanaka)，鈴木　雄太(Yuuta Suzuki)，
堀川　順生(Junsei Horikawa)
豊橋技科大・工・知識情報

P2-f02 **モルモット大脳皮質聴覚野背尾側領域の尾側に位置するベルト領域の特徴**
**Characterization of belt regions caudal to the dorsocaudal field in guinea pig auditory cortex**
西村　方孝(Masataka Nishimura)，宋　　文杰(Wen-Jie Song)
熊本大院・医・知覚生理

P2-f03 **聴覚系の上オリーブ核群における NB-2 の発現**
**Expression of NB-2 in the superior olivary complex of the auditory system.**
豊島　　学[1](Manabu Toyoshima)，桜井　都衣[1](Kunie Sakurai)，島崎久仁子[2](Kuniko Shimazaki)，
霜田　　靖[1](Yashushi Shimoda)，渡邉　和忠[1](Kazutada Watanabe)
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P2-f04 **視床網様核から内側膝状体への投射様式**
**Topography of thalamic reticular nucleus projections to the medial geniculate body**
木村　晃久(Akihisa Kimura)，堂西　倫弘(Tomohiro Donishi)，井辺　弘樹(Hiroki Imbe)，
玉井　靖彦(Yasuhiko Tamai)
和歌山県立医科大学・医・第一生理

P2-f05 **聴覚中枢における両耳間時間差・強度差の相互作用：心理物理学的アプローチ**
**Interaction between interaural time and intensity differences in the central auditory system: A psychophysical approach**
古川　茂人(Shigeto Furukawa)
日本電信電話株式会社・ＮＴＴ　ＣＳ基礎研・人間情報研究部

P2-f06 **聴覚皮質ニューロンの音反応性にみられる環境刺激による変調**
**Modulatory effects of environmental stimuli on the acoustic responsiveness of auditory cortical neurons**
小島　久幸[1,2](Hisayuki Ojima)，田岡　三希[1](Miki Taoka)，入來　篤史[1,2](Atsushi Iriki)
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P2-f07 **ラット内耳の蝸牛外側壁線維細胞の生後変化**
**Developmental changes in cochlear spiral ligament fibrocytes of rat inner ear**
長嶋　玲子[1](Reiko Nagashima)，務台　英樹[2](Hideki Mutai)，松永　達雄[2](Tatsuo Matsunaga)，
荻田喜代一[1](Kiyokazu Ogita)
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P2-f08 **マウス蝸牛有毛細胞における酸感受性イオンチャネル ASIC1b の発現**
**Acid-sensing ion channel-1b in the stereocilia of mammalian cochlear hair cells**
鵜川　眞也(Shinya Ugawa)，石田　雄介(Yusuke Ishida)，植田　高史(Takashi Ueda)，
島田　昌一(Shoichi Shimada)
名古屋市立大学・大学院医学研究科・機能組織学

P2-f09 **ラットの海馬 CA1 野から側頭葉聴覚野への多シナプス性投射**
**Multisynaptic pathways from CA1 of the hippocampus to the temporal auditory cortex in the rat**
山崎　吉之[1](Yoshiyuki Yamazaki)，石塚　典生[1](Norio Ishizuka)，塚田　　稔[2](Minoru Tsukada)，
高田　昌彦[1](Masahiko Takada)
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P2-f10 Virtual reality によって作り出された optic flow が眼球運動と歩行に与える影響
Effects of optic flow produced by virtual reality on ocular movement and locomotion
西池　季隆[1](Suetaka Nishiike)，大山　晴三[2](Seizo Ohyama)，渡邊　洋[3](Hiroshi Watanabe)，
松岡　克典[3](Katsunori Matsuoka)，武田　憲昭[2](Noriaki Takeda)，原田　保[1](Tamotsu Harada)
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P2-f11 Juxtacellular 法および in vivo patch clamp 法による哺乳類下丘神経回路の研究
The local circuits of mammalian inferior colliculus studied by juxtacellular staining and in vivo whole cell recording
小野　宗範(Munenori Ono)，笠井　昌俊(Masatoshi Kasai)，大森　治紀(Harunori Ohmori)
京都大学院・医・神経生物

P2-f12 頭部垂直回転刺激に対する小脳小節・虫部垂プルキンエ細胞の発射応答パターン
Firing patterns of Purkinje cells in the cerebellar nodulus and uvula during vertical head rotation in awake cats.
北間　敏弘[1](Toshihiro Kitama)，嶋宮　民安[1](Tamiyasu Shimamiya)，佐藤　悠[2](Yu Sato)
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P2-f13 聴覚空間地図：スナネズミの中脳神経核間の比較
Auditory space map: Comparison of the three midbrain nuclei of gerbils
牧　勝弘(Katuhiro Maki)，古川　茂人(Shigeto Furukawa)
日本電信電話（株）・NTT CS基礎研

P2-f14 周波数変調音に対する覚醒ネコ一次聴覚野の持続性反応
Sustained responses to frequency modulated sound in cat primary auditory cortex
秦　嶺(Ling Qin)，王　京宇(Jingyu Wang)，佐藤　悠(Yu Sato)
山梨大学・第二生理

## 体性感覚
## Somatosensory System

P2-f15 一本ヒゲ刺激方向弁別における毛帯路系の役割
A role of the lemniscal pathway in the single-whisker directional discrimination in rats
中村　晋也(Shinya Nakamura)，鳴海　毅亮(Takaaki Narumi)，吉里信一郎(Shin-ichiro Yoshizato)，
筒井健一郎(Ken-Ichiro Tsutsui)，飯島　敏夫(Toshio Iijima)
東北大院・生命・脳情報処理

P2-f16 ノルアドレナリン系の活性化は視床後内側腹側核のシグナル / ノイズ比を上昇させる
Noradrenergic activation amplifies signal-to-noise ratios in the VPM thalamus
平田　昭夫(Akio Hirata)，Manuel A Castro-Alamancos
Department of Neurobiology and Anatomy, Drexel University College of Medicine

P2-f17 マウスバレル野切片の視床刺激応答の経験依存的可塑性
Experience-dependent plasticity induced by whisker trimming in thalamocortical responses of slices obtained from the mouse barrel cortex
渡邉　健児(Kenji Watanabe)，鎌谷　大樹(Daiki Kamatani)，菱田　竜一(Ryuichi Hishida)，
工藤　雅治(Masaharu Kudoh)，澁木　克栄(Katsuei Shibuki)
新潟大・脳研・システム脳生理

P2-f18 インビボパッチクランプ記録法を用いた炎症性ラット一次体性感覚野の解析
*In vivo* patch clamp analysis of synaptic responses evoked in primary somatosensory cortex in inflamed rats
古賀　浩平(Kohei Koga)，塩川　浩輝(Hiroaki Shiokawa)，水野　雅晴(Masaharu Mizuno)，
土井　篤(Atsushi Doi)，高瀬 - 水口洋子(Hiroko Takase-Mizuguchi)，古江　秀昌(Hidemasa Furue)，
吉村　恵(Megumu Yoshimura)
九州大学院・医・統合生理

P2-f19 **末梢神経の自発発火阻止によって誘発されるマウス体性感覚野応答の増強**
**Sensitization of somatosensory cortical responses after blockade of spontaneous peripheral nerve firing in mice.**
駒形　成司[1](Seiji Komagata)，陳　　山林[2](Shanlin Chen)，工藤　雅治[1](Masaharu Kudoh)，
柴田　　実[2](Minoru Shibata)，澁木　克栄[1](Katsuei Shibuki)
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P2-f20 **脊髄マイクログリアにおけるTLR3の活性化と神経因性疼痛**
**Activation of Toll-like receptor 3 in spinal microglia contributes to tactile allodynia after nerve injury**
小畑　浩一(Koichi Obata)，野口　光一(Koichi Noguchi)
兵庫医科大学・解剖学第２講座

P2-f21 **脊髄アストロサイトにおけるTAK1の発現上昇とアロディニア**
**TAK1 upregulation in spinal astrocytes contributes to tactile allodynia after nerve injury**
桂　　弘和[1](Hirokazu Katsura)，小畑　浩一[1](Koichi Obata)，阪上　雅史[2](Masafumi Sakagami)，
野口　光一[1](Koichi Noguchi)
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P2-f22 **脊髄後角介在ニューロンにおけるプロスタグランジンの役割**
**Functional analyses of the prostaglandin (PGE2) receptors in the interneurons of the spinal dorsal horn**
川手　豊子(Toyoko Kawate)，竹田　　扇(Sen Takeda)
山梨大院・医・解剖学

P2-f23 **リン酸化NFkBは１次および高次感覚ニューロンの活動マーカーとして利用できる**
**Phospho-NFkB is a suitable marker for activated primary and higher order sensory neurons.**
松村　　潔[1](Kiyoshi Matsumura)，西谷　典浩[1](Norihiro Nishitani)，三井健太郎[1](Kentarou Mii)，
田村　雅行[1](Masayuki Tamura)，細川　　浩[2](Hiroshi Hosokawa)，小林　茂夫[2](Shigeo Kobayashi)
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P2-f24 **力覚に必要な一次運動野の神経活動**
**Is it enough for neuronal excitability of MI to produce force perception?**
宝田　雄大[1](Yudai Takarada)，泰羅　雅登[2](Masato Taira)，福山　秀直[3](Hidenao Fukuyama)，
美馬　達哉[3](Tatsuya Mima)，中塚　晶博[3](Masahiro Nakatsuka)，阿部　十也[3](Mitsunari Abe)
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P2-f25 **On selectivity of laser-heat irradiation in activation of cutaneous nociceptors**
Wan-Ting Tseng，Yen Chen-Tung
Institude of Zoology, National Taiwan University, Taiwan, Taipei

P2-f26 **c-fos expression in the catecholaminergic neurons of the brainstem after eccentric rotation of rats**
Yulin Dong，FX Zhang，YQ Li
Department of Anatomy, The Fourth Military Medical University

P2-f27 **ラットの頭部仰角に関連した神経活動**
**Neural activity of rats related to head elevation angle**
大久保達夫[1](Tatsuo Okubo)，谷口　徳恭[2](Noriyuki Taniguchi)，深山　　理[1](Osamu Fukayama)，
鈴木　隆文[1](Takafumi Suzuki)，満渕　邦彦[1,2](Kunihiko Mabuchi)
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P2-f28 **マウス三叉神経の３つの枝の感覚線維の数と太さの計測**
**Measurement of the size and number of the sensory fibers in three branches of the trigeminal nerve in the mouse**
吉本　正美(Masami Yoshimoto)，山本　直之(Naoyuki Yamamoto)，小澤　一史(Hitoshi Ozawa)
日本医科大・医・解剖学（生体構造学）

P2-f29 ラットのヒゲを用いた物体認識における神経メカニズム
Population coding of tactile information of object in rat's whiskerring behavior
武井　央仁[1](Hirohito Takei)，藤田　一寿[1](Kazuhisa Fujita)，樫森与志喜[2](Yoshiki Kashimori)
[1]電気通信大学情報システム学研究科，[2]電気通信大学量子物質工学科

P2-f30 三叉神経核群中間核から主感覚核への抑制性入力
Inhibitory input from the interpolar nucleus of the spinal trigeminal complex to the principal trigeminal nucleus
古田　貴寛[1](Takahiro Furuta)，Elena Timofeeva[2]，岡本_古田敬子[1](Keiko Okamoto-Furuta)，
Martin Deschenes[2]，中村　公一[1,3](Kouichi Nakamura)，金子　武嗣[1,3](Takeshi Kaneko)
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P2-f31 オレキシンによる三叉神経中脳路核ニューロン活動の調節
Orexin modulates neuronal activities in rat mesencephalic trigeminal sensory neurons
中山希世美(Kiyomi Nakayama)，中村　史朗(Shiro Nakamura)，井上　富雄(Tomio Inoue)
昭和大・歯・口腔生理

P2-f32 手指と口の運動中に発火するニホンザル第二体性感覚野ニューロン
Neurons that become active during hand and mouth movements in the hand region of the secondary somatosensory cortex of the macaque monkey
田岡　三希[1](Miki Taoka)，田中美智雄[1](Michio Tanaka)，小島　久幸[1](Hisayuki Ojima)，
日原さやか[1,2](Sayaka Hihara)，入來　篤史[1,2](Atsushi Iriki)
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## ブレイン・マシン／コンピュータ・インターフェイス
## BMI/BCI

P2-f33 ラット海馬のマルチニューロン活動を用いた行動推測
Behavioral inference based on hippocampal multi-neuronal activities of rats
野村　真樹[1,2](Masaki Nomura)，櫻井　芳雄[1,3](Yoshio Sakurai)，青柳富誌生[1,2](Toshio Aoyagi)
[1]CREST・科学技術振興機構，[2]京都大学情報学研究科，[3]京都大学文学研究科

P2-f34 前頭連合野ニューロン集団の単一試行活動に基づく多次元意思決定の予測
Prediction of multi-dimensional decision-making based on the single-trial activities of a population of prefrontal neurons
松本　有央[1](Narihisa Matsumoto)，長谷川良平[1,2](Ryohei P Hasegawa)
[1]産総研・脳神経，[2]米国国立衛生研

P2-f35 方位推定アルゴリズム獲得のための電極開発
Development of an electrodes array for a homogeneous sampling of orientation tuned cells in the visual cortex
圓山　由子(Yoshiko Maruyama)，伊藤　浩之(Hiroyuki Ito)
京都産業大院・工・情報通信

P2-f36 マルチユニットレコーディングに基づく細胞外多細胞活動のモデリング
Modeling of extracellular multiple neuronal activities based on the multi-unit recording
久保　貴嗣[1](Takashi Kubo)，片山　統裕[1](Norihiro Katayama)，虫明　元[2](Hajime Mushiake)，
菊池　修[1](Osamu Kikuchi)，辛島　彰洋[1](Akihiro Karashima)，中尾　光之[1](Mitsuyuki Nakao)
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P2-f37 聴性定常反応 EEG を用いた注意配分推定の研究
Study of attention assignment estimation by Auditory Steady State Response EEG
大貫　泰(Yasushi Oonuki)，前山　健(Takeshi Maeyama)，岡田　浩之(Hiroyuki Okada)，
佐々木　寛(Hiroshi Sasaki)，大森　隆司(Takashi Omori)
玉川大学・工

P2-f38 脳波を用いたBCIシステムのためのオンラインデータ処理プラットフォームの開発
Development of on-line data processing platform for EEG-based BCI
Susila I Putu, 加納慎一郎(Shin'ichiro Kanoh), 宮本浩一郎(Koichiro Miyamoto),
吉信 達夫(Tatsuo Yoshinobu)
東北大学大学院工学研究科電子工学専攻

P2-f39 上肢の到達運動と姿勢を再現できるブレインマシンインターフェース
A brain-machine interface for predicting both arm-reaching movements and postures
廣瀬 秀顕[1,4](Hideaki Hirose), 崔 圭完[2](Kyuwan Choi), 筒井健一郎[1](Ken-Ichiro Tsutsui),
櫻井 芳雄[3,4](Yoshio Sakurai), 小池 康晴[2,4](Yasuharu Koike), 飯島 敏夫[1,4](Toshio Iijima)
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## 自律神経系 2
## Autonomic Nervous System 2

P2-f40 脳室内投与CRFによる交感神経 - 副腎髄質系賦活における視床下部脊髄路での一酸化窒素合成酵素の役割
Role of nitric oxide synthase-containing hypothalamic paraventricular-spinal neurons in the CRF-induced activation of the central sympatho-adrenal outflow in rats
臼井 大介[1,2](Daisuke Usui), 山口奈緒子[1](Naoko Yamaguchi), 岡田尚志郎[1](Shoshiro Okada),
清水 孝洋[1](Takahiro Shimizu), 脇口 宏[2](Hiroshi Wakiguchi), 横谷 邦彦[1](Kunihiko Yokotani)
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P2-f41 延髄網様体ニューロンの伝達の可塑的変化におけるニューロキニン1受容体の役割
Neurokinin 1 receptor plastically changes excitability of neurons in reticular formation of rat medulla oblongata
氷見 直之[1](Naoyuki Himi), 古我 知成[2](Tomoshige Koga), 辻岡 克彦[1](Katsuhiko Tsujioka)
[1]川崎医大・生理学, [2]川崎医療福祉大 リハビリテーション

P2-f42 乳幼期ラットの背内側延髄の5-HT遊離に対する$CO_2$吸入の効果
Effects of $CO_2$ inhalation on 5-HT release in the dorsomedial medulla oblongata in infant rats
金丸みつ子(Mitsuko Kanamaru), 本間 生夫(Ikuo Homma)
昭和大・医・第二生理

P2-f43 ラットの覚醒 / あくび反応におけるコルチコトロピン放出因子ニューロンの役割
The role of corticotropin-releasing factor neurons on arousal/yawning response of rats
久保田夏子(Natsuko Kubota), 柳田 信也(Shinya Yanagita), 北 一郎(Ichiro Kita)
首都大学東京大学院・人間健康科学・行動生理

P2-f44 吻側延髄網様体小細胞部ニューロンの心拍同期活動に対する舌神経刺激の効果と吻側延髄腹外側部への投射
The effect of lingual nerve stimulation on the cardiac-related activity of the parvicellular reticular formation neurons, and their projections to the rostral ventrolateral medulla
石塚 健一(Kenichi Ishizuka), Oskutyte Diana, 佐藤 義英(Yoshihide Satoh),
村上 俊樹(Toshiki Murakami)
日本歯科大学・新潟生命歯学部・生理学講座

P2-f45 新生ラットin vitro標本における前吸息性(Pre-I)及び吸息性ニューロンのリズム形成に対する延髄左右離断の影響
Effects of midline sagittal sectioning of medulla on pre-inspiratory and inspiratory neuron activity in vitro
鬼丸 洋(Hiroshi Onimaru), 本間 生夫(Ikuo Homma)
昭和大・医・第二生理

P2-f46 横隔膜下食道の筋層間神経節に直接投射する迷走神経背側運動核ニューロンのシナプス構成
Ultrastructure of the myenteric ganglion of the subdiaphragmatic esophagus and its vagal efferent terminals from the dorsal motor nucleus of the vagus (DMV) of the rat
早川　徹(Tetsu Hayakawa)，桑原　佐知(Sachi Kuwahara)，前田　誠司(Seishi Maeda)，
田中　宏一(Koichi Tanaka)，関　真(Makoto Seki)
兵庫医大第一解剖

P2-f47 ラット脳室内投与したNMDAは脳内トロンボキサン$A_2$を介して副腎髄質からカテコールアミンを分泌する
Brain thromboxane $A_2$ is involved in the centrally administered NMDA-induced adrenomedullary outflow in rats
岡田尚志郎(Shoshiro Okada)，山口奈緒子(Naoko Yamaguchi)，清水　孝洋(Takahiro Shimizu)，
新井　淳一(Jyunichi Arai)，横谷　邦彦(Kunihiko Yokotani)
高知大・医・薬理

P2-f48 室傍核へ投射する脳弓下器官ニューロンに対する正中視索前核ニューロンのシナプス
Synaptic contacts between median preoptic neurons and subfornical organ neurons projecting to the paraventricular hypothalamic nucleus
河野　史[1](Hitoshi Kawano)，増子　貞彦[2](Sadahiko Masuko)，段　平国[2](Ping-Guo Duan)
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P2-g01 ラット孤束核交連亜核から横隔神経核へのグルタミン酸作動性神経路
Glutamatergic pathways from the commissural subnucleus of the nucleus of the solitary tract to the phrenic nucleus in the rat
横田　茂文(Shigefumi Yokota)，津森登志子(Toshiko Tsumori)，岡　達郎(Tatsuro Oka)，
安井　幸彦(Yukihiko Yasui)
島根大学・医学部・解剖学講座神経形態学

P2-g02 8-OH DPATの延髄縫線核呼吸性ニューロンの発火様式に及ぼす影響
Effects of 8-OH DPAT on firing rates of respiratory neurons in raphe nuclei
細貝　正江(Masae Hosogai)，松尾　聡(Satoshi Matsuo)
鳥取大学　医学部　機能形態統御学講座適応生理学分野

P2-g03 げっ歯類の体温調節におけるエストロゲン、生物時計の役割
Physiological role of sex hormones and biological clock in thermoregulation in rodents
永島　計(Kei Nagashima)，狩野　真清(Masumi Kano)，内田　有希(Yuki Uchida)
早稲田大学・人間科学・統合生理

P2-g04 ネコの前脳基底部刺激中の大脳皮質血流反応
Response of cortical cerebral blood flow during stimulating the basal forebrain in cats
堀田　晴美(Harumi Hotta)，内田　さえ(Sae Uchida)，柴　加奈子(Kanako Shiba)
東京都老人総合研究所　自律神経部門

## ストレス
## Stress

P2-g05 海馬におけるストレス反応の分子機構に関する研究
An investigation of the stress-responsive molecular system in the hippocampus
車地　暁生(Akeo Kurumaji)，石井　澄和(Sumikazu Ishii)，伊藤　卓(Takashi Ito)，
西川　徹(Toru Nishikawa)
東京医歯大院・精神行動医科学

P2-g06 創造的芸術活動はストレスを調節する
Creative art activities regulate stress
福井　一(Hajime Fukui)，豊島久美子(Kumiko Toyoshima)，久田　清人(Kiyoto Kuda)，
福光　佐今(Sakon Fukumitsu)，脇田　宗孝(Munetaka Wakita)
奈良教育大学教育学部

P2-g07 新規環境下でのラット海馬における亜鉛のユニークな応答
Unique response of extracelluar zinc in the ventral hippocampus against novelty stress
武田　厚司(Atsushi Takeda)，菅野　慎吾(Shingo Kannno)，安藤　正樹(Masaki Ando)，
櫻田　直美(Naomi Sakurada)，南　　彰(Akira Minami)，奥　　直人(Naoto Oku)
静岡県大・薬・医薬生命化学分野

P2-g08 ラット幼若期のストレス負荷は成長後の情動ストレスに対する皮質 - 辺縁系シナプス応答性を変化させる
Early postnatal stress alters the synaptic response in the cortico-limbic system underlying regulation of emotional expression in adult rats
松本真知子[1](Machiko Matsumoto)，小関　裕代[1,2](Hiroyo Koseki)，山口　　拓[2](Taku Yamaguchi)，
泉　　剛[2](Takeshi Izumi)，吉岡　充弘[2](Mitsuhiro Yoshioka)，富樫　広子[1,2](Hiroko Togashi)
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P2-g09 Neurobehavioral and neurochemical changes caused by dichlorvos: Role of forced swim stress
Reyaz Waris Ansari[1,2]，Nishi Srivastava[1]，Kavita Seth[1]，Vinay K. Khanna[1]，Fakhrul Islam[2]，
Ashok K Agrawal[1]
[1]ITRC, Lucknow, India，[2]Jamia Hamdard , India

P2-g10 Effects of neonatal maternal separation (MS) on peri-pubertal onset of aggressive behavior in male mice
Mumeko C Tsuda，Mariko Nakata，Kota Watai，Sonoko Ogawa
Univesity of Tsukuba, Tsukuba, Japan

P2-g11 チューイングによるストレス緩和効果の神経科学的解析
Chewing-related neuronal mechanism under stress condition
金　　完哉[1,2](Wanjae Kim)，橘　　篤導[2,5](Atsumichi Tachibana)，大塚　剛郎[2,4](Takero Otsuka)，
平野　好幸[2,6](Yoshiyuki Hirano)，久保　金弥[3](Kin-ya Kubo)，小野　弓絵[2,5](Yumie Ono)，
中村　浩幸[7](Hiroyuki Nakamura)，唐沢　延幸[1](Nobuyuki Karasawa)，小野塚　実[2,5](Minoru Onozuka)，
渡邊　和子[1,2](Kazuko Watanabe)
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P2-g12 コーヒー香気の抗不安および抗ストレス作用について
Anxiolytic and anti-stress effects of coffee aroma in mice
丹原香菜子[1](Kanako Tambara)，田中　淳一[2](Junichi Tanaka)，服部　幸雄[1](Yukio Hattori)，
林　　泰資[1](Yasushi Hayashi)
[1]ノートルダム清心女子大・人間生活・食品栄養，[2]鳴門教育大・授業開発

P2-g13 月経前気分不快障害 (PMDD) における心理的ストレスに対する反応性低下
Impaired reactivity to mental stress in premenstrual dysphoric disorder (PMDD) patients
中本百合江[1](Yurie Nakamoto)，佐藤弥都子[2](Mitsuko Sato)，三輪　雅子[1](Masako Miwa)，
村上　正人[2](Masato Murakami)，吉井　光信[1](Mitsunobu Yoshii)
[1]東京都精神研・精神生物学研究分野，[2]日本大学医学部第一内科

P2-g14 自発および強制運動時の心理的変化に関連する脳内神経活動の比較
The comparison of acute spontaneous and forced exercise on activation of several brain areas related to psychological responses
柳田　信也(Shinya Yanagita)，雨宮誠一朗(Seiichiro Amemiya)，鈴木　聡子(Satoko Suzuki)，
加藤　裕美(Yumi Kato)，久保田夏子(Natsuko Kubota)，北　　一郎(Ichiro Kita)
首都大学東京大学院・人間健康科学

P2-g15 咬み合せが悪いと海馬で神経細胞死が引き起こされる
Involvement of glucocorticoid response in hippocampal changes in aged SAMP8 mice with occlusal disharmony
久保　金弥[1](Kin-ya Kubo)，山田　幸子[2](Yukiko Yamada)，荒川　容子[2](Yoko Arakawa)，
飯沼　光生[2](Mitsuo Iinuma)，岩久　文彦[1](Fumihiko Iwaku)，田村　康夫[2,3](Yasuo Tamura)，
渡邊　和子[3](Kazuko Watanabe)，小野塚　実[4](Minoru Onozuka)
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P2-g16 AD 変異型プレセニリン 1 過剰発現マウスの海馬神経前駆細胞は慢性ストレスにより分化抑制される
Chronic stress arrests the maturation of hippocampal neuronal progenitors in mice overexpressing an AD type presenilin 1 (PS1).
國本　正子(Shohko Kunimoto)，河野　朋子(Tomoko Kohno)，井上　高良(Takayoshi Inoue)，
和田　圭司(Keiji Wada)，中村　　俊(Shun Nakamura)
国立精神神経センター・神経研究所・診断研究部

P2-g17 主観的ストレスは、DEX/CRH テストで測定したコルチゾールやアルギニン・バゾプレッシン値と関連する
The combined DEX/CRH test as a marker of subjective stress in non-clinical population
堀　　弘明[1,2](Hiroaki Hori)，岡部　繁男[2](Shigeo Okabe)，功刀　　浩[1](Hiroshi Kunugi)
[1]国立精神・神経センター神経研究所疾病研究第三部，[2]東京医科歯科大学大学院医歯学総合研究科細胞生物学教室

P2-g18 若齢マウスの隔離飼育による異常行動の可逆性
Reversibility of behavior abnormalities induced by isolation housing in young mice
折田　将成[1](Masanari Orita)，首藤　　誠[2,3](Makoto Shuto)，杉山　千絵[1,3](Chie Sugiyama)，
倉本　展行[1](Nobuyuki Kuramoto)，米田　幸雄[3](Yukio Yoneda)，荻田喜代一[1](Kiyokazu Ogita)
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P2-g19 ストレス脆弱性モデルラットにおけるマイクロＲＮＡを介したグルココルチコイド受容体の発現調節異常
Involvement of microRNA-mediated downregulation of glucocorticoid receptor in vulnerability to stress
内田　周作(Shusaku Uchida)，西田　　朗(Akira Nishida)，原　久美子(Kumiko Hara)，
末次　正知(Masatomo Suetsugi)，藤本美智子(Michiko Fujimoto)，渡辺　義文(Yoshifumi Watanabe)
山口大院・医・高次脳機能病態学

P2-g20 ２時間の騒音はストレスホルモンの上昇なしにうつ反応を減弱する
Loud white noise attenuates depressive response without affecting glucocorticoid levels in rats
坂口　俊輔(Shunsuke Sakaguchi)，水野　雅晴(Masaharu Mizuno)，粟生　修司(Shuji Aou)
九工大院・生命体工学・脳情報

P2-g21 静電気パルスは意識に上らない弱い驚愕反射を伴って呼吸ペースに影響する
Electrostatic pulse stimulation may alter respiration pace by unconsciously weak startle reflex
安田　達弘(Tatsuhiro Yasuda)
フリー

P2-g22 急性ストレス時における Glucocorticoid を介した海馬 neuropsin mRNA の増加
Acute stress increased neuropsin mRNA level via glucocorticoid pathway in the hippocampus
原田　暁子(Akiko Harada)，駒井　章治(Shoji Komai)，石川　保幸(Yasuyuki Ishikawa)，
塩坂　貞夫(Sadao Shiosaka)
奈良先端大・バイオサイエンス・細胞構造学

## 睡眠、生体リズム
## Sleep and Biological Rhythms

P2-g23 位相応答曲線の様々なノイズ環境下での統計的推定
Statistical estimation of phase response curves under the various types of noisy environments
中江　健[1](Ken Nakae)，伊庭　幸人[2](Yukito Iba)，青柳富誌生[1](Toshio Aoyagi)
[1]京都大学・情報学研究科，[2]統計数理研究所・モデリング研究系

P2-g24 Rhythmic control of AANAT translation by hnRNP Q in circadian melatonin production
Tae-Don Kim, Kyung-Chul Woo, Sungchan Cho, Dae-Cheong Ha, Sungkey Jang, Kyong-Tai Kim
Department of Life Science, POSTECH, Pohang, Korea

P2-g25 Neuronal activity in the paraventricular nucleus during penile erection in unanaesthetized rats.
Juan-Carlos Toledo[1], Yoshimasa Koyama[2], Yukihiko Kayama[1]
[1]Department of Physiology 2, Fukushima Medical University, [2]Fukushima University

P2-g26 体温調節、温冷感覚に与える性周期の影響
Menstrual cycle affects thermal regulation and comfort
安原　祥[1](Saki Yasuhara)，岩間　愛音[1](Aine Iwama)，狩野　真清[1](Masumi Kanou)，
中村真由美[2](Mayumi Nakamura)，永島　計[1,3](Kei Nagashima)
[1]早稲田大学・人間科学学術院・統合生理学，[2]早稲田大学 スポーツ科学学術院，[3]早稲田大学 先端科学・健康医療融合機構

P2-g27 BMAL1 蛋白質修飾の体内時計における役割
Role of post-translational modification of BMAL1 in mammalian circadian clock
田丸　輝也(Teruya Tamaru)，高松　研(Ken Takamatsu)
東邦大・医・第2生理

P2-g28 延髄網様核—橋網様核連絡と筋緊張維持機構
Differential effect of nuclei Gigantocellularis and Magnocellularis stimulation on muscular activity under the absence of pontine regulation.
児玉　亨[1](Tohru Kodama)，本多　芳子[1](Yoshiko Honda)，Jerome Siegel[2]，Yuan-Yang Lai[2]
[1]東京都神経科学総合研究所・心理学研究部門，[2]カリフォルニア大学

P2-g29 わが国における子どもたちの睡眠健康および睡眠時歯ぎしり
Sleep health and sleep bruxism in the children of Japan
諏訪　幸子[1](Sachiko Suwa)，白川修一郎[4](Shuichirou Shirakawa)，笹栗　健一[1,2](Kenichi Sasaguri)，
高原　円[1,2,3](Madoka Takahara)，駒田　陽子[4](Yoko Komada)，小野塚　実[2,3](Minoru Onozuka)，
佐藤　貞雄[1,2](Sadao Sato)
[1]神奈川歯科大学大学院・成長発達歯科学講座，[2]神奈川歯科大学・高次脳・口腔科学研究センター，[3]神奈川歯科大学・生体機能講座生理学分野，[4]国立精神・神経センター　精神保健研究所

P2-g30 睡眠状態移行期における脳波律動と大脳皮質血行動態の内因性相関
Intrinsic correlates of sleep state transition in EEG rhythms and cerebral hemodynamics
内田真理子[1](Mariko Uchida)，田中　尚樹[1,2](Naoki Tanaka)，佐藤　大樹[1,2](Hiroki Sato)，
牧　敦[1,2](Atsushi Maki)
[1]科学技術振興機構・CREST，[2]日立製作所・基礎研究所

P2-g31 動脈圧受容器が腎交感神経活動のサーカディアンリズム形成に果たす役割
Role of arterial baroreceptor in circadian rhythm of renal sympathetic nerve activity in rats
網野　由香(Yuka Amino)，齋藤めぐみ(Megumi Saito)，吉本　光佐(Misa Yosimoto)，三木　健寿(Kenju Miki)
奈良女子大学大学院・人間文化研究科・統御生理

P2-g32 ラットの睡眠レム期における海馬 CA1 領域の血流量変化
Local cerebral blood flow in the hippocampus during REM sleep in rats
三木　健寿(Kenju Miki)，畑　　知子(Tomoko Hata)，阿部　淳子(Junko Abe)，吉本　光佐(Misa Yoshimoto)
奈良女子大学院・人間文化研究科・統御生理

P2-g33 行動状態依存的な呼吸パターンと海馬情報処理モードの同期的変化
Behavioral state-dependent simultaneously change between the respiratory pattern and information processing mode in hippocampus
真部　寛之(Hiroyuki Manabe)，森　　憲作(Kensaku Mori)
東京大院・医・細胞分子生理

P2-g34 睡眠状態の指標としてのラット新皮質および海馬におけるマルチユニット神経活動
Multi-neuronal dynamic in rat neocortex and hippocampus as a measure of natural sleep
姜　　時友(Siu Kang)，礒村　宜和(Yoshikazu Isomura)，竹川　高志(Takashi Takekawa)，
加藤　英之(Hideyuki Cateau)，深井　朋樹(Tomoki Fukai)
理化学研究所・BSI・脳回路機能理論

P2-g35 PACAP ノックアウトマウスの非視覚光受容における異常
PACAP deficient mice exhibit light parameter dependent abnormalities of nonvisual photoreception
礒島　康史[1,2](Yasushi Isojima)，川口ちひろ[3](Chihiro Kawaguchi)，新谷　紀人[3](Norihito Shintani)，
畑中　道賢[3](Michiyoshi Hatanaka)，郭　　暁宏[3](Xiaohong Guo)，永井　克也[2](Katsuya Nagai)，
橋本　　均[3,4](Hitoshi Hashimoto)，馬場　明道[3](Akemichi Baba)
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P2-g36 ラット海馬スライスにおけるカルバコール誘導β振動の生成に関与するグルタミン酸受容体とギャップ結合
The glutamatergic receptors and gap junction involving the generation of carbachol-induced beta oscillation in rat hippocampal slices
太田　好徳(Yoshinori Ohta)，橋本あゆみ(Ayumi Hashimoto)，夏目季代久(Kiyohisa Natsume)
九州工業大学大学院・生命体工学研究科・脳情報専攻

P2-g37 β-エンドルフィンはハムスターの冬眠維持期の体温を制御する
β-Endorphin regulates body temperature of Syrian hamster during maintenance phase of hibernation.
田村　　豊[1](Yutaka Tamura)，新谷　光輝[2](Mitsuteru Shintani)，門田麻由子[1](Mayuko Monden)，
塩見　浩人[1,2](Hirohito Shiomi)
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P2-g38 ヒスタミン受容体欠損マウスとその表現系
Histamine receptors genes knockout mice and their phenotypes
櫻井　映子(Eiko Sakurai)，邵　　和(He Shao)，倉増　敦朗(Astuo Kuramasu)，
岡村　信行(Nobuyuki Okamura)，谷内　一彦(Kazuhiko Yanai)
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## 学習、記憶 2
## Learning and Memory 2

P2-g39 咀嚼活動によるストレス性記憶障害の改善：行動生理学的検討
Ameliorative effect of biting on the stress-attenuated hippocampal memory process: a Morris Water Maze study
三宅真次郎[1,4](Shinjiro Miyake)，小野　弓絵[2,4](Yumie Ono)，片岡　　烈[1,4](Tsuyoshi Kataoka)，Wanjae Kim[3,4]，
笹栗　健一[1,4](Kenichi Sasaguri)，渡邊　和子[3,4](Kazuko Watanabe)，佐藤　貞雄[1,4](Sadao Sato)，
小野塚　実[2,4](Minoru Onozuka)
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P2-g40 咀嚼活動によるストレス性記憶障害の改善：海馬 CA1-LTP による検討
Ameliorative effect of biting on the stress-attenuated hippocampal memory process: a CA1-LTP study.
小野　弓絵[1,4](Yumie Ono)，三宅真次郎[2,4](Shinjiro Miyake)，片岡　烈[2,4](Tsuyoshi Kataoka)，
笹栗　健一[2,4](Ken-ichi Sasaguri)，佐藤　貞雄[2,4](Sadao Sato)，渡邊　和子[3,4](Kazuko Watanabe)，
小野塚　實[1,4](Minoru Onozuka)
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P2-g41 自由行動下ラット背側縫線核の報酬遅延条件におけるセロトニン濃度変化
Increased serotonin efflux in the dorsal raphe nucleus of rats working for delayed rewards
宮崎佳代子[1](Kayoko Miyazaki)，宮崎　勝彦[1](Katsuhiko Miyazaki)，銅谷　賢治[1,2](Kenji Doya)
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P2-g42 運動記憶の固定化が運動学習のどの時点でおこるかについての実験的考察
Temporal window for consolidation of memory trace of adaptation of mouse optokinetic response eye movement
岡本　武人[1,2](Takehito Okamoto)，永雄　総一[1,3](Soichi Nagao)
[1]理研・脳センター・運動学習制御，[2]群馬大院・医・高次細胞機能，[3]SORST, JST

P2-g43 多価不飽和脂肪酸の長期食餌投与がラット海馬神経細胞に与える影響
Effects of long term supplementation of PUFA on rat hippocampal neuron
樫八重泰斗[1](Yasuto Kashiyae)，深谷　泰亮[1](Taisuke Fukaya)，紺谷　昌仙[2](Masanori Kontani)，
河島　洋[2](Hiroshi Kawashima)，木曽　良信[2](Yoshinobu Kiso)，榊原　学[1](Manabu Sakakibara)
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P2-g44 自由行動下ラット背側縫線核セロトニン神経の報酬および無報酬に対する遅延期間における活動
Activity of serotonergic neurons in the dorsal raphe nucleus of freely moving rats during reward and non-reward delay period
宮崎　勝彦[1](Katsuhiko Miyazaki)，宮崎佳代子[1](Kayoko Miyazaki)，銅谷　賢治[1,2](Kenji Doya)
[1]沖縄大学院大学先行的研究事業・神経計算ユニット，[2]ATR脳情報研究所

P2-g45 大脳皮質の主要な解剖学的特徴と一致する BESOM モデル
BESOM model consistent with the main anatomical characteristics of cerebral cortex
一杉　裕志(Yuuji Ichisugi)
産業技術総合研究所

P2-g46 培養ニューラルネットワーク時空間ダイナミクスの長期間測定法
Long-term measurement for spatiotemporal dynamics of cultured neural networks
伊東　大輔(Daisuke Ito)
北大院・工・応用物理

P2-g47 霊長類の前庭動眼反射の長期適応の記憶痕跡の場
Effects of bilateral flocculus shutdown on the primate long-term adaptation of horizontal vestibulo-ocular reflex
永雄　総一[1,2](Soichi Nagao)，安西　真理[1](Mari Anzai)
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P2-g48 再認記憶のゆらぎの神経機構
Neural mechanism of fluctuated recognition memory
橋本　照男[1](Teruo Hashimoto)，臼井　信男[2](Nobuo Usui)，泰羅　雅登[2](Masato Taira)，
小嶋　祥三[1](Shozo Kojima)
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P2-h01 多試行報酬スケジュール課題遂行中の前部島皮質と後部島皮質における単一ニューロン活動の違い
Differences between single neuronal responses in anterior and posterior insula during multi-trial reward schedules
水挽　貴至[1,2](Takashi Mizuhiki)，Barry_J Richmond[3]，設楽　宗孝[1,2](Munetaka Shidara)
[1]筑波大学大学院人間総合科学研究科，[2]産業技術総合研究所脳神経情報部門，[3]米国精神衛生研究所

P2-h02 海馬 CA1 野における嗅内皮質及び CA3 入力の統合様式
Visualization of the convergence of entorhinal and hippocampal CA3 input onto CA1 neurons.
梶原 利一[1](Riichi Kajiwara), Floris_G. Wouterlood[2], Anupam Sah[2], Amber_J. Boekel[2], Luciënne_T.G. Baks-te Bulte[2], Menno_P. Witter[2,3]
[1]産業技術総合研究所・脳神経情報, [2]Dept Anat, VU Univ Med Ctr, Amsterdam, Netherlands, [3]Ctr Biol of Memory, NTNU, Trondheim, Norway

P2-h03 鳥類の大脳神経細胞選択的な *in vivo* 遺伝子導入法の確立
Establishment of *in vivo* gene transfer system into newly-hatched chick brain
廣瀬 直樹[1](Naoki Hirose), 片桐 幸子[1](Sachiko Katagiri), 山口 真二[1](Shinji Yamaguchi), 藤本 康之[1](Yasuyuki Fujimoto), 松島 俊也[2](Toshiya Matsushima), 高野 達哉[1](Tatsuya Takano), 本間 光一[1](Koichi Homma)
[1]帝京大・薬, [2]北大院・理

P2-h04 線虫 *C. elegans* において、新規加水分解酵素 AHO-3 は温度と餌条件の連合学習に関与する
Novel hydrolase AHO-3 is required for associative learning between cultivation temperature and feeding state in *C. elegans*
西尾 奈々[1](Nana Nishio), 毛利 亮子[2](Akiko Mohri), 児玉 英志[1](Eiji Kodama), 木村幸太郎[3](Kotaro Kimura), 久原 篤[1](Atsushi Kuhara), 森 郁恵[1,4](Ikue Mori)
[1]名大・院理・生命, [2]現所属: テキサス大・医, [3]現所属: 遺伝研・総研大, [4]CREST

P2-h05 ゴキブリにおける状況依存的な嗅覚学習訓練
Context-dependent olfactory conditioning in cockroaches
佐藤 千尋[1](Chihiro Sato), 松本 幸久[1](Yukihisa Matsumoto), 佐倉 緑[2](Midori Sakura), 渡邊 英博[1](Hidehiro Watanabe), 水波 誠[1](Makoto Mizunami)
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P2-h06 中脳黒質ドーパミンニューロンは線条体に広範囲かつ高密度な軸索終末を形成する
Nigrostriatal dopamine neurons form wide and highly dense axon terminals in the striatum of rat: single-cell study
松田 和郎[1](Wakoto Matsuda), 古田 貴寛[2](Takahiro Furuta), 中村 公一[2](Kouichi Nakamura), 金子 武嗣[2](Takeshi Kaneko), 新井 良八[1](Ryohachi Arai)
[1]滋賀医大・医・解剖・生体機能形態学, [2]京都大院・医・高次脳形態学

P2-h07 小脳核 GABA 受容体はマウス瞬目反射条件づけの獲得と保持に重要な役割を果たしている。
GABA receptors play roles in acquisition and retention of mouse eyeblink conditioning in the cerebellar nuclei.
坂本 敏郎(Toshiro Sakamoto), 新崎 智子(Tomoko Arasaki), 遠藤 昌吾(Shogo Endo)
沖縄科学技術研究基盤整備機構 記憶と学習の分子神経生物

P2-h08 Ras-MAPK 経路を負に制御する分子 SCOP の長期記憶形成への関与
Proteolytic degradation of SCOP contributes to activation of ERK/MAPK and memory formation
清水貴美子[1](Kimiko Shimizu), 深田 吉孝[2](Yoshitaka Fukada), 岡田 雅人[3](Masato Okada), 永井 克也[4](Katsuya Nagai), Daniel Storm[1]
[1]ワシントン大学、薬学部, [2]東京大院、理、生化, [3]大阪大、微研, [4]ANBAS Corporation,Osaka, Japan

P2-h09 ラット膨大後皮質内の線維連絡
Intrinsic connections of the retrosplenial cortex in the rat
柴田 秀史(Hideshi Shibata)
東京農工大学院・共生科学技術・獣医解剖

P2-h10 Improvement of memory in *Drosophila* by specific point mutations in PKA
Junjiro Horiuchi, Daisuke Yamazaki, Minoru Saitoe
Tokyo Metropolitan Institute for Neuroscience, Tokyo, Japan

P2-h11 コカインによる条件性場所選好と海馬シータ波との関係
Relationship between cocaine-induced conditioned place preference and hippocampal theta rhythm
高野 裕治[1,2](Yuji Takano), 廣中 直行[2](Naoyuki Hironaka)
[1]専修大学大学院・文・心理, [2]科学技術振興機構下條潜在脳機能プロジェクト

P2-h12 電気生理学的に調べたラット海馬 CA3 野における興奮ー抑制シナプスの幾何学的分布と順序的活性化
Electrophysiologicaly probed geometric correlation of E/I synapses and directional activation of the rat hippocampal CA3 area
冨永 貴志[1,2,4](Takashi Tominaga), 市川 道教[2](Michinori Ichikawa), Guosong Liu[3,4]
[1]徳島文理大・香薬・病態生理, [2]理研脳センター, [3]清華大学記憶学習研究所 北京 中国, [4]PCLM RIKEN-MIT 米国

P2-h13 カルシンテニン / アルカデインの線虫ホモログ CASY-1 は、種々の学習に必須である
CASY-1, a homolog of Calsyntenin/Alcadein, is essential for multiple forms of learning in *C. elegans*
池田 大祐[1](Daisuke D. Ikeda), Yukan Duan[2], 松木 正尋[1](Masahiro Matsuki),
國友 博文[1](Hirofumi Kunitomo), Edward M. Hedgecock[2], 飯野 雄一[1](Yuichi Iino)
[1]東京大・遺伝子実験施設, [2]Dept. of Biol., Johns Hopkins Univ., Baltimore, USA

P2-h14 シータ位相コードによる物ー場所連合の階層的ネットワークにおける想起過程
Memory recall of object-place associations in a hierarchical network self-organized by theta phase coding
佐藤 直行(Naoyuki Sato), 山口 陽子(Yoko Yamaguchi)
理化学研究所・脳科学総合研究センター・創発知能ダイナミクス研究チーム

P2-h15 第二言語の統語規則獲得の指標となる下前頭回左側優位の構造的非対称性 : VBM による研究
The leftward structural asymmetry in the inferior frontal gyrus as an indicator of acquiring syntax in second language: A VBM study
名内 存人[1,2](Arihito Nauchi), 平野 和由[2,3](Kazuyoshi Hirano), 村石 幸正[2,3](Yukimasa Muraishi),
酒井 邦嘉[1,2](Kuniyoshi_L Sakai)
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P2-h16 Molecular mechanisms of decision making in Caenorhabditis elegans
Paola Jurado
Nagoya University, Japan

## 言語とコミュニケーション
## Language and Communication

P2-h17 m系列変調談話に対する脳波応答の相関関数分析
Correlation analysis of scalp EEG responses to spoken sentences modulated by M-sequence
竹市 博臣[1,3](Hiroshige Takeichi), 小山 幸子[1,2,3](Sachiko Koyama), Andrzej Cichocki[1]
[1]理化学研究所, [2]北海道大学, [3]科学技術振興機構

P2-h18 音声の感情認知に関わる神経基盤ー事象関連 fMRI による研究
Neural correlates of emotional voice recognition: an event-related functional magnetic resonance imaging study
加藤 誠(Makoto Kato), 南 哲人(Tetsuto Minami), 田中 靖人(Yasuto Tanaka),
村田 勉(Tsutomu Murata)
(独)情報通信研究機構・未来ICT研究センター・バイオICTグループ

P2-h19 多点電極皿（MED）を用いた鳴鳥の音声学習に必須な脳領域の長期増強の研究
Study of long-term potentiation (LTP) in the song bird brain regions essential for vocal learning using multi-electrode dish (MED)
別所　親房(Chikafusa Bessho)，山本　達也(Tatsuya Yamamoto)
京都産業大・理・物理

P2-h20 上昇音および下降 FM 音に対するスナネズミの反応
Behaveoral responses to frequency modulated sounds in Mongolian gerbils: upward FM vs. downward FM
長田　裕輝(Yuki Nagata)
同志社大学院・工・知識工

P2-h21 漢字単語の読みにおける音韻情報の意味処理への影響
Phonological and lexico-semantic processing during kanji word reading: An MEG study
魏　　強[1,2](Qiang Wei)，井原　　綾[1](Aya Ihara)，早川　友恵[1,3](Tomoe Hayakawa)，
村田　　勉[1](Tsutomu Murata)，藤巻　則夫[1,2](Norio Fujimaki)
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P2-h22 視覚単語認知の意味アクセスにおける２つの異なる脳ネットワーク
Two distinct neural networks for semantic access during visual word recognition
Hyeonjeong Jeong[1]，杉浦　元亮[1,2](Motoaki Sugiura)，佐々　祐子[1,4](Yuko Sassa)，
宮本　正夫[3](Tadao Miyamoto)，堀江　　薫[3](Kaoru Horie)，佐藤　　滋[3](Shigeru Sato)，
川島　隆太[1](Ryuta Kawashima)
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P2-h23 演奏行為の観察における下前頭回の活動
IFG activity in observing music performance
脇田　真清[1,2](Masumi Wakita)
[1]京都大・霊長研・行動神経，[2]科学技術振興機構

P2-h24 GABA 抑制性機構に制御されるキンカチョウ歌学習の臨界期
Early GABA function regulates sensory critical period during birdsong learning.
杉山（矢崎）陽子[1,2](Yoko Yazaki-Sugiyama)，Jason Kushner[3]，Neal A Hessler[2,3]，
ヘンシュ貴雄[1,2](Takao K Hensch)
[1]CREST，科学技術振興機構，[2]理化学研究所　脳研　神経回路発達研究チーム，[3]理化学研究所　脳研　発声行動機構研究チーム

P2-h25 日本語における基本語順の探索：NIRS を用いた前頭葉機能計測
Flexible word order in Japanese and its effects on frontal activation: A NIRS study
西村　幸香[1](Y Nishimura)，杉崎　鉱司[2](K Sugisaki)，服部　範子[2](N Hattori)，井口　　靖[2](Y Inokuchi)，
西村　嘉洋[1](Y Nishimura)，小川眞理子[2](M Ogawa)，岡崎　祐士[1,3](Y Okazaki)，滝　　和郎[1](W Taki)，
山本　哲朗[1](T Yamamoto)，吉田　悦子[2](E Yoshida)，綾野　誠紀[2](Seiki Ayano)
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P2-h26 語彙的曖昧性解消における神経メカニズム
Neural mechanisms of the lexically ambiguity resolution - An MEG study
井原　　綾[1](Aya Ihara)，早川　友恵[1,2](Tomoe Hayakawa)，魏　　強[1,3](Qiang Wei)，
宗綱　信治[1,3](Shinji Munetsuna)，藤巻　則夫[1,3](Norio Fujimaki)
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P2-h27 構音様式による脳賦活の変化
Changes of the brain activity related to the differences in manner of articulation.
内田　信也[1,2,3](Shinya Uchida)，三浦　直樹[2,4](Naoki Miura)，中村　克樹[3,4](Katsuki Nakamura)，
佐藤　和則[1](Kazunori Sato)，福田　　寛[1](Hiroshi Fukuda)，川島　隆太[2,5](Ryuta Kawashima)
[1]東北大・加齢研・機能画像医学，[2]東北大・加齢研・脳機能開発，[3]国立精神・神経センター・神経研・モデル動物開発，[4]CREST・科学技術振興機構，[5]RISTEX・科学技術振興機構

P2-h28 **fMRIによる日本語文の再分析処理に関わる脳活動の研究**
**Reanalysis in Japanese sentence comprehension: An fMRI study**
生田　奈穂[1](Naho Ikuta)，横山　悟[1](Satoru Yokoyama)，
Hyeonjeong Jeong[1]，杉浦　元亮[2](Motoaki Sugiura)，堀江　薫[3](Kaoru Horie)，
佐藤　滋[3](Shigeru Sato)，川島　隆太[1](Ryuta Kawashima)
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P2-h29 **対面コミュニケーションの脳活動**
**Difference in neural distributions between face-to-face and remote situations in a communicative game**
関口　達彦[1](Tatsuhiko Sekiguchi)，飯田　公司[2](Koji Iida)，川畑　秀明[3](Hideaki Kawabata)，
板井　司郎[2](Shiro Itai)，渡辺　貴文[2](Takabumi Watanabe)，三輪　敬之[2](Yoshiyuki Miwa)
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P2-h30 **階層的自己組織化を用いた意味記憶のカテゴリー特異性の検討**
**A multilayer SOM for category specific semantic memory impairments**
浅川　伸一(Shin-ichi Asakawa)
東京女子大学

P2-h31 **外国語の語彙処理に関する縦断的研究：fMRIを用いて**
**A longitudinal fMRI study of neural plasticity in the second language lexical processing**
横山　悟[1](Satoru Yokoyama)，Jungho Kim[3,4]，内田　信也[1](Shin-ya Uchida)，
岡本　英行[1](Hideyuki Okamoto)，Chen Bai[6]，遊佐　典昭[5](Noriaki Yusa)，宮本　正夫[2](Tadao Miyamoto)，
吉本　啓[2](Kei Yoshimoto)，堀江　薫[2](Kaoru Horie)，佐藤　滋[2](Shigeru Sato)，
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## アルツハイマー病、他の認知症、老化2
## Alzheimer Disease, Other Dementia, Aging 2

P2-h32 **培養神経細胞のアミロイドβ蛋白取り込みにおけるRAGEの関与**
**Role of receptor for advanced glycation end products in neuronal uptake of amyloid β-peptide**
福崎　笑子[1](Emiko Fukuzaki)，田熊　一敞[1](Kazuhiro Takuma)，溝口　博之[1](Hiroyuki Mizoguchi)，
David M. Stern[2]，Shirley ShiDu Yan[3]，山田　清文[1](Kiyofumi Yamada)
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P2-h33 **学習機能減衰を示す老齢カニクイザルの海馬歯状回における新生ニューロン数の減少**
**Dramatic decrease in new neurons in the dentate gyrus of learning attenuated aged monkeys**
相澤　憲[1](Ken Aizawa)，揚山　直英[2](Naohide Ageyama)，横山ちひろ[3](Chihiro Yokoyama)，
寺尾　恵治[2](Keiji Terao)，久恒　辰博[1](Tatsuhiro Hisatsune)
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P2-h34 **小胞体ストレスにおけるアミロイド前駆体タンパク質C末端断片の役割に関する研究**
**The role of C-terminus of amyloid precursor protein under endoplasmic reticulum stress**
高橋　啓太(Keita Takahashi)，新留　徹広(Tetsuhiro Niidome)，木原　武士(Takeshi Kihara)，
杉本　八郎(Hachiro Sugimoto)
京都大院・薬・創薬神経

P2-h35 **アポリポ蛋白Eアイソフォーム依存的コレステロール搬出機構の検討**
**Isoform dependent effect of apolipoprotein E on cholesterol efflux**
源川　博久[1](Hirohisa Minagawa)，キョウ建生[1](Jian-Sheng Gong)，中村　俊行[1](Toshiyuki Nakamura)，
鄒　鵬[1](Kun Zou)，Sissel Lund-Katz[2]，Michael Phillips[2]，斎藤　博幸[3](Hiroyuki Saito)，
道川　誠[1](Makoto Michikawa)
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P2-h36 PI3K/AKT/GSK3β経路はγセクレターゼ複合体の発現調節に関わる
PI3K/AKT/GSK3β pathway regulates the expression of gamma secretase complex.
青柳 信寿[1](Nobuhisa Aoyagi), 植村 健吾[1](Kengo Uemura), 葛谷 聡[2](Akira Kuzuya),
木下 彩栄[3](Ayae Kinoshita), 高橋 良輔[1](Ryousuke Takahashi)
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P2-h37 アミロイドβ蛋白の神経細胞傷害におけるミクログリアの役割
The role of microglia on the neurotoxicity of Aβ
土井由紀子(Yukiko Doi), 牧 祐輝(Yuki Maki), 水野 哲也(Tetsuya Mizuno),
竹内 英之(Hideyuki Takeuchi), 錫村 明生(Akio Suzumura)
名古屋大学 環境医学研究所 神経免疫分野

P2-h38 N279K 変異タウトランスジェニックマウス（SJLB）を用いた認知症関連蛋白の探索
Causative candidates for the dementing diseases analysed by SJLB mice
谷口 泰造[1](Taizo Taniguchi), 鷹野 正興[3](Masaoki Takana), 尾谷三枝子[3](Mieko Otani),
松山 正剛[4](Shogo Matsuyama)
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P2-h39 γ-セクレターゼ阻害薬 DAPT, L-685,458 は Wnt シグナルを抑制する
DAPT and L-685,458, γ-secretase inhibitors perturb Wnt signaling
中山 泰亮(Taisuke Nakayama), 大河内正康(Masayasu Okochi), 田上 真次(Shinji Tagami),
柳田 寛太(Kanta Yanagida), 西富 晃平(Kouhei Nishitomi), 田坂志都子(Shizuko Tasaka), 姜 経緯
(Jingwei Jiang), 森 康治(Kohji Mori), 森原 剛史(Takashi Morihara), 田中 稔久(Toshihisa Tanaka),
工藤 喬(Takashi Kudo), 武田 雅俊(Masatoshi Takeda)
大阪大院・医・精神医学

P2-h40 膜ミクロドメイン依存性の新規アミロイド前駆体蛋白質代謝制御機構
A new membrane microdomain-dependent regulatory mechanism of amyloid precursor protein processing
櫻井 隆[1,2](Takashi Sakurai), 金子 貢巳[1](Kumi Kaneko), 奥野弥佐子[1](Misako Okuno),
和田 浩司[1](Koji Wada), 樫山 拓[2](Taku Kashiyama), 貫名 信行[1](Nobuyuki Nukina)
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P2-h41 加齢依存的な学習障害を呈するヒトタウのトランスジェニックマウス脳における形態学的解析
Morphological analysis of human tau transgenic mice showing age-dependent learning disorders
福田 哲也[1](Tetsuya Fukuda), 木村 哲也[1](Tetsuya Kimura), 新井 哲朗[2](Tetsuaki Arai),
山下 俊治[1](Shunji Yamashita), 溝呂木達也[1](Tatsuya Mizoroki), 朴 正美[1](Yung-Mi Park),
村山美由紀[1](Miyuki Murayama), 佐原 成彦[1](Naruhiko Sahara), 秋山 治彦[2](Haruhiko Akiyama),
高島 明彦[1](Akihiko Takashima)
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P2-h42 認知症を伴う筋萎縮性側索硬化症の大脳白質病変に関する検討：TDP-43 陽性封入体はユビキチンではなく p62 と共存する
White matter lesions in the brain with amyotrophic lateral sclerosis with dementia: TDP-43 immunopositive inclusion is not co-localized with ubiquitin but p62
日地 正典[1,2](Masanori Hiji), 高橋 哲也[2](Tetsuya Takahashi), 伊藤 聖[1](Hijiri Ito),
山下 拓史[2](Hiroshi Yamashita), 和泉 唯心[1](Yuishin Izumi), 郡山 達男[2](Tatsuo Kohriyama),
山村 安弘[1](Yasuhiro Yamamura), 松本 昌泰[2](Masayasu Matsumoto)
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P2-h43 リノール酸誘導体 DCP-LA は学習・記憶障害を改善する
The linoleic acid derivative DCP-LA ameliorates impairment of learning and memory
矢口 貴博(Takahiro Yaguchi), 山本 英幸(Hideyuki Yamamoto), 藤川 浩一(Hirokazu Fujikawa),
永田 徹(Tetsu Nagata), 山本 悟史(Satoshi Yamamoto), 西崎 知之(Tomoyuki Nishizaki)
兵庫医科大学・医・生理学2

P2-h44 アルツハイマー病モデルネズミの老化促進機構における GABA 活性の解析
GABA-related acceleration of aging process in Alzheimer's model mouse
吉池 裕二[1](Yuji Yoshiike), 木村 哲也[1](Tetsuya Kimura), 山下 俊治[1](Shunji Yamashita),
古舘 宏之[2](Hiroyuki Furudate), 福田 哲也[1](Tetsuya Fukuda), 溝呂木達也[1](Tatsuya Mizoroki),
村山美由紀[1](Miyuki Murayama), 高島 明彦[1](Akihiko Takashima)
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P2-h45 小胞体関連分解 ERAD に関与する HRD1 は APP によるアポトーシス抑制する
HRD1 involved in ER-associated degradation (ERAD) suppresses amyloid precursor protein-induced apoptosis
金子 雅幸[1](Masayuki Kaneko), 小池 洋[2](Hiroshi Koike), 大村 友博[1](Tomohiro Omura),
大熊 康修[1](Yasunobu Okuma), 野村 靖幸[3](Yasuyuki Nomura)
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P2-h46 A natural scavenger of peroxynitrites, rosmarinic acid, protects against impairment of memory induced by Abeta (25-35)
Tursun Alkam[1], Atsumi Nitta[1], Hiroyuki Mizoguchi[1], Akio Itoh[1], Toshitaka Nabeshima[1,2]
[1]Dept. Neuropsychopharmacol. & Hosp. Pharm, Nagoya University, Nagoya, Japan, [2]Japanese Drug Organization of Appropriate Use and Research, Nagoya 468-0069, Japan

P2-h47 ピルビン酸のラット海馬培養切片の生存維持に対する効果とβアミロイド細胞毒性に対する保護効果について
Protective effect of pyruvate on survival of hippocampal slice culture and against beta-amyroid toxicity.
高田 俊宏(Toshihiro Takata), 王 曉楠(Xiaonan Wang), 桜井 孝(Takashi Sakurai),
横野 浩一(Koichi Yokono)
神戸大学医学部付属病院 老年内科

P2-h48 Reticulon 3 と BACE1 の相互作用メカニズムの解析
Analysis of the mechanism of the physical interaction between reticulon 3 and BACE1
荒木 亘[1](Wataru Araki), 久米 秀明[1](Hideaki Kume), 村山紀代子[1](Kiyoko Murayama),
亀谷富由樹[2](Fuyuki Kametani)
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P2-j01 中性子小角散乱及び動的光散乱法を用いた酸化ストレスによるタウの初期的凝集過程の研究
Small-angle neutron scattering (SANS) and dynamic light scattering (DLS) analyses of the initial aggregation process of tau by oxidative stress
内藤 幸雄[1](Sachio Naito), 池田 進[1](Susumu Ikeda), 清水 裕彦[1](Hirohiko Shimizu),
望月 秀樹[2](Hideki Mochizuki), 安田 徹[2](Toru Yasuda), 水野 美邦[1](Yoshikuni Mizuno),
古坂 道弘[3](Michihiro Furusaka), 藤原 悟[4](Satoru Fujiwara), 鈴木 淳市[4](Junichi Suzuki),
和田 圭司[5](Keiji Wada)
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P2-j02 MPC, a newly synthesized compound, improves learning and memory deficits of neurodegenerative animal models and protects SH-SY5Y cells from neurotoxicities
Geon Ho Lee
Department of Pharmacy, Seoul national university , Seoul, Korea

## 神経筋疾患 Neuromuscular Diseases

P2-j03 α-dystroglycan のプロセッシングに関する検討
Characterization of the processing of α-dystroglycan
斉藤 史明(Fumiaki Saito), 新井 祐子(Yuko Arai), 清水 輝夫(Teruo Shimizu),
松村喜一郎(Kiichiro Matsumura)
帝京大学医学部神経内科

P2-j04 遺伝性神経痛性筋萎縮症を引き起こすSept9の点変異はSept4とSept11の相互作用を変化させ、Rho/Rhotekinシグナル伝達を妨害する
Sept9 sequence alterations causing HNA are associated with altered interactions with Sept4/11 and resistance to Rho/Rhotekin signaling
須藤 香織(Kaori Sudo), 伊東 秀記(Hidenori Ito), 岩本 郁子(Ikuko Iwamoto), 森下 理香(Rika Morishita), 浅野 富子(Tomiko Asano), 永田 浩一(Koh-ichi Nagata)
愛知県心身障害者コロニー 発達障害研究所

P2-j05 カエル腓腹筋の筋収縮に対する低出力レーザー照射効果
Effects of low power laser irradiation on muscle contraction of the frog gastrocnemius
木暮 信一(Shinichi Kogure), 蛭子 法子(Noriko Ebisu), 久保 富洋(Tomihiro Kubo), 松田 芳樹(Yoshiki Matsuda)
創価大学大学院・工・生命情報

P2-j06 尾部懸垂による筋萎縮はジストロフィン複合体から細胞質内に移行したnNOSが産生するNOにより引き起こされる
Nitric oxide production results in disuse-induced muscle atrophy through dislocation of neuronal nitric oxide synthase
鈴木 直輝[1](Naoki Suzuki), 本橋 紀夫[2](Norio Motohashi), 鈴木 友子[2](Yuko Suzuki), 糸山 泰人[1](Yasuto Itoyama), 青木 正志[1](Masashi Aoki), 武田 伸一[2](Shin'ichi Takeda)
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## 依存、乱用
## Addiction and Abuse

P2-j07 NMDA受容体ε4サブユニット欠損マウスにおいてPCP投与で誘発される遺伝子発現パターンの異常性
PCP-induced abnormal gene expression pattern in NMDAR ε 4 KO mice
山本 秀子[1](Hideko Yamamoto), 亀ヶ谷悦子[1](Etsuko Kamegaya), 萩野 洋子[1](Yoko Hagino), 巽 国子[1,2](Kuniko Tatsumi), 田村 浩平[1,2](Kohei Tamura), 安本 智[1,2](Satoshi Yasumoto), 山本 敏文[1,2](Toshifumi Yamamoto), 三品 昌美[3](Masayoshi Mishina), 古閑比佐志[4](Hisashi Koga), 池田 和隆[1](Kazutaka Ikeda)
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P2-j08 線条体の構造とそれに関連する行動に与えるGli3の影響
Role of the zinc-finger transcriptional factor Gli3 in determining striatal structure and striatal-based behavior
佐郡 和人[1](Kazuto Sakoori), 純 元山[2](Motoyama Jun), Niall P. Murphy[1]
[1]理研・脳センター・マーフィー研究ユニット, [2]理研・脳センター・元山研究ユニット

P2-j09 摂食障害の前頭葉機能の検討：近赤外線スペクトロスコピーを用いて
Frontal lobe function in eating disorder:a multichannel near-infrared spectroscopy study
須田 真史(Masashi Suda), 上原 徹(Toru Uehara), 伊藤 誠(Makoto Ito), 佐藤 利正(Toshimasa Sato), 福田 正人(Masato Fukuda), 三國 雅彦(Masahiko Mikuni)
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P2-j10 報酬手がかり刺激に関連したマウスの新しい情動観察モデル
A new model to evaluate affective states associated with reward-predicting cues in mice
Barbara Cagniard, Niall P. Murphy
独立行政法人理化学研究所 神経回路メカニズム研究グループ

P2-j11 条件付け場所嗜好と条件付け場所嫌悪の獲得及び発現の際のカテコールアミンの活動の検討
Catecholamine activity during drug conditioned place preference and aversion
Adam Weitemier, Niall P. Murphy
独立行政法人理化学研究所 脳科学総合研究センター

P2-j12 メタンフェタミンによるドパミン終末毒性には小胞体ストレスが関与する
Methamphetamine induced dopamine terminal toxicity involves endoplasmic reticulum stress
入江 康至[1](Yasuyuki Irie), 宮 冬樹[2](Fuyuki Miya), 角田 達彦[2](Tatsuhiko Tsunoda),
佐伯万騎男[3](Makio Saeki), 上崎 善規[3](Yoshinori Kamisaki), 金村 米博[4](Yonehiro Kanemura),
三木 直正[5](Naomasa Miki), 平 英一[1](Eiichi Taira)
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P2-j13 The expression of cocaine-induced behavioral sensitization is blocked by microinjection of CART peptide 55-102 into the nucleus accumbens.
Hyung Shin Yoon, Seungwoo Kim, Hye Kyung Park, Jeong-Hoon Kim
Department of Physiology, Brain Korea 21 Project for Medical Sciences, Yonsei University College of Medicine, Seoul, South Korea

P2-j14 Epinephrine inhibits analgesic tolerance to intrathecal morphine while increases the expression of CaM kinase II$\alpha$
Leila Satarian, Mohammad Javan, Yaghoub Fathollahi
Department of Physiology, School of Medical Sciences, Tarbiat Modares University, Tehran, Iran.

P2-j15 ダイアモンド電極による脳内ドパミン測定
Diamond microelectrodes for dopamine detection in the brain
吉見 建二[1,2](Kenji Yoshimi), 鈴木あかね[3](Akane Suzuki), 大山 彦光[1,2](Genko Oyama),
籠橋 麻紀[1,2](Maki Kagohashi), 服部 信孝[1](Nobutaka Hattori), 中里 泰三[2,4](Taizo Nakazato),
栄長 泰明[3](Yasuaki Einaga), 北澤 茂[2,4](Shigeru Kitazawa)
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## 痛覚 2
## Pain 2

P2-j16 Protease-activated receptor 2 による TRPA1 活性増強
Sensitization of TRPA1 by PAR-2 through PLC/PIP2-dependent mechanisms
戴 毅[1](Yi Dai), 王 勝蘭[1](Shenglan Wang), 富永 真琴[2](Makoto Tominaga),
野口 光一[1](Koichi Noguchi)
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P2-j17 Bradykinin による TRPA1 活性の増強
Potentiation of TRPA1 activity by Bradykinin
王 勝蘭[1](Shenglan Wang), 戴 毅[1](Yi Dai), 富永 真琴[2](Makoto Tominaga),
野口 光一[1](Koichi Noguchi)
[1]兵庫医大・医・解剖, [2]岡崎統合バイオサイエンス・生命環境・細胞生理

P2-j18 神経因性疼痛における脊髄マイクログリアに発現する P2Y12 受容体の関与
P2Y12 receptor expressed in spinal microglia contributes to neuropathic pain
小林希実子(Kimiko Kobayashi), 福岡 哲男(Tetsuo Fukuoka), 山中 博樹(Hiroki Yamanaka),
野口 光一(Koichi Noguchi)
兵庫医科大学・解剖学第2講座

P2-j19 筋痛動物モデルにおけるイオンチャネルの関与
Involvement of ion channels in experimental models of muscle pain
藤井 優子(Yuko Fujii), 尾崎 紀之(Noriyuki Ozaki), 杉浦 康夫(Yasuo Sugiura)
名古屋大学大学院医学系研究科機能組織学

P2-j20 LPAの小脳延髄槽、下歯槽神経、三叉神経節への投与がフォンフライテストとc-Fos発現に及ぼす効果
Effects of LPA injection into the cisterna magna, trigeminal ganglion and inferior alveolar nerve on the Von Frey test and c-Fos expression
酒井　勇介(Yusuke Sakai)，杉生　真一(Shinichi Sugiyo)，下田　隆史(Takashi Shimoda)，
正脇　綾(Aya Masawaki)，森谷　正之(Masayuki Moritani)，吉田　篤(Atsushi Yoshida)，
竹村　元秀(Motohide Takemura)
大阪大院・歯・第二解剖

P2-j21 トラマドールはオピオイド受容体活性化やモノアミン取り込み阻害と無関係に神経伝導ブロックを起こす
Tramadol produces nerve conduction block in a manner independent of opioid receptor activation and monoamine uptake inhibition
香月　亮[1,2](Ryo Katsuki)，藤田　亜美[2](Tsugumi Fujita)，水田恒太郎[2](Kotaro Mizuta)，
柳　涛[2](Tao Liu)，中塚　映政[2](Terumasa Nakatsuka)，熊本　栄一[2](Eiichi Kumamoto)
[1]嬉野医療センター，[2]佐賀大・医・生体構造機能学

P2-j22 疼痛制御機構に関する研究（第67報）：オピオイドによる視床ナトリウムチャネルの制御
Studies on pain control system (Rept. 67): Opioids regulate sodium currents in rat cultured thalamic neurons
橋本　敬輔[1](Keisuke Hashimoto)，成田　年[1](Minoru Narita)，天野　託[2](Taku Amano)，
鈴木　勉[1](Tsutomu Suzuki)
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P2-j23 関節不動化ラットの脊髄後角における疼痛関連分子の免疫組織化学的変化
Immunohistochemical changes of pain-related molecules in the spinal dorsal horn of the joint immobilized rat
田中健二朗[1](Kenjiro Tanaka)，大迫　洋治[1](Yoji Osako)，牛田　享宏[3](Takahiro Ushida)，
西上　智彦[2](Tomohiko Nishigami)，由利　和也[1](Kazunari Yuri)
[1]高知大・医・解剖，[2]高知大学医学部整形外科学講座，[3]愛知医科大学付属学際的痛みセンター

P2-j24 疼痛制御機構に関する研究（第66報）：神経障害性疼痛によるモルヒネ精神依存不形成機構における内因性オピオイド神経系の関与
Studies on pain control system (Rept. 66): Involvement of endogenous opioidergic system in the suppression of the morphine-induced rewarding effect under the neuropathic pain-like state
新倉　慶一(Keiichi Niikura)，成田　年(Minoru Narita)，成田　道子(Michiko Narita)，
中邨　篤史(Atsushi Nakanura)，葛巻　直子(Naoko Kuzumaki)，南條　加奈(Kana Nanjo)，
倉橋　香菜(Kana Kurahashi)，小林　泰久(Yasuhisa Kobayashi)，鈴木　雅美(Masami Suzuki)，
鈴木　勉(Tsutomu Suzuki)
星薬大・薬・薬品毒性

P2-j25 再生下歯槽神経が関与する三叉神経脊髄路核ニューロン活動
Modulation of trigeminal spinal subnucleus caudalis neuronal activity following regeneration of transected inferior alveolar nerve in rats
岩田　幸一(Koichi Iwata)，齋藤　仁子(Kimiko Saito)，人見　涼露(Suzuro Hitomi)，
坪井　美行(Yoshiyuki Tsuboi)
日本大学・歯・生理

P2-j26 In vivo パッチクランプ法を用いた神経因性疼痛モデルラット脊髄膠様質におけるセロトニン抑制系可塑的変化の解析
In vivo patch-clamp analysis of plastic changes in descending serotonergic inhibition of noxious mechanical transmission to substantia gelatinosa neurons through 5-HT1A receptors in ovariectomized rats
大森　優[1,2](Yu Omori)，古江　秀昌[1](Hidemasa Furue)，伊藤　彰敏[1](Akitoshi Ito)，
倉石　泰[2](Yasushi Kuraishi)，吉村　恵[1](Megumu Yoshimura)
[1]九大院・医・統合生理，[2]富山大院・薬・応用薬理

P2-j27 シナプス前 TRPA1 の活性化によって成熟ラット脊髄膠様質細胞にグルタミン酸の放出が増強する
Presynaptic TRPA1 activation enhances glutamate release onto substantia gelatinosa neurons of the adult rat spinal cord
小杉 雅史(Masafumi Kosugi)，中塚 映政(Terumasa Nakatsuka)，藤田 亜美(Tsugumi Fujita)，
青山 貴博(Takahiro Aoyama)，熊本 栄一(Eiichi Kumamoto)
佐賀大学医学部生体構造機能学講座・神経生理学分野

P2-j28 神経障害性疼痛下における脳内グリア細胞新生の変化
Changes in new generation of glial cells under the neuropathic pain like state
葛巻 直子[1](Naoko Kuzumaki)，成田 年[1](Minoru Narita)，成田 道子[1](Michiko Narita)，
新倉 慶一[1](Keiichi Niikura)，天野 託[2](Taku Amano)，鈴木 勉[1](Tsutomu Suzuki)
[1]星薬科大学・薬品毒性，[2]広島大学大学院医歯薬学総合研究科・神経・精神薬理学教室

## 疾患モデル
## Animal Models

P2-j29 *XPG* 遺伝子欠損マウスの病理学的解析
Pathological analysis of *XPG*-deficient mice
中根 裕信[1](Hironobu Nakane)，廣田 誠一[2](Seiichi Hirota)，塩見 忠博[3](Tadahiro Shiomi)，
大浜 栄作[4](Eisaku Ohama)，亀家 俊夫[1](Toshio Kameie)，稲賀すみれ[1](Sumire Inaga)，
名黒 知徳[1](Tomonori Naguro)
[1]鳥取大・医・ゲノム形態学分野，[2]兵庫医科大学 医学部 病院病理学，[3]放射線医学総合研究所 安全研究センター 低線量生体影響プロジェクト，[4]鳥取大学 医学部 脳神経病理学部門

P2-j30 βガラクトシダーゼ欠損症の神経変性における Trk 受容体の機能異常
Dysregulation of Trk receptor signaling in β-galactosidase-deficient mouse brain
高村 歩美[1](Ayumi Takamura)，檜垣 克美[1](Katsumi Higaki)，松田潤一郎[2](Junichiro Matsuda)，
飯田 真巳[3](Masami Iida)，鈴木 義之[4](Yoshiyuki Suzuki)，難波 栄二[1](Eiji Nanba)
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P2-j31 鉄代謝の微細変化がもたらす神経変性疾患の発症または増悪機構の解析
Trivial change of iron metabolism can cause or deteriorate neurodegenerative symptoms
坂田 真一[1](Shin-ichi Sakata)，中川 朋子[1](Tomoko Nakagawa)，大橋 聡[2](Satoshi Ohashi)，
波田野 琢[2](Taku Hatano)，吉見 健二[2](Kenji Yoshimi)，服部 信孝[2](Nobutaka Hattori)，
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P2-j32 遺伝的不適合による神経発達異常
Aberrant neurological development caused by genetic incompatibility
梅森 十三[1](Juzoh Umemori)，近藤 亮太[1](Ryouta Kondou)，湯浅 茂樹[2](Shigeki Yuasa)，
小出 剛[1](Tsuyoshi Koide)
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P2-j33 マウス行動実験のためのプロトコールデータベースの開発
Development of database for experimental parameters of behavior analyses in mice
若菜 茂晴[1](Shigeharu Wakana)，山田 郁子[1](Ikuko Yamada)，和田由美子[1,4](Yumiko Wada)，
古瀬 民生[1](Tamio Furuse)，高雄 啓三[2](Keizo Takao)，中西 和男[2](Kazuo Nakanishi)，
高橋 阿貴[3](Aki Takahashi)，小出 剛[3](Tsuyoshi Koide)，宮川 剛[2](Tsuyoshi Miyakawa)，
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P2-j34 多系統萎縮症モデルマウスにおける神経細胞内α-synuclein 複合体の動態解析
Analysis of α-synuclein and the associated protein in MSA mouse model
中山貴美子(Kimiko Nakayama)，鈴木 康予(Yasuyo Suzuki)，矢澤 生(Ikuru Yazawa)
国立長寿医療センター・研究資源有効利用室

P2-j35 Micro PET を用いた片頭痛病態モデルの評価
Evaluation of pathophysiological features of migraine by micro PET study using rats
崔　翼龍[1](Yilong Cui)，和田　康弘[1](Yasuhiro Wada)，大和　正典[2](Masanori Yamato)，
田村　泰久[1](Yasuhisa Tamura)，片岡　洋祐[1,2](Yosky Kataoka)，渡辺　恭良[1,2](Yasuyoshi Watanabe)
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P2-j36 Elevated 5-HT expression of spinal motoneurons of ataxic mutant mice, Pogo
Nam-Seob Lee[1], Jin-Seong Kim[1], Jae-Min Kim[1], Jin-A Son[1], Yoshiki Takeuchi[2], Young-Gil Jeong[1]
[1]Department of Anatomy, College of Medicine, Konyang University, Daejeon, Republic of Korea,
[2]Department of Anatomy and Neurobiology, Faculty of Medicine, Kagawa University, Kagawa, Japan

P2-j37 Schnurri-2 欠失マウスの精神疾患様行動異常
Deletion of Schnurri-2 causes multiple behavioral abnormalities related to psychiatric disorders in mice.
高雄　啓三[1,2](Keizo Takao)，山崎　信幸[1](Nobuyuki Yamasaki)，高木　豪[3](Tsuyoshi Takagi)，
石井　俊輔[3](Shunsuke Ishii)，宮川　剛[1,2](Tsuyoshi Miyakawa)
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P2-j38 alpha-CaMKII ヘテロノックアウトにより引き起こされる行動異常と未成熟歯状回
Alpha-CaMKII deficiency causes dysregulated behaviours and immature dentate gyrus
山崎　信幸[1](Nobuyuki Yamasaki)，前川　素子[2](Motoko Maekawa)，小林　克典[3](Katsunori Kobayashi)，
梶井　靖[4](Yasushi Kajii)，前田　純[5](Jun Maeda)，相馬　美歩[2](Miho Soma)，高雄　啓三[1](Keizo Takao)，
神崎　康治[4](Kouji Kanzaki)，鈴木　秀典[3](Hidenori Suzuki)，樋口　真人[5](Makoto Higuchi)，
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P2-j39 ニューレグリン - 1 トランスジェニックマウスの認知行動変化：遺伝的バックグラウンドの影響
Alterations in cognitive behaviors of neuregulin-1 transgenic mice: An influence of genetic backgrounds
加藤　泰介[1](Taisuke Kato)，水野　誠[1,2](Makoto Mizuno)，尾崎美和子[3](Miwako Ozaki)，
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P2-j40 ErbB1 遺伝子改変動物の作製およびその行動解析
Establishment and behavioral analysis of conditional transgenic mouse lines carrying mutant ErbB1 gene.
今野　公博[1](Kimihiro Konno)，熊谷　春乃[1](Haruno Kumagai)，荒木　一明[1](Kazuaki Araki)，
糸原　重美[2](Shigeyoshi Itohara)，那波　宏之[1](Hiroyuki Nawa)
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P2-j41 イボテン酸投与ラットにおけるチロシン受容体阻害剤の効果
Antipsychotic effects of ErbB1 tyrosine kinase inhibitors on behavioral deficits in the neonatal hippocampal lesion
水野　誠[1](Makoto Mizuno)，川村　宏樹[2](Hiroki Kawamura)，江田　岳誉[2](Takeyoshi Eda)，
朴　英善[2](Ying-Shan Piao)，那波　宏之[1,2](Hiroyuki Nawa)
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P2-j42 中枢神経特異的カテプシン D 欠損による神経性セロイドリポフスチン蓄積症類似症状の誘導
Central nervous system specific deficiency of lysosomal cathepsin D induces a phenotype resembling neuronal ceroid lipofuscinosis.
柴田　昌宏(Masahiro Shibata)，小池　正人(Masato Koike)，内山　安男(Yasuo Uchiyama)
大阪大院・医・神経生物学形態学

P2-j43 PTSD 発症に関連する海馬内遺伝子
Expression of a gene in response to traumatic experience in murine hippocampus
玉置　啓祐(Keisuke Tamaki)，前田紗弥香(Sayaka Maeda)，米田　幸雄(Yukio Yoneda)
金沢大院・自然科学（薬）・薬物学

P2-j44 頭部外傷後のラット海馬歯状回における過興奮と長期増強の障害に対するエダラボンの改善作用
Edaravone attenuates post-traumatic hyperactivity and impairment of LTP of excitatory synaptic transmission in rat hippocampal dentate gyrus
山下　伸[1,2](Shin Yamashita)，蓮尾　博[1](Hiroshi Hasuo)，徳富　孝志[2](Takashi Tokutomi)，
重森　稔[2](Minoru Shigemori)，赤須　崇[1](Takashi Akasu)
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## イメージング 2
## Imaging 2

P2-j45 ラット大脳皮質の光学的膜電位多部位記録測定領野内からの脳波測定を目的とした透明電極の開発
Transparent electrode for recording electrocorticogram from optical sampling area of the multiple-site optical detection of neural activity in the rat cerebral cortex
廣田　秋彦[1](Akihiko Hirota)，吉田　勇気[2](Yuki Yoshida)，藤田　恭久[2](Yasuhisa Fujita)，
伊藤　眞一[1](Shin-ichi Ito)
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P2-j46 蛍光グルコーストレーサを用いた黒質網様部のリアルタイムグルコース取り込み可視化
Real-time imaging of glucose uptake in the substantia nigra pars reticulata using a fluorescent glucose tracer
山田　勝也[1,4](Katsuya Yamada)，武尾　照子[3](Teruko Takeo)，高綱　将史[1](Masafumi Takatsuna)，
菅　世智子[2](Sechiko Suga)
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P2-j47 重粒子線照射した脳における COX2 タンパク質と Edema 形成機構の関連
Edema and cyclooxygenase-2 protein expression in the brain after local irradiation with carbon ion beams
孫　学智[1](Xuezhi Sun)，Rui Zhang[2]，Chun Cui[2]，澤田　和彦[2](Kazuhiko Sawada)，
久野　節二[3](Setusji Hisano)，福井　義浩[2](Yoshihiro Fukui)，米原　英典[1](Hidenori Yonehara)
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P2-j48 薬物アセスメントのための単一神経モデル細胞からのドーパミン放出モニタリング
Single cell-based monitoring of dopamine release from a nerve model cell using a microelectrode for drug assessment
篠原　寛明(Hiroaki Shinohara)，石井　友也(Tomoya Ishii)
富山大院・理工・生命情報システム

P2-k01 後口動物神経系のグリオキシル酸による蛍光可視化
Glyoxylic acid induced fluorescence imaging of the larval nervous system of deuterostomia
矢澤　徹[1](Toru Yazawa)，霜田　幸雄[2](Yukio Shimoda)
[1]首大・都市教養・理工系・生命科学，[2]東京女子医科大学　総研

P2-k02 ハイスループットスクリーニングによる新規蛍光性グルタミン酸プローブの開発
Generation of novel fluorescent glutamate indicators by high-throughput screening system
飯沼　将(Sho Iinuma)，中西　絢美(Ayami Nakanishi)，並木　繁行(Shigeyuki Namiki)，
坂本　寛和(Hirokazu Sakamoto)，廣瀬　謙造(Kenzo Hirose)
名古屋大院・医・細胞生理

P2-k03 シナプス伝達可視化のための高性能蛍光性グルタミン酸プローブの開発
Development of the high performance glutamate indicator for visualization of neurotransmission
中西　絢美(Ayami Nakanishi)，飯沼　　将(Sho Iinuma)，並木　繁行(Shigeyuki Namiki)，
坂本　寛和(Hirokazu Sakamoto)，廣瀬　謙造(Kenzo Hirose)
名古屋大・医・細胞生理

P2-k04 ラット皮質線条体における膜電位感受性色素による神経伝達イメージング
neuronal activity imaging in the rat cortico-striatum pathway using voltage-sensitive dye
稲次　基希[1](Motoki Inaji)，佐藤　勝重[2](Katsushige Sato)，成相　　直[1](Tadashi Nariai)，
百瀬－佐藤容子[2](Yoko Momose-Sato)，大野喜久郎[1](Kikuo Ohno)
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P2-k05 脳損傷モデルマウスにおける脳移行性骨髄細胞の活性化と分化のイメージング
In vivo imaging indicates activation and differentiation of brain-migrated immature bone marrow cells in an artificial brain injury
小野　健治(Kenji Ono)，山本　奈穂(Naho Yamamoto)，鈴木　弘美(Hiromi Suzuki)，
神澤　孝夫(Takao Kanzawa)，澤田　　誠(Makoto Sawada)
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P2-k06 マウス海馬 CA1 領域におけるシータバースト刺激後のセリンプロテアーゼ in vivo リアルタイムイメージング
Real time in vivo imaging of serine protease activity after theta-burst stimulation in the CA1 area of the mouse hippocampus
田村　英紀[1](Hideki Tamura)，David C Ng[2]，徳田　　崇[2](Takashi Tokoda)，中川　琢磨[2](Takuma Nakagawa)，
水野　太郎[2](Taro Mizuno)，太田　　淳[2](Jun Ohta)，塩坂　貞夫[1](Sadao Shiosaka)
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P2-k07 改良型膜電位感受性蛍光タンパク質の開発と特性評価
Engineering and characterization of an enhanced fluorescent protein voltage sensor
武藤　弘樹(Hiroki Mutoh)，Dimitar Dimitrov，You He，Walther Akemann，Thomas Knopfel
理化学研究所　脳科学総合研究センター　神経回路ダイナミクス研究チーム

P2-k08 ファイバー式リアルタイム共焦点顕微鏡による多細胞シグナル解析法
Analysis for multiple cellular signals by fiber-coupled realtime confocal microscopy
須々木 - 土屋礼美[1](Remi Susuki-Tsuchiya)，櫻井　孝司[1](Takashi Sakurai)，三池　神也[2](Shinya Miike)，
橋本　欣和[2](Yoshikazu Hashimoto)，山宮　広之[3](Hiroyuki Sanguu)，秋山　　喬[3](Takashi Akiyama)，
山本　清二[1](Seiji Yamamoto)，寺川　　進[1](Susumu Terakawa)
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P2-k09 Real-time monitoring of l-glutamate release from C6 glioma cells using a modified enzyme-luminescence method and its application to drug assessment
S. M. Z. Hossain，H. Shinohara
Life , Information and System Sci., University of Toyama

P2-k10 X 線マイクロ CT 技術を用いた脳神経細胞の 3 次元再構築
3-dimensional reconstruction of neurons in the brain by using X-ray micro-CT technology
水谷　治央[1](Haruo Mizutani)，武田　佳彦[2](Yoshihiro Takeda)，百生　　敦[2](Atsushi Momose)，
高木　利久[1](Toshihisa Takagi)
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P2-k11 クロマフィン細胞の開口分泌機構におけるミオシン及びミオシン ATPase の役割
Roles of Myosin and myosin-ATPase for exocytosis in chromaffin cells
関口　瑞季[1,2](Mizuki Sekiguchi)，林　　光紀[1](Mitsunori Hayashi)，松田　冬香[1](Fuyuka Matsuda)，
笹川　展幸[1](Nobuyuki Sasakawa)，熊倉鴻之助[1](Konosuke Kumakura)
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P2-k12 **2細胞間の同期性にもとづくネットワーク特性の解析**
**Complex topology underlies pairwise spike synchronization**
高橋　直矢[1](Naoya Takahashi)，佐々木拓哉[1](Takuya Sasaki)，松木　則夫[1](Norio Matsuki)，
池谷　裕二[1,2](Yuji Ikegaya)
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P2-k13 **多ニューロンスパイク列計量法**
**Metrics for multineuronal spike trains**
宇佐美　篤(Atsushi Usami)，松木　則夫(Norio Matsuki)，池谷　裕二(Yuji Ikegaya)
東京大院・薬・薬品作用

P2-k14 **海馬多シナプス回路における情報演算の再構築**
**Remodeling of information computation by hippocampal polysynaptic circuits**
木村　梨絵[1](Rie Kimura)，松木　則夫[1](Norio Matsuki)，池谷　裕二[1,2](Yuji Ikegaya)
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## 神経情報学 Neuroinformatics

P2-k15 **聴覚誘発波N100mピークの発生メカニズム－複数振動子の位相同期**
**Phase locking among multiple oscillators as a mechanism for generating the auditory evoked peak of N100m**
浜田　隆史(Takashi Hamada)
産業技術総合研究所・関西センター

P2-k16 **スパイクソーティングにおける特徴抽出とクラスタリングの最適手法**
**A spike sorting method with optimal feature extraction and clustering**
竹川　高志(Takashi Takekawa)，姜　　時友(Siu Kang)，礒村　宜和(Yoshikazu Isomura)，
深井　朋樹(Tomoki Fukai)
理研・脳総研

P2-k17 **細胞固有のスパイク時系列パターンの領野間比較**
**Comparison of cell-specific spike patterns among different cortical areas**
松野　奈苗(Nanae Matsuno)，篠本　　滋(Shigeru Shinomoto)
京都大院・理

P2-k18 **ベイズ統計に基づいた海馬CA1錐体細胞の摂動応答計測**
**Perturbation response measurements in hippocampal CA1 pyramidal neuron based on Bayesian statistics**
太田　桂輔[1](Keisuke Ota)，青西　　亨[1,2](Toru Aonishi)，渡部　重夫[3](Shigeo Watanabe)，
宮川　博義[3](Hiroyoshi Miyakawa)，大森　敏明[2,4](Toshiaki Omori)，岡田　真人[2,5](Masato Okada)
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P2-k19 **C.elegansの温度走性・化学走性に関与するニューラルネットワークのシナプス極性予測**
**A prediction of synaptic polarities of neural network regulating thermotaxis and chemotaxis in C.elegans**
祝田　尚之(Naoyuki Iwaita)，山本　真二(Shinji Yamamoto)，若林　篤光(Tokumitsu Wakabayashi)，
新貝　鋤蔵(Ryuzo Shingai)
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P2-k20 **パーキンソン病患者の視床下核におけるマルチニューロン活動および局所電場電位の解析**
**An analysis of multi-neuronal activities and local field potentials in the subthalamic nucleus in Parkinson's disease**
岡本　　剛[1](Tsuyoshi Okamoto)，宮城　　靖[1](Yasushi Miyagi)，合原　一幸[2,3](Kazuyuki Aihara)，
飛松　省三[4](Shozo Tobimatsu)
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P2-k21 線虫 Caenorhabditis elegans の電位依存性チャネルのパラメータ探索
Determination of parameters of voltage-gated channel of *Caenorhabditis elegans*
坂田　和実(Kazumi Sakata)，小栗栖太郎(Tarou Ogurusu)
岩手大学・工・福祉システム

P2-k22 スパイクデータのヒストグラムの最適化
Optimization of a histogram of spike data
島崎　秀昭(Hideaki Shimazaki)，篠本　　滋(Shigeru Shinomoto)
京都大学院・理・物理学専攻

P2-k23 Fine spatio-temporal interactions in multielectrode LFP signals
Gustavo S. Santos[1], Masaki Arisaka[4], Takafumi Higashi[1], Tohru Ozaki[3], Dietmar Plenz[2], Hiroyuki Nakahara[1]
[1]Lab. For Integrated Theoretical Neurosci., RIKEN BSI, Japan, [2]Lab. of Systems Neurosci., Porter Neurosci. Res. Center, NIMH/NIH, USA, [3]Inst. of Statistical Math., Tokyo, Japan, [4]Dept. of Math. Informatics, Grad. Sch. of Info. Sci. and Tech., U. Tokyo, Japan

P2-k24 On a novel measure for synchrony and its application to EEG
Justin Dauwels[1], Francois Vialatte[2], Andrzej Cichocki[2]
[1]Amari Research Unit, RIKEN Brain Science Institute, Wako-shi,Saitama,Japan, [2]Laboratory for Advanced Brain Signal Processing, RIKEN Brain Science Institute

P2-k25 A fast in-silico protocol for identification of anti epileptic drug leads
Suyambu Kesava Vijayan Ramaswamy, Nanda Ghoshal
Structural Biology and Bioinformatics Division, Indian Institute of Chemical Biology

Core Time Odd numbers : 11:00 ～ 12:00
Even numbers : 13:00 ～ 14:00

## イオンチャンネル、興奮性膜
## Ion Channels and Excitable Membranes

P3-a01 電位依存性カルシウムチャネル gamma-5 および 7 は AMPA 型受容体と複合体を形成している
Association of gamma-5 and gamma-7 proteins with AMPA receptors
板倉 誠[1](Makoto Itakura), 菅谷津貴子[1](Tsukiko Sugaya), 山森 早織[1](Saori Yamamori),
深谷 昌弘[2](Masahiro Fukaya), 渡辺 雅彦[2](Masahiko Watanabe), 高橋 正身[1](Masami Takahashi)
[1]北里大学・医・生化学, [2]北海道大学・医・解剖

P3-a02 海馬ＣＡ１錐体細胞尖端樹状突起における周波数依存的応答特性
Frequency-dependent response property of the dendrites in the hippocampal CA1 pyramidal cells
渡辺 秀典[1](Hidenori Watanabe), 坪川 宏[2](Hiroshi Tsubokawa), 相原 威[3](Takeshi Aihara),
塚田 稔[1](Minoru Tsukada)
[1]玉川大学・脳科学研究所, [2]東北大学 大学院情報科学研究科 応用情報科学専攻, [3]玉川大学工学部

P3-a03 ラット後部帯状回皮質２層における遅延発火性錐体細胞
Specialized late-spiking pyramidal neurons in layer 2 of the rat retrosplenial cortex
黒谷 亨(Tohru Kurotani), 宮下 俊雄(Toshio Miyashita), 一戸 紀孝(Noritaka Ichinohe),
Kathleen Rockland
理研・BSI・脳皮質機能構造研究チーム

P3-a04 ラット体性感覚野 Fast-spiking 細胞におけるスパイク生成とリズミックな活動電位振動にコンダクタンス入力信号が及ぼす影響
Influence of conductance-input signal on spike generation and stability of rhythmic activity in rat somatosensory fast-spiking neurons
舘野 高(Takashi Tateno)
大阪大院・基礎工・機能創成

P3-a05 プロポフォールによるラット大脳皮質 GABA 作動性抑制伝達増強作用
Effects of propofol on tonic GABAergic inhibition in rat cerebral cortex
山本 純偉[1](Sumii Yamamoto), 山田 順子[2](Junko Yamada), 古川 智範[2](Tomonori Furukawa),
田中 誠[1](Makoto Tanaka), 福田 敦夫[2](Atsuo Fukuda)
[1]筑波大学大学院人間総合科学研究科, [2]浜松医科大学生理学第一講座

P3-a06 エストロジェン受容体遺伝子プロモータートランスジェニックラット海馬ニューロンの電位依存性カリウム電流に対するエストロジェンの修飾
Estrogen modulates voltage dependent potassium currents in the EGFP-tagged neurons in the hippocampus of estrogen receptor gene promoter transgenic rats
濱田 知宏(Tomohiro Hamada), Hongli Shan, 佐久間康夫(Yasuo Sakuma)
日本医大・医・生理学第一

P3-a07 PSD-95 は NR1/NR2A サブタイプ NMD 受容体の MK-801 への感受性を変える
PSD-95 modulates the sensitivity of NR1/NR2A subtype NMDA receptor channels for a NMDA receptor antagonist, MK801.
山田 康枝(Yasue Yamada), 乾 誠(Makoto Inui)
山口大院・医・分子薬理

P3-a08 小型後根神経節ニューロンでの痛覚伝搬における電位依存性ナトリウムチャネルの機能的役割
Functional roles of voltage-gated $Na^+$ channels for pain sensation in different subpopulations of small dorsal root ganglion neurons
中本 千泉[1,2](Chizumi Nakamoto), 松富 智哉[1](Tomoya Matsutomi), 柿村 順一[1](Jun-ichi Kakimura),
鄭 泰星[1](Taixing Zheng), 緒方 宣邦[1](Nobukuni Ogata)
[1]広島大院・医歯薬・神経生理, [2]日本学術振興会特別研究員

P3-a09 スーパーオキサイドは電位依存性 $Na^+$ チャネル電流の不活性化を遅延する
Superoxide retards the inactivation of voltage-gated $Na^+$ currents
竹内 啓太(Keita Takeuchi), 吉井 清哲(Kiyonori Yoshii)
九工大院・生命体・脳情報

P3-a10 乳児部分てんかんに関連する変異型ナトリウムチャネルの機能解析
SCN1A missense mutation associated with infantile partial epilepsy
大守 伊織[1](Iori Ohmori), 大内田 守[2](Mamoru Ouchida), 小林 勝弘[3](Katsuhiro Kobayashi),
大塚 頌子[3](Yoko Ohtsuka), 清水 憲二[2](Kenji Shimizu), 西木 禎一[1](Teiichi Nishiki),
富澤 一仁[1](Kazuhito Tomizawa), 松井 秀樹[1](Hideki Matsui)
[1]岡山大院・医・細胞生理, [2]岡山大院・医・分子遺伝, [3]岡山大院・医・発達神経病態学

P3-a11 ミクログリアにおける電位依存性プロトンチャネル VSOP の解析
Analysis of a voltage-gated proton channel, VSOP in microglia
大河内善史[1](Yoshifumi Okochi), 澤田 誠[4](Makoto Sawada), 佐々木真理[1,2](Mari Sasaki),
岩崎 広英[1,2](Hirohide Iwasaki), 岡村 康司[1,2,3](Yasushi Okamura)
[1]岡崎統合バイオサイエンスセンター・神経分化研究部門, [2]自然科学研究機構・生理学研究所, [3]総合研究大学大学院, [4]名古屋大学環境医学研究所 生体適応・防御研究部門・脳機能分野

P3-a12 オピオイドによるカエル坐骨神経の活動電位抑制
Inhibition by opioids of compound action potentials in frog sciatic nerves
水田恒太郎(Kotaro Mizuta), 藤田 亜美(Tsugumi Fujita), 香月 亮(Ryo Katsuki), 柳 涛(Tao Liu),
朴 蓮花(Lian-Hua Piao), 岳 海源(Hai-Yuan Yue), 中塚 映政(Terumasa Nakatsuka),
熊本 栄一(Eiichi Kumamoto)
佐賀大・医・生体構造機能学

P3-a13 三叉神経中脳路核ニューロンにおけるhチャネルとグルタミン酸受容体チャネル間の相互作用
Interaction between h-channels and glutamate receptor channels in the mesencephalic trigeminal neurons
佐藤 元(Hajime Sato), 齋藤 充(Mitsuru Saito), 豊田 博紀(Hiroki Toyoda),
姜 英男(Youngnam Kang)
大阪大院・歯・高次脳口腔機能学

P3-a14 海馬錐体細胞の左右非対称性
Left-right asymmetry of mouse hippocampal pyramidal neurons
篠原 良章[1](Yoshiaki Shinohara), 重本 隆一[2](Ryuichi Shigemoto)
[1]理化学研究所, [2]生理学研究所

P3-a15 アクティブゾーンタンパク質 RIM1 と電位依存性カルシウムチャネルの機能的相互作用
Active zone protein RIM1 functionally associates with presynaptic VDCCs
清中 茂樹[1](Shigeki Kiyonaka), 三木 崇史[1](Takafumi Miki), 野中 美央[2](Mio Nonaka),
瓜生 幸嗣[1](Yoshitsugu Uriu), 若森 実[1,6](Minoru Wakamori), 森 恵美子[1](Emiko Mori),
原 雄二[1](Yuji Hara), Michel De Waard[4], 板倉 誠[5](Makoto Itakura), 高橋 正身[5](Masami Takahashi),
尾藤 晴彦[2](Haruhiko Bito), Kevin Campbell[3], 森 泰生[1](Yasuo Mori)
[1]京都大院・工・合成生物, [2]東京大院・医・神経性化学, [3]アイオワ大学・医, [4]インセルムU607, [5]北里大院・医・神経生物学, [6]東北大院・歯・薬理

P3-a16 錐体・桿体ジストロフィー遺伝変異 RIM1 による電位依存性カルシウムチャネルの機能変化解析
The RIM1 mutation associated with an autosomal dominant cone-rod dystrophy, CORD7, alters effects of RIM1 on VDCC currents
三木 崇史[1](Takafumi Miki), 清中 茂樹[1](Shigeki Kiyonaka), 瓜生 幸嗣[1](Yoshitugu Uriu),
若森 実[1,4](Minoru Wakamori), 森 恵美子[1](Emiko Mori), 板倉 誠[2](Makoto Itakura),
高橋 正身[2](Masami Takahashi), Kevin Campbell[3], 森 泰生[1](Yasuo Mori)
[1]京都大院・工・合成生物化学, [2]北里大学・医・生化学, [3]アイオワ大学, [4]東北大学・歯・薬理

P3-b01 C2Bを共通ドメインとして有するRIMファミリーによる電位依存性カルシウムチャネルの機能修飾
Functional impact of RIM family sharing $C_2B$ domain on gating of voltage-dependent $Ca^{2+}$ channels
瓜生 幸嗣[1](Yoshitsugu Uriu), 若森 実[1,2](Minoru Wakamori), 清中 茂樹[1](Shigeki Kiyonaka),
三木 崇史[1](Takafumi Miki), 森 恵美子[1](Emiko Mori), 森 泰生[1](Yasuo Mori)
[1]京都大院・工・合成生物, [2]東北大院・歯・薬理

P3-b02 KCNQ チャネルにおけるサブユニットの認識および会合の機構
Mechanisms underlying subunit recognition and channel assembly in KCNQ channels
中條 浩一[1](Koichi Nakajo), 久保 義弘[1,2](Yoshihiro Kubo)
[1]生理学研究所・神経機能素子, [2]戦略的創造研究推進事業 発展研究

P3-b03 Groggy 変異ラットの Purkinje 細胞から記録された $Ca^{2+}$ チャネル電流の増大
Increased $Ca^{2+}$ channel currents in cerebellar Purkinje cells of the ataxic Groggy rat
金子 周司[1](Shuji Kaneko), 田中 健太[1](Kenta Tanaka), 白川 久志[1](Hisashi Shirakawa),
徳田 智子[2](Satoko Tokuda), 芹川 忠夫[2](Tadao Serikawa)
[1]京都大院・薬・生体機能, [2]京都大院・医・動物実験施設

P3-b04 プレシナプスアクティブゾーンタンパク質による電位依存性 P/Q 型カルシウムチャネルの不活性化過程の機能修飾
Effect of RIM 1 on inactivation of neuronal voltage-dependent calcium channels
若森 実[1,2](Minoru Wakamori), 瓜生 幸嗣[2](Yoshitsugu Uriu), 三木 崇史[2](Takafumi Miki),
清中 茂樹[2](Shigeki Kiyonaka), 森 泰生[2](Yasuo Mori)
[1]東北大院・歯・口腔生物, [2]京都大院・工・合成生物化学

P3-b05 Activity-dependent relief of G-protein-dependent inhibition of presynaptic P/Q-type $Ca^{2+}$ channels
Masahiro Kaneko, Tomoyuki Takahashi
Dept. Neurophys., Univ. Tokyo Grad. Sch. Med., Tokyo, Japan

P3-b06 Effect of ciprofloxacin and biphenylacetic acid on voltage-gated ion channels in cultured rat hippocampal neurons
Ryoko Hirakawa, Parto Khansari, Leanne Coyne, Robert F. Halliwell
TJL School of Pharmacy and Health Science, University of the Pacific, Stockton, CA, USA

P3-b07 Identification of the Ginsenoside Interaction Site in Human Kv1.4 Channels
Jun-Ho Lee[1], Byung-Hwan Lee[1], Sun-Hye Choi[1], In-Soo Yoon[1], Joon-Hee Lee[1], Mi Kyung Pyo[1], Sang Min Jeong[1], Woo-Sung Choi[1], Sang-Mok Lee[1], Hyewhon Rhim[2], Seung-Yeol Nah[1]
[1]Dpt of Physiology, College of Vet. Medicine, Konkuk Univ., SEOUL, KOREA, [2]Biomedical Res. Center, KIST, Seoul Korea

P3-b08 Expression and localization of TREK-1 channel in Schwann cells
Dawon Kang, Eun-Jin Kim, Gyu-Tae Kim, Chang-Gi Hur, Jae-Yong Park, Seong-Geun Hong, Jaehee Han
Department of Physiology, Gyeongsang Nati. University, Jinju, South Korea

## シナプス可塑性 2
## Synaptic Plasticity 2

P3-b09 シナプス前終末におけるサイレントシナプスの賦活化ー海馬苔状線維における開口放出増強メカニズム
Activation of presynaptically silent synapses - one of underlying mechanisms enhancing mossy fiber transmission in the hippocampus
引間　卓弥[1](Takuya Hikima)，荒木　力太[2](Rikita Araki)，石塚　　徹[1](Toru Ishizuka)，
八尾　　寛[1](Hiromu Yawo)
[1]東北大学院・生命科学・脳機能解析，[2]理研・脳科学総合研究センター・神経回路ダイナミックス研究センター

P3-b10 HPC-1/syntaxin 1A ノックアウトマウスの長期増強における cAMP 情報伝達系の減衰
cAMP signaling cascade is reduced in hippocampal LTP of HPC-1/syntaxin 1A knock-out mice
三嶋　竜弥[1](Tatsuya Mishima)，藤原　智徳[1](Tomonori Fujiwara)，小藤　剛史[2](Takefumi Kofuji)，
赤川　公朗[1](Kimio Akagawa)
[1]杏林大・医・細胞生理，[2]杏林大・医・RI研究部門

P3-b11 脊髄損傷後に RGM 阻害が皮質脊髄路軸索のシナプス形成を高める
RGMa inhibition enhances synapse formation of the cortico-spinal axons after spinal cord injury
京藤　聡弘(akihiro kyoto)，山下　俊英(Toshihide Yamashita)
千葉大学大学院医学研究院神経生物学

P3-b12 Vesl-1S 蛋白の樹状突起棘へのとりこみは入力特異的である
Trapping of the Vesl-1S protein into dendritic spines is input-specific
岡田　大助[1](Daisuke Okada)，小澤　史子[1](Fumiko Ozawa)，井ノ口　馨[1,2](Kaoru Inokuchi)
[1]三菱化学生命科学研究所、記憶形成・精神疾患研究グループ，[2]CREST, JST

P3-b13 マウス視覚野のテレンセファリンはプラスミンにより限定分解される
Proteolytic cleavage of telencephalin by plasmin in mouse visual cortex
俣賀　宣子[1](Nobuko Mataga)，James C Poyer[1]，森　　憲作[3](Kensaku Mori)，
吉原　良浩[2,4](Yoshihiro Yoshihara)，ヘンシュ貴雄[1,4](Takao K Hensch)
[1]理研・脳センター・神経回路発達，[2]理研・脳センター・シナプス分子機構，[3]東大・医・細胞分子生理，
[4]CREST・JST

P3-b14 コルチコステロイドは海馬神経細胞のスパイン形態を急性的に変化させる
Rapid morphological changes of dendritic spines induced by corticosteroids in rat hippocampus
小松崎良将[1,2,3](Yoshimasa Komatsuzaki)，北條　泰嗣[2,3](Yasushi Hojo)，川戸　　佳[2,3](Suguru Kawato)
[1]日本大学・文理・物理生命システム科，[2]東大院・総合文化・広域科学，[3]CREST・バイオインフォマティクス・JST

P3-b15 神経因性疼痛ラット扁桃体中心核におけるシナプス可塑的変化形成過程
Time course of the neuropathic pain-evoked synaptic potentiation in the central amygdala of the rat
高橋由香里(Yukari Takahashi)，池田　　亮(Ryo Ikeda)，井村　泰子(Taiko Imura)，加藤　総夫(Fusao Kato)
東京慈恵会医科大院・総合医科研・神経生理

P3-b16 吸入麻酔薬イソフルランは *in vivo* 海馬短期シナプス可塑性を修飾する
Isoflurane bidirectionally modulates the hippocampal paired-pulse responses *in vivo*.
橘　かおり(Kaori Tachibana)，森本　裕二(Yuji Morimoto)
北大院・医・麻酔・周術期医学

P3-b17 海馬における経験依存的なスパインの可塑性
Experience-dependent spine plasticity in the hippocampus
北西　卓磨(Takuma Kitanishi)，野村　　洋(Hiroshi Nomura)，池谷　裕二(Yuji Ikegaya)，
松木　則夫(Norio Matsuki)，山田　麻紀(Maki K Yamada)
東京大院・薬・薬品作用

P3-b18 ＤＨＥＡＳの急性投与はラット海馬スライスにおけるＮＭＤＡ受容体非依存性ＬＴＰ誘導を促進する
Acute administration of the neurosteroid DHEAS primes the induction of NMDA-receptor independent LTP in rat hippocampal slices
徐　玉霞[1](Yuxia Xu)，陳　玲[2](Ling Chen)，曽我部正博[1,3,4](Masahiro Sokabe)
[1]名大医・生理，[2]中国・南京・南医大・生理，[3]SORST・細胞力覚JST，[4]生理研・分子生理

P3-b19 海馬 CA1 領域における神経ステロイドの急性作用
Neurosteroids rapidly modulate synaptic plasticity of hippocampus at CA1 region.
大石　悠貴[1](Yuuki Ooishi)，川戸　佳[2](Suguru Kawato)
[1]東京大院・理・物理，[2]東京大院・総合文化

P3-b20 海馬 - 前頭前野路のＮＭＤＡ受容体依存性シナプス可塑性における逆Ｕ字型ドーパミンＤ１受容体作用
Inverted-U dopamine D1 receptor actions in NMDA receptor-dependent plasticity at hippocampal-prefrontal synapses in vivo
瀧田　正寿(Masatoshi Takita)
産業技術総合研究所　人間福祉医工学RI

P3-b21 恐怖条件付け神経回路での不均等なシナプス可塑性
Spatiotemporal asymmetry of associative synaptic plasticity in fear conditioning pathways
辛　龍文[1](ろんぶん しん)，Vadim Bolshakov[2]
[1]独立行政法人　放射線医学総合研究所，[2]マックレーン病院、ハーバード大学

P3-b22 Perineuronal nets の神経保護作用と可塑性に関する研究
Functional significance of perineuronal nets; neuroprotection and plasticity
中村　充(Michiru Nakamura)，中野　恵子(Keiko Nakano)，宮田　清司(Seiji Miyata)
京都工芸繊維大学・応用生物学部門

P3-b23 シナプス形成期の慢性ＡＰ５処理が海馬培養細胞グルタミン酸受容体電流にもたらす効果
Effects of chronic blockade of NMDA receptors during synaptogenesis on glutamate receptor-mediated currents in cultured hippocampal neurons
加藤　健一[1,2](Kenichi Kato)，白尾　智明[2](Tomoaki Shirao)，高橋　秀人[2](Hideto Takahashi)，
関野　祐子[1,2,3](Yuko Sekino)
[1]CREST・JST，[2]群馬大学医学系研究科高次細胞機能学，[3]東京大学医科学研究所神経ネットワーク

P3-b24 KCl 刺激によるラフト構成脂質と蛋白組成の変化
Alteration of lipid and protein composition in raft under continuous KCl stimulation
天沼　崇[1](Takashi Amanuma)，大須賀　壮[2](Soh Osuka)，大嶋恵理子[2](Eriko Oshima)，
田口　良[3](Ryo Taguchi)，平林　義雄[2](Yoshio Hirabayashi)，尾崎美和子[4,5,6](Miwako Ozaki)
[1]神歯大院・歯・矯正，[2]理化学研究所，[3]名古屋市大・薬，[4]生命医療工学研究所，[5]早稲田大学　先端バイオ研究所，
[6]早稲田・オリンパス バイオサイエンス研究所

P3-b25 分散培養系における機能的シナプス結合不均一性の発生依存的変化
Developmental change of heterogeneous distribution of the functional synaptic connections in a rat hippocampal dissociated neurons
清原　藍[1,2](AI KIYOHARA)，工藤　卓[1](Suguru N. Kudoh)，田口　隆久[1](Takahisa Taguchi)
[1]独）産総研、RICE，[2]大阪大学大学院 理学研究科 化学専攻

P3-b26 アルツハイマー病治療薬 ZSET1446 がマウス海馬に与える影響
Effects of ZSET1446 on hippocampal spine density in female mice
滝口　雅人[1,2](Masahito Takiguchi)，山口　芳正[3](Yoshimasa Yamaguchi)，林　しん治[2](Shinji Hayashi)
[1]早稲田大院・先進理工・生物，[2]横浜市大・理・内分泌，[3]全薬工業株式会社

## 受容体、輸送体 2
## Receptors and Transporters 2

P3-b27 **Developmental changes of presynaptic glycine receptor expression on hippocampal mossy fiber boutons**

Hisahiko Kubota[1,2], Henrik Alle[2], Heinrich Betz[1], Jeorg R.P. Geiger[2]
[1]Department of Neurochemistry, Max-Planck Institute for Brain Research, Frankfurt am Main, Germany, [2]Independent Hertie Research Group, Max-Planck Institute for Brain Research

P3-b28 **分化させたNG108-15細胞でのセロトニンによる細胞内 $Ca^{2+}$ 濃度上昇反応の性質について**
**Characteristics of $Ca^{2+}$ responses induced by serotonin in differentiated NG108-15 cells**

市田　成志[1](Seiji Ichida)，吉田　　繁[2](Shigeru Yoshida)
[1]近畿大・薬・生化，[2]近畿大学理工学部

P3-b29 **脂質二分子膜へ再構築されたグルタミン酸受容体のＡＦＭによる構造観察**
**AFM structural observation of an ionotropic glutamate receptor reconstituted into artificial lipid bilayer**

河西奈保子[1](Nahoko Kasai)，Chandra_S Ramanujan[2]，藤本　一朗[3](Ichiro Fujimoto)，
島田　明佳[1](Akiyoshi Shimada)，John_F Ryan[2]，鳥光　慶一[1,4](Keiichi Torimitsu)
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P3-b30 **匂い分子の分子内炭化水素鎖は嗅覚レセプタS6のシグナリングのオン／オフのスイッチングをコントロールすると思われる**
**Intramolecular hydrocarbon chain of odorant may control switching between off and on signaling states of the odorant receptor S6**

佐藤　孝明(T Sato)，浜名　　洋(H Hamana)，廣野　順三(J Hirono)
（独）産総研　セルエンジニアリング研究部門

P3-b31 **AMPA型グルタミン酸受容体の動態の光学的解析**
**Optical analysis of AMPA receptor motility in cultured hippocampal neurons.**

清末　和之(Kazuyuki Kiyosue)，亀山　仁彦(Kimihiko Kameyama)
産業技術総合研究所　脳神経情報研究部門

P3-b32 **乳酸アシドーシスは培養アストロサイトにおける水チャネルアクアポリン4の発現を増加させる**
**Lactic acid increases expression of aquaporin 4 in rat astrocytes**

森島　徹朗[1](Tetsuro Morishima)，祖父江和哉[1](Kazuya Sobue)，飯田　裕子[1](Yuko Iida)，
大原健太郎[2](Kentaro Ohara)，高柳　猛彦[1](Takehiko Takayanagi)，平手　博之[1](Hiroyuki Hirate)，
青山　峰芳[2](Mineyoshi Aoyama)，有馬　　一[1](Hajime Arima)，藤田　義人[1](Yoshihito Fujita)，
津田　喬子[1](Takako Tsuda)，勝屋　弘忠[1](Hirotada Katsuya)，浅井　清文[2](Kiyofumi Asai)
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P3-b33 **マウス網膜P2X受容体の生後発達**
**Developmental change of the distribution of P2X purinoceptors in the mouse retina**

霜田　幸雄[1](Yukio Shimoda)，重松　康秀[1](Yasuhide Shigematsu)，金田　　誠[2](Makoto Kaneda)
[1]東京女子医科大学・総合研究所，[2]慶應義塾大・医・生理

P3-b34 **ホヤAMPA受容体のクローニングと機能解析**
**Cloning and characterization of an ascidian AMPA receptor**

平井　志伸[1,2](Shinobu Hirai)，西野　敦雄[3](Atsuo Nishino)，高橋　弘樹[4](Hiroki Takahashi)，
岡村　康司[3](Yasushi Okamura)，岡戸　晴生[1](Haruo Okado)
[1]東京都神経科学総合研究所・分子神経生理，[2]東京医科歯科大学・医歯学総合研究科・細胞生物学研究室，[3]自然科学研究機構・生理学研究所・岡崎バイオサイエンスセンター　時系列生命現象研究領域　神経分化研究部門，[4]基礎生物学研究所・形態形成研究部門

P3-b35 シナプス形成における Notch シグナルの役割
The role of Notch signaling in synaptogenesis
林　ゆかり[1](Yukari Hayashi)，富田　泰輔[1](Taisuke Tomita)，Raphael Kopan[2]，
岩坪　　威[1](Takeshi Iwatsubo)
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P3-b36 RGM のシグナル伝達機構
The intracellular signal of RGM
羽田　克彦(Katsuhiko Hata)，山下　俊英(Toshihide Yamashita)
千葉大・医・神経生物学

P3-b37 ラット前頭部セロトニン線維における VGLUT3 発現
The existence of two distinct types of serotonergic fibers revealed by VGLUT3 expression in the rat brain
首藤　文洋(F. Shutoh)，伊奈　鮎香(A. Ina)，吉田さちね(S. Yoshida)，今野　仁子(J. Konno)，T Wu，
久野　節二(S. Hisano)
筑波大院・人間総合・感性認知脳

P3-b38 代謝型グルタミン酸受容体および $GABA_B$ 受容体間の相互作用の異種発現系を用いた研究
Interaction between the metabotropic glutamate receptor 1$\alpha$ and the $GABA_B$ receptor studied in the heterologous expression system
松下　真一[1](Shinichi Matsushita)，立山　充博[1,2](Michihiro Tateyama)，久保　義弘[1,2,3](Yoshihiro Kubo)
[1]総合研究大学院大学　生命科学研究科　生理科学専攻，[2]生理学研究所　神経機能素子研究部門，[3]SORST, JST

P3-b39 鳩中枢神経系における小胞性グルタミン酸トランスポーターの遺伝子配列および分布
Molecular sequence and distribution of vesicular glutamate transporter mRNA in the pigeon central nervous system
Mohammad Rafiqul Islam，齋藤正一郎(Shouichiro Saito)，阿閉　泰郎(Yasuro Atoji)
岐阜大学大学院・連合獣医学研究科・獣医解剖

P3-b40 VGAT ノックアウトマウスに見られる運動機能の変容
Altered motor function in vesicular GABA transporter knockout mice
斉藤　憲史[1,2,3](Kenzi Saito)，西丸　広史[4](Hiroshi Nishimaru)，柿崎　利和[1,3](Toshikazu Kakizaki)，
中里　洋一[1](Yoichi Nakazato)，林　遼太郎[5](Ryotaro Hayashi)，福本　　学[5](Manabu Fukumoto)，
柳川右千夫[1,3](Yuchio Yanagawa)
[1]群馬大学，[2]総研大，[3]SORST，[4]産総研，[5]東北大

P3-b41 生後松果体発達過程における VGLUT の発現解析
Expression analysis of VGLUT during the postnatal maturation of rat pineal gland
吉田さちね(S Yoshida)，伊奈　鮎香(A Ina)，今野　仁子(J Konno)，小笠原清基(K Ogasawara)，T Wu，
首藤　文洋(F Shutoh)，野上　晴雄(H Nogami)，久野　節二(S Hisano)
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P3-b42 温刺激に対する培養細胞の細胞内 $Ca^{2+}$ 応答
Warm temperature-activated $Ca^{2+}$ response in cultured fibroblasts, neuronal cells and keratinocytes
小田 - 望月紀子[1](Noriko Mochizuki-Oda)，楠野　智幸[1](Tomoyuki Kusuno)，山下　晴史[1](Haruhito Yamashita)，
高森　康晴[2](Yasuharu Takamori)，山田　久夫[2](Hisao Yamada)，山田　廣成[1](Hironari Yamada)
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## 情報伝達、変換、修飾 2
## Signal Transduction and Modulation 2

P3-b43 酸化型ガレクチン―1によるマクロファージ内の mRNA 発現調節
Effect of oxidized galectin-1 on mRNA expression in macrophage
川崎　陽久[1](Haruhisa Kawasaki)，古川　幸枝[2](Yukie Kogawa)，笹栗　健一[2](Kenichi Sasaguri)，
佐藤　貞夫[2](Sadao Sato)，井上　宏子[3](Hiroko Inoue)，堀江　秀典[4](Hidenori Horie)
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P3-b44 カイニン酸誘発てんかんモデルマウスにおけるヒストン修飾の関与
Histone modifications involved in kainate-induced status epilepticus
谷浦　秀夫(Hideo Taniura)，Judy Sng，米田　幸雄(Yukio Yoneda)
金沢大・自然研・分子作用

P3-b45 AMPA 受容体のシナプス後膜への輸送に BDNF による PKA と ERK の活性化が関与する
Essential role of activation of PKA and ERK by BDNF for AMPA receptor translocation to postsynaptic sites
中田　博子[1,2](Hiroko Nakata)，中村　　俊[1](Shun Nakamura)
[1]国立精神・神経セ・神経研，[2]東京都精神研

P3-b46 合成サイクリック ADP リボースアナログによる神経腫瘍細胞内 $Ca^{2+}$ 濃度上昇の増強効果
Amplification of depolarization-induced and ryanodine-sensitive cytosolic $Ca^{2+}$ elevation by synthetic carbocyclic analogues of cyclic ADP-ribose in NG108-15 neuronal cells
橋井美奈子[1](Minako Hashii)，周東　　智[2](Satoshi Shuto)，東田　陽博[1](Haruhiro Higashida)
[1]金沢大院・医・脳細胞遺伝，[2]北海道大院・薬学研究院・創薬有機化学講座

P3-b47 エタノールとアセトアルデヒドによる NG108-15 細胞の CREB の活性化
Activation of CREB by ethanol and acetaldehyde in NG108-15 cells.
森田　淳子(Junko Morita)，井上　宏子(Hiroko Inoue)
早稲田大院・先進理工・電気・情報生命

P3-b48 p35 のリン酸化による Cdk5-p35 の活性化および膜結合性の制御
Regulation of the membrane association and kinase activity of Cdk5-p35 by phosphorylation of p35
佐藤　　巨[1](Ko Sato)，朱　　英善[1](Ying-Shan Zhu)，斎藤　太郎[1](Taro Saito)，
四本　健介[1](Kensuke Yotsumoto)，浅田　明子[1](Akiko Asada)，長谷川成人[2](Masato Hasegawa)，
久永　真市[1](Shin-ichi Hisanaga)
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P3-c01 Casein kinase 1 (CK1) による Rho GTPase の機能調節に関する解析
Characterization of novel CK1-mediated phosphorylation of SCS-binding Rho GTPases in vitro
川上　文貴(Fumitaka Kawakami)，鈴木　敢三(Kanzo Suzuki)，大槻　健蔵(Kenzo Ohtsuki)
北里大院・医療系・分子生体情報

P3-c02 ラット脳における tau protein と tau proetin 結合因子の CK1 による SCS 依存的なリン酸化の特徴
Characterrization of SCS as a potent activator for the CK1-mediated phosphorylation of tau protein and its associated proteins from rat brain
鈴木　敢三(Kanzo Suzuki)，川上　文貴(Fumitaka Kawakami)，佐々木　悠(Hisashi Sasaki)，
大槻　健蔵(Kenzo Ohtsuki)
北里大学医療系研究科・分子生体情報学

P3-c03 コンピュータシミュレーションによる神経栄養因子様活性物質、ゲニピンの標的分子の解析：神経型NO合成酵素のテトラヒドロビオプテリン結合領域とゲニピン関連化合物の相互作用
Computer simulation of target molecule of genipin with neurotrofic factor-like activity: Interaction of genipin-related compounds and tetrahydrobiopterin binding domain in neuronal NO synthase
千葉　賢三[1,2](Kenzo Chiba)，山崎眞津美[1](Matsumi Yamazaki)，鈴木　宏一[1](Hirokazu Suzuki)，
澤西　啓之[1,2](Hiroyuki Sawanishi)
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P3-c04 PC12細胞を分化誘導した時のc-fos遺伝子プロモーター活性化におけるPKAの関与
Activation of the *c-fos* promoter is modulated by PKA in PC12 cells.
杉山　　崇(Takashi Sugiyama)，鈴木　浩文(Hirobumi Suzuki)
オリンパス（株）・基礎技術部

P3-c05 小脳抑制性介在ニューロンのmGluR1興奮性応答におけるチロシンキナーゼの役割
Tyrosin kinase is involved in the mGluR1-mediated inward current in the cerebellar molecular layer interneurons
久保田英雄[1](Hideo Kubota)，廣野　守俊[2](Moritoshi Hirono)，小幡　邦彦[2](Kunihiko Obata)
[1]東京医歯大・医病・材料部，[2]理化学研究所・脳科学総合研究センター・神経回路メカニズム研究グループ

P3-c06 マウス終脳の特定の介在神経におけるPKCαおよびPKCγの細胞発現パターンの解析
Distinct cellular expression of PKCα and PKCγ in particular interneurons of the mouse telencephalon.
三浦会里子(Eriko Miura)，窪田　圭志(Yoshiyuki Kubota)，渡辺　雅彦(Masahiko Watanabe)
北大院・医・解剖発生

P3-c07 シグナル伝達経路の代数的解析法
Algebraic method for the analysis of signaling crosstalk
菊地　進一[1](Shinichi Kikuchi)，松原　嘉哉[1](Yoshiya Matsubara)，杉本　昌弘[1,2](Masahiro Sugimoto)，
岡　浩太郎[1,3](Kotaro Oka)，冨田　　勝[1](Masaru Tomita)
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P3-c08 ガンマ型およびイプシロン型ジアシルグリセロールキナーゼの神経細胞における局在について
Localization of diacylglycerol kinase γ and - ε in neurons
八月朔日泰和(Yasukazu Hozumi)，後藤　　薫(Kaoru Goto)
山形大学・医・組織細胞生物学

P3-c09 シナプス膜由来界面活性剤不溶性低比重画分の脂質組成
Lipid components in the detergent-resisitant membrane microdomain from rat synaptic plasma membrane.
前川　昌平[1](Shohei Maekawa)，松浦　大介[1](Daisuke Matsuura)，田口　勝敏[1](Katsutoshi Taguchi)，
八木澤　仁[2](Hitoshi Yagisawa)
[1]神戸大院・理・生物，[2]兵庫県立大学理学研究科生体情報

P3-c10 ラット脳シナプス膜微小領域におけるホスホリパーゼCβ1の局在とその解析
Localization of phospholipase Cβ1 on detergent-resistant membrane microdomains prepared from synaptic plasma membrane of rat brain
田口　勝敏[1](Katsutoshi Taguchi)，熊ノ郷晴子[2](Haruko Kumanogoh)，中村　　俊[2](Shun Nakamura)，
前川　昌平[1](Shohei Maekawa)
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P3-c11 線条体ニューロンにおけるspinophilin Ser94残基リン酸化調節機構：DARPP-32依存性および非依存性経路の考察
DARPP-32-dependent regulation of spinophilin Ser94 phosphorylation in neostriatal neurons of direct and indirect pathways
上松　　謙[1,2,3](Ken Uematsu)，前田　久雄[2](Hisao Maeda)，西　　昭徳[1](Akinori Nishi)
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## グリア、グリア-ニューロン相互作用 2
## Glia and Glia-Neuron Interaction 2

P3-c12 **Neurofibrillary tangles in Lafora disease: Functional link between laforin and tau proteins**
Rajat Puri[1], Kazuhiro Yamakawa[2], Ganesh Subramaniam[1]
[1]Department of Biological Sciences and Bioengineering, Indian Institute of Technology, Kanpur, India, [2]Laboratory for Neurogenetics, RIKEN Brain Science Institute, Wako-shi, Japan

P3-c13 **Modulation by brain natriuretic peptide of rectify outward K+ current on mouse Schwann cells**
Xiao-Wei Fei
School of Life Sciences, Fudan University, Shanghai, China

P3-c14 **Lysophosphatidic acid stimulates astrocyte proliferation through $LPA_1$**
Yuki Sogo[1], Shinya Shano[1], Ryutaro Moriyama[1], Jerold Chun[2], Nobuyuki Fukushima[1]
[1]Department of Life Science, Kinki University, Japan, [2]The Scripps Research Institute, Ja Lolla, CA

P3-c15 **Signaling mechanism inducing deleterious factors in microglia**
Yukihisa Yosino[1], Yoko Tohyama[1], Shinichi Kohsaka[2], Tadashi Kurihara[1], Kazuyuki Nakajima[1,2]
[1]Department of Bioengineer, University of Soka, Tokyo, Japan, [2]National Institute of Neuroscience

P3-c16 **The response of microglia and astrocytes to the infected cortical neurons induced by a coronavirus**
Wan Zhu Bai[1], Yan Chao Li[1], N Hirano[2], T Tohyama[3], T Hashikawa[1]
[1]Brain Science Institute, RIKEN, Japan, [2]Dept. of Vet. Microbiol., Iwate Univ., [3]Center for EM & Bio-Imaging Res., Iwate Med. Univ.

P3-c17 **一酸化窒素がアストロサイトからのカルシウム依存的グルタミン酸放出増加を調節する**
**Nitric oxide mediates cytokine-induced enhancement of $Ca^{2+}$-dependent Glu release from astrocytes**
井田　智章[1](Tomoaki Ida), 中村　洋一[2](Yoichi Nakamura), 小崎　俊司[2](Shunji Kozaki), 居原　秀[1](Hideshi Ihara)
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P3-c18 ***in vivo* におけるアストロサイトの自発的カルシウム活動は大脳皮質の第 1 層と第 2/3 層とで異なる**
**Spontaneous calcium activities of astrocytes *in vivo* differ between cortical layer 1 and layer 2/3**
高田　則雄(Norio Takata), 平瀬　肇(Hajime Hirase)
理研・BSI

P3-c19 **アストロサイトによる孤束核シナプス伝達物質放出の調節**
**Astrocyte-mediated regulation of synaptic glutamate release in the NTS**
井村　泰子(Taiko Imura), 長谷川奈海(Nami Hasegawa), 和光　未加(Mika Wako), 加藤　総夫(Fusao Kato)
慈恵医大　神経生理

P3-c20 **培養下のアストロサイトに発現する機能的な低温／メントール受容体**
**Functional cold/menthol receptor expressed in cultured astrocytes**
小倉　明彦[1](Akihiko Ogura), 佐野　耕介[2](Kosuke Sano), 冨永－吉野恵子[1](Keiko Tominaga-Yoshino)
[1]大阪大院・生命機能・脳神経工学, [2]大阪大・理

P3-c21 **ニューロンの刺激はミクログリアをより神経保護的に変換する**
**Neuronal stimulation leads microglia to more neurotrophic cells**
中嶋　一行[1](Kazuyuki Nakajima), 遠山　陽子[1](Yoko Tohyama), 前田　秀一[1](Shyuichi Maeda), 高坂　新一[2](Shinichi Kohsaka), 栗原　正[1](Tadashi Kurihara)
[1]創価大・工・生命情報, [2]国立精神・神経センター、神経研・代謝

P3-c22 オリゴデンドロサイトにおける膜結合型エストロゲンレセプターの局在と生理機能解析
The localization and non-genomic function of the membrane-associated estrogen receptor in oligodendrocytes
和田（平原）幸恵[1,2](Yukie Wada-Hirahara)，Wen Gao[2]，Dina_N. Arvanitis[2]，松田　賢一[1](Ken-ichi Matsuda)，河田　光博[1](Mitsuhiro Kawata)，Joan_M. Boggs[2]
[1]京都府立医科大学・解剖学教室　・生体構造科学部門，[2]Molecular Structure and Function, Hospital for Sick Children, Toronto, Canada

P3-c23 ミクログリアにおけるカタラーゼの発現誘導を介したアポトーシス抑制作用
Hydrogen peroxide at a low concentration protects BV-2 cells against hydrogen peroxide-induced apoptosis through the production of catalase.
吉岡　靖啓(Yasuhiro Yoshioka)，山室　晶子(Akiko Yamamuro)，笠井　淳司(Atsushi Kasai)，前田　定秋(Sadaaki Maeda)
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P3-c24 ミクログリアの一酸化窒素産生におけるネガティブフィードバック調節機構
Negative feedback regulation of NO production in LPS-activated microglia.
武田　存央(Nobuo Takeda)，吉岡　靖啓(Yasuhiro Yoshioka)，山室　晶子(Akiko Yamamuro)，笠井　淳司(Atsushi Kasai)，前田　定秋(Sadaaki Maeda)
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P3-c25 新生仔ラット脳幹におけるオキシトシン受容体の分布
Distribution of oxytocin receptors in the neonatal brain stem
村田　祐造(Yuzo Murata)，李　　明子(Ming-Zi Li)，増子　貞彦(Sadahiko Masuko)
佐賀大学・医・生体構造機能学

P3-c26 三叉神経節内での GABA を介した神経伝達機構
Local GABAergic system in trigeminal ganglion
早崎　　華[1](Hana Hayasaki)，相馬　義郎[2](Yoshiro Sohma)，渡辺　正仁[1](Masahito Watanabe)，大槻　勝紀[1](Yoshinori Otsuki)
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P3-c27 バーグマングリアに発現する AMPA 型グルタミン酸受容体の輸送様式
The mode of AMPA-type glutamate receptor trafficking in Bergmann glia
深谷　昌弘(Masahiro Fukaya)，渡辺　雅彦(Masahiko Watanabe)
北海道大院・医・解剖発生

P3-c28 アレキサンダー病モデルマウスの解析
Analysis of Alexander disease model mice
李　　海雄[1,2](Hae Ung Lee)，田中　謙二[2](Kenji Tanaka)，池中　一裕[1,2](Kazuhiro Ikenaka)
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P3-c29 CTAB/SDS-PAGE を用いたミエリンタンパク質の解析
Analysis of myelin proteins using CTAB/SDS-PAGE
宮城　雄大(Yudai Miyagi)，山口　宜秀(Yoshihide Yamaguchi)，馬場　広子(Hiroko Baba)
東京薬大院・薬・機能形態

P3-c30 VIP/VPAC2 システムを介した反応性アストロサイトの機能制御機構
The functional regulatory mechanism in reactive astrocytes via VIP/VPAC2 system
西本　美香[1,2](Mika Nishimoto)，古田　晶子[2](Akiko Furuta)，和田　圭司[2](Keiji Wada)
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## RNAの制御と機能、翻訳制御
## Regulation and Function of RNA, Translational Regulation

P3-c31 ジストロフィータイプ1におけるSERCA1の異状スプライシング
Dysfunction of MBNL1 involved in aberrant splicing of SERCA1 in myotonic dystrophy type 1
関屋　裕史[1](Hiroshi Sekiya)，日野真一郎[1](Shin-ichiro Hino)，中森　雅之[2](Masayuki Nakamori)，
高橋　正紀[2](Masanori P Takahashi)，今泉　和則[1](Kazunori Imaizumi)
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P3-c32 脳特異的タンパク質メチル化酵素PRMT8の解析
Analysis of a brain specific protein arginine N-methyltransferase 8 (PRMT8) in the mouse brain
幸坂　葵(Aoi Kousaka)，種子田　艶(Tsuya Taneda)，小山　佳久(Yoshihisa Koyama)，
宮田　信吾(Shingo Miyata)，森　泰丈(Yasutake Mori)，遠山　正彌(Masaya Tohyama)
大阪大院・医・神経機能形態

P3-c33 Dynamic localization of eIF4E mRNA in dendrites of cultured rat hippocampal neurons
Il Soo Moon
Department of Anatomy, College of Medicine, Dongguk University, Gyeongju, Korea

## 神経幹・前駆細胞と細胞分化，移植3
## Neural Stem/Progenitor Cells and Cellular Differentiation, Transplantation 3

P3-c34 Cystaitn Cによるグリア細胞分化制御
Cystatin C regulates glial differentiation
成瀬　雅衣(Masae Naruse)，長谷川明子(Akiko Hasegawa)，竹林　浩秀(Hirohide Takebayashi)，
等　誠司(Seiji Hitoshi)，池中　一裕(Kazuhiro Ikenaka)
自然科学研究機構　生理学研究所　分子神経生理

P3-c35 持続的磁場曝露に伴う神経系前駆細胞の増殖能変動
Regulation of proliferation by sustained magnetism in cultured neural progenitor cells
石岡　佑吉(Yukichi Ishioka)，神戸　悠輝(Yuki Kambe)，中道　範隆(Noritaka Nakamichi)，
谷浦　秀夫(Hideo Taniura)，米田　幸雄(Yukio Yoneda)
金沢大院・自然科学（薬）・薬物学

P3-c36 Sox6の神経幹・前駆細胞における発現および機能解析
Sox6 is expressed in mouse embryonic neural stem/progenitor cells (NSPCs) and contributes to their maintenance and/or survival
大多　茂樹[1](Shigeki Ohta)，岡野　栄之[2](Hideyuki Okano)，河瀬　斌[3](Takeshi Kawase)，
戸田　正博[1,3](Masahiro Toda)
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P3-c37 マウス神経発生におけるLGN遺伝子の役割
Roles of the LGN gene in the mouse neural development
塩井　剛[1,2](Go Shioi)，今野大治郎[1,2](Daijiro Konno)，下向　敦範[1,2](Atsunori Shitamukai)，
森　麻子[1](Asako Mori)，松崎　文雄[1,2](Fumio Matsuzaki)
[1]理研・CDB・非対称細胞分裂，[2]JST, CREST

P3-c38 マウス神経系前駆細胞におけるc AMPシグナルによる増殖の制御
Regulation by cAMP signals of proliferative activity in murine neural progenitor cells
Juliet_O. Makanga，吉田　浩平(Kohei Yoshida)，福井　正樹(Masaki Fukui)，
中道　範隆(Noritaka Nakamichi)，米田　幸雄(Yukio Yoneda)
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P3-c39 unc5h4 および netrin-4 は培養下においてマウス皮質第４層ニューロンの生存および突起成長を制御する
Unc5h4 and netrin-4 regulate survival and neurite outgrowth of mouse neocortical layer IV neurons in culture
竹本　誠(Makoto Takemoto)，服部　祐紀(Yuki Hattori)，山本　亘彦(Nobuhiko Yamamoto)
大阪大院・生命機能

P3-c40 *Pax6* ヘテロ接合変異ラットにおける神経分化の亢進
Premature neurogenesis in the *Pax6* heterozygous mutant cerebral cortex
梅田　稔子[1](Toshiko Umeda)，野村　真[1](Tadashi Nomura)，大隅　典子[1,2](Noriko Osumi)
[1]東北大院・医・形態形成解析分野，[2]CREST, JST

P3-c41 低分子量 G タンパク質 RhoG によるマウス大脳皮質の神経前駆細胞の増殖制御
RhoG regulates neural progenitor cell proliferation in mouse cerebral cortex
藤本　聡志(Satoshi Fujimoto)，根岸　学(Manabu Negishi)，加藤　裕教(Hironori Katoh)
京都大院・生命科学・生体システム学

P3-c42 登上線維ニューロンの発生における *Ptf1a* の役割
Origin of climbing fiber neurons and their developmental dependence on *Ptf1a*
山田　真弓[1](Mayumi Yamada)，寺尾　真美[1](Mami Terao)，寺島　俊雄[2](Toshio Terashima)，
藤山　知之[1](Tomoyuki Fujiyama)，川口　義弥[3](Yoshiya Kawaguchi)，鍋島　陽一[1](Yo-ichi Nabeshima)，
星野　幹雄[1](Mikio Hoshino)
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P3-c43 GABA シグナルによる神経系前駆細胞の増殖能と分化能の制御
Modulation of proliferation and differentiation by GABAergic signals in murine neural progenitor cells
小澤　秀介(Shusuke Ozawa)，福井　正樹(Masaki Fukui)，吉田　浩平(Kouhei Yoshida)，
中道　範隆(Noritaka Nakamichi)，米田　幸雄(Yukio Yoneda)
金沢大院・自然科学・薬物

P3-c44 神経芽腫細胞株 Neuro2a における N 末端則関連遺伝子 Ntan1 の強制発現
Transient overexpression of Ntan1 gene relevant to N-end rule in murine neuroblastoma Neuro2a cell line
亀島　優太(Yuta Kameshima)，谷浦　秀夫(Hideo Taniura)，木村　仁美(Satomi Kimura)，
中道　範隆(Noritaka Nakamichi)，米田　幸雄(Yukio Yoneda)
金沢大院・自然科学・薬物

P3-c45 Runx1 は脊髄神経節ニューロンの適切な細胞分化に必要である
Runx1 is required for the proper cell fate specification of dorsal root ganglion neuron
吉川　雅朗(Masaaki Yoshikawa)，先崎　浩次(Kouji Senzaki)，横溝　智雅(Tomomasa Yokomizo)，
高橋　智(Satoru Takahashi)，尾崎　繁(Shigeru Ozaki)，志賀　隆(Takashi Shiga)
筑波大学大学院 人間総合科学研究科

P3-c46 p27Kip1 に対する RNA 干渉法を用いた蝸牛支持細胞の分裂・増殖誘導 - 器官培養系での検討 -
RNA interference for p27kip1 induce supporting cell proliferation in organotypic cultures of postnatal mammalian cochlea
小野　和也(Kazuya Ono)，小島　憲(Ken Kojima)，中川　隆之(Takayuki Nakagawa)，
松本　昌宏(Masahiro Matsumoto)，伊藤　壽一(Juichi Ito)
京都大学大学院医学研究科・耳鼻咽喉科・頭部外科

P3-c47 神経上皮細胞間ギャップ結合の消失と神経分化
Decoupling of gap junctions between neuroepithelial cells for neuronal differentiation
山下　勝幸(Masayuki Yamashita)
奈良医大・第一生理

P3-c48 ヒト神経前駆細胞トランスクリプトームにおける血清の影響
A drastic change in gene expression profile of human neural progenitor cells under serum-free and serum-containing culture conditions
佐藤　準一(Jun-Ichi Satoh)，三澤多真子(Tamako Misawa)，尾林　信哉(Nobuya Obayashi)，
住吉　健太(Kenta Sumiyoshi)，大隅貴美子(Kimiko Oosumi)，天竺桂弘子(Hiroko Tabunoki)
明治薬科大学・薬・バイオインフォマティクス

P3-d01 マウス神経幹細胞生存・増殖機構における p38 MAP kinase の役割
Roles of p38 MAP kinase in mouse neural progenitor cells survival and proliferation
浜之上　誠[1](Makoto Hamanoue)，佐藤健一郎[2](Kenichiro Sato)，高松　研[1](Ken Takamatsu)
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## 細胞移動、突起伸展、回路網形成 3
## Migration, Axonal and Dendritic Outgrowth, Network Formation 3

P3-d02 系統的免疫染色及び siRNA に基づく成長円錐のマーカー分子群の同定
Systematic immunostaining and RNAi experiments for finding the novel molecular marker candidates of the growth cone.
野住　素広[1,2](Motohiro Nozumi)，仁木加寿子[1](Kazuko Takahashi-Niki)，五十嵐道弘[1,2](Michihiro Igarashi)
[1]新潟大・医歯学・分子機能学，[2]同・超域研究機構

P3-d03 Cxcl12/Cxcr4 ケモカインシグナルは嗅覚プラコード形成と嗅神経投射に必要である
Cxcl12/Cxcr4 chemokine signaling is required for placode assembly and sensory axon pathfinding in the zebrafish olfactory system
宮坂　信彦[1,2](Nobuhiko Miyasaka)，Holger Knaut[3]，吉原　良浩[1,2](Yoshihiro Yoshihara)
[1]理研・脳センター・シナプス分子機構，[2]CREST, JST，[3]Dept. of Mol. and Cell. Biol., Harvard Univ., USA

P3-d04 菱脳運動神経の回路形成における Rho/Rho-kinase シグナル伝達系の役割
Key role of Rho/Rho-kinase signaling in hindbrain motor axon patterning
小林　憲太[1](Kenta Kobayashi)，増田　知之[2](Tomoyuki Masuda)，高橋　将文[3](Masanori Takahashi)，
宮崎　純一[4](Jun-ichi Miyazaki)，中川　匡弘[5](Masahiro Nakagawa)，八木沼洋行[2](Hiroyuki Yaginuma)，
大隅　典子[3](Noriko Osumi)，貝淵　弘三[6](Kozo Kaibuchi)，小林　和人[1](Kazuto Kobayashi)
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P3-d05 リーリンとアルカリフォスファターゼの融合タンパク質を用いた、リーリン受容体の機能的発現部位の決定
Localization of functional reelin receptors as revealed by alkaline phosphatase-reelin fusion protein
内田　貴之[1](Takayuki Uchida)，馬場　敦[1](Atsushi Baba)，宮田　卓樹[2](Takaki Miyata)，
仲嶋　一範[3](Kazunori Nakajima)，服部　光治[1](Mitsuharu Hattori)
[1]名古屋市立大院・薬・病態生化，[2]名大・医・解剖，[3]慶応大・医・解剖

P3-d06 大脳皮質錐体細胞の樹状突起形成とシナプス形成におけるセロトニン 1A および 2A 受容体の作用
Roles of $5\text{-HT}_{1A}$ and $5\text{-HT}_{2A}$ receptors in the dendrite formation and synaptogenesis of cerebral cortex pyramidal neurons
金丸千沙子(Chisako Kanamaru)，先崎　浩次(Kouji Senzaki)，志賀　隆(Takashi Shiga)
筑波大学院・人間総合科学研究科

P3-d07 R-Ras は ILK-GSK-3β 経路を介して軸索決定を制御する
R-Ras controls axon specification upstream of GSK-3β through integrin-linked kinase
生沼　泉(Izumi Oinuma)，加藤　裕教(Hironori Katoh)，根岸　学(Manabu Negishi)
京都大学院・生命・生体システム学

P3-d08 Cdc42 活性化制御因子 Zizimin1/Dock9 による海馬神経細胞の樹状突起形成制御
The Cdc42 activator Zizimin1/Dock9 regulates dendritic development in hippocampal neurons
倉本 和也(Kazuya Kuramoto), 根岸 学(Manabu Negishi), 加藤 裕教(Hironori Katoh)
京都大院・生命科学・生体システム学

P3-d09 脳形成に必須の分泌タンパク質リーリンの分泌と機能における、C 末端領域の機能解析
The exremly conserved C-terminal region of Reelin is not nsecessary for secretion but is required for efficient activation of downstream signaling
中野 良美[1](Yoshimi Nakano), 河野 孝夫[1](Takao Kohno), 日比 輝正[1](Terumasa Hibi),
河野 志織[1](Shiori Kohno), 馬場 敦[1](Atsushi Baba), 御子柴克彦[2](Katsuhiko Mikoshiba),
仲嶋 一範[3](Kazunori Nakajima), 服部 光治[1](Mitsuharu Hattori)
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P3-d10 神経細胞における受容体型タンパクチロシンホスファターゼβアイソフォームの機能
Different functions of two RPTPβ isoforms in neurons
横山 彰太[1](Showta Yokoyama), 前田 信明[2](Nobuaki Maeda), 宮田 清司[1](Seiji Miyata)
[1]京都工芸繊維大学・応用生物学, [2]東京都神経研・分子発生生物学

P3-d11 tPA-plasmin システムによる細胞外マトリクス分解とバソプレッシン神経の構造的可塑性
tPA-plasmin-dependent degradation of extracellular matrix causes structural plasticity in vasopressin neurons
宮田 清司[1](Seiji Miyata), 今村 友樹[1](Youki Imamura), 松尾 理[2](Osamu Matsuo)
[1]京都工芸繊維大学・応用生物学部門, [2]近畿大学医学部第2生理学

P3-d12 Crk is required for Reelin-mediated hippocampal dendrite outgrowth
Tohru Matsuki, Albéna Pramatarova, Brian_W. Howell
Neurogenetics Branch, NINDS, Bethesda, USA

P3-d13 発生期大脳皮質の皮質板上層部で優位に発現する分子の検索と機能解析
Screening and functional analyses of the molecules that are preferentially expressed in the upper cortical plate of the developing cerebral cortex
刀川夏詩子[1](Kashiko Tachikawa), 佐々木慎二[1](Shinji Sasaki), 前田 卓哉[1](Takuya Maeda),
仲嶋 一範[1,2](Kazunori Nakajima)
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P3-d14 音響インピーダンス顕微鏡を用いた、発達期小脳のL型カルシウムチャネル分布の観察
Observation on developing cerebellar cortex using acoustic impedance microscope and characterization of L-type Calcium channel distribution
正木 志保[1](Shiho Masaki), 福士絵梨奈[1](Erina Fukushi), 森島 寿貴[1](Toshitaka Morishima),
松田 厚範[1](Atsunori Matsuda), 小林 和人[2](Kazuto Kobayashi), 穂積 直裕[3](Naohiro Hozumi),
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P3-d15 DRG 感覚神経の分化と生存における転写制御因子 NZF ファミリーの役割
Neural zinc finger (NZF) transcription factors regulate development and survival of dorsal root ganglia neurons
亀山 俊樹(Toshiki Kameyama), 松下 文雄(Fumio Matsushita), 丸野内 棣(Tohru Marunouchi),
角川 裕造(Yuzo Kadokawa)
藤田保健衛生大・総医研・応用細胞

P3-d16 CRMP5 ノックアウトマウスにおける表現型解析
Phenotype analysis of *crmp5*-deficient mice
宮崎 真里[1](Mari Miyazaki), 宇賀神 梢[1](Kozue Ugajin), 山下 直也[1](Naoya Yamashita),
中村 史雄[1](Fumio Nakamura), Pappachan Kolattukudy[2], 五嶋 良郎[1](Yoshio Goshima)
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P3-d17 CRMP2 の S522 のリン酸化は樹上突起の形成に関与する
Phosphorylation of CRMP2 at S522 is required for proper dendritic patterning of layer V cortical neurons in vivo
内田　穣[1](Yutaka Uchida), 大島登志男[2](Toshio Ohshima), 山下　直也[1](Naoya Yamashita),
中村　史雄[1](Fumio Nakamura), Papachan Kolattukudy[3], Jerome Honnorat[4],
五嶋　良郎[1,5](Yoshio Goshima)
[1]横浜市大院・医・分子薬理神経生物学, [2]理研・脳センター・発生神経生物, [3]Univ. Cent. Florida, [4]INSERM U433, [5]CREST

P3-d18 小脳前核神経細胞移動に於ける Calmodulin 1 の役割
The role of *calmodulin 1* in the migration of precerebellar neurons
小林　裕明(Hiroaki Kobayashi), 皿谷　俊祐(Shunsuke Saragai), 内藤　篤志(Atsushi Naito),
川内　大輔(Daisuke Kawauchi)
大阪大院・生命機能

P3-d19 がん環境下における神経突起の伸展抑制について
Inhibition of axonal outgrowth in the tumor environment: Involvement of class 3 semaphorins
Vachkov Ivan[1,2], 高倉　伸幸[2,3](Nobuyuki Takakura), 加藤　聖[1](Satoru Kato)
[1]金沢大院・医・脳情報分子, [2]金沢大・がん研・細胞分化, [3]大阪大・微生物病研・情報伝達

P3-d20 移動中のグリオブラストーマ細胞における $[Ca^{2+}]_i$ と $[Cl^-]_i$ の変動解析
Measurements of changes in $[Ca^{2+}]_i$ and $[Cl^-]_i$ during glioblastoma cell movement
熊田　竜郎(Tatsuro Kumada), 福田　敦夫(Atsuo Fukuda)
浜松医大・医・生理学第一

P3-d21 α-1 アドレナリン受容体アゴニストの神経前駆細胞の細胞遊走に対する影響
Induction of cell migration of neural progenitor cells *in vitro* by α-1 adrenergic receptor stimulation
平本　豪志(Takeshi Hiramoto), 渡辺　康裕(Yasuhiro Watanabe)
防衛医大・薬理

P3-d22 成長円錐におけるタンパク質翻訳因子 eEF2 を介したカルシウム依存性の突起伸長制御
Calcium dependent-regulation of neurite outgrowth mediated by a protein translational factor eEF2 within nerve growth cones
飯塚　朗[1](Akira Iizuka), 池谷　真澄[1](Masumi Iketani), 仙石くみこ[1](Kumiko Sengoku),
中村　史雄[1](Fumio Nakamura), 松下　正之[2](Masayuki Matsushita), Angus Nairn[3],
五嶋　良郎[1,4](Yoshio Goshima), 竹居光太郎[1,4](Kohtaro Takei)
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P3-d23 成長円錐内のカルシウム結合タンパク質 NCS-1 は神経突起伸長と成長円錐形態を独立に制御する
Neuronal calcium sensor-1 in nerve growth cones independently regulates both the neurite outgrowth and growth cone morphology
池谷　真澄[1](Masumi Iketani), 今泉　千尋[1](Chihiro Imaizumi), Andreas Jeromin[2],
中村　史雄[1](Fumio Nakamura), 御子柴克彦[3,4](Katsuhiko Mikoshiba), 五嶋　良郎[1,5](Yoshio Goshima),
竹居光太郎[1,5](Kohtaro Takei)
[1]横浜市大院・医・分子薬理神経, [2]テキサス大・学習&記憶センター, [3]東大・医科研・脳神経, [4]理研・脳総研・発生神経, [5]科技振機構・CREST

P3-d24 運動系神経回路形成の分子機構：＂ミッフィー＂変異マウスに学ぶ EphA4 シグナル伝達
How does EphrinB3/EphA4 signaling regulate motor-circuit formation?
岩里　琢治[1](Takuji Iwasato), 加藤　裕教[2](Hironori Katoh), 西丸　広史[3](Hiroshi Nishimaru),
石川　幸雄[2](Yukio Ishikawa), 井上　治久[2](Haruhisa Inoue), 斉藤　芳和[1](YM Saito), 安藤れい子[1](R Ando),
岩間　瑞穂[1](M Iwama), 高橋　良輔[2](Ryosuke Takahashi), 根岸　学[2](Manabu Negishi),
糸原　重美[1](Shigeyoshi Itohara)
[1]理化学研究所・脳科学総合研究センター, [2]京都大学, [3]産総研

P3-d25 CRMP1 は *in vivo* において Semaphorin3A シグナルを媒介してスパイン成熟を調節する
CRMP1 regulates spine maturation through mediating Semaphorin3A signaling *in vivo*
山下　直也[1](Naoya Yamashita)，森田　麻[1](Asa Morita)，内田　穣[1](Yutaka Uchida)，
大島登志男[2](Toshio Ohshima)，中村　史雄[1](Fumio Nakamura)，Jerome Honnorat[3]，
Pappachan Kolattukudy[4]，五嶋　良郎[1](Yoshio Goshima)
[1]横浜市大院・医・分子薬理神経生物学，[2]理研・脳センター，[3]INSERM U433，[4]Biomol. Sci., Univ. of Central Florida

## 細胞接着因子
## Cell Adhesion Molecules

P3-d26 IgLON 接着因子の海馬神経のシナプス形成における役割
IgLON adhesion molecules participate in synapse formation of hippocampal neurons
橋本　隆[1](Takasi Hashimoto)，前川　昌平[2](Shohei Maekawa)，宮田　清司[1](Seiji Miyata)
[1]京都工芸繊維大学・応用生物学部門，[2]神戸大学自然科学研究科

P3-d27 マウス生後脳におけるカドヘリン6遺伝子発現調節機構
Regulatory mechanisms of Cdh6 gene expression in the postnatal mouse brain
井上　高良(Takayoshi Inoue)，井上由紀子(Yukiko Inoue)，浅見　淳子(Junko Asami)，
泉　仁美(Hitomi Izumi)，中村　俊(Shun Nakamura)
精神神経セ、神経研

P3-d28 マウス海馬神経細胞におけるプロトカドヘリン-αの局在解析
Localization of Protocadherin-α protein in the mouse hippocampal neuron.
岡山　厚(Atsushi Okayama)，金子　涼輔(Ryosuke Kaneko)，八木　健(Takeshi Yagi)
大阪大学　大学院生命機能研究科

P3-d29 発生期大脳皮質における protocadherin9 の層特異的発現と細胞内局在
Lamina-specific expression and subcellular localization of protocadherin9 in the developing cortex
松居亜寿香(Asuka Matsui)，吉岡　真友(Mayu Yoshioka)，山本　亘彦(Nobuhiko Yamamoto)
大阪大院・生命機能

P3-d30 免疫グロブリンスーパーファミリーに属する NEPH ファミリー分子群の後脳における発現
Expression of NEPH family of immunoglobulin superfamily in the hindbrain
西田　和彦[1](Kazuhiko Nishida)，竹内　春樹[2](Haruki Takeuchi)，坂野　仁[2](Hitoshi Sakano)，
村上富士夫[1](Fujio Murakami)
[1]大阪大・生命機能，[2]東京大院・理・生物化学

P3-d31 Protocadherin-αファミリーは感覚運動ゲーティングと恐怖条件付け学習を制御する
Protocadherin-α family is required for sensorimotor gating and spatial learning
福田　絵美[1](Emi Fukuda)，濱田　俊[2](Shun Hamada)，平林　敬浩[1](Takahiro Hirabayashi)，
八木　健[1](Takeshi Yagi)
[1]大阪大院・生命機能，[2]福岡女子大・人間環境

P3-d32 マウス嗅神経軸索投射におけるプロトカドヘリンαファミリーの役割
Protocadherin-α family is required for early establishment and maintenance of the olfactory axonal projection
長谷川園子[1](Sonoko Hasegawa)，濱田　俊[2](Shun Hamada)，雲出　佑[1,3](You Kumode)，
平林　敬浩[1](Takahiro Hirabayashi)，Peter Mombaerts[3]，八木　健[1](Takeshi Yagi)
[1]大阪大院・生命機能，[2]福岡女子大，[3]ロックフェラー大

P3-d33 末梢神経損傷後の後根神経節および脊髄後角における細胞間接着因子 CHL1 の発現変化
Alteration of the cell adhesion molecule CHL1 expression in a specific subset of primary afferent neurons and dorsal horn following nerve injury
山中　博樹(Hiroki Yamanaka)，大久保正道(Masamichi Okubo)，野口　光一(Koichi Noguchi)
兵庫医科大学　解剖学第二講座

P3-d34 脳に特異的に発現するコンドロイチン硫酸プロテオグリカン、ニューログリカンCのスプライシングバリアントについて
The fifth splice variant of neuroglycan C, a CNS-specific transmembrane chondroitin sulfate proteoglycan
青野　幸子(Sachiko Aono)，鈴木　敦史(Atsushi Suzuki)，時田　義人(Yoshihito Tokita)，
中西　圭子(Keiko Nakanishi)，伊田みちる(Michiru Ida)，松井ふみ子(Fumiko Matsui)，
大平　敦彦(Atsuhiko Oohira)
愛知県コロニー・研究所・周生期

P3-d35 シーターバースト刺激により誘導される細胞接着分子L1の動態
Molecular dynamics of cell adhesion molecule L1 induced by theta burst stimulation in rat hippocampus
相川　義勝(Yoshikatsu Aikawa)，渡邊　正知(Masatomo Watanabe)，伊藤　康一(Kouichi Itoh)
徳島文理大学・香川薬学部・分子薬理学

P3-d36 細胞接着分子の後シナプス部局在の in vivo 可視化
Postsynaptic accumulation of cell adhesion molecules imaged in vivo
高坂　洋史[1](Hiroshi Kohsaka)，高須　悦子[1](Etsuko Takasu)，能瀬　聡直[1,2](Akinao Nose)
[1]東京大院・理・物理，[2]東京大院・新領域・複雑理工

P3-d37 発達期ラット孤束核におけるニューロカンの発現
Expression of neurocan in the rat nucleus of tractus solitarius during postnatal development
根岸　義勝(Yoshikatsu Negishi)，河合　良訓(Yoshinori Kawai)
慈恵医大院・医・解剖1

P3-d38 小脳神経回路におけるプロトカドヘリンアルファのアイソフォーム発現と機能解析
Protocadherin-alpha family in the olivo-cerebellar system; their isoform expression and loss of function analysis
雲出　佑[1](You Kumode)，濱田　俊[1,2](Shun Hamada)，木津川尚史[1](Takashi Kitsukawa)，
橋本　浩一[1](Kouichi Hashimoto)，宮崎　太輔[3](Taisuke Miyazaki)，狩野　方伸[1](Masanobu Kano)，
渡辺　雅彦[3](Masahiko Watanabe)，八木　健[1](Takeshi Yagi)
[1]大阪大院・生命機能・時空生物，[2]福岡女子大学，[3]北海道大学医学部

P3-d39 ラット感覚神経路のグリア系細胞におけるギセリンの発現
Expression of gicerin in glial cells of the rat sensory nervous pathway
近藤ゆき子[1](Yukiko Kondo)，立川　英一[1](Eiichi Tachikawa)，水間　謙三[1](Kenzo Mizuma)，
遠山稿二郎[2](Koujiro Tohyama)，平　英一[1](Eiichi Taira)
[1]岩手医大・医・薬理，[2]岩手医大・医・バイオイメージングセンター

## 神経細胞死、アポトーシス2
## Neuronal Death and Apoptosis 2

P3-d40 グルタミン酸毒性に対するAMPAの保護効果：PI3K/Akt及びGSK3,caspase-3の関与
AMPA protects cultured neurons against glutamate cytotoxicity via activation of phosphatidylinositol-3 kinase /Akt and inactivation of GSK3$\beta$ and caspase-3
西本　高明(Takaaki Nishimoto)，木原　武士(Takeshi Kihara)，新留　徹広(Tetsuhiro Niidome)，
杉本　八郎(Hachiro Sugimoto)
京都大院・薬・創薬神経科学

P3-d41 ラット神経幹細胞におけるトリブチルスズの影響
Effects of tributyltin on rat neural stem cells
鈴木　純子(Junko Suzuki)，石堂　正美(Masami Ishido)
国立環境研究所

P3-d42 プロサイモシンα1はネクローシスを神経栄養因子による保護可能なアポトーシスへ細胞死スイッチする
Prothymosin-alpha1 switches the neuronal necrosis to neurotrophin-reversible apoptosis
高濱　和弘(Kazuhiro Takahama)，藤田　亮介(Ryousuke Fujita)，植田　弘師(Hiroshi Ueda)
長崎大・院医歯薬学総合・分子薬理

P3-d43 プロサイモシンα1によるGLUT表在化促進を介した神経性ネクローシス保護機構
Prothymosin-alpha1 prevents neuronal necrosis by reversing the decreased membrane localization of GLUT
藤田　亮介(Ryousuke Fujita)，植田　弘師(Hiroshi Ueda)
長崎大・院医歯薬学総合・分子薬理

P3-d44 NG108-15細胞におけるスタウロスポリンで誘発されるアポトーシスの解析
Staurosporine-induced apoptosis in NG108-15 cells.
鍬形　友輔(Yusuke Kuwagata)，井上　宏子(Hiroko Inoue)
早稲田大院・先進理工・電気・情報生命

P3-d45 ゲニピンの神経栄養効果：Neuro2a細胞におけるtunicamycin誘引細胞毒性への神経防護作用
Neurotrophic effect of genipin: Neuroprotective action on tunicamycin-induced cytotoxicity in Neuro2a cells
田中　聖之[1,2](Masayuki Tanaka)，山崎眞津美[1](Matsumi Yamazaki)，千葉　賢三[1,2](Kenzo Chiba)
[1]北陸大学・薬学部・生化学，[2]北陸大・フロンティア

P3-d46 グルタミン酸誘発性神経細胞死におけるRNA分解の関与の可能性
Possible involvement of RNA degradation in glutamate-induced neuronal death
杉山　千絵[1,2](Chie Sugiyama)，倉本　展行[1](Nobuyuki Kuramoto)，米田　幸雄[2](Yukio Yoneda)，
荻田喜代一[1](Kiyokazu Ogita)
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P3-d47 MAGのシグナル伝達におけるRap1の関与
Rap1 is involved in the signal transduction of myelin-associated glycoprotein
谷口　順子[1](Junko Taniguchi)，藤谷　昌司[1,2,3](Masashi Fujitani)，遠藤　光晴[1](Mitsuharu Endo)，
久保　武一[1](Takekazu Kubo)，藤谷　真弓[1](Mayumi Fujitani)，羽田　克彦[1](Katsuhiko Hata)，
山口　淳[1](Atsushi Yamaguchi)，Freda D Miller[2,3]，David R Kaplan[2,3]，山下　俊英[1](Toshihide Yamashita)
[1]千葉大院・医・神経生物，[2]Hospital for Sick Children，[3]University of Toronto

P3-d48 TRPV1はバニロイドや低pHにより惹起される培養大脳皮質神経細胞死を媒介する
TRPV1 mediates vanilloids and low pH-induced neurotoxicity in rat cortical cultures
白川　久志(Hisashi Shirakawa)，山岡とも子(Tomoko Yamaoka)，三平　和明(Kazuaki Sanpei)，
中川　貴之(Takayuki Nakagawa)，金子　周司(Shuji Kaneko)
京都大院・薬・生体機能解析

P3-e01 神経型グルタミン酸トランスポーターは、システイントランスポーターとして機能している
Neuronal glutamate transporter EAAT3 functions as cysteine transporter in neurons
氷見　敏行(Toshiyuki Himi)，川口真以子(Maiko Kawaguchi)
武蔵野大・薬・安全性

P3-e02 ニュータイプの小胞体ストレスセンサーBBF2H7は、傷害を受けた神経細胞で発現する
A new type of ER stress transducer, BBF2H7 expresses in injured neurons
近藤　慎一[1](Shinichi Kondo)，原　英彰[2](Hideaki Hara)，今泉　和則[1](Kazunori Imaizumi)
[1]宮崎大・医・解剖分子細胞生物，[2]岐阜薬科大・生体機能分子

P3-e03 神経細胞内TAGE (toxic AGEs) 形成と神経障害に及ぼす影響
Intracellular TAGE (toxic AGEs) trigger neuronal degeneration
佐藤　隆[1](Takashi Sato)，下垣内徳子[1](Noriko Shimogaito)，瀧野　純一[1](Jun-ichi Takino)，
山岸　昌一[2](Sho-ichi Yamagishi)，竹内　正義[1](Masayoshi Takeuchi)
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P3-e04 カルノシン酸誘とカルノソールによる神経細胞保護作用
Carnosic acid and carnosol as nuroprotective electrophilic compounds
泉　正訓[1](Masanori Izumi)，佐藤　拓己[1,2](Takumi Satoh)，犬飼　有紀[1](Yuki Inukai)，
堤　庸昴[1](Yasutaka Tutumi)，中山　直人[1](Naoto Nakayama)，小坂　邦夫[2](Kunio Kosaka)，
伊藤　健[3](Ken Itoh)
[1]岩手大・工・福祉システム，[2]長瀬産業研究開発センター，[3]弘前大・医・高度先進

P3-e05 カルノシン酸のKeap1/Nrf2経路を介した脳保護作用
A new aspect of carnosic acid as a neuroprotective electrophilic compound: activation of the Keap1/Nrf2 pathway
佐藤　拓己[1](Takumi Satoh)，小坂　邦夫[2](Kunio Kosaka)，伊東　健[3](Ken Itoh)
[1]岩手大学・工・福祉システム，[2]長瀬産業研究開発センター，[3]弘前大・医・高度先進

P3-e06 グルコース枯渇／再還流誘発性神経細胞死におけるNADPH oxidaseの関与
NADPH oxidase is not directly involved in neuronal death induced by hypoglycemia-glucose reperfusion
長澤　一樹(Kazuki Nagasawa)，角田　大智(Taichi Kakuda)，藤本　貞毅(Sadaki Fujimoto)
京都薬大・衛生化学

## 成体ニューロン新生
## Adult Neurogenesis

P3-e07 成体海馬におけるGFAP陽性神経前駆細胞のニューロン分化機構の解析
Analysis of neuronal differentiation of GFAP-expressing neural precursors in the adult mice hippocampus
劉　印明[1](Yinming Liu)，塩田　清二[2](Seiji Shioda)，石　龍徳[1](Tatsunori Seki)
[1]順天堂大學・医・解剖二，[2]昭和大学医学部解剖一

P3-e08 海馬での神経新生に関わる脳障害による顆粒下層での分子メカニズムの変化
Injury-related molecular profiling of the subgranular zone in young and aged brain, relative to neurogenesis
青山　峰芳[1,2](Mineyoshi Aoyama)，Sherry Grissom[4]，Julia Gohlke[3]，浅井　清文[1](Kiyofumi Asai)，
G_Jean Harry[2]
[1]名古屋市立大院・医・分子神経生物学，[2]Lab of Neurobiology, NIEHS,NIH,USA，[3]Lab of Mol Toxicology, NIEHS,NIH,USA，[4]Microarray Group, NIEHS,NIH,USA

P3-e09 ROCKはβ-catenin/TCF pathwayを介してSH-SY5Y細胞の細胞周期を調節する
Rho-associated coiled-coil kinase (ROCK) regulates cell cycle of SH-SY5Y cell through β-catenin/TCF pathway
朴　秀賢(Shuken Boku)，中川　伸(Shin Nakagawa)，
鈴木　克治(Katsuji Suzuki)，増井　拓哉(Takuya Masui)，木原　舞(Mai Kihara)，井上　猛(Takeshi Inoue)，
小山　司(Tsukasa Koyama)
北大院・医・精神医学

P3-e10 幼若期長期隔離飼育マウスの神経新生抑制と行動発達異常
Social isolation rearing impairs neurogenesis and behavioral development
衣斐　大祐(Daisuke Ibi)，田熊　一敞(Kazuhiro Takuma)，小池　宏幸(Hiroyuki Koike)，
溝口　博之(Hiroyuki Mizoguchi)，山田　清文(Kiyofumi Yamada)
金沢大院・自然科学・薬物治療

P3-e11 脳内自己刺激が成体ネズミの細胞新生に及ぼす影響
Intracranial self-stimulation enhances neurogenesis in the rodent brain
高橋　太郎[1](Taro Takahashi)，Yuyan Zhu[1]，清水-岡部千草[2](Chigusa Shimizu-Okabe)，
畑　敏道[1](Toshimichi Hata)，中原大一郎[1](Daiichiro Nakahara)
[1]浜松医科大・医・心理，[2]徳島文理大・薬・薬理

P3-e12 マウス嗅球顆粒細胞サブタイプ除去後の新生顆粒細胞の補償的組み込み
Increased incorporation of ablated subtype of newborn granule cells in the mouse olfactory bulb
村田 航志[1](Koshi Murata), 中西 重忠[2](Shigetada Nakanishi), 小林 和人[3](Kazuto Kobayashi),
森 憲作[1](Kensaku Mori), 山口 正洋[1](Masahiro Yamaguchi)
[1]東京大院・医・細胞分子生理, [2]大阪バイオサイエンス研究所 システムズ生物学部門, [3]福島県立医科大学附属生体情報伝達研究所 生体機能研究部門

P3-e13 成獣ラットの脳のグリア系細胞におけるラミン・サブタイプの構成パターンの解析
Immunohistochemical analysis of nuclear lamins in the glial cells of adult rat brain
高森 康晴(Yasuharu Takamori), 森 徹自(Tetsuji Mori), 若林 毅俊(Taketoshi Wakabayashi),
山田 久夫(Hisao Yamada)
関西医大・医・第一解剖

P3-e14 Long-term effects of voluntary running on neurogenesis of adult mice
内田 克哉(Katsuya Uchida), 井樋 慶一(Keiichi Itoi)
東北大院・情報・情報生物

P3-e15 生後海馬に存在するアストロサイト様神経前駆細胞のニューロン分化過程の解析
Direct evidence for neuronal differentiation of astrocyte-like progenitors in the postnatal hippocampus
難波 隆志[1,2](Takashi Namba), 並木 秀男[2](Hideo Namiki), 石 龍徳[1](Tatsunori Seki)
[1]順天堂大学医学部第二解剖学講座, [2]早稲田大学大学院理工学研究科生命理工学専攻

P3-e16 糖尿病ラットの認知機能障害と海馬における IGF-1mRNA 発現、神経新生能について
Cognitive impairments in diabetic rats in relation with IGF-1 and hippocampal neurogenesis
鬼頭 昭三[1](Shozo Kito), 新郷 明子[2](Akiko S. Shingo)
[1]順心会 幸生リハビリテーション病院, [2]兵庫大学 健康科学部

P3-e17 生後脳神経新生における Ephrin-Eph シグナルの機能
The function of Ephrin-Eph signaling in postnatal neurogenesis
原 芳伸[1,2](Yoshinobu Hara), Jonas Frisen[3], 大隅 典子[1,2](Noriko Osumi)
[1]東北大院・医・創生応用, [2]戦略的創造研究推進事業 (CREST), 科学技術振興機構 (JST), [3]Dept, Cell, Mol, Biol, Karolinska Ins, Sweden

P3-e18 成体マウス海馬における新生ニューロンの軸索の形態学的解析
Morphological analysis of axons of newly generated granule cells in adult mouse hippocampus
井出 陽子[1](Yoko Ide), 藤山 文乃[2](Fumino Fujiyama), 古田 敬子[2](Keiko Furuta),
玉巻 伸章[3](Nobuaki Tamamaki), 金子 武嗣[2](Takeshi Kaneko), 久恒 辰博[1](Tatsuhiro Hisatsune)
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P3-e19 ラット脳海馬における神経新生の口腔環境因子による影響
Cell proliferation in the rat hippocampal formation under the conditions of dysfunctional mastication
青木 宏道[1](H. Aoki), 堀 紀雄[1](N. Hori), 木本 克彦[1,3](K. Kimoto), 豊田 實[1](M. Toyoda),
小野塚 実[2,3](M. Onozuka)
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[3]神奈川歯科大学 高次脳・口腔科学研究センター

P3-e20 ドレブリン E は成体脳内において新生神経細胞の移動停止に伴い細胞体から消失する
Cessation of neuronal migration in adult rodent olfactory bulb is paralleled with the disappearance of drebrin E from the cell body
花村 健次(Kenji Hanamura), 宋 明橋(Mingqiao Song), 児島 伸彦(Nobuhiko Kojima),
関野 祐子(Yuko Sekino), 白尾 智明(Tomoaki Shirao)
群馬大院・医・高次細胞機能

P3-e21 Δ FosB は興奮毒性による脳障害後のマウス海馬歯状回において神経新生を促進する
Δ FosB promotes neurogenesis in the dentate gyrus of mouse hippocampus after brain damage caused by excitotoxicity
湯通堂紀子(Noriko Yutsudo)，本田 - 大西陽子(Yoko Honda-Ohnishi)，大西　克典(Yoshinori Ohnishi)，
中別府雄作(Yusaku Nakabeppu)
九州大学・生体防御医学研究所・脳機能制御学分野

P3-e22 新規バナジウム化合物は Akt と ERK を介して脳虚血後の海馬歯状回顆粒細胞下層における神経新生を亢進する。
A novel vanadyl compound enhances neurogenesis via Akt and ERK activation in the hippocampal subgranular zone after mouse focal cerebral ischemia
塩田　倫史[1](Norifumi Shioda)，福永　浩司[1,2](Kohji Fukunaga)
[1]東北大院・薬・薬理，[2]東北大学21世紀COEプログラム

P3-e23 培養海馬スライスから二次培養されたβIII-tubulin 発現細胞の有糸分裂
Mitotic hippocampal cells expressing βIII-tubulin in secondary cultures from organotypic slices
坂口　卓也[1,2,3](Takuya Sakaguchi)，大野　英治[1,2,3](Eiji Ohno)
[1]加計学園細胞病理学研究所，[2]倉敷芸術科学大学 生命科学部 生命科学科，[3]倉敷芸術科学大学大学院 産業科学技術研究科

P3-e24 成体ラット運動ニューロン損傷後に出現する内在性神経前駆細胞
Proliferation of neural stem/progenitor cells in adult rats after facial nerve avulsion
渡部　和彦(Kazuhiko Watabe)，高澤　隆紀(Takanori Takazawa)，米倉　真一(Shinichi Yonekura)
東京都神経科学総合研究所・分子神経病理

P3-e25 Galectin-1 は興奮毒性後の海馬歯状回における神経新生を促進する
Galectin-1 promotes neurogenesis in the dentate gyrus of mouse hippocampus after brain damage caused by excitotoxicity
梶谷　康介[1,2](Kosuke Kajitani)，門屋　利彦[3](Toshihiko Kadoya)，堀江　秀典[4](Hidenori Horie)，
Françoise Poirier[5]，中別府雄作[1](Yusaku Nakabeppu)
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## 体性運動 3
## Somatomotor System 3

P3-e26 マウス新生児脊髄反射経路から Renshaw 細胞への抑制性シナプス入力
Inhibition of Renshaw cells during spinal reflexes in the neonatal mouse spinal cord
西丸　広史[1](Hiroshi Nishimaru)，柳川右千夫[2](Yuchio Yanagawa)
[1]産総研・脳神経情報・脳遺伝子，[2]群馬大・院・医・遺伝発達行動学

P3-e27 低周波領域における筋緊張と自律神経活動の同期および姿勢制御に対する影響
Postural muscle tone synchronizes with automatic nervous activity in very low frequency domain
江　　依法[1](Yifa Jiang)，長崎　幸雄[2](Sachio Nagasaki)，木村　英紀[1](Hidenori Kimura)
[1]独立行政法人理化学研究所・生物制御システム研究チーム，[2]岐阜大学・医学部

P3-e28 皮質線条体終末シナプス前 AMPA 受容体による小胞放出確率の増強
Presynaptic AMPA receptors on the corticostriatal terminals enhance the release probability of the synaptic vesicles
田中　琢真[1](Takuma Tanaka)，藤山　文乃[1](Fumino Fujiyama)，野村　真樹[2](Masaki Nomura)，
青柳富誌生[2,3](Toshio Aoyagi)，金子　武嗣[1,2](Takeshi Kaneko)
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P3-e29 ラット脊髄前角ニューロンのパッチクランプ解析
Patch-clamp analysis of membrane properties of bursting neurons in the young adult rat ventral horn in vitro
久光　涼子(Ryoko Hisamitsu)，古江　秀昌(Hidemasa Furue)，吉村　　恵(Megumu Yoshimura)
九州大学・医・統合生理

P3-e30 視覚ターゲットの移動がリーチング運動に及ぼす影響
Asymmetric adjustments of human target-reaching by target switch
渋谷　　賢[1](Satoshi Shibuya)，五十嵐一峰[2](Kazutaka Igarashi)，佐野　秀仁[2](Hideto Sano)，
高橋　雅人[2](Masahito Takahashi)，里見　和彦[2](Kazuhiko Satomi)，大木　　紫[1](Yukari Ohki)
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P3-e31 運動可能性判断の脳内神経機序
Neural correlates of estimating graspability
廣瀬　智士[1,2](Satoshi Hirose)，羽倉　信宏[1,4](Nobuhiro Hagura)，松村　道一[1](Michikazu Matsumura)，
内藤　栄一[2,3](Eiichi Naito)
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P3-e32 GABA を含有するγ-運動ニューロン
GABA-containing γ-motor neurons
伊藤　哲史[1](Tetsufumi Ito)，日置　寛之[2](Hiroyuki Hioki)，中村　公一[2,3](Kouichi Nakamura)，
金子　武嗣[2,3](Takeshi Kaneko)，野条　良彰[1](Yoshiaki Nojyo)
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P3-e33 鳩の中間外套中間部の線維連絡
Fiber connections of the intermediate mesopallium in the pigeon
阿閉　泰郎(Yasuro Atoji)
岐阜大・応用生物・獣医解剖

P3-e34 ラット新生仔脳における線条体出力ニューロンの軸索投射を解析する
Single cell tracing of striatofugal neurons in neonatal rat brain
中村　公一[1](Kouichi Nakamura)，藤山　文乃[1](Fumino Fujiyama)，古田　貴寛[1](Takahiro Furuta)，
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P3-e35 空圧刺激 - レバー放し応答を用いたラット選択反応時間タスク
A new choice reaction-time task in the rat using air-puff stimuli and lever-release responses
金子　秀和[1](Hidekazu Kaneko)，田村　　弘[2](Hiroshi Tamura)，川島　貴弘[3](Takahiro Kawashima)，
鈴木　慎也[1](Shinya S. Suzuki)
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P3-e36 箸操作に関わる脳機構：PET 研究
Functional brain areas involved in the use of chopsticks: a PET study
木下　　博[1](Hiroshi Kinoshita)，津田　勇人[2](Hayato Tsuda)，青木　朋子[3](Tomoko Aoki)，
奥　　直彦[4](Naohiko Oku)，畑澤　　順[1](Jyun Hatazawa)，木村　泰之[1](Yasuyuki Kimura)，
梶本　勝文[1](Katsufumi Kajimoto)，榎本　圭祐[1](Keisuke Enomoto)
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P3-e37 筋電図と相関するサル感覚運動野の律動性活動の機能結合
Functional connectivity of sensorimotor oscillatory activities associated with electromyogram in the monkey
美馬　達哉[1](Tatsuya Mima)，島津　秀紀[2](Hideki Shimazu)，磯村　宜和[3,4](Yoshikazu Isomura)，
福山　秀直[1](Hidenao Fukuyama)，逵本　　徹[5](Toru Tsujimoto)
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P3-e38 到達運動の視覚 - 運動変換の学習が視野の動きに誘発される追従性腕応答へ与える影響
Effect of visuomotor adaptation for arm reaching on the manual following response induced by visual motion
西條　直樹[1](Naoki Saijo)，五味　裕章[1,2](Hiroaki Gomi)
[1]NTT CS基礎研 人間情報，[2]ERATO 下條潜在脳 JST

P3-e39 前庭系への電流刺激により生ずる視覚誘導性腕反射応答のゲイン変化
Gain modulation of manual following response associated with galvanic vestibular stimulation
門田　浩二[1](Koji Kadota)，五味　裕章[1,2](Hiroaki Gomi)
[1]JST-ERATO下條潜在脳機能プロジェクト，[2]NTTコミュニケーション科学基礎研究所

P3-e40 視覚誘導性腕応答におけるゲイン修飾メカニズムは追従性眼球応答でも共有されるか？
Do the similar quick visuomotor controls of arm and eye share a gain modulation caused by the spatial dissociation of gaze and pointing?
安部川直稔[1](Naotoshi Abekawa)，五味　裕章[1,2](Hiroaki Gomi)
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P3-e41 視覚誘導性腕応答と追従眼球運動の時空間周波数特性について
Spatiotemporal tuning of short-latency manual and ocular following responses associated with sudden visual motion in monkeys
竹村　文[1,5](Aya Takemura)，安部川直稔[2](Naotoshi Abekawa)，山根　茂[3](Shigeru Yamane)，
河野　憲二[4](Kenji Kawano)，五味　裕章[2,5](Hiroaki Gomi)
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## 視覚 3
## Visual System 3

P3-e42 網膜における V-ATPase の局在：プロトンフィードバック仮説を支持する根拠
Localization of V-ATPase in the retina: evidence for proton feedback hypothesis
城宝　浩[1](H Jouhou)，中野　健二[2](K Nakano)，石川　修司[2](S Ishikawa)，佐野　孝一[2](K Sano)，
宍戸　隆男[2](T Shishido)，金子　章道[3](A Kaneko)，山田　雅弘[1](M Yamada)
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P3-e43 アマクリン細胞の機能を説明する網膜神経回路モデル
A retinal circuit model accounting for functions of amacrine cells
林田　祐樹(Yuki Hayashida)，Murat Saglam，村山　伸樹(Nobuki Murayama)
熊本大学大学院自然科学研究科

P3-e44 リアルな映像が眼球運動と脳血流に与える影響
Motion picture effects to eye movement and cerebral blood flow
飯島　淳彦[1,2](Atsuhiko Iijima)，木竜　徹[3](Tohru Kiryu)，鵜飼　一彦[2](Kazuhiko Ukai)，
戸田　春男[1](Haruo Toda)，板東　武彦[1](Takehiko Bando)
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P3-e45 複雑な光景の中での視覚探索課題においてサッカードの終端位置を決定する因子
Determining factors for endogenous saccadic eye movement during visual search in complex scene
勝亦　憲子[1,2](Noriko Katsumata)，Michel Vidal-Naquet[1]，谷藤　学[1](Manabu Tanifuji)
[1]理化学研究所　脳総合研究センター，[2]東京医科歯科大学大学院　医歯学総合研究科　脳神経機能外科学分野

P3-e46 色彩と輝度の方位に依存した知覚相互作用
Perceptional orientation-dependent interaction between color and luminance
花沢　明俊(Akitoshi Hanazawa)
九州工業大学・院生命体工学

P3-e47 高密度電極アレイによるサル下側頭葉視覚連合野の視覚刺激応答の空間パターンの計測
Functional maps of visual responses with a multiple-electrode array in macaque inferotemporal cortex
谷藤　学[1](Manabu Tanifuji)，宮川　尚久[1](Naohisa Miyakawa)，David Blake[2]，Michael Merzenich[3]
[1]理研脳科学総合研究センター，[2]ジョージア医科大学，[3]カルフォルニア大学サンフランシスコ校

P3-e48 網膜桿体視細胞のネットワークモデル
A mathematical model of retinal rod photoreceptor network
宮本　侑典(Yusuke Miyamoto)，神山　斉己(Yoshimi Kamiyama)
愛知県立大・情報

P3-f01 運動からの３次元知覚に対する頭頂及び外側後頭側頭領域の異なった役割
Different roles of the parietal and lateral occipito-temporal cortex for 3D perception from motion
山本　哲也[1,2](Tetsuya Yamamoto)，高橋　成子[3](Shigeko Takahashi)，花川　隆[4,5](Takashi Hanakawa)，
浦山　慎一[4](Shin-ichi Urayama)，麻生　俊彦[4](Toshihiko Aso)，福山　秀直[4](Hidenao Fukuyama)，
江島　義道[6](Yoshimichi Ejima)
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P3-f02 X型網膜神経節細胞の時空間処理モデル
A model of spatiotemporal processing in X retinal ganglion cell
古澤　優希(Yuki Furusawa)，神山　斉己(Yoshimi Kamiyama)
愛知県立大・情報

P3-f03 脳梁結合をもつ初期視覚野細胞の受容野構造
Receptive field structures of transcallosally connected neurons in the cat's visual cortex
朝田　雄介(Yusuke Asada)，眞田　尚久(Takahisa M Sanada)，大澤　五住(Izumi Ohzawa)
大阪大院・生命機能

P3-f04 図方向決定における周辺結合領域と刺激形状の特徴
Characteristics of surround modulation and stimulus shape for consistent determination of border ownership
西村　悠(Haruka Nishimura)，辻　義尚(Yoshihisa Tsuji)，酒井　宏(Ko Sakai)
筑波大院・システム情報工学・コンピュータサイエンス

P3-f05 網膜への遠心性投射ニューロンの自発的活動
Voluntary activity of neurons projecting centrifugally to the retina
大野　裕史(Hiroshi Ohno)，内山　博之(Hiroyuki Uchiyama)
鹿児島大学・工学部

P3-f06 視覚的運動によって誘導される色彩拡散
Perceptual color spreading induced by visual motion
原　千晶(Chiaki Hara)，花沢　明俊(Akitoshi Hanazawa)
九工大院・生命体

P3-f07 網膜のバースト発火による情報伝達は種間で保存されている
Burst ISI coding is conserved across species
石井　俊行[1,2](Toshiyuki Ishii)，細谷　俊彦[1](Toshihiko Hosoya)
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P3-f08 chick終脳における刷り込み成立に伴う神経回路活性の解析
Analysis of neuronal activation in the telencephalon in the visually imprinted chicks
中森　智啓[1](Tomoharu Namkamori)，佐藤　勝重[2](Katsushige Sato)，田中　光一[1](Kohichi Tanaka)，
浜崎　浩子[1](Hiroko Ohki-Hamazaki)
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P3-f09 経頭蓋電気刺激を用いた麻酔下マウス皮質間結合の活性化
Transcranial electrical stimulation for activating cortico-cortical connections in anesthetized mice
菱田　竜一(Ryuichi Hishida)，工藤　雅治(Masaharu Kudoh)，澁木　克栄(Katsuei Shibuki)
新潟大・脳研・システム脳生理

P3-f10 MEG/EEG を再現するための neural mass model を元にした Multiple generators model の構築
Multiple generators model based on neural mass model for MEG/EEG
成瀬　　康[1](Yasushi Naruse)，眞溪　　歩[1,2](Ayumu Matani)，宮脇　陽一[2,3](Yoichi Miyawaki)，
岡田　真人[1,4](Masato Okada)
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P3-f11 運動透明視における奥行き知覚とフリッカー知覚の関係
Relation between depth and flicker perception in motion transparency
三澤　秀明(Hideaki Misawa)，花沢　明俊(Akitoshi Hanazawa)
九州工業大学　大学院生命体工学研究科

P3-f12 視覚性運動検出に刺激の大きさが与える影響について
Effect of the stimulus size on the human brain response to visual motion
浦川　智和[1](Tomokazu Urakawa)，金桶　吉起[1,2](Yoshiki Kaneoke)，田中　絵実[1,2](Emi Tanaka)，
柿木　隆介[1,2](Ryusuke Kakigi)
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P3-f13 運動透明視の知覚コスト：心理実験とモデル解析
Perceptual cost for motion transparency: psychophysics and model analysis
渡部　　修(Osamu Watanabe)，鈴木　有人(Naoto Suzuki)
室蘭工大・工・情報

P3-f14 視差エネルギー計算を用いた両眼ロボットビジョンの実時間輻輳眼球運動制御
Real-time vergence control of binocular robot vision employing disparity energy computation in visual cortex
下ノ村和弘[1](Kazuhiro Shimonomura)，八木　哲也[2](Tetsuya Yagi)
[1]大阪大・MEIセンター，[2]大阪大院・工・電気電子情報

## 嗅覚、味覚、化学感覚 2
## Olfaction, Taste, Chemical Senses 2

P3-f15 嗅上皮付き単離脳標本を用いた前梨状皮質における嗅覚表現の研究
Odor representation in the anterior piriform cortex of an *in vitro* isolated whole brain with the olfactory epithelium
清水　　章(Akira Shimizu)，石川　享宏(Takahiro Ishikawa)，筒井健一郎(Ken-Ichiro Tsutsui)，
飯島　敏夫(Toshio Iijima)
東北大学大学院・生命科学研究科

P3-f16 受動的嗅覚と能動的嗅覚によって測定された嗅覚神経磁界応答
Olfactory neuromagnetic responses measured by passive and active olfaction
外池　光雄[1](Mitsuo Tonoike)，山口　雅彦[2](Masahiko Yamaguchi)，三分一史和[1](Fumikazu Miwakeichi)
[1]千葉大院・工・メディカルシステム，[2]産総研・関西センター・人間福祉医工学

P3-f17 線虫 *C. elegans* におけるアルカリｐＨ感覚に関わる神経の解析
Neurons responsible for *C. elegans* chemosensation to pH
村山　　孝(Takashi Murayama)，丸山　一郎(Ichiro Maruyama)
沖縄科学技術研究基盤整備機構・分子神経科学ユニット

P3-f18 線虫 *C. elegans* の嗅覚順応にネプリライシン NEP-2 が関与する
The neprilysin gene *nep-2* is involved in olfactory adaptation in *C. elegans*
山田 康嗣[1](Koji Yamada), 広津 崇亮[2](Takaaki Hirotsu), 松木 正尋[1](Masahiro Matsuki),
国友 博文[1](Hirofumi Kunitomo), 飯野 雄一[1](Yuichi Iino)
[1]東京大学 遺伝子実験施設, [2]九州大学 理学 生物科学

P3-f19 線虫の化学走性行動の2つの戦略
Behavioral strategy of salt chemotaxis in *C. elegans*
飯野 雄一(Yuichi Iino)
東京大学遺伝子実験施設

P3-f20 マウス茸状乳頭由来味細胞のグルタミン酸に対する応答
Glutamate responses of mouse fungiform taste cells with action potentials
村田 芳博(Yoshihiro Murata), 吉田 竜介(Ryusuke Yoshida), 安尾 敏明(Toshiaki Yasuo),
安松 啓子(Keiko Yasumatsu), 重村 憲徳(Noriatsu Shigemura), 二ノ宮裕三(Yuzo Ninomiya)
九州大院・歯・口腔機能解析

P3-f21 単一味蕾中に存在する活動電位を発生する味受容細胞の応答解析
Taste receptor cells generating action potentials within a single taste bud of mouse fungiform papillae
吉田 竜介(Ryusuke Yoshida), 村田 芳博(Yoshihiro Murata), 安尾 敏明(Toshiaki Yasuo),
安松 啓子(Keiko Yasumatsu), 重村 憲徳(Noriatsu Shigemura), 二ノ宮裕三(Yuzo Ninomiya)
九州大院・歯・口腔機能解析

P3-f22 幼児期マウスにおける味蕾細胞の電位依存性電流
Voltage-gated currents of taste bud cells in infant mice.
岩本 正史(Masafumi Iwamoto), 大坪 義孝(Yoshitaka Ohtubo), 吉井 清哲(Kiyonori Yoshii)
九州工業大学大学院生命体工学研究科脳情報専攻

P3-f23 ENaC 遺伝子多型性とアミロライド感受性塩味応答との関与
Polymorphisms of ENaC subunits relate to mouse strain differences in amiloride sensitive salt responses
重村 憲徳[1](Noriatsu Shigemura), 大栗 弾宏[1](Tadahiro Ohkuri), 貞光 千春[1](Chiharu Sadamitsu),
安松 啓子[1](Keiko Yasumatsu), 吉田 竜介[1](Ryusuke Yoshida), Gary K Beauchamp[2],
Alexander A Bachmanov[2], 二ノ宮裕三[1](Yuzo Ninomiya)
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P3-f24 マウス鼓索神経における塩、酸、苦味物質による甘味応答の修飾
Modulations of sweet taste responses by salts, acids and bitter compounds in the mouse chorda tympani nerve
安松 啓子[1](Keiko Yasumatsu), 吉田 竜介[1](Ryusuke Yoshida), Robert F. Margolskee[2],
二ノ宮裕三[1](Yuzo Ninomiya)
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P3-f25 マウス茸状乳頭および軟口蓋味蕾における細胞型の定量的比較
Quantitative differences between taste buds in mouse fungiform papillae and soft palates
大坪 義孝(Yoshitaka Ohtubo), 吉井 清哲(Kiyonori Yoshii)
九州工業大学院・生命体工学科・脳情報専攻

P3-f26 マウス鼓索神経のグルマリン感受性甘味応答の温度による修飾
Temperature modulation of gurmarin sensitive sweet responses in the mouse chorda tympani nerve
大栗 弾宏(Tadahiro Ohkuri), 楠原 庸子(Yoko Kusuhara), 安松 啓子(Keiko Yasumatsu),
二ノ宮裕三(Yuzo Ninomiya)
九州大院・歯・口腔機能解析学

P3-f27 GAL4 エンハンサートラップ系統を用いて可視化されたショウジョウバエ味覚受容神経の投射先の構造
Organization of the projection targets of the *Drosophila* gustatory receptor neurons visualized with GAL4 enhancer-trap strains
宮崎 隆明[1,2](Takaaki Miyazaki), 伊藤 啓[1,2](Kei Ito)
[1]東大院・新領域・情報生命, [2]東大・分生研・細胞機能情報セ

P3-f28 昆虫の嗅覚系神経経路におけるセロトニン免疫反応性神経の系統間比較
Phylogenetic comparison of serotonin-immunoreactive neurons in the insect olfactory neural systems
岩野 正晃[1,2](Masaaki Iwano), 伊藤 啓[1](Kei Ito)
[1]東京大学・分子細胞研・高次構造, [2]科学技術振興機構

P3-f29 アデニリルサイクラーゼ遺伝子 AC78C の甘味受容における関与
Transmembrane adenylyl cyclase, encoded by *AC78C*, is required for sugar perception in *Drosophila*
上野 耕平[1](Kohei Ueno), 城所 良明[2,3](Yoshiaki Kidokoro)
[1]群馬大院・医・遺伝発達行動学, [2]群馬大学・医・生体調節研究所, [3]UCLA

P3-f30 ショウジョウバエの幼虫キノコ体の嗅覚二次中枢であるカリックスにおける神経結合様式
Connectivity in the larval mushroom body calyx, a secondary olfactory center of Drosophila
増田（中川）リリア[1](Liria M. Masuda-Nakagawa), 田中 暢明[1](Nobuaki K. Tanaka), 伊藤 啓[1](Kei Ito), Cahir J. O'Kane[2]
[1]東京大学・分生研, [2]Department of Genetics, University of Cambridge, UK

P3-f31 *Lhx2* 遺伝子欠損マウスにおけるクラス依存的な嗅覚受容体遺伝子発現異常の解析
Differential impact of Lhx2 deficiency on expression of class I and class II odorant receptor genes in mouse
廣田 順二[1,2](Junji Hirota), 大村 雅代[2](Masayo Omura), Peter Mombaerts[2]
[1]大阪府立大院・理・生物科学, [2]The Rockefeller University, New York, USA

## 内臓感覚
## Viscerosensory System

P3-f32 膀胱支配後根神経節神経における TRPM8 の発現
Expression of TRPM8 in rat dorsal root ganglion neurons innervating the urinary bladder
松吉ひろ子[1](Hiroko Matsuyoshi), 辰巳 晃子[1](Kouko Tatsumi), 横山 昇平[1](Shohei Yokoyama), 眞部 孝幸[1](Takayuki Manabe), 吉村 直樹[2](Naoki Yoshimura), 和中 明生[1](Akio Wanaka)
[1]奈良県立医科大学・医学部・解剖学第二講座, [2]ピッツバーグ大学・医学部・泌尿器科

P3-f33 ラット舌咽および迷走神経における BDNF 含有 一次知覚ニューロンの分布
Brain-derived neurotrophic factor-immunoreactive neurons in the rat vagal and glossopharyngeal sensory ganglia; co-expression with other neurochemical substances
閻 忠智(Zhongzhi Yan), 市川 博之(Hiroyuki Ichikawa), 寺山 隆司(Ryuji Terayama), 山合友一郎(Tomoichiro Yamaai), 杉本 朋貞(Tomosada Sugimoto)
岡山大学大学院医歯薬学総合研究科 口腔機能解剖学分野

P3-f34 過敏な内臓知覚の認知的修飾過程におけるヒスタミンニューロンの役割
Role of histaminergic neurons in cognitive control of hypersensitive visceral perception
渡辺 諭史(Satoshi Watanabe), 服部 朝美(Tomomi Hattori), 金澤 素(Motoyori Kanazawa), 鹿野 理子(Michiko Kano), 福土 審(Shin Fukudo)
東北大学院・医・行動医学

P3-f35 **Immunohistochemical mapping of NPR-A in the brainstem of macaca fascicularis**

Essam M. Abdelalim[1,2,3], Abdel-Hamid K. Osman[3], Tatsuyuki Takada[2], Ryuzo Torii[2], Ikuo Tooyama[1]
[1]Molecular Neuroscience Research Center, Shiga Univ. of Medical science, Otsu, Japan, [2]Research Center For Animal Life Science, Shiga Univ of Medical science, Otsu, Japan, [3]Dep. of Cytology & Histology, Suez Canal Univ., Egypt

P3-f36 **ラット視索上核大細胞性神経分泌細胞におけるシナプス入力の浸透圧感受性の検討**
**Osmosensitivity in the synaptic inputs to the magnocellular neurosecretory cells in the supraoptic nucleus of rat**

横山　徹(Toru Yokoyama)，斎藤　健(Takeshi Saito)，藤原　広明(Hiroaki Fujihara)，
上田　陽一(Yoichi Ueta)
産業医科大学医学部第１生理学

P3-f37 **逆行性トレーサーおよび、ゴルジ染色を用いたメダカ内側縦束の観察**
**Injection of biocytin and golgi staining of medaka brain**

大矢　環(Tamaki Ohya)，林　しん治(Shinji Hayashi)
横浜市立大学・総合理学研究科

P3-f38 **ラット脳におけるオレキシンニューロンの生後発生に関する免疫組織化学的観察**
**Immunohistochemical observation of the postnatal development of the orexin neuron in the rat brain.**

澤井　信彦(Nobuhiko Sawai)，小澤　一史(Hitoshi Ozawa)
日本医科大院・医・生体制御形態科学

P3-f39 **発達期小脳における甲状腺ホルモン依存性遺伝子群の同定：甲状腺ホルモンによる細胞周期・細胞死制御の可能性**
**Identification of thyroid hormone dependent genes in the developing mouse cerebellum: effect of thyroid hormone on cell proliferation and death**

高橋　理貴[1,2](Masaki Takahashi)，根岸　隆之[1](Takayuki Negishi)，田代　朋子[1](Tomoko Tashiro)
[1]青山学院大院・理工・生命科学，[2](独)日本学術振興会

P3-f40 **ラットの不安関連行動に対するPrRP投与の効果**
**Effects of prolactin-releasing peptide administration upon anxiety-related behavior in rats**

高柳　友紀(Yuki Takayanagi)，尾仲　達史(Tatsushi Onaka)
自治医大・医・生理学

P3-f41 **神経系におけるグルココルチコイド受容体とNF κ Bの相互作用**
**Glucocorticoid receptor interaction with nuclear factor κ B in the nervous system**

藤川　和世(Kazuyo Fujikawa)，西　真弓(Mayumi Nishi)，河田　光博(Mitsuhiro Kawata)
京都府立医科大学生体構造科学

P3-f42 **視床下部と脳下垂体におけるCD38の免疫組織学的解析**
**Immunohistochemistry of CD38 in hypothalamus and pituitary**

神山　裕子[1](Yuko Kamiyama)，藤田　慶大[1](Kyota Fujita)，清家　稔博[1](Toshihiro Seike)，
城戸　瑞穂[2](Mizuho Kido)，田中　輝男[2](Teruo Tanaka)，東田　陽博[3](Haruhiro Higashida)，
野田　百美[1](Mami Noda)
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P3-f43 **海馬におけるエストロゲンのスパイン制御作用**
**Effect of estrogen on spine regulation in hippocampus**

村上　元[1,2](Gen Murakami)，釣木澤朋和[1](Tomokazu Tsurugizawa)，畑中　悠佑[1](Yuusuke Hatanaka)，
小松崎良将[1](Yoshimasa Komatsuzaki)，小南　思郎[3](Shirou Kominami)，木本　哲也[1](Tetsuya Kimoto)，
川戸　佳[1,2](Suguru Kawato)
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P3-f44 ラット前視床下部ニューロンに対するghrelinの作用
Effects of ghrelin on neuronal activity of anterior hypothalamic area in rats
金　主賢[1](Juhyon Kim)，牧　新太郎[1](Shintaro Maki)，中島　一樹[1](Kazuki Nakajima)，
大村　裕[2](Yutaka Oomura)，佐々木和男[1](Kazuo Sasaki)
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P3-f45 遺伝子欠損マウスを用いたオキシトシン受容体の体温調節機構に関する解析
Cold-sensitive phenotype in mice lacking oxytocin receptor
笠原　好之[1](Yoshiyuki Kasahara)，高柳　友紀[2](Yuki Takayanagi)，吉田　匡秀[1](Masahide Yoshida)，
河田　輝雄[3](Teruo Kawada)，井樋　慶一[4](Keiichi Itoi)，西森　克彦[1](Katsuhiko Nishimori)
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P3-f46 ラット海馬における女性ホルモンおよび男性ホルモン合成
Neurosteroids synthesis in the rat hippocampus
木本　哲也(Tetsuya Kimoto)，肥後　心平(Shimpei Higo)，北條　泰嗣(Yasushi Hojo)，
石井　寛高(Hirotaka Ishii)，川戸　佳(Suguru Kawato)
東京大院・総合文化・広域科学・生命環境科学系

P3-f47 GnRH-EGFPトランスジェニックラットを用いた性腺刺激ホルモン放出ホルモン（GnRH）含有ニューロンの形態的特徴に関する研究
Morphological characteristics of gonadotropin-releasing hormone (GnRH) neurons in the preoptic area of GnRH-EGFP transgenic rat
薛ほうがん[1](Hao-Gang Xue)，山本　直之[1](Naoyuki Yamamoto)，加藤　昌克[2](Masakatsu Kato)，
楊　春英[1](Chun-Ying Yang)，佐久間康夫[2](Yasuo Sakuma)，小澤　一史[1](Hitoshi Ozawa)
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P3-f48 エストラジオールによる培養視床下部細胞での熱ショック蛋白質の発現変化―プロテオミクス解析法を用いた検討―
Increase of heat shock proteins by treatment of cultured hypothalamic cells with 17beta-estradiol
大谷一金子律子[1](Ritsuko Ohtani-Kaneko)，岩倉　聖[1](Takashi Iwakura)，村上妃呂子[2](Hiroko Murakami)，
菅家　美恵[2](Mie Kannke)，加藤　智啓[2](Tomohiro Kato)
[1]東洋大学生命科学部，[2]聖マリアンナ医科大学難治研

P3-g01 終神経GnRHニューロンにおけるGABA作動性シナプスの神経生理学的解析
Neurophysiological analysis of GABAergic synapses in the terminal nerve GnRH neurons
中根　亮(Ryo Nakane)，岡　良隆(Yoshitaka Oka)
東京大院・理・生物科学

P3-g02 統合失調症関連神経ペプチドPACAPの海馬歯状回における機能解析
Function analysis of Pituitary adenylate cyclase-activating polypeptide associated with schizophrenia in hippocampal dentate gyrus
山田　浩平[1](Kohei Yamada)，松崎　伸介[1,3](Shinsuke Matsuzaki)，服部　剛志[1,3](Tsuyoshi Hattori)，
橋本　均[2](Hitoshi Hashimoto)，新谷　紀人[2](Norihito Shintani)，馬場　明道[2](Akemichi Baba)，
遠山　正彌[1,3](Masaya Tohyama)
[1]大阪大院・医・神経機能形態学講座，[2]大阪大院・薬・神経薬理学分野，[3]子どものこころの分子統御機構研究センター

P3-g03 噛み合わせが悪くなると大脳辺縁系と視床下部の内因性オピオイドの産生が増加する
Dysfunctional mastication increases dynorphin A-like and β-endorphin-like immunoreactivities in the amygdala and hypothalamus
山田健太朗[1,4](Kentaro Yamada)，岩本真由子[2,4](Mayuko Iwamoto)，片岡　烈[2,4](Tsuyoshi Kataoka)，
笹栗　健一[2,4](Ken-ichi Sasaguri)，山本　利春[3,4](Toshiharu Yamamoto)，佐藤　貞雄[2,4](Sadao Sato)，
小野塚　實[1,4](Minoru Onozuka)
[1]神歯大院・歯・生理，[2]神歯大・歯・矯正，[3]神歯大・歯・生物，[4]神歯大・高次脳・口腔科学研究センター

P3-g04 エストロゲン受容体ノックアウトマウスおよびアロマターゼノックアウトマウスにおける分界条床核主核の形態学的性差の消失
Elimination of sex differences in the principal nucleus of the bed nucleus of the stria terminalis (BSTp) in estrogen receptor-$\alpha$ and -$\beta$ and aromatase knockout mice
塚原　伸治[1](Shinji Tsukahara)，渡井　浩太[2](Kota Watai)，黒田　淑子[2](Yoshiko Kuroda)，
小澤　貴明[2](Takaaki Ozawa)，福士　碧沙[2](Aosa Fukushi)，肖　　凱[2](Kai Xiao)，
津田夢芽子[2](Mumeko Tsuda)，戸田　勝巳[3](Katsumi Toda)，藤巻　秀和[1](Hidekazu Fujimaki)，
小川　園子[2](Sonoko Ogawa)
[1]国立環境研究所・環境リスク研究センター，[2]筑波大学，[3]高知大学

P3-g05 メダカにおいて性的二型を示すメタスチンニューロン群
Sexually dimorphic metastin neurons in Medaka brain
松本　真司[1](Shinji Matsumoto)，赤染　康久[1](Yasuhisa Akazome)，山本　直之[2](Naoyuki Yamamoto)，
大久保範聡[3](Kataaki Okubo)，山田　俊児[4](Shunji Yamada)，束村　博子[4](Hiroko Tsukamura)，
前多敬一郎[4](Kei-Ichiro Maeda)，岡　　良隆[1](Yoshitaka Oka)
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P3-g06 海馬におけるエストロゲン受容体の局在と作用の解析
Synaptic estrogen receptors in principal neurons of hippocampus
向井　秀夫[1,2](Hideo Mukai)，村上　　元[1,2](Gen Murakami)，
小南　思郎[3](Shiro Kominami)，肥後　心平[1,2](Shimpei Higo)，北條　泰嗣[1,2](Yasushi Hojo)，
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P3-g07 ラット過労モデルにおける下垂体中間葉細胞の変性
Accumulated fatigue induces over-activation and subsequent degeneration of melanotrophs in rat pituitary gland
小川登紀子[1,3](Tokiko Ogawa)，小西　博之[1,3](Hiroyuki Konishi)，田中　雅彰[2,3](Masaaki Tanaka)，
桐生 - 瀬尾寿美子[1,3](Sumiko Kiryu-Seo)，中込　咲綾[1,3](Saya Nakagomi)，渡辺　恭良[2,3](Yasuyoshi Watanabe)，
木山　博資[1,3](Hiroshi Kiyama)
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P3-g08 硬骨魚脳を用いた視床下部外（終神経）ゴナドトロピン放出ホルモン（GnRH）ニューロン単離培養系
Isolated primary culture of the terminal nerve (TN)- GnRH neurons derived from the brain of a tropical teleost
阿部　秀樹(Hideki Abe)，岡　　良隆(Yoshitaka Oka)
東京大・院理・生物科学

P3-g09 オキシトシン受容体 -Venus knock-in マウスによるオキシトシン受容体発現ニューロンの可視化
Oxytocin receptor-Venus knock-in mice enable direct visualization of oxytocin receptor-expressing neurons
吉田　匡秀[1](Masahide Yoshida)，高柳　友紀[2](Yuki Takayanagi)，尾仲　達史[2](Tatushi Onaka)，
西森　克彦[1](Katuhiko Nishimori)
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## 摂食調節
## Regulation of Food Intake

P3-g10 亜鉛欠乏ラットの食塩欲求に味覚は関与しない
Taste may not affect salt appetite in zinc deficient rats
裕　　哲崇[1](Noritaka Sako)，勝川　秀夫[1](Hideo Katsukawa)，中島　清人[2](Kiyohito Nakashima)，
杉村　忠敬[1](Tadataka Sugimura)
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P3-g11 遺伝子導入マウスにおける皮質線維終末とメラノコルチン４受容体発現視床ニューロンとのシナプス構築
Synaptic organization between cortical axon terminals and Melanocortin 4 receptor-expressing thalamic neurons of the transgenic mouse
津森登志子(Toshiko Tsumori)，中村佐和子(Sawako Nakamura)，横田　茂文(Shigefumi Yokota)，
安井　幸彦(Yukihiko Yasui)
島根大・医・解剖学講座・神経形態学

P3-g12 マウス視床下部における扁桃体終末と摂食関連ペプチド含有ニューロンおよびメラノコルチン４レセプター発現ニューロンとのシナプス構築
Synaptic organization between amygdaloid axon terminals and feeding-related peptide containing or melanocortin 4 receptor-expressing neurons in the mouse hypothalamus.
中村佐和子(Sawako Nakamura)，津森登志子(Toshiko Tsumori)，横田　茂文(Shigehumi Yokota)，
安井　幸彦(Yukihiko Yasui)
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P3-g13 siRNA を用いた視床下部弓状核 NPYmRNA ノックダウンによる摂食行動の減少
Reduction of feeding behavior by *in vivo* knockdown of NPY mRNA using siRNA in hypothalamic arcuate nucleus
仁木　剛史(Takeshi Niki)，椎谷　友博(Tomohiro Shiiya)，樋口　宗史(Hiroshi Higuchi)
新潟大院・医・薬理

P3-g14 オランザピンによる体重増加と視床下部での細胞分裂との関わり
Olanzapine increases body weight and cell mitotic activity in the hypothalamus of mice
山内　崇平[1](Takahira Yamauchi)，辰巳　晃子[2](Kouko Tatsumi)，牧之段　学[1](Manabu Makinodan)，
松吉ひろ子[2](Hiroko Matsuyoshi)，岸本　年史[1](Toshifumi Kishimoto)，和中　明生[2](Akio Wanaka)
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P3-g15 ラットの高嗜好性溶液摂取行動における側坐核 shell 部カンナビノイド１型受容体の役割
Cannabinoid type 1 receptors in the nucleus accumbens shell are involved in the intake of palatable solutions
篠原　祐平[1](Yuhei Shinohara)，志村　　剛[1](Tsuyoshi Shimura)，山本　　隆[1,2](Takashi Yamamoto)
[1]大阪大学院・人間科学・行動生理，[2]大阪大学院・歯学

P3-g16 WD-repeat protein 6 は視床下部におけるエネルギーセンシングと関連している
WD-repeat protein 6 is involved in nutrient sensing in rat hypothalamus
千葉　卓哉(Takuya Chiba)，井上　大輔(Daisuke Inoue)，水野　　綾(Aya Mizuno)，
小松　利光(Toshimitsu Komatsu)，林田　隆広(Takahiro Hayashida)，林　　洋子(Hiroko Hayashi)，
朴　　盛浚(Seongjoon Park)，山座　治義(Haruyoshi Yamaza)，樋上　賀一(Yoshikazu Higami)，
下川　　功(Isao Shimokawa)
長崎大院・医歯薬・探索病理

P3-g17 レプチンによって誘導される遺伝子のグレリン受容体の発現に対する機能的役割
A novel leptin-induced transcript in the expression of growth hormone secretagogue-receptor
小森　忠祐[1](Tadasuke Komori)，土井　麻子[2](Asako Doi)，古田　浩人[2](Hiroto Furuta)，
南條輝志男[2](Kishio Nanjo)，仙波恵美子[1](Emiko Senba)，森川　吉博[1](Yoshihiro Morikawa)
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P3-g18 油脂 binge 摂食によるラット側坐核のドーパミン応答変化
Fat binge eating alters dopamine response in rat nucleus accumbens
成清　公弥(Kimiya Narikiyo)，粟生　修司(Shuji Aou)
九工大院・生命体・脳情報

P3-g19 パルミチン酸は下垂体ゴナドトロフ株化細胞 LβT2 細胞において長鎖脂肪酸受容体 GPR120 の発現量を増加させる
Palmitate-induced increase in long-chain fatty acid receptor GPR120 mRNA level in LβT2 pituitary gonadotrope cells
森山隆太郎(Ryutaro Moriyama), 能勢　和浩(Kazuhiro Nose), 小浜　梓(Azusa Kohama),
福嶋　伸之(Nobuyuki Fukushima)
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## 学習、記憶 3
## Learning and Memory 3

P3-g20 小脳運動学習の分子機構：内在性カンナビノイド受容体および代謝型グルタミン酸受容体シグナリングの役割について
Molecular basis of associative eyeblink conditioning: New player involved in motor memory formation
岸本　泰司[1,2](Yasushi Kishimoto), 桐野　豊[1](Yutaka Kirino), 狩野　方伸[2](Masanobu Kano)
[1]徳島文理大院・香川薬・生物物理, [2]大阪大・医・細胞神経科学

P3-g21 マウス味覚嗜好学習の神経基盤としての扁桃体の役割
The role of amygdala as a neural substrate for conditioned taste preference learning in mice
宮本　武典[1](Takenori Miyamoto), 船木　美保[1](Miho Funaki), 北澤　美保[1](Miho Kitazawa),
吉沢　光[1](Hikaru Yoshizawa), 藤原　宏子[1](Hiroko Eda-Fujiwara), 佐藤　亮平[2](Ryohei Satoh),
八十島安伸[3](Yasunobu Yasoshima)
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P3-g22 Altered levels of BDNF protein following prenatal auditory stimulation
S Chaudhury, T C Nag, S Wadhwa
Department of AnatomyAIIMS, New Delhi, India

P3-g23 Hypobaric hypoxia induces neuronal damage and impair spatial memory in rat brain
Panchanan Maiti, Shashi Bala Singh, G Ilavazhagan
Applied Physiology Division, Delhi, India

P3-g24 摂食による脳可塑性の活性化
Facilitation of brain plasticity during food intake
大村　裕[1](Yutaka Oomura), 粟生　修司[2](Shuji Aou), 福永　浩司[3](Kouji Fukunaga),
森口　繁樹[3](Sigeki Moriguchi), 佐々木和男[4](Kazuo Sasaki)
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P3-g25 Dopamine D1 receptors in neural responses to rewards of amygdala and accumbens neurons
Anh H. Tran[1,6], Teruko Uwano[2], Tatsuo Kimura[1,6], Motoya Katsuki[4], Ryoi Tamura[2], Etsuro Hori[3,6],
Hisao Nishijo[3,6], Taketoshi Ono[1,5]
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P3-g26 Distinctive neuronal networks and biochemical pathways in appetitive and aversive memory in *Drosophila* larvae
Ken Honjo, Asami Tanaka, Katsuo Furukubo-Tokunaga
Grad. Schl. Life & Envir. Sci., Univ. Tsukuba

P3-g27 Involvement of a *Drosophila* transient receptor potential ion channel gene *painless* in long-term courtship memory

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P3-g28 Comprehensive behavioral characterization of mice lacking ICER, an endogenous antagonist of CREB

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P3-g29 多数および少数刺激セット条件の再認記憶の再学習に及ぼすサル嗅周皮質摘除の効果
Effect of perirhinal cortex lesion on relearning of recognition memory tasks with large and small sets of stimuli in monkey

靱負　正雄[1,2](Masao Yukie)，大沢　康隆[2](Yasutata Osawa)
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P3-g30 CREB の内在性アンタゴニスト ICER の過剰発現は恐怖記憶を阻害する
Overexpression of ICER, an endogenous antagonist of CREB, impairs fear memory

荒井　路子[1](M. Arai)，G. Borlikova[1]，児島　伸彦[2](N. Kojima)，遠藤　昌吾[1](S. Endo)
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P3-g31 ラット瞬目反射学習での同側小脳依存性
Dependency on the ipsilateral cerebellum in rat eyeblink conditioning

堀内　嵩大[1,2](Takahiro Horiuchi)，川原　茂敬[2](Shigenori Kawahara)
[1]東京大院・薬・神経生物物理，[2]富山大・工・物質生命科学

P3-g32 ラットにおける聴覚情報の短期記憶は視覚情報の短期記憶に比べて優れている
In the rat, auditory short-term memory is better than visual short-term memory

筒井　雄二(Yuji Tsutsui)，西澤　佳代(Kayo Nishizawa)，福士　紗織(Saori Fukushi)
福島大・共生システム理工学類・心理

P3-g33 サルの道具使用の概念形成の萌芽
Signs of generalization of tool-use in monkeys

平井　直樹[1](Naoki Hirai)，佐々木成人[2](Shigeto Sasaki)，内藤　公卿[2](Kimisato Naito)，
本郷　利憲[2](Toshinori Hongo)
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P3-g34 視覚弁別課題における OLETF の学習障害
Learning deficit of OLETF in visual discrimination task

菅　　理江[1](Rie Suge)，長谷川　元[2](Hajime Hasegawa)，島津　智一[3](Tomokazu Shimazu)
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P3-g35 光トポグラフィによる記憶の組織化に関わる脳機能の検討
Study of brain activation related to memory organization by Near-infrared spectroscopy

宮崎　　淳[1](Atsushi Miyazaki)，松井　三枝[1](Mie Matsui)，奈良原光隆[1](Mitsutaka Narahara)，
小林　恒之[2](Tsuneyuki Kobayashi)，西条　寿夫[2](Hisao Nishijo)
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P3-g36 ランダム遅延ネットワークにおける位相差検出
Detection of phase difference in random delay recurrent networks

山本　哲也(Tetsuya Yamamoto)
都立産技高専

P3-g37 種を超えて保存されている新規因子 TTX-8 の神経における機能解析
Studies on neuronal function of a novel and conserved protein TTX-8
宮良 晶子(Akiko Miyara), 太田 茜(Akane Ohta), 大河内善史(Yoshifumi Okochi),
奥村 将年(Masatoshi Okumura), 高野 良(Ryo Takano), 久原 篤(Atsushi Kuhara),
森 郁恵(Ikue Mori)
名大院・理・生命理学

P3-g38 オウム目鳥類の脳における聴覚記憶に関連した神経活性
Neuronal activation related to auditory memory in parrot brain
藤原 宏子[1](Hiroko Eda-Fujiwara), 佐藤 亮平[2](Ryohei Satoh), Johan J. Bolhuis[3],
M A (Thijs) Zandbergen[3], 宮本 武典[1](Takenori Miyamoto)
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P3-g39 線虫を用いた感覚情報の統合過程の遺伝学的解析
Genetic analyses of the sensory integration process in the neuronal circuits in *C. elegans*
新海 陽一(Yoichi Shinkai)
九州大院・理・生物科学

P3-g40 線虫の化学走性学習に異常を示す変異体の単利と解析
A suppressor screen for genes that regulate salt chemotaxis learning in *C. elegans*
安達 健[1](Takeshi Adachi), 富岡 征大[1](Masahiro Tomioka), 國友 博文[1](Hirofumi Kunitomo),
大河内善史[2,3](Yoshifumi Okochi), 森 郁恵[2](Ikue Mori), 飯野 雄一[1](Yuichi Iino)
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P3-g41 インターロイキン 1 の発現はミクログリア活性化による学習記憶障害の引き金となる
Involvement of IL-1 in LPS-induced learning and memory deficits
田中佐知子[1](Sachiko Tanaka), 近藤 宏明[1](Hiroaki Kondoh), 塩田 清二[2](Seiji Shioda),
沼澤 聡[1](Satoshi Numazawa), 吉田 武美[1](Takemi Yoshida)
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P3-g42 ラットの時間弁別行動に影響を及ぼすニコチン濃度の検討
Study of nicotine dose effects on temporal discrimination in rats
坂田 省吾(Shogo Sakata), 服部 稔(Minoru Hattori)
広島大院・総合科学・行動科学

P3-g43 オレキシンは脳内自己刺激における報酬効果を抑制する
Orexin attenuates the rewarding effect of intracranial self-stimulation in rats
畑 敏道[1](Toshimichi Hata), Jihuan Chen[2], 高橋 太郎[1](Taro Takahashi), Shuibo Gao[1],
石田 康[3](Yasushi Ishida), 伊達 紫[4](Yukari Date), 中原大一郎[1](Daiichiro Nakahara)
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P3-g44 MPK-1 MAP キナーゼの活性が線虫 *C. elegans* の味覚順応を調節する
Activity of MPK-1 MAP kinase regulates gustatory adaptation in *C. elegans*
江本由香子[1](Yukako Emoto), 大島 靖美[2](Yasumi Ohshima), 石原 健[1](Takeshi Ishihara),
古賀 誠人[1](Makoto Koga)
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P3-g45 ミツバチ嗅覚学習の長期記憶形成に関わるシグナル分子
CNG channel, calmodulin and CaMKII underlie olfactory long-term memory formation in the honeybee
松本 幸久[1](Yukihisa Matsumoto), Jean-Christophe Sandoz[2], Jean-Marc Devaud[2],
水波 誠[1](Makoto Mizunami), Martin Giurfa[2]
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## 認知 2
## Cognition 2

P3-g46 **前頭連合野背側部の帰納的推論への関与**
**Involvement of the dorsolateral prefrontal cortex in inductive reasoning**
佐藤　祐太(Yuta Sato)，山田　宗和(Munekazu Yamada)，飯島　敏夫(Toshio Iijima)，
筒井健一郎(Ken-Ichiro Tsutsui)
東北大院・生命科学・脳情報処理

P3-g47 **刺激呈示前におけるベータ帯域活動は刺激呈示後の顔処理時の特徴統合の成否を予測する.**
**Oscillatory beta activity predicts successful feature binding in face processing**
松本　　敦[1](Atsushi Matsumoto)，飯高　哲也[2](Tetsuya Iidaka)
[1]生理学研究所・心理生理，[2]名古屋大学・医・精神生物

P3-g48 **知覚あいまい性における同期皮質ネットワーク活動：MEG による研究**
**Synchronized cortical network activity of perceptual ambiguity: a magnetoencephalographic study**
南　　哲人[1](Tetsuto Minami)，村田　　勉[1](Tsutomu Murata)，矢野　史郎[1](Shiro Yano)，
宗綱　信治[1](Shinji Munetsuna)，鈴木　良次[1,2](Ryoji Suzuki)
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P3-h01 **興奮 / 抑制バランス入力の揺動成分を時間積分する神経回路モデル**
**Neural mechanisms for temporal integration of the fluctuating component of a balanced input**
岡本　　洋[1,2](Hiroshi Okamoto)，深井　朋樹[1](Tomoki Fukai)
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P3-h02 **Predictive control of saccadic eye movements during errors**
Sharika K.M., Aditya Murthy
NBRC, Manesar, India

P3-h03 **Does covert inhibition potentiate online inhibitory control?**
Aditya N. Murthy, Vishal Kapoor
National Brain Research Centre, Manesar, India

P3-h04 **Model-based reward prediction in the primate prefrontal cortex**
Xiaochuan Pan[1], Kosuke Sawa[2], Masamichi Sakagami[1]
[1]Research Institute, Tamagawa University, [2]Department of Psychology, Senshu University

P3-h05 **サル扁桃核内で異なる情動情報の表象**
**Differences in representation of emotional information among nuclei of monkey amygdala**
倉岡　康治[1,2](Koji Kuraoka)，中村　克樹[1,2](Katsuki Nakamura)
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P3-h06 **報酬に基づく意思決定における線条体アセチルコリン信号の役割**
**Cholinergic signal in the striatum during multi-step choice task for reward**
井之川　仁(Hitoshi Inokawa)，山田　　洋(Hiroshi Yamada)，木村　　實(Minoru Kimura)
京都府立医科大学 神経生理

P3-h07 **サルによる反復グループ逆転課題の遂行と帰納的推論**
**Performance of monkeys in a repeated group-reversal task requiring inductive reasoning.**
山田　宗和(Munekazu Yamada)，佐藤　祐太(Yuta Sato)，飯島　敏夫(Toshio Iijima)，
筒井健一郎(Ken-Ichiro Tsutsui)
東北大院・生命科学研究科・脳情報処理

P3-h08 バイオロジカルモーションによる視覚誘発脳磁図
Brain magnetic responses to biological motion
吉山 顕次[1](Kenji Yoshiyama), 中村 昭範[1](Akinori Nakamura),
Kersten Diers[1], 文堂 昌彦[1](Masahiko Bundo), 小野健太郎[1](Kentaro Ono), 森 理也[2](Yoshiya Mori),
服部 英幸[1](Hideyuki Hattori), 伊藤 健吾[1](Kengo Ito)
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P3-h09 ラット線条体における報酬確率の表現
Representation of reward probability in the rat striatum
小山 佳(Kei Oyama), 玄蕃 恵理(Eri Gemba), 飯島 敏夫(Toshio Iijima), 筒井健一郎(Ken-Ichiro Tsutsui)
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P3-h10 視覚ワーキングメモリの符号化, 保持, 検索に及ぼす COMT Val[158]Met 遺伝子多型の影響
Effect of catechol-*O*-methylferase (COMT) Val[158]Met genotype on encoding, maintenance, and retrieval of visual working memory
近藤 洋史[1](Hirohito M. Kondo), 野村 理朗[1,2](Michio Nomura), 柏野 牧夫[1,3,4](Makio Kashino)
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P3-h11 空間性、非空間性視覚刺激に応答する神経活動のサル前頭連合野背外側部における空間分布パターン
Spatial activation patterns of spatial and non-spatial visual information in primate DLPFC
新田 統昭(Motoaki Nitta), 船橋新太郎(Shintaro Funahashi)
京都大院・人環・共生人間

P3-h12 Go/no-go conditional discrimination task における前頭前野における活動
Prefrontal activities under a go/no-go conditional discrimination task
則武 厚(Atsushi Noritake), 小泉 昌司(Masashi Koizumi), 坂上 雅道(Masamichi Sakagami)
玉川大学・学術・脳科学研究施設

P3-h13 遅延自由選択課題における前頭連合野ニューロン活動
The effect of random delay length on prefrontal delay-period activity during free-choice ODR tasks
渡邉 慶(K Watanabe), 船橋新太郎(S Funahashi)
京都大学・人環・認知行動

P3-h14 手の身体モデルが対象物認知に及ぼす影響
Internal model of the human hand affects object recognition
片山 正純(Masazumi Katayama), 中山泰治郎(Taijirou Nakayama), 川治 誠明(Masaaki Kawaji)
福井大院・工・知能システム

P3-h15 音色のカテゴリー知覚の聴覚誘発 N1m 潜時への反映
Auditory evoked N1m latency reflects categorical perception of timbre
水落 智美[1](Tomomi Mizuochi), 湯本 真人[2](Masato Yumoto), 狩野章太郎[3](Shotaro Karino),
伊藤 憲治[4](Kenji Itoh), 山川 恵子[4](Keiko Yamakawa), 加我 君孝[1,3](Kimitaka Kaga)
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P3-h16 時間弁別課題中のサル線条体の手掛かり期の神経細胞活動
Cue response of monkey striatal neurons during a duration discrimination task
千葉 惇(Atsushi Chiba), 生塩 研一(Ken-ichi Oshio), 稲瀬 正彦(Masahiko Inase)
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P3-h17 マカクザルにおける頭頂葉から前頭前皮質への多シナプス性入力様式
Organization of multisynaptic inputs from parietal cortex to prefrontal cortex in macaque monkeys
平田 快洋[1](Yoshihiro Hirata), 宮地 重弘[2](Shigehiro Miyachi), 井上 謙一[1](Ken-ichi Inoue),
今西美知子[1](Michiko Imanishi), 高田 昌彦[1](Masahiko Takada)
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P3-h18 タスクチェンジを用いた把持位置の知覚と行動の独立性の解明
Independence of percepion and action by using a task change strategy
藤田　貴大(Takahiro Fujita)，前田　佳延(Yoshinobu Maeda)，片山　正純(Masazumi Katayama)
福井大院・工・知能システム

P3-h19 運動抑制における前頭前野機能の側方性
Laterality of human prefrontal cortex function during motor inhibition
楯　　勇人[1](Hayato Tabu)，美馬　達哉[2](Tatsuya Mima)，麻生　俊彦[2](Toshihiko Aso)，
澤本　伸克[2](Nobukatsu Sawamoto)，高橋　良輔[1](Ryousuke Takahashi)，福山　秀直[2](Hidenao Fukuyama)
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## 社会的行動
## Social Behavior

P3-h20 社会的隔離飼育がオススナネズミの社会行動および脳内神経栄養因子発現に与える影響
Effects of isolation rearing on the development of social behavior and central neurotrophin levels in male Mongolian gerbils
下鶴　倫人[1](Michito Shimozuru)，菊水　健史[2](Takefumi Kikusui)，武内ゆかり[1](Yukari Takeuchi)，
森　　裕司[1](Yuji Mori)
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P3-h21 C57BL/6J と MSM 系統から作出されたコンソミックマウス系統を用いた攻撃行動の遺伝学的解析
Genetic analysis of inter-male aggression using consomic mouse strains established from C57BL/6J and MSM
高橋　阿貴[1](Aki Takahashi)，富原　一哉[2](Kazuya Tomihara)，城石　俊彦[1](Toshihiko Shiroishi)，
小出　　剛[1](Tsuyoshi Koide)
[1]国立遺伝学研究所，[2]鹿児島大学

P3-h22 兵隊シロアリにおけるカースト特異的神経改変
Soldier-specific neural modification in termites
石川　由希(Yuki Ishikawa)，三浦　　徹(Toru Miura)
北海道大学大学院・環境科学院・生態遺伝学講座

P3-h23 母親の養育行動と ADP リボシルシクラーゼ活性とオキシトシン濃度との相関
ADP-ribosyl cyclase activity and oxytocin levels in relation to mouse maternal behavior
Olga Lopatina，小泉　恵太(Keita Koizumi)，Natalia Schnayder，劉　　鴻翔(Hong-Xiang Liu)，
金　　鐸(Duo Jin)，横山　　茂(Shigeru Yokoyama)，橋井美奈子(Minako Hashii)，
Mohammad Saharul Islam，Sarwat Amina，東田　陽博(Haruhiro Higashida)
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P3-h24 CD38 とサイクリック ADP リボースに非依存的なバゾプレシン遊離と社会認識行動
CD38-and cyclic ADP-ribose-independent vasopressin secretion in social behavior of mice
劉　　鴻翔(Hong Xiang Liu)，金　　鐸(Duo Jin)，Olga Lopatina，Natalia Schnayder，
平井　宏和(Hirokazu Hirai)，東田　陽博(Haruhiro Higashida)
金沢大・21世紀COE革新脳科学プログラム

P3-h25 ギャンブル課題におけるコモンマーモセットの意思決定
Decision-making of the common marmoset in gambling task
徳野　博信(Hironobu Tokuno)，田中いく子(Ikuko Tanaka)
東京都神経科学総合研究所

P3-h26 霊長類前頭前皮質ニューロンによる社会行動の自発的なカテゴライズ化
Spontaneous categorization of social behaviors in primate prefrontal cortical neurons
綱田　丈二[1,2](Joji Tsunada)，澤口　俊之[1,2](Toshiyuki Sawaguchi)
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P3-h27 **Pointing 行動に関連する運動前野ニューロン**
**Premotor neurons related to pointing behavior**
神代　真里[1,2,4,5](Mari Kumashiro)，佐藤　暢哉[3,4,5](Nobuya Sato)，勝山　成美[5](Narumi Katsuyama)，
泰羅　雅登[3,4,5](Masato Taira)，中村　克樹[1,3](Katsuki Nakamura)
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P3-h28 **内省未来志向のコーピングスタイルは右側頭領域の活動が関与している。**
**The right temporal region contributes to the self-prospective direction of stress coping style**
関口　敦[1,2](Atsushi Sekiguchi)，杉浦　元亮[1,3](Motoaki Sugiura)，横山　悟[1,4](Satoru Yokoyama)，
神原　利宗[1,4](Toshimune Kanbara)，生田　奈穂[1,5](Naho Ikuta)，佐藤　滋[4](Shigeru Satou)，
堀江　薫[4](Kaoru Horie)，川島　隆太[1](Ryuta Kawashima)
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P3-h29 **習慣的な反応の抑制の成功に関わる前頭前野の活動：fMRI 研究**
**Prefrontal cortex activities in the successful inhibition of habitual responses: an fMRI study**
門田　宏(Hiroshi Kadota)，中島八十一(Yasoichi Nakajima)，関口　浩文(Hirofumi Sekiguchi)，
河野　豊(Yutaka Kohno)，宮崎　真(Makoto Miyazaki)，神作　憲司(Kenji Kansaku)
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P3-h30 **自己呈示動機の神経基盤：fMRI 研究**
**Building a good reputation in others' eyes: the neural basis of the self-presentation motivation**
出馬　圭世[1,2](Keise Izuma)，齋藤　大輔[1,3](Daisuke Saito)，定藤　規弘[1,2,3,4](Norihiro Sadato)
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P3-h31 **お気に入りのブランド選びの神経機構**
**Neural mechanisms for preference-based decision making**
磯尾　綾子[1,2](Ayako Isoo)，上野　賢一[3](Kenichi Ueno)，田中　啓治[1](Keiji Tanaka)，
程　康[1](Kang Cheng)
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P3-h32 **味刺激による下前頭回の活性化**
**Ventrolateral prefrontal response to taste stimuli**
岡本　雅子(Masako Okamoto)，和田　有史(Yuji Wada)，續木　大介(Daisuke Tsuzuki)，Archana Singh，
檀　一平太(Ippeita Dan)
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P3-h33 **物 / 行動と背景状況の社会的関係認知：ｆＭＲＩによる検討**
**An fMRI study: Processing of item-situation relationship**
涌澤　圭介[1,2](Keisuke Wakusawa)，杉浦　元亮[1,3](Motoaki Sugiura)，
佐々　祐子[1,4](Yuko Sassa)，Hyeonjeong Jeong[1,5]，堀江　薫[6](Kaoru Horie)，佐藤　滋[6](Shigeru Sato)，
横山　浩之[2](Hiroyuki Yokoyama)，土屋　滋[2](Shigeru Tsuchiya)，川島　隆太[1,4](Ryuta Kawashima)
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P3-h34 **報酬－罰 Go/No-go 課題遂行中の前頭前野の活動と 5-HT2A 遺伝子多型性**
**5-HT2A receptor gene polymorphism modulates activation for monetary reward in the human ventrolateral frontal lobe during Go/No-go task**
野村　理朗[1,2](Michio Nomura)，近藤　洋史[2](Hirohito M. Kondo)，柏野　牧夫[2,3,4](Makio Kashino)
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P3-h35 ラットにおける回避学習の社会的伝達は学習経験に依存する
Social transmission in avoidance learning is dependent on learning experience in rats
増田　明(Akira Masuda)，粟生　修司(Shuji Aou)
九工大院・生命体工・高次脳機能

P3-h36 ミツバチと脊椎動物で保存されたUrbilateria遺伝子の脳機能進化への関与
The Urbilaterian genes implicated to underlie the evolution of advanced brain functions
門脇　辰彦[1](Tatsuhiko Kadowaki)，松井　利晃[1](Toshiaki Matsui)，土本麻友美[1](Mayumi Tsuchimoto)，
Wyder Stefan[2]，Zdobnov Evgeny[2]
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## パーキンソン病とその類縁疾患
## Parkinson Disease and Related Disorders

P3-h37 マグネシウム欠損飼育マウスにおけるMPTP神経毒性の感受性増大について
Enhanced susceptibility to MPTP neurotoxicity in magnesium-deficient C57BL/6 mice
稲中　牧子[1](Makiko Inaka)，室山　明子[1](Akiko Muroyama)，寺内　彩[1](Aya Terauchi)，
森　厚詞[2](Atsushi Mori)，杉野春日子[3](Haruhiko Sugino)，丸中　良典[3](Yoshinori Marunaka)，
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P3-h38 パーキンソン病モデルにおけるパラコートの細胞毒性に対する植物性エストロゲンの抑制効果。
Preventive effects of phytoestrogens on the paraquat-induced toxicities in the cellular model of Parkison disease.
久宝　真一(Shin-ichi Kyuhou)
関西医科大学・医・生理学第2講座

P3-h39 パーキンソン病視床β帯域活動と固縮の量的関係
Quantitative relationship of parkinsonian thalamic β-band activities with rigidity
大島　知一(Tomokazu Oshima)，楢林　洋介(Yohsuke Narabayashi)
楢林神経内科クリニック・楢林記念神経学ラボラトリー

P3-h40 ニューロシンはα-シヌクレインのNAC regionを切断する
Neurosin cleaves the NAC region of α-synuclein
笠井　高士[1](Takashi Kasai)，徳田　隆彦[1](Takahiko Tokuda)，水野　敏樹[1](Toshiki Mizuno)，
中川　正法[1](Masanori Nakagawa)，渡邊　義久[2](Yoshihisa Watanabe)，山口　希[2](Nozomi Yamaguchi)，
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P3-h41 ニコチン受容体刺激によるドーパミン作動性神経保護
Nicotinic receptor stimulation is protective for dopaminergic neuron.
竹内　啓喜[1](Hiroki Takeuchi)，青柳　信寿[1](Nobuhisa Aoyagi)，山川健太郎[4](Kentaro Yamakawa)，
高橋　良輔[1](Ryosuke Takahashi)，下濱　俊[2](Shun Shimohama)，赤池　昭紀[3](Akinori Akaike)，
澤田　秀幸[4](Hideyuki Sawada)，北村　佳久[5](Yoshihisa Kitamura)，谷口　隆之[5](Takashi Taniguchi)
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P3-h42 ドパミンキノン神経毒性におけるグルタチオン合成関連分子のグリアでの変化
Glutathione synthesis-related molecule in astrocytes in dopamine quinone neurotoxicity
宮崎　育子[1](Ikuko Miyazaki)，穂積　宏彰[2](Hiroaki Hozumi)，三好　耕[1](Ko Miyoshi)，
浅沼　幹人[1](Masato Asanuma)
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P3-h43 ドパミンキノン誘発神経障害に対するメタロチオネインの保護効果
Protective effects of metallothionein against dopamine quinone-induced neurotoxicity
浅沼　幹人[1](Masato Asanuma)，宮崎　育子[1](Ikuko Miyazaki)，三好　耕[1](Ko Miyoshi)，
穂積　宏彰[2](Hiroaki Hozumi)，十川　紀夫[3](Norio Sogawa)
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P3-h44 拘束ストレスはC57BL/6マウスにおけるMPTP神経毒性を増大する
MPTP neurotoxicity is enhanced by a restraint stress in C57BL/6 mice
和田　太輔[1,2](Daisuke Wada)，森　厚詞[1](Atsushi Mori)，中井　哲[1](Satoru Nakai)，
栗原　直樹[1](Naoki Kurihara)，大橋　聡[1](Satoshi Ohashi)，押村　光雄[2](Mitsuo Oshimura)，
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P3-h45 α-synucleinによる細胞脆弱性はカテコラミン代謝の抑制によって回避される
Inhibition of catecholamine metabolism reduces α-synuclein related cytotoxicity in PC12 cells
伊藤　悟(Satoru Ito)
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P3-h46 MPTP処理後MPTP高感受性マウスと低感受性マウスで認められ変化：行動、グリア、サイトカインに関する検討
Comparison between high and low MPTP sensitive mice after MPTP systematic treatment: alterations of behavior, glia and cytokines
安田ゐう子[1](Yuko Yasuda)，立石　成人[1](Narito Tateishi)，品川　理佳[2](Rika Shinagawa)，
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P3-h47 霊長類を用いたパーキン遺伝子治療
Advances in parkin gene therapy -a primate study-
安田　徹[1](Toru Yasuda)，井上　謙一[2](Kenichi Inoue)，山崎　吉之[2](Yoshiyuki Yamazaki)，
仁平　友子[1](Tomoko Nihira)，高田　昌彦[2](Masahiko Takada)，島田　隆[3](Takashi Shimada)，
望月　秀樹[1](Hideki Mochizuki)，水野　美邦[1](Yoshikuni Mizuno)
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P3-h48 メダカにおける*parkin*ノックアウトモデルの作成
Development of a Parkinson disease model by *parkin* gene knockout in medaka
小林　芳人[1](Yoshito Kobayashi)，高橋　良輔[1](Ryosuke Takahashi)，谷口　善仁[2](Yoshihito Taniguchi)，
武田　俊一[2](Shunichi Takeda)
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P3-j01 $MPP^+$又は6-OHDAによるCdk5/p35の分解制御と神経細胞死
Involvement of p35 Cdk5 activator metabolism in $MPP^+$ or 6-OHDA-induced neuronal cell death
遠藤　良(Ryo Endo)，斎藤　太郎(Taro Saito)，浅田　明子(Akiko Asada)，久永　真市(Shin-ichi Hisanaga)
首都大学東京　理工学研究科　生命科学専攻

P3-j02 ドパミンアゴニストの6-OHDA由来小胞体ストレス抑制作用の分子機序解明
Molecular basis of anti-apoptotic effect of D2 agonists against 6-OHDA-induced ER stress
田中　健一(Ken-ichi Tanaka)，小郷　裕也(Hiroya Ogo)，東　幸司(Koji Azuma)，
難波　雄(Takeshi Nanba)，八木　崇夫(Takao Yagi)
就実大・薬・薬物治療
（＊P3-j02は発表者の都合により，9月11日にP2-k26として発表されます。）

P3-j03 Phosphorylated alpha-synuclein contributes to accumulation of ubiquitinated proteins
Guanghui Wang[1], Chao Liu[1], Erkang Fei[1], Nali Jia[1], Hongfeng Wang[1], Ruisong Tao[1], Atsushi Iwata[2], Nobuyuki Nukina[2]
[1]Department of Neurobiology, University of Science & Technology of China, Anhui, China, [2]RIKEN Brain Science Institute, Saitama, Japan

P3-j04 Microglial migration is up-regulated by alpha-synuclein-mediated induction of CD44 and MT1-MMP in Parkinson's disease
Kim Seonghan, Seo-Hyun Cho, Ka Young Kim, Yoo-Hun Suh
Department of Pharmacology, Seoul National University, College of Medicine, Seoul, Korea

P3-j05 Genetic Polymorphism in susceptibility genes for PD
Madhu Singh[1], R Shukla[2], V_K Khanna[1], D Parmar[1]
[1]ITRC, Lucknow, India, [2]KGMU, India

P3-j06 核内受容体 Nurr1 は中脳黒質ドーパミンニューロンの機能維持に必須である
Nurr1 is essential for maintenance of the dopaminergic phenotype in the nigro-striatal dopaminergic neurons
伊藤　岳人[1](Takehito Ito), 村松　慎一[2](Shin-ichi Muramatsu), 小澤　敬也[3](Keiya Ozawa),
Daniel Metzger[4], Pierre Chambon[4], 一瀬　宏[1](Hiroshi Ichinose)
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P3-j07 MPTP 処置 C57BL/6 マウスのドパミン神経変性に対する水溶化 $CoQ_{10}$ の保護効果
Protective effect of water-soluble coenzyme $Q_{10}$ on dopaminergic neurodegeneration in MPTP-treated C57BL/6 mice
松島　大章[1](Hiroaki Matsushima), 杉山　朋美[1](Tomomi Sugiyama), 内之倉史朗[1](Shiro Uchinokura),
福田紗弥香[1](Sayaka Fukuda), 吉村　育生[2](Ikuo Yoshimura), 光本　泰秀[1](Yasuhide Mitsumoto)
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P3-j08 PC12 細胞においてドパミン, L-ドパ, キノン体は凝集体形成を促進する
Enhanced aggregate formation by dopamine, L-dopa, and quinone in PC12 cells
中曽　一裕(Kazuhiro Nakaso), 伊藤　悟(Satoru Ito), 吉本　祐子(Yuko Yoshimoto),
今村　恵子(Keiko Imamura), 竹島多賀夫(Takao Takeshima), 中島　健二(Kenji Nakashima)
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## 中毒と炎症
## Neurotoxicity and Inflammation

P3-j09 アジュバント関節炎ルイスラットにおける亜鉛摂取の効果
Effects of zinc by food on adjuvant arthritis in Lewis rats
笠井　聖仙(Masanori Kasai), 七田　将人(Masato Shichita), 児崎　達哉(Tatsuya Kozaki)
鹿児島大・理・生命化学

P3-j10 フロン代替化学物質 1 －ブロモプロパンのシナプス受容体、細胞内シグナル伝達および局所回路への影響
Effects of 1-bromopropane on neurotrasmitter receptors, intracellular signalings and feedback inhibition in the CNS
笛田由紀子(Yukiko Fueta), 吉田　安宏(Yasuhiro Yoshida), 上野　晋(Susumu Ueno),
石田尾　徹(Toru Ishidao), 保利　一(Hajime Hori)
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P3-j11 エタノールによる中枢神経細胞の障害は焙煎米糠及び大豆抽出エキスで緩和される
Cellular damages on the CNS neurons treated with ethanol are alleviated by roasted rice bran and soybean extracts
塩崎　元子[1](Motoko Shiozaki), 早川　直哉[1](Naoya Hayakawa), 竹内　翔一[1](Syoichi Takeuchi),
和田　清孝[3](Kiyotaka Wada), 内山　安男[2](Yasuo Uchiyama), 後藤　隆洋[1](Takahiro Gotow)
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P3-j12 アストロサイトとミクログリアにおける EAATs 発現の差異
Comparison research on excitatory amino acid transporters between astrocyte and microglia
梁　剣峰(Jianfeng Liang), 竹内　英之(Hideyuki Takeuchi), 川ノ口　潤(Jun Kawanokuchi),
薗部　佳史(Yoshifumi Sonobe), 土井由紀子(Yukiko Doi), 八幡　泉(Izumi Yawata), 金　世杰(Shijie Jin),
王　金岩(Jinyan Wang), 水野　哲也(Tetsuya Mizuno), 錫村　明生(Akio Suzumura)
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## てんかん
## Epilepsy

P3-j13 ラット海馬スライスにおいてアセチルコリン受容体の活性化がてんかん様発火に与える影響
The effect of acetylcholine receptor activation on the epileptogenesis in rat hippocampal slices
橋本あゆみ(Ayumi Hashimoto)，夏目季代久(Kiyohisa Natume)
九州工業大学大学院・生命体工学研究科・脳情報専攻

P3-j14 Neuronal activities in the parietal cortex of an EL and DDY mouse
Jiro Suzuki[1,2], N Ozawa[2], YL Murashima[2], T Shinba[2], M Yoshii[2]
[1]Sannou Institute of Psychiatry and Psychology, Tokyo, Japan, [2]Tokyo Institute of Psychiatry

P3-j15 ENU ミュータジェネシスにより作製された Scn1a 遺伝子変異ラット
Scn1a mutant rats established by gene-driven ENU mutagenesis
真下　知士[1](Tomoji Mashimo)，徳田　智子[1](Satoko Tokuda)，鶴見東志子[1](Toshiko Tsurumi)，
滝沢　明子[1](Akiko Takizawa)，柳原　克彦[2](Katsuhiko Yanagihara)，平林　真澄[3](Masumi Hirabayashi)，
庫本　高志[1](Takashi Kuramoto)，芹川　忠夫[1](Tadao Serikawa)
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P3-j16 Ca チャネルミュータントマウスにおける経路依存的 NMDA 受容体電流の増加
Pathway-specific enhancement of NMDA-mediated currents in $Ca^{2+}$-channel mutant mice, *tottering*
濱田 - 佐々木幸恵[1](Sachie Hamada-Sasaki)，井上　剛[2](Tsuyoshi Inoue)，井本　敬二[2](Keiji Imoto)
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P3-j17 GABAA 受容体輸送と IP3 結合能を有するタンパク（PRIP-1）欠損マウスのフェノタイプ解析とその発症機構の検討
Phenotype analysis of PRIP1-Knock out mouse
上野　伸哉[1](Shinya Ueno)，朱　剛[2](Gang Zhu)，森　文秋[2](Fumiaki Mori)，
若林　孝一[2](Kouichi Wakabayashi)，吉田　淑子[3,4](Shukuko Yoshida)，上村雄一郎[5](Yuichiro Kamimura)，
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P3-j18 神経型一酸化窒素合成酵素依存的な恒常的一酸化窒素は抗痙攣作用を有する
A higher sensitive pentylenetetrazole-indued seizures in nNOS deficient mice
渡邊　正知(Masatomo Watanabe)，相川　義勝(Yoshikatsu Aikawa)，伊藤　康一(Kouichi Itoh)
徳島文理大学・香川薬・分子薬理

P3-j19 ENU 誘発突然変異により作出された痙攣、無動発作を示す突然変異マウスの遺伝解析
Genetic analysis of an ENU-induced mutant that shows convulsive seizure and immobility
古瀬　民生[1](Tamio Furuse)，和田由美子[1](Yumiko Wada)，山田　郁子[1](Ikuko Yamada)，
桝屋　啓志[1](Hiroshi Masuya)，金田　秀貴[1](Hideki Kaneda)，小林喜美男[1](Kimio Kobayashi)，
三浦　郁生[1](Ikuo Miura)，串田　知子[1](Tomoko Kushida)，渋川　陽子[1](Yoko Shibukawa)，
兼子　幸一[2](Koichi Kaneko)，若菜　茂晴[1](Shigeharu Wakana)，城石　俊彦[1](Toshihiko Shiroishi)
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P3-j20 ウサギ海馬のてんかん様発作波誘発閾値に対する電気凝固の効果
Effects of electrical coagulation on the threshold of electrically induced paroxysmal discharge in the rabbit hippocampus CA1
松田　芳樹(Yoshiki Matsuda)，斉藤　伸明(Nobuaki Saito)，高橋　聡史(Satoshi Takahashi)，
木暮　信一(Shinichi Kogure)
創価大・工・生命情報工

P3-j21 若年性ミオクロニーてんかんから見つかったEFHC1 short isoformの変異
EFHC1 short isoform-specific mutations in patients with epilepsy
鈴木 俊光[1,2](Toshimitsu Suzuki), Antonio V. Delgado-Escueta[3], 山川 和弘[1](Kazuhiro Yamakawa)
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P3-j22 てんかん発火活動によるinterictal波周波数調節
The frequency of interictal activity was modulated by the generation of the activity per se
緒方 元気(Genki Ogata), 夏目季代久(Kiyohisa Natsume)
九州工業大学大学院・生命体工学研究科

P3-j23 連続ウェーブレット変換と人工ニューラルネットワークを用いたてんかん発作脳波の特徴抽出と発作頻度の解析
Detection and classification of epileptic seizure using continuous wavelet transform and artificial neural network
都丸 千夏[1](Chinatsu Tomaru), 飯島 一憲[1](Kazunori Iijima), 大和田-真壁恭子[1](Kyoko Owada-Makabe),
坪田 裕司[2](Yuji Tsubota)
[1]群馬高専・物質工学, [2]大阪河崎リハ大・生理学

P3-j24 てんかんモデルラットにおけるエリスロポエチンのてんかん抑制効果
Erythropoietin prevents the epileptogenesis and generalization in the epilepsy model rats
近藤 聡彦(Akihiko Kondo), 新郷 哲郎(Tetsuro Shingo), 蔵本 智士(Satoshi Kuramoto),
安原 隆雄(Takao Yasuhara), 上利 崇(Takashi Agari), 三好 康之(Yasuyuki Miyoshi),
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## 発達障害 Developmental Disorders

P3-j25 PQBP1の細胞周期への関連性について
PQBP1 and cell cycle arrest
高橋 慧子(Keiko Takahashi), Hong Luo, 田川 一彦(Kazuhiko Tagawa),
安達(玉盛)三美(Mimi Tamamori-Adachi), 北嶋 繁孝(Shigetaka Kitajima), 岡澤 均(Hitoshi Okazawa)
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P3-j26 PACAP遺伝子欠損マウスの異常行動発現における環境因子の役割
Involvement of environmental factors in the expression of psychomotor abnormalities in PACAP knock-out mice
石濱 壽紘[1](Toshihiro Ishihama), 吾郷由希夫[1](Yukio Ago), 新谷 紀人[2](Norihito Shintani),
角田 享也[3](Michiya Kakuda), 橋本 均[2,3](Hitoshi Hashimoto), 馬場 明道[2](Akemichi Baba),
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P3-j27 胎生期にアルコール投与されたラットの注意機能障害
Attention deficits induced by prenatal alcohol exposure in rats
田丸 政男[1](Masao Tamaru), 福居 玲奈[1](Rena Fukui), 平田ゆかり[2](Yukari Hirata)
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P3-j28 注意欠陥/多動性障害モデルラットが示す注意機能障害におけるニコチン受容体の関与
The role of nicotinic acetylcholine receptors on attentional dysfunction of an animal model for attention-deficit/hyperactivity disorder
山口 拓[1](Taku Yamaguchi), 富樫 廣子[1,2](Hiroko Togashi), 大村 優[1](Yu Ohmura),
泉 剛[1](Takeshi Izumi), 松本真知子[1,2](Machiko Matsumoto), 吉岡 充弘[1](Mitsuhiro Yoshioka)
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P3-j29 広汎性発達障害の眼球運動
Eye movements in Pervasive developmental disorder (PDD)
福島　順子[1](Junko Fukushima)，七戸　夏子[2](Natsuko Shichinohe)，福島　菊郎[2](Kikuro Fukushima)
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P3-j30 甲状腺ホルモン受容体ノックアウトマウスにおける行動学的、形態学的異常の検討
Behavioral and histological examinations in TR-alpha or TR-beta deficient mice
定松　美幸[1](Miyuki Sadamatsu)，金井　裕彦[2](Hirohiko Kanai)，加藤　進昌[3](Nobumasa Kato)
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P3-j31 TSC2-Rheb-mTOR 経路はスパインの形態を制御する
TSC2-Rheb-mTOR pathway regulates dendritic spine morphology
杉浦　弘子[1](Hiroko Sugiura)，安田　新[1](Shin Yasuda)，吉村　好之[2](Yoshiyuki Yoshimura)，
竹宮　孝子[1](Takako Takemiya)，山内　卓[2](Takashi Yamauchi)，山形　要人[1](Kanato Yamagata)
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P3-j32 プルキンエ細胞の細胞質に局在する FoxP2 の結合蛋白 CtBPs
CtBPs are binding proteins of the novel isoform of FoxP2 lacking forkhead domain
藤田恵理子[1](Eriko Fujita)，松崎　鮎美[1,2](Ayumi Matsuzaki)，田辺　裕子[1](Yuko Tanabe)，
笠原　忠[2](Tadashi Kasahara)，桃井真里子[3](Mariko Momoi)，桃井　隆[1](Takashi Momoi)
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P3-j33 マウスプルキンエ細胞質に局在する新規 Foxp2 の同定
Identification of a novel isoform of Foxp2 located in the cytoplasm of mouse developing Purkinje cells
松崎　鮎美[1,2](Ayumi Matsuzaki)，田辺　裕子[1](Yuko Tanabe)，藤田恵理子[1](Eriko Fujita)，
笠原　忠[2](Tadashi Kasahara)，桃井　隆[1](Takashi Momoi)
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P3-j34 脳における RA175/SynCAM 結合蛋白の解析
Brain Proteins with PDZ domains associated with RA175/SynCAM
田辺　裕子[1](Yuko Tanabe)，松崎　鮎美[1](Ayumi Matsuzaki)，藤田恵理子[1](Eriko Fujita)，Giulo Piluso[2]，
大野　茂男[3](Shigeo Ohno)，石浦　章一[4](Shouichi Ishiura)，Alaa Hussein[5]，Vencenzo Nigro[2]，
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P3-j35 Perinatal BPA exposure impairs synaptic architectures and functions in the hippocampus of offspring
Ling Chen[1]，Masahiro Sokabe[2,3]
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## 虚血
## Ischemia

P3-j36 神経細胞における虚血再灌流時のミトコンドリア ATP 感受性 K チャンネル開口による効果
Effects of Mitochondrial ATP-sensitive K Channel Opening during ischemia/reperfusion in neurons
松岡　信広[1](Nobuhiro Matsuoka)，山田　茂行[1](Shigeyuki Yamada)，雫石　正明[1](Masaaki Shizukuisi)，
風間　富栄[1](Tomiei Kazama)，小林　靖[2](Yasushi Kobayashi)，石田　英之[3](Hideyuki Ishida)
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P3-j37 虚血負荷に対するエチルピルビン酸の脳保護作用はグリア細胞に依存する：$^{31}$P-NMR による検討
Neuroprotective effects of ethyl pyruvate on brain energy metabolism after ischemia depend on glial cells: a $^{31}$P-NMR study
徳丸　治[1](Osamu Tokumaru)，黒木　千尋[1](Chihiro Kuroki)，北野　敬明[2](Takaaki Kitano)，
西丸　直子[1](Naoko Nisimaru)，横井　功[1](Isao Yokoi)
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P3-j38 ミトコンドリア膜透過性亢進抑制薬の評価
Mitochondrial permeability transition: evaluation of putative "pore blockers"
諸田　沙織[1,2](Saori Morota)，Magnus_J Hansson[3]，Roland Mansson[3]，工藤　佳久[1](Yoshihisa Kudo)，
石井　脩夫[2](Nagao Ishii)，Eskil Elmer[3]，内野　博之[2](Hiroyuki Uchino)
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P3-j39 Identification and characterization of protein marker(s) for diagnosis and prognosis of acute Ischemic stroke patients
R S Kashyap[1]，A Nayak[1]，P Deshpande[1]，HJ Purohit[2]，GM Taori[1]，HF Daginawala[1]
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P3-j40 低酸素脳虚血によるラット海馬プロテオグリカン発現の変化
Expression of the proteoglycan in rat hippocampus with hypoxia-ischemia insult
山口　昌俊(Masatoshi Yamaguchi)，池ノ上　克(Tsuyomu Ikenoue)
宮崎大学・医・産婦人科

P3-j41 実験的脳梗塞ラット急性期における HMGB1 の発現について
The expression of high mobility group box 1 (HMGB1) after acute cerebral infarction in rats
松田　史代[1](Fumiyo Matuda)，菊池　清志[2](Kiyoshi Kikuchi)，川原　幸一[2](Ko-ichi Kawahara)，
榊間　春利[3](Harutoshi Sakakima)，丸山　征郎[2](Ikuro Maruyama)，吉田　義弘[1](Yoshihiro Yoshida)
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P3-j42 ラット中大脳動脈一次閉塞モデルにおけるヘパラナーゼの発現
Expression of heparanase in rat brain after transient middle cerebral artery occlusion
高橋　寿明[1](Hisaaki Takahashi)，松本　裕明[2](Hiroaki Matsumoto)，Anna Smirkin[1]，
今井　嘉紀[1](Yoshinori Imai)，田中　潤也[1](Junya Tanaka)
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P3-j43 リコピンを摂取したネズミでは脳虚血負荷後の海馬神経細胞のアポトーシスが抑制される
Lycopene uptake to Mongolian gerbil attenuates apoptosis in hippocampus induced by ischemia
藤田　公和[1](Kimikazu Fujita)，芳本　信子[2](Nobuko Yoshimoto)，加藤　寿章[3](Toshiaki Kato)，
山根　理学[4](Masataka Yamane)，稲熊　隆博[4](Takahiro Inakuma)，永田　豊[1](Yutaka Nagata)，
宮地　栄一[1](Eiichi Miyachi)
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P3-j44 NG2 プロテオグリカンを発現するマクロファージ様細胞の虚血脳における役割
Roles of macrophage-like cells expressing NG2 proteoglycan in ischemic rat brain
田中　潤也[1](Junya Tanaka)，松本　洋明[2](Hiroaki Matsumoto)，高橋　寿明[1](Hisaaki Takahashi)，
Anna Smirkin[1]，今井　嘉紀[1](Yoshinori Imai)，久門　良明[2](Yoshiaki Kumon)，大西　丘倫[2](Tkanori Ohnishi)
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P3-j45 一過性脳虚血後の梗塞巣形成への 1 型代謝型グルタミン酸受容体を介した NMDA 受容体チロシンリン酸化の関与
Involvement of tyrosine phosphorylation of the NMDA receptor via metabotropic glutamate receptor 1 in infarction after middle cerebral artery occlusion and reperfusion
室冨　和俊(Kazutoshi Murotomi)，高木　教夫(Norio Takagi)，高柳　元(Gen Takayanagi)，
竹尾　聰(Satoshi Takeo)
東京薬大・薬・分子細胞病態薬理

P3-j46 Change and translocation of HSP60 in gerbil hippocampal CA1 region induced by transient ischemia and its protective effect against ischemic damage

In Koo Hwang[1], Ki-Yeon Yoo[2], Jung Hoon Choi[1], In Se Lee[1], Yeo Sung Yoon[1], Je Kyung Seong[1], Moo Ho Won[2]
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P3-j47 Time dependent progesterone-nouroprotection against ischemic brain damages

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## 分子生物学的、細胞生物学的方法
## Molecular and Cellular Biological Techniques

P3-j48 トランスジェニックマウスを利用した逆行性シナプス輸送トレーサーの開発
Inducible expression of retrograde transynaptic genetic tracer in mice

佐野　裕美[1,2](Hiromi Sano), 横井　峰人[1,2](Mineto Yokoi)
[1]京大・医・先端領域, [2]CREST・科学技術振興機構

P3-k01 アメフラシ感覚ニューロンへの光活性化アデニル酸シクラーゼの機能移植
Functional transplant of photoactivated adenylyl cyclase into *Aplysia* mechanosensory neurons

長濱　辰文[1](Tatsumi Nagahama), 鈴木　武士[2](Takeshi Suzuki), 吉川　伸哉[3](Shinya Yoshikawa), 伊関　峰生[2](Mineo Iseki)
[1]東邦大・薬・生物物理, [2]科学技術振興機構・さきがけ／基生研, [3]福井県立大・海洋生物資源

P3-k02 マウス青斑核において選択的に発現する遺伝子転写産物の同定と性格づけ
Identification and characterization of transcripts selectively expressed in the mouse locus coeruleus (LC)

中村　浩章[1](Hiroaki Nakamura), 横川　健[1](Ken Yokokawa), 石井　祥之[1](Yoshiyuki Ishii), 佐藤　靖史[2](Yasufumi Sato), 小林　和人[3](Kazuto Kobayashi), 井樋　慶一[1](Keiichi Itoi)
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P3-k03 簡便型光照射分子機能阻害法の適用
Simple, easy and long-term protein ablation method: an application of fluorophore-assisted light inactivation to cell. explant and organotypic cultures of the nervous system

山口めぐみ[1](Megumi Yamaguchi), 佐藤　泰史[1,2](Yasufumi Sato), 池谷　真澄[1](Masumi Iketani), 中村　史雄[1](Fumio Nakamura), 五嶋　良郎[1,2](Yoshio Goshima), 竹居光太郎[1,2](Kohtaro Takei)
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P3-k04 改変狂犬病ウイルスベクターを用いた神経回路の二重標識
Dual viral transneuronal tracing using recombinant rabies virus vectors

大原　慎也[1,2](Shinya Ohara), 井上　謙一[3](Ken-ichi Inoue), 山田　真広[1](Masahiro Yamada), 筒井健一郎[1](Ken-Ichiro Tsutsui), 飯島　敏夫[1,2](Toshio Iijima)
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P3-k05 複数閾値を用いた新たな細胞計数法
A novel cell count method with multiple thresholds

和田　真[1,2](Makoto Wada), 谷口　達樹[3](Tatsuki Taniguchi), 北澤　茂[1,2,4](Shigeru Kitazawa)
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P3-k06 高効率マイクロインジェクション法の開発
An efficient gene delivery system into cells using a nano-size silicon needle

原　央子[1,2](Chikako Hara), 館山　清彦[3](Kiyohiko Tateyama), 岡野　栄之[1](Hideyuki Okano), 宮脇　敦史[2](Atsushi Miyawaki)
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P3-k07 サブタイプ特異的リガンドセンサー：神経伝達物質受容体結合タンパク質の利用
Subtype-specific ligand sensors: utilization of neurotransmitter binding proteins
久保　泰[1](Tai Kubo)，小林寿珠子[1](Suzuko Kobayashi)，野口　悠紀[1](Yuuki Noguchi)，Bong-Kiun Kaang[2]
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P3-k08 The morphogenesis and trans-neuronal transfer of swine hemagglutinating encephalomyelitis virus in the central nervous system
Yan-chao Li[1]，Wan Zhu Bai[1]，Norio Hirano[2]，Koujiro Tohyama[1,3]，Tsutomu Hashikawa[1]
[1]Laboratory for Neural Architecture, RIKEN Brain Science Institute, [2]Dept. Veterinary Microbiology, Iwate Univ., Morioka, Japan, [3]Bio-Imaging Center, Iwate Medical Univ., Morioka, Japan

P3-k09 Investigate the function of neural circuits in Drosophila using light-activating ion channels
Wei Zhang
Institute of Neuroscience, Chinese Academy of Sciences, Shanghai, China

## その他
## Others

P3-k10 Non-parametric resampling-based methods for functional NIRS studies
Archana K. Singh[1]，Masako Okamoto[1]，James B. Cole[2]，Ippeita Dan[2]
[1]Sensory and cognitive Science Lab, NFRI, Tsukuba, Japan, [2]National Food Research Institute

P3-k11 昔の日本・中国で「脳」はどのようなものと捉えられていたか：漢字「脳」の意味から考える
What was the brain for historical Japanese or Chinese; Examination of the Chinese letter (Kanji) of the brain in literature
香山　雪彦[1](Yukihiko Kayama)，Robert W. Doty[2]
[1]福島県立医大・医・神経生理，[2]ロチェスター大学（米国）

P3-k12 TMS/EEG における ICA を用いたアーチファクト除外手法の確立
Independent component analysis applied to the removal of artifacts in combined TMS/EEG recordings
竹内　成生(Shigeki Takauchi)，中島八十一(Yasoichi Nakajima)，門田　宏(Hiroshi Kadota)，
関口　浩文(Hirofumi Sekiguchi)，神作　憲司(Kenji Kansaku)
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P3-k13 Alkmaion, ca 500 BC, discovers that mind is in the brain
Robert W. Doty
Dept Neurobiology & Anatomy, Univ Rochester, Rochester, New York, U.S.A.

P3-k14 メチコバール（MTB）の培養ラット胎児への影響
Effects of methycobal(MTB) on rat embryos cultured in vitro
横山　篤[1,2](Atsushi Yokoyama)，秋田　正治[3](Masaharu Akita)
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# 抄録 Abstracts


## プレナリーレクチャー
Plenary Lectures

## 時実利彦記念賞受賞講演
Tokizane Award Lecture

## 塚原仲晃記念賞受賞講演
Tsukahara Award Lectures

## シンポジウム
Symposia

## PL1 Glycine neurotransmission: from molecules to mice and disease

Heinrich Betz

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Glycine is a major inhibitory neurotransmitter in the mammalian CNS. Upon $Ca^{2+}$-triggered release from glycinergic nerve terminals, glycine causes postsynaptic inhibition by binding to strychnine-sensitive glycine receptors (GlyRs) which exist in different ( $\alpha$ 1- $\alpha$ 4) isoforms. Synaptic localization of GlyRs requires the scaffolding protein gephyrin which also is found at GABAergic synapses. Glycinergic neurotransmission is terminated by the reuptake of glycine into glycinergic nerve terminals and neighbouring glial cells via the $Na^+$-/$Cl^-$-dependent glycine transporters, GlyT1 and GlyT2. We have used gene targeting to dissect the physiological functions of distinct GlyR subtypes and GlyT isoforms. Accordingly, $\alpha$ 1 GlyRs are mainly involved in motor control, whereas $\alpha$ 3 GlyRs are crucial for pain sensitization. Inactivation of the gephyrin gene causes lethality due to a loss of postsynaptic GlyR (and $GABA_A$ receptor) clustering. Similarly, ablation of the GlyT1 and GlyT2 genes is postnatally lethal. Our results underline the vital roles of glycinergic neurotransmission and mutant mice deficient in distinct GlyR and establish GlyT isoforms as animal models of hereditary human disorders caused by mutations in GlyR, gephyrin and GlyT genes.

## PL2 Signaling networks that control synapse development and cognitive function

Michael E. Greenberg

*Neurobiology Program Children's Hospital and Harvard Medical School, Boston, USA*

Experience plays a crucial role in the development of the nervous system by promoting the maturation, and elimination of synapses. We have found that synaptic stimulation by promoting calcium influx into neurons regulates a complex gene program that controls aspects of synapse development. We have identified a signaling network that conveys the calcium signal from the site of entry to the nucleus where transcription is activated. Of the activity-regulated genes, brain derived neurotrophic factor (BDNF) is one of the best characterized. Studies of BDNF gene regulation indicate the presence of a repressor complex bound to the BDNF promoter prior to stimulation that becomes replaced by an activator complex in stimulated cells. Mutations in MeCP2 a component of the BDNF repressor complex lead to Rett Syndrome an X linked human disorder marked by severe cognitive and motor impairment. Mutations in additional components of the activity-dependent signaling network also lead to cognitive impairment in humans indicating a critical role for this gene program in controlling normal cognitive function.

## PL3 Frontal lobe mechanisms for rules and control

Earl K. Miller

*The Picower Institute for Learning and Memory, RIKEN-MIT Neuroscience Research Center, and Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology*

What controls your thoughts? How do you focus attention? How do you know how to act while dining in a restaurant? This is cognitive control, the ability to organize thought and action around goals. Results from our laboratory have shown that neurons in the prefrontal cortex and related brain areas have properties commensurate with a role in "executive" brain function. They are involved in directing attention, in recalling stored memories, predicting reward value, and they integrate the diverse information needed for a given goal. Perhaps most importantly, they transmit acquired knowledge. Their activity reflects learned task contingencies, concepts and rules. In short, they seem to underlie our internal representations of the "rules of the game". This may provide the foundation for the complex behavior of primates, in whom this structure is most elaborate.

## SL1 Metabolism of amyloid $\beta$ peptide and Alzheimer's disease

Takaomi C. Saido

*Laboratory for Proteolytic Neuroscience, RIKEN Brain Science Institute, Wako-shi, Japan*

The conversion of normal brain aging to Alzheimer's disease (AD) via a transition state is a continuous and chronic process caused primarily by accumulation of amyloid $\beta$ peptide (A $\beta$) in the brain. A $\beta$ is constantly produced from its precursor and immediately catabolized under normal conditions, whereas dysmetabolism of A $\beta$ seems to lead to pathological deposition. We have focused our attention on elucidation of the unresolved mechanism of A $\beta$ catabolism *in vivo*. We first identified neprilysin as a major A $\beta$ -degrading enzyme, the aging-dependent reduction of which seems to account for sporadic AD pathogenesis. Neprilysin stands out among all the candidate clearance mechanisms because only neprilysin can degrade A $\beta$ at synapses. We then discovered that somatostatin activates neuronal neprilysin, suggesting that somatostatin receptor subtype(s) may be a relevant pharmacological target. We also found that calpain is involved in AD pathology. Combining medications targeting at up-, mid- and down-stream processes of disease cascade shall be the most effective strategy for prevention and treatment of AD.

## SL2 In pursuit of the brain mechanism of reinforcement learning

Kenji Doya

*Okinawa Institute of Science and Technology, ATR Computational Neuroscience Laboratories*

Reinforcement learning is a theoretical framework for learning novel behaviors on the basis of reward signals. We have been tackling the dual problems of how we can realize robust and flexible reinforcement learning as the brain does, and how the brain realizes reinforcement learning by its circuit and molecular machinery. Pioneering works by W. Schultz, J. Wickens, and O. Hikosaka inspired us with a hypothesis on how the cortico-basal ganglia circuit can realize reward prediction and action selection. We have been fortunate to work with M. Kimura on monkey experiments to test the hypothesis, with a positive result. Our experiences in robot experiments taught us the critical roles of 'meta-parameters' that regulate the learning process, and lead us to a hypothesis on how meta-parameters can be controlled by neuromodulators, most notably serotonin. We are testing the hypothesis in human brain imaging with S. Yamawaki and rat recording experiments we freshly started in Okinawa. Our future goal is to give a computational account on why the brain came up with the circuit and molecular mechanisms to realize reinforcement learning.

## SL3 Innovations in fluorescence imaging of brain functions using fluorescent proteins

Atsushi Miyawaki

*RIKEN BSI*

Understanding mechanisms by which neurons process and integrate synaptic inputs, as well as how these mechanisms are modified by activity, is a central challenge in neuroscience. The striking progress in genome science and gene technology has led to numerous discoveries and the development of new technologies, including fluorescence imaging using fluorescent proteins. I have developed genetically-encoded probes by fusing fluorescent proteins to functional proteins that are involved in physiological signaling. The probes are introduced into an intact organism and targeted to specific tissues, cell types, or subcellular compartments, thereby allowing specific signals to be extracted more efficiently than was previously possible. Of particular interest are neuronal mechanisms that may be responsible for regulating signal localization and for controlling the transitions from local to global, and from transient to constitutive signals. I will discuss how the probes have advanced our understanding of the spatio-temporal regulation of biological functions inside neurons and brains, and how these approaches will continue to improve due to various features of fluorescent proteins.

## S1A-A1 Distinct excitatory and inhibitory subnetworks of the cortical microcircuit

Yasuo Kawaguchi
*Div Cerebral Circuitry, NIPS, Okazaki*

Cortical circuits are characterized by selective synaptic connections between individual neurons. We have used physiological and morphological data to explore neuronal connectivity within the cortical network. In so doing, we identified two subtypes of pyramidal cells based on their subcortical projection sites and firing patterns, as well as several nonpyramidal cell types based on their physiology, biochemical expression patterns and morphological characteristics. We then investigated the synaptic connectivity patterns between these neuron subtypes using electrophysiological and electronmicroscopic analysis. We found in pyramidal neurons that intracortical connectivity is strongly correlated with the extracortical projection, and also that nonpyramidal neurons provide GABAergic innervation specifically to pyramidal cell spines also receiving thalamocortical input. Nonpyramidal neurons themselves show subtype specificity in their excitatory and inhibitory drives, as well as postsynaptic target selection. These observations suggest the existence of several distinct intracortical excitatory and inhibitory pathways that subserve the larger cortical circuit.

## S1A-A2 Disynaptic inhibition between neocortical pyramidal cells

Henry Markram
*EPFL Brain Mind Institute, Lausanne, Switzerland*

Reliable activation of inhibitory pathways is essential for maintaining the balance between excitation and inhibition during cortical activity. Little is known about the activation of these pathways at the level of the local neocortical microcircuit. We report a disynaptic inhibitory pathway among neocortical pyramidal cells (PCs). Inhibitory responses were evoked in layer 5 PCs following stimulation of individual neighboring PCs with trains of action potentials. The probability for inhibition between PCs was more than twice that of direct excitation, and inhibitory responses increased as a function of rate and duration of presynaptic discharge. Simultaneous somatic and dendritic recordings indicated that inhibition originated from PC apical and tuft dendrites. Multineuron whole-cell recordings from PCs and interneurons combined with morphological reconstructions revealed the mediating interneurons as Martinotti cells. Martinotti cells received facilitating synapses from PCs and formed reliable inhibitory synapses onto dendrites of neighboring PCs. We describe this feedback pathway and propose it as a central mechanism for regulation of cortical activity.

EU, HFSP

## S1A-A3 A novel form of inhibitory connections between nearby neocortical pyramidal cells

Yukio Komatsu
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We found that individual spikes in one pyramidal cell reliably evoked large IPSCs in nearby pyramidal cells using dual whole-cell recording from layer 2/3 cells in mouse visual cortex. Pharmacological inhibition of kainate receptors blocked these IPSCs, whereas it did not affect EPSCs from pyramidal cells to inhibitory cells, suggesting that these IPSCs are polysynaptic but not mediated by the excitatory synapses from pyramidal cells to the somatodendritic domain of inhibitory cells. Thus, we tested the possibility that IPSCs are mediated by axo-axonic synapses from axon terminals of the source pyramidal cell to nerve terminals of inhibitory cells which connect to the target pyramidal cell. Analysis of mIPSCs suggested the presence of kainate receptors on inhibitory terminals, and iontophoretic application of glutamate to those terminals surrounding pyramidal cell soma produced IPSCs. Immunocytochemical analysis supported the presence of such connections. Thus, pyramidal cells likely exert strong inhibitory effects on nearby pyramidal cells via the direct activation of nerve terminals of inhibitory cells.

KAKENHI

## S1A-A4 Neuronal signaling patterns in the developing mouse visual cortex in vivo

Arthur Konnerth
*Institute of Neuroscience, Technical University Munich, Germany*

We investigated neuronal signaling in layer 2/3 neurons of the mouse primary visual cortex just prior and after eye opening, which occurs usually around postnatal day 14 (P14). For this purpose we used two-photon Ca2+ imaging and whole-cell recordings in anesthetized mice. We found that even before eye opening (starting with P11) brief light flashes effectively evoked Ca2+ transients in individual neurons. Remarkably, this early responses had a wave-like character, as they engaged each time the majority of the neurons. The fraction of responding cells was highest (90%) at the youngest ages and continuously decreased with development.Already at the day of eye opening, a remarkable fraction of layer 2/3 cells were orientation and direction selective. Neurons with different orientation selectivity were randomly distributed within the cortical layer. Another group of neurons responded to changes in luminance, but not to moving gratings. Overall, our results demonstrate that already at early stages of development a high proportion of layer 2/3 visual cortex neurons respond with a high degree of specificity to visual stimuli.

## S1A-A5 Inhibitory circuit control of critical period plasticity

Takao K. Hensch
*RIKEN BSI*

Local GABAergic circuits trigger a critical period for binocular vision in early postnatal life. Parvalbumin-positive large basket cells innervate somatic synapses containing the $\alpha$1-subunit of GABAA receptors, specifically optimized in number to drive experience-dependent changes. The spillover of GABA at these nascent boutons is strictly controlled by an excess of GABA transporter (GAT1) in the pre-critical period to ensure that plasticity occurs at the appropriate late postnatal age. Morphological maturation of these interneurons is further regulated by the passage of a homeoprotein, Otx2, along the visual pathway. With age, these cells become enwrapped in peri-neuronal nets of extracellular matrix molecules, whose disruption resets ocular dominance plasticity in adulthood. Competition detected by fast-spiking circuits is ultimately implemented at an extracellular locus by proteases which cleave cell adhesions to permit spine pruning and axonal rewiring. Understanding the excitatory-inhibitory balance for circuit development may lead to novel therapies and training strategies for rehabilitation, recovery from brain injury, and lifelong learning in adulthood.

RIKEN BSI, JST, HFSP, KAKENHI (17021047)

## S1A-B1 Primate model of attention deficit/ hyperactivity disorders (ADHD)

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[2]*Primate Research Institute, Kyoto University, Inuyama, Japan*

ADHD is one of the prevalent childhood psychiatric disorders. Behavioral characteristics include hyperactivity and inattention, suggesting prefrontal (PFC) contribution to ADHD. ADHD is also known as dopamine (DA) related dysfunctions, in that methylphenidate is an effective drug for the treatment of ADHD. PFC has strong DA innervation and DA-related drugs modulate PFC-related cognitive functions. These observations suggest that DA-related dysfunction in PFC at an early developmental stage is a biological cause of ADHD. To prove this, we injected 6-OHDA bilaterally into dorsal PFC of infant monkeys to destroy DA innervation and examined whether monkeys exhibited hyperactivity. 6-OHDA injected monkeys showed significantly increased spontaneous activity compared with non-injected monkeys. Oral administration of methylphenidate to injected monkeys reduced spontaneous activity. These results indicate that monkeys having injection of 6-OHDA in PFC would be a candidate for an animal model of ADHD.

KAKENHI (17650101, 17021002)

## S1A-B2 Changes in behavior and brain activity by acute and diurnal mental fatigue: PET study with macaque monkeys

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We examined effects of acute and diurnal mental fatigue on the behavior and brain activity using rhesus monkeys. We trained monkeys to perform conditional visual discrimination task and continuous simple reaction task for acute and diurnal fatigue, respectively. Monkeys were forced to continue the task intermittently for several hours for acute fatigue, and they were deprived sleep at one-night (SD) for diurnal fatigue. We measured the regional cerebral blood flow (rCBF), and binding activity of [$^{11}$C]Ro15-4513 and [$^{11}$C]Ro15-1788 to GABAA receptor using PET. In acute fatigue, RT was delayed dependent on the progression of time, and decreases of rCBF in the anterior cingulate and some regions were observed. In diurnal fatigue, RT was also prolonged, and BPs of [$^{11}$C]Ro15-4513, but not of [$^{11}$C]Ro15-1788, were increased in some limbic structures. These results strongly indicate neuronal involvements in the deterioration of behavioral performance by acute and diurnal mental fatigue.

Molecular Imaging Program (MEXT)

## S1A-B3 Effects of early experience on visual performance

Yoichi Sugita

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Infant monkeys were reared under special visual environments for a year to examine the effects of early experience on the development of higher visual functions. Four monkeys were reared under monochromatic illuminations. They were able to perform color matching. But, they had severe deficits in color constancy; their color vision was very much wavelength-dominated. Another 4 monkeys were reared under the illumination of 10 Hz stroboscopic lights so that they could not perceive visual motion. Their performance was quite poor for speed or direction matching. Furthermore, they had severe deficits in perceiving global motion. A third group was reared without being exposed to any faces. After the deprivation period, the monkeys were exposed for the first time to either human or monkey faces. Soon thereafter they electively discriminated only the kind of face to which they had received exposure. Furthermore, the ability to iscriminate the non-exposed species of face were not restored. These results indicate distinct influences of these different aspects of visual experience, and suggest the existence of sensitive period for higher visual functions.

CREST, JST

## S1A-B4 Gene expression profiles in two areas of monkey temporal cortex

Noritaka Ichinohe, Marie Wintzer, Toshio Miyashita, Kathleen Rockland

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Two areas defined within temporal cortex are: TE, a unimodal visual area, and perirhinal cortex (PRh), a multimodal limbic area. These areas are distinguished by connectivity, electrophysiological properties, and behavioral and neurochemical profiles. In humans, PRh is preferentially susceptible to seizures, Alzheimer's disease, and Parkinson's disease. Gene expression profiling offers a new approach for investigating basic and clinical issues in the context of area specialization. By using layer specific samples from TE and PRh, and Affymetrix GeneChip, we identified (> 1.5 fold) about 500 genes which are more strongly expressed in PRh than in TE. Functional annotation of this list using Gene Ontology vocabulary and Ingenuity Pathways Analysis revealed overrepresentation of GO categories related to development, growth, extracellular matrix, gylcosaminoglycan degradation and actin/cytoskelton. These suggest a greater degree of plasticity or retained immaturity in PRh. Genes which have been implicated in the several human diseases mentioned above were also in this list.

KAKENHI 18020032

## S1A-B5 Nonhuman primate models of normal social behavior, neurodevelopmental disorders and functional reorganization following early brain Damage

David G Amaral

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We have developed a program of studies to evaluate the emergence of social behavior in animals that have received lesions of the amygdala, hippocampus or orbitofrontal cortex either as adults or as two-week-old infants. Work on the amygdala has led to the conclusion that the amygdala is a critical structure for appropriate detection of environmental dangers. However, the amygdala does not appear to be an essential component of the social brain. Based on experience in evaluating social behavior following brain lesions, we have begun to develop nonhuman primate models of autism. I will discuss our early efforts that deal with a potential immune etiology of autism. Finally, work on the role of the hippocampal formation in memory function has indicated that while lesions in adult primates lead to irreversible impairments in spatial memory, similar lesions in neonatal animals result in almost complete restoration of spatial memory function. The nonhuman primate is an ideal animal model for the analysis of functions important for normal and pathological human cognition.

NIH

## S1A-C1 Impaired decision making in individuals with Parkinson's disease

Masaru Mimura

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Parkinson's disease (PD) is primarily characterised by a movement disorder. However, recent studies have suggested that PD individuals may present with a wide range of cognitive and/or affective problems including impaired social cognition. The present study used Iowa Gambling Task and demonstrated that PD individuals display risk taking tendency in decision making process. Compared with healthy controls, decision making of PD individuals was biased toward high-risk and high-return, but disadvantageous choices. The ability to make beneficial judgments solely correlated with ability to appropriately attribute internal mental-state to other people, as shown in a mind-reading task. In contrast, decision making ability in PD was not associated with effective executive function, intelligence or depressed mood. These findings offer a new perspective on cognitive impairment of PD individuals that affect their social living. Risk taking tendency in PD may be attributed to dysfunction of the amygdala or ventromedial frontal cortex, which is probably caused by dopamine depletion in subcortical structures including the striatum and/or basal ganglia.

## S1A-C2 Impaired recognition of the facial expressions in patients with Parkinson's disease

Shinichi Koyama[1], Mitsuru Kawamura[2], Masaru Mimura[2], Masamichi Sakagami[3], Atsunobu Suzuki[4], Hackjin Kim[5], Peter Bossaerts[5]

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Although Parkinson's disease (PD) is primarily thought to be a dysfunction of the motor system, PD patients have difficulties with the recognition of the facial expressions. My recent studies demonstrated that their facial-expression recognition was impaired at an early stage of PD, even before the development of the motor symptoms. On the other hand, a recent study by our group showed that the recognition of the facial expression was normal in patients with Autosomal-Recessive Juveline Parkinson's disease (ARJP), in which the nigrostriatal dopaminergic system was selectively impaired whereas the mesocorticolimbic dopaminergic system was relatively intact. Taken together, our studies suggest that the mesocorticolimbic dopaminergic system plays an important role in the recognition of the facial expressions, and the system may be impaired at an early stage of PD.

KAKENHI (17022035, 18020027), CREST

## S1A-C3 Novel neuropsychological evidence for multidimensionality of facial expression recognition

Atsunobu Suzuki[1]

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The ability to recognize others' emotions from facial expressions constitutes an important basis for our social interaction. My colleagues and I recently developed a refined behavioral test to measure this ability as "sensitivities to emotions" embedded in ambiguous facial expressions, and examined individual, age-related, and disease-related (Parkinson's disease) differences. The results revealed interesting dissociations among the sensitivities to different emotions: (1) the sensitivity to happiness was uncorrelated with the sensitivities to negative emotions, (2) the sensitivity to anger was selectively reduced in the elderly than in the young, and (3) the sensitivity to disgust was selectively impaired in patients with Parkinson's disease. Our findings provide strong support for a classical but still controversial view that the ability to recognize facial expressions is multidimensional, and that effects of aging and disease are specific to certain dimensions.

Grant-in-Aid for JSPS Fellows

## S1A-C4 Functionally and temporally dissociable neural processes underlying social decision-making on faces

Hackjin Kim[1], Mitsuru Kawamura[2], Masamichi Sakagami[3], Masaru Mimura[2], Shinichi Koyama[2], Atsunobu Suzuki[4], Peter Bossaerts[1]

*[1]Division of Humanities and Social Sciences, Caltech, Pasadena, USA., [2]Showa Univ., [3]Tamagawa Univ., [4]University of Illinois*

Reading emotional states from others' faces and evaluating people in terms of preference are probably two most fundamental social decisions we make almost everyday. Increasing pieces of evidence suggest that subcortical and cortical brain structures play functionally distinctive roles in processing faces, and display significant interactions with each other during social decision-making on faces. We recently developed a novel temporally extended decision-making paradigm and found that the neural processes in the nucleus accumbens and the orbitofrontal cortex can be temporally and functionally dissociable while subjects are engaged in simple preference decisions on faces. The findings support a model in which rapid, automatic engagement of the subcortical systems such as the amygdala and the nucleus accumbens can read crude but essential signals from faces and convey them to the prefrontal cortex for further sophisticated analysis.

## S1A-C5 Human Imagination in Financial Markets with Insiders

Peter L. Bossaerts[1,2], Antoine Bruguier[1], Steve Quartz[1]

*[1]Computational and Neural Systems, California Institute of Technology, Pasadena (CA), USA, [2]Swiss Finance Institute*

How do humans try to understand the behavior of complex systems? We ask this question in the context of financial markets with insiders. We advance here that humans anthropomorphize financial markets with insiders, reading intentions and beliefs in the order flow, and hence, applying Theory of Mind to predict future price changes. No such anthropomorphizing is hypothesized for markets without insiders. While plenty of casual evidence exists that traders indeed use Theory of Mind language to describe market behavior, we show here that markets with insiders, in contrast with markets without insiders, engage the main brain structures involved in Theory of Mind tasks. In addition, we provide preliminary evidence that Theory of Mind capacity correlates with trading success.

US National Science Foundation Grant SES-0527491

## S1A-D1 Differential organization and remodeling of the glutamatergic postsynaptic specializations between excitatory and inhibitory neurons

Shigeo Okabe

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Neuronal network is regulated by the balance of excitatory and inhibitory neuron activity. Glutamatergic synapses onto excitatory pyramidal neurons in the hippocampus are formed on the surface of dendritic spines. Maintenance of PSDs in pyramidal neurons is dependent on the integrity of actin filaments, which are highly enriched in spines. In turn, most of the GABAergic interneurons in the hippocampus lack dendritic spines but form PSDs within dendritic shafts. We performed time-lapse imaging of postsynaptic scaffolds in interneurons combined with ultrastructural analysis and genetic manipulation of key regulators of microtubule-dependent motility. The results indicate interneuron-specific regulation of PSDs, including intimate interactions with microtubules, retrograde microtubule-dependent motility, and its regulation by Lis1 and Ndel1, two essential components of dynein-dependent motility. Our observation suggests fundamental differences in the organization of the PSDs and their motility between two major neuron subtypes in the hippocampus.

KAKENHI 18200025

## S1A-D2 Finding the presynaptic organizers

Hisashi Umemori

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Precise assembly of synapses is critical to process information underlying all neural activity, and thus for proper functioning of the nervous system. Synapses are formed by signaling between the presynaptic neuron and its specific postsynaptic target. Target-derived "presynaptic organizers" promote local differentiation of axons into functional nerve terminals at sites of synaptic contact (reviewed in Fox and Umemori, J. Neurochem., 2006). We have purified and identified such presynaptic organizers. One of them, FGF22, is a critical presynaptic organizer in the cerebellum in vivo (Umemori et al., Cell, 2004). We also found that multiple target-derived cues act sequentially to organize presynaptic differentiation at the neuromuscular junction, with FGF7/10/22, laminin $\beta 2$, and collagen IV playing predominant roles in induction, maturation and maintenance of the motor nerve terminal, respectively (Fox et al., Cell, 2007). We are currently investigating how multiple organizers coordinate synaptic connections in the brain to understand the temporal and spatial specificity of synapse formation that is required for organizing the functional circuits.

MBNI, BSSP, Klingenstein, Mallinckrodt

## S1A-D3 Cbln family proteins-characterization of anterogradely transported molecules regulating synapse formation and plasticity

Michisuke Yuzaki

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Cbln1, a member of the C1q/tumor necrosis factor (TNF) superfamily, is secreted from cerebellar granule cells. Cbln1 plays two crucial roles at granule cell-Purkinje cell synapses: the matching and maintenance of pre- and postsynaptic structures; and the induction of long-term depression, a synaptic plasticity model underlying cerebellar motor learning. Nevertheless, molecular mechanisms by which Cbln1 functions have remained unclear. We found that Cbln1 and other Cbln family members, Cbln2-Cbln4, were expressed in immature and mature neurons, which often send axons to principal neurons in various brain regions. In addition, we found that recombinant Cblns specifically bound to the postsynaptic dendrites of target neurons. Like other C1q/TNF family proteins, Cbln family members formed a homo- and heteromeric oligomers; such oligomeric states were crucial for their specific binding and biological activities. These results indicate that Cblns represent a new class of anterogradely transported molecule, which regulates synapse development and plasticity in distinct subsets of neurons.

## S1A-D4 Development and plasticity of functional odor inputs into the olfactory bulb

Venkatesh N Murthy, Dinu F Albeanu, William J Tyler, Tomokazu F Sato, Edward R Soucy, Markus Meister

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Olfactory sensory axons expressing a particular odorant receptor project to about two glomeruli in each olfactory bulb in adult mice. To examine the postnatal development of functional synaptic inputs to the olfactory bulb, we imaged odor-evoked responses in transgenic mice expressing the presynaptic reporter synaptopHluorin in olfactory sensory neurons (Neuron 42:9). Functional odor maps in neonatal animals were very similar to those in adults, and functionally identified glomeruli were found in predictable locations on the dorsal surface. To determine if sensory experience affects the organization of synaptic inputs to the bulb, we performed unilateral naris occlusion at birth. Sensory deprivation led to strengthening of sensory synapses. Experiments are underway to examine whether naris occlusion alters functional odor maps. Our results indicate that sensory inputs to the glomeruli develop early in the mouse olfactory bulb and that the strength of inputs can be altered by experience.

Harvard University Startup Funds

## S1A-E1 Physiologcal roles of Apaf-1-mediated caspase signaling in neural development

Masayuki Miura

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Macrochaetes are external sensory organs located on the notum and are typical structures of the Drosophila peripheral nervous system. Dapaf-1 mutant flies have one extra sensory organ precursor (SOP) cell on each side. Certain number of SOP cells are selected among cells of proneural clusters without cell death, and basal caspase-3 like activity observed in this cluster is required for determining the number of SOP cells in the scutellum. Genetic study indicated IKKε determine the level of caspase-inhibitor DIAP1, and thus regulate the caspase activity. Apaf-1-mediated caspase activation is also observed in olfactory development in mice. At late embryonic stage, caspase-3 was activated in many of mature olfactory sensory neurons (OSNs), especially in the anterior region of the olfactory epithelium. Apaf-1-defcient mice abolished this caspase-3 activation in OSNs. Caspase-3-active OSN did not show typical sign of apoptosis indicating caspase-3 may execute functions for neuronal development other than cell death.

## S1A-E2 Early motoneuron death in the cervical cord of the avian embryo occurs to eliminate a specific subgroup of motoneurons.

Hiroyuki Yaginuma

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In the chick embryo spinal cord, motoneurons (MNs) undergo programmed cell death (PCD) at two periods. One is observed at relatively later stages (E6-E10) and involves all segments of the spinal cord. The other occurs at earlier stages (E4-E5) and only in the cervical spinal cord. To determine the identity of the dying MNs in the early cervical PCD, we examined expression of subgroup-specific MN markers (Isl1, Isl2, Lim3 and MNR2) in the dying MNs. Quantitative analysis revealed that PCD occurs only in a subgroup of MNs that express Isl1, Isl2 and MNR2 but do not express Lim3 ($Lim3^-$). This subgroup appeared in the dorsolateral region of the ventral horn by E4 after losing Lim3 expression and disappeared by E5. When MNs were rescued by Bcl2 overexpression, this $Lim3^-$ subgroup remained. We observed that similar MNs appear also in the thoracic region and develop into MNs that innervate intercostal muscles. These results suggest that the early cervical PCD occurs to eliminate the $Lim3^-$ MN subgroup that correspond to MNs innervating intercostal muscles.

KAKENHI (16500223, 18500266)

## S1A-E3 Caspase-independent neuronal death triggered by abolition of neurotrophic signals

Hideki Enomoto

*RIKEN Center for Developmental Biology*

The regulation of neuronal survival and death by neurotrophic factors plays a central role in the sculpting of the nervous system, but the identity of survival signals for developing enteric neurons remains obscure. We demonstrate here that conditional ablation of GFR $\alpha$1, the high affinity receptor for GDNF, in mice during late gestation induces rapid and widespread neuronal death in the colon, leading to colon aganglionosis reminiscent of Hirschsprung disease. Enteric neuron death induced by GFR $\alpha$1 inactivation is not associated with the activation of common cell death executors, caspase-3 or -7, and lacks the morphological hallmarks of apoptosis, such as chromatin compaction and mitochondrial pathology. Consistent with these in vivo observations, neither caspase inhibition nor Bax deficiency blocks death of colon-derived enteric neurons induced by GDNF deprivation. This study reveals an essential role for GFR $\alpha$1 for the survival of enteric neurons and suggests that caspase-independent death can be triggered by abolition of neurotrophic signals.

## S1A-E4* Pael-R transgenic mouse crossed with parkin null mouse displays persistent endoplasmic reticulum stress, reduction in complex I activity and dopaminergic neuronal death

Ryosuke Takahashi[1], Hua-Qin Wang[1], Yuzuru Imai[2], Haruhisa Inoue[1], Ayane Kataoka[3], Sachiko Iita[3], Nobuyuki Nukina[3]

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Parkin is responsible for autosomal recessive juvenile parkinsonism (AR-JP). We identified Pael-R as a substrate of parkin, whose accumulation is thought to induce unfolded protein response (UPR), leading to dopaminergic neuronal loss. To create an animal model of AR-JP, we generated parkin-KO/Pael-R-TG mice. parkin-KO/Pael-R-TG mice exhibited early and progressive loss of dopaminergic neurons without inclusion bodies formation, recapitulating the pathological features of AR-JP. Evidence of activation of UPR was observed throughout the life time. Moreover, complex I activity was reduced at later stages. These findings indicate that persistent induction of unfolded protein stress is presumably underlying dopaminergic neuronal death and that dysfunction of mitochondrial complex I might be involved in the progression of PD.

## S1A-E5* Identification of prothymosin-alpha1, the necrosis-apoptosis switch molecule under the in vitro ischemia condition

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*Div. of Mol. Pharmacol. & Neurosci., Nagasaki Univ. Grad. Sch. of Biomed. Sci., Nagasaki, Japan*

A stroke delivers a double whammy. First, cells near the clot begin to perish from necrosis, which is triggered by ATP scarcity. Later, more distant cells start dying through apoptosis. We initially identified a nuclear protein, prothymosin-alpha1 (ProT $\alpha$), as a key protein inhibiting necrosis. ProT $\alpha$ inhibited necrosis of cultured neurons by preventing rapid loss of cellular ATP levels by reversing the decreased membrane localization of glucose transporters, but caused apoptosis through up-regulation of pro-apoptotic $Bcl_2$-family proteins. The apoptosis caused by ProT $\alpha$ was further inhibited by growth factors including brain-derived neurotrophic factor. Knockdown of PKC $\beta_{II}$ expression prevented this cell death mode switch. Taken together, these results suggest that ProT $\alpha$ is an extracellular signal protein that acts as a cell death mode switch and could be a promising candidate for preventing brain strokes with the help of known apoptosis inhibitors.

KAKENHI 13470490, 15390028, 17025031

## S1A-E6 Participation of autophagy in pyramidal neuron death of the hippocampus after hypoxic ischemic brain injury

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It has been shown that the lysosomal system including autophagy is activated in the CA1 pyramidal neurons of gerbil hippocampus after brief forebrain ischemia. The mechanism underlying neuronal death in hypoxic ischemic (H/I) brain injury is excitotoxicity, while the cell death mode associated with ischemic brain injury remains controversial. We examined neuron death in the pyramidal layers of the hippocampus in neonatal and adult mice after H/I brain injury and found that the activation of caspase-3 occurred in the neonatal hippocampus within 24 hr after H/I injury but not in the adult hippocampus, as evidenced by western blotting. Immunohistochemical observations demonstrated that 35% of damaged neurons were immunopositive for cleaved caspase-3 in the neonatal hippocampus, while autophagy was highly induced in pyramidal neurons of both neonatal and adult hippocampus. These lines of data indicate that autophagy may play an important role in pyramidal neuron death in the hippocampus after H/I brain injury.

Grant-in-Aid for Creative Scientific Research (16GS0315)

## S1A-F1 An epileptic disease with genomic imprinting disorder: Angelman syndrome

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Angelman syndrome (AS) is a neurodevelopmental disorder characterized by severe mental retardation, epilepsy, ataxic movement and characteristic behavior including easily evoked laughter. AS is caused by the loss of function of the imprinted *UBE3A* gene, which is only expressed from the maternally derived allele in neurons. *UBE3A* encodes the putative ubiqutin protein ligase E3A, although its role remains unclear. *UBE3A* is located in 15q11-q13 that comprises an imprinted domain containing two maternally expressed genes including *UBE3A*, and several paternally expressed genes. Imprinting state of the entire 15q11-q13 imprinting domain is regulated by the imprinting center (IC) that is located upstream of the imprinted *SNURF-SNRPN* gene, and is controlled through epigenetic process. The dysfunction of IC due to microdeletions or epimutations could cause imprinting defects and is responsible for a few cases of AS. AS is an extraordinary model which bridges genomic imprinting and neurodevelopment.

## S1A-F2 A severe mental retardation disease with defect in chromatin remodling: ATR-X syndrome

Takahito Wada

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X-linked $\alpha$-thalassemia/ mental retardation syndrome (ATR-X, OMIM #301040) is a syndromic form of X-linked mental retardation (XLMR), and characterized by severe mental retardation in males, $\alpha$-thalassemia, dysmorphic facies, genital abnormalities, skeletal abnormalities and characteristic posture and/or behavior. ATR-X is caused by a mutation in the *ATRX* gene at Xq13.3, which is also involved in a non-syndromic form of XLMR in females, as well as in males. The *ATRX* gene encodes ATRX protein, which consist of two functionally important domains: a PHD-like zinc finger domain and a SNF2-like ATPase domain, where disease-causing missense mutations are clustered. ATRX is a member of the SNF2 family of ATPase/helicase proteins, and thought to regulate transcription of a discrete set of target genes by modifying chromatin at target loci. However, the $\alpha$-globin genes are the only confirmed targets of ATRX. The manner by which ATRX affects gene expression remains to be clarified.

The research grants (18A-5) for Nervous and Mental Disorders from the Ministry of Health, Labour and Welfare, Japan.

## S1A-F3 Cerebral gigantism with histone metyl transferase abnormality : Sotos syndrome

Naomichi Matsumoto

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Sotos syndrome (OMIM #117550) is a childhood overgrowth syndrome characterized by accelerated growth, typical craniofacial features and learning impairment. Sotos syndrome is caused by haploinsufficiency of the NSD1 gene at 5q35.2 - q35.3. Functional roles of NSD1 are yet largely unknown. A major role of NSD1 seems to lie in the transcriptional repression of chromatin through the histone methyltransferase activity of its SET domain. In two patients with a Sotos syndrome phenotype, abnormalities in the imprinted region on 11p15, which is associated with Beckwith-Wiedemann syndrome, were recently found. Although additional patients have yet to be reported, it is interesting to hypothesize that the histone methyltransferase activity of NSD1 could be a role playing factor in establishing the parental imprint in this region. Imprinting status of the 11p15 region in our series of patients with confirmed NSD1 abnormality is currently being investigated. Furthermore another functional aspect of NSD1 regarding cell growth, which we have recently found, will be presented.

KAKENHI (18659094)

## S1A-F4 Overview of epigenetic disorders in neurodevelopmental diseases

Takeo Kubota, Chunshu Yang, Masaki Soutome, Kazushi Endoh

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What we learned from the human genome project is not only importance of the gene structure, but also importance of the gene regulation to maintain human health. Epigenetics is one of the gene regulation systems, which is based on 'beyond the DNA sequence' (DNA and chromosomal histone modifications). It has recently been found that failures of steps of the epigenetic system are associated with distinct neurodevelopmental diseases. One exmaple is Rett syndrome, an autistic disorder caused by abnormality in a methylated-DNA binding protein. Together with other recent findings that DNA methylation and accompanied histone modifications can be altered and then restored after birth, we reasoned that some autistic patients, caused by improper epigenetic modifications, can be treatable epigenetically. Update findings in epigenetic studies in neurodevelopmental diseases will be presented in this symposium.

KAKENHI (Houga 17659311); KOSEIKAKEN (18Shi-4); TV Yamanashi Foundation

## S1A-F5 What we do for neurodevelopmental diseases with epigenetic disorders

Shinji Fushiki, Takeshi Yaoi, Keiko Nakamura, Kyoko Itoh

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Epidemiological evidence suggests that environmental exposures early in development have a role in susceptibility to disease in later life, including neurobehavioral disorders. Animal studies indicate that prenatal and early postnatal environmental factors can result in altered epigenetic programming and subsequent changes in the risk of developing disease. We have demonstrated that prenatal exposures to environmental chemicals affect brain development, accompanied with changes in gene transcription. We have thus started to solve the question what genes exhibit changes in the DNA methylation status after the exposure to chemicals. By using restriction landmark genomic scanning with a methylation-sensitive enzyme (RLGS), we have found that approximately 0.5% of RLGS spots obtained from the embryonic brain-derived genomic DNA appeared and disappeared after the exposure. These data suggest that environmental chemicals could influence the DNA methylation status of genomic loci, which may result in changes in gene transcriptional regulation.

KAKENHI (15390334, 17022032)

## S1A-G1 The development of amygdala and emotional neural circuit

Shigeki Yuasa, Miho Soma

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Amygdala is involved in the processing of emotional information and the control of emotional expression. Defective amygdalar development was found in the human developmental disorders such as autism. Developmental mechanism of amygdala has not been adequately elucidated in spite of the importance for the study of the pathogenesis of such disorders. Accordingly, we have investigated the mode of cell migration and nuclear formation of the amygdala. We applied the EGFP gene transfer into the amygdalar origins in order to trace the migration of amygdalar neurons to the destination and identified the nuclear formation by using their molecular markers. Furthermore, we have examined the involvement of transcription factors such as Pax6 in the amygdalar development by transfer of siRNA expression vector to amygdalar origin. We have also examined the development of main amygdalar output, stria terminalis. Based on these results, the significance of factors controlling the amygdalar development will be discussed in regard to the pathogenesis of defective development of higher brain functions.

NIBIO Grant 05-32

## S1A-G2 Regulation of amygdala functions by NMDAR tyrosine phosphorylation

Takanobu Nakazawa, Mina Delawary, Tadashi Yamamoto

*Division of Oncology, Institute of Medical Science, University of Tokyo*

Phosphorylation of neural proteins in response to a diverse array of external stimuli is one of the main mechanisms underlying dynamic changes in neural circuitry. The NMDAR subunits are phosphorylated by NMDAR complex-associated protein kinases to regulate receptor localization, current flow through the receptor, and downstream signaling events. These phosphorylation events are thought to be essential mechanisms underlying dynamic changes in neural circuitry. One of the NMDAR subunits, the NR2B subunit is tyrosine phosphorylated in brain, with Tyr-1472 its major phosphorylation site. Using mice with a knock-in mutation at the phosphorylation site, Tyr1472Phe (YF/YF mice), we found that YF/YF mice showed impaired induction of amygdaloid long-term potentiation and fear-related learning. In the elevated plus-maze test, we found that YF/YF mice showed reduced time spent in open arm. Thus, NR2B tyrosine phosphorylation regulates amygdala functions. In this presentation, we will focus on molecular mechanisms of regulation of amygdala functions by NR2B tyrosine phosphorylation.

KAKENHI (17013021)

## S1A-G3 Impaired amygdala GABAergic synapses and enhanced emotional behavior in dystrophin-deficient mdx mice

Masayuki Sekiguchi

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The scaffolding proteins in GABAergic synapse are expected to be required for formation or maintenance of normal neural circuits underlying emotional behaviors. Here we report a role of dystrophin, an actin-binding scaffold selectively localized to a subset of GABAergic synapses, in the emotional behavior. In dystrophin-deficient mdx mice, noradrenaline (NE)-induced GABAergic IPSCs were attenuated in the basolateral nucleus of the amygdala (BLA) pyramidal neurons, whereas NE normally induced action potential in a subset of BLA interneurons. In the mdx BLA, the number of GABAergic synapses was reduced and some GABAergic synapses exhibited aberrant ultrastructural morphology. Mdx mice exhibited dramatically enhanced unconditioned fear in response to insignificant stimuli for wild-type mice. These results indicate that dystrophin, a responsible protein for Duchenne muscular dystrophy, is essential for the integrity of GABAergic synapses required for the information processing on unconditioned fear.

## S1A-G4 Modulation of hippocampal synaptic plasticities by the activities of the amygdala

Norio Matsuki, Yuji Ikegaya

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Synaptic plasticity in the hippocampus is a fundamental feature of brain function involved in the encoding of information, and the capability of synapses to express plasticity is itself activity-dependent. Numerous studies have shown that various drugs and physiological substances including neurotrophic factors, cytokines are effective in modulating the plasticity. However, little is known about interaction between the hippocampus and other brain nuclei. The amygdala transfers emotion to various brain regions including hippocampus and there are two kinds of projections to the hippocampus from the basolateral amygdala (BLA), direct and indirect pathway via the entorhinal cortex. The modulation is not uniform and ventral hippocampus receives more dense direct innervation, which is sensitive to stress. Activities of the indirect pathway can modulate bidirectionally medial perforant path - dentate gyrus synapses. These results suggest a high-order dimension of heterosynaptic modulations of hippocampal synaptic plasticity.

## S1A-H1 Cerebellar development transcriptome database

Teiichi Furuichi

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To elucidate the genetic basis of mouse cerebellar development, we combine our gene expression profiling research and bioinformatics research by generating a neuroscience database, Cerebellar Development Transcriptome Database (CDT-DB). The postnatal development of mouse cerebellum is programmed by thousands of wide varieties of genes, which exhibit differential expression patterns in time and space during this period. We collect large datasets on spatio-temporal gene expression profiles by fluorescent differential display, GeneChip microarray, *in situ* hybridization analyses, etc. We not only systematize all these lines of the information with annotation in terms of their gene ontology and their functional properties in cerebellar development, but also correlate them to the corresponding information at relevant public database websites. As the result, the synergy from combining neuroscience and informatics accelerates our research progress toward a better understanding of the cerebellar development. Furthermore, sharing our data over the internet would be beneficial to our neuroscience community. **CDT-DB http://www.cdtdb.brain.riken.jp*

RIKEN

## S1A-H2 Large scale analysis of gene expression in CNS: a rapidly expanding collaborative landscape

Robert W. Williams

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The completion of GeneNetwork (www.genenetwork.org) and the Allen Brain Atlas (ABA) now makes it possible to study the expression of most genes in the CNS at high spatial and genetic resolution. This pair of remarkable resources are complementary: The ABA provides detailed high resolution *in situ* images for a single strain of mouse, whereas GeneNetwork provides extensive quantitative data on differences in expression for 100 strains for major CNS regions (cerebellum, hippocampus, striatum, eye, and cortex). Their combination with major genomic resources (RIKEN, NCBI, UCSC) gives rise to a rich online collaborative environment. These neuroinformatics knowledgebases, together with the Mouse Brain Atlasing Toolkit, greatly improve the efficiency with which we can generate hypotheses relevant to neurodegeneration, psychiatric disease, and addiction research.

NCRR (U01NR 105417), NIAAA (U01AA13499, U24AA13513), NIDA, NIMH, and NIAAA (P20-DA 21131)

## S1A-H3 Neuron-Glia Platform: Neuroinformatics at the cellular and local network level.

Hiroyoshi Miyakawa

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At the cellular level and local network level, an immense number of experimental findings have been accumulated. With regard to the basic principle on neural information processing, however, we have not achieved much during the last 100 years since Cajal's neuron doctrine. We need to share knowledge and new ideas, and integrate them. To do so, we proposed to give mathematical model descriptions to new findings and ideas, and build the Neuron-Glia Platform, an infrastructure to share models and data. Model description would not only help sharing findings, but would help intense collaboration of experimental and theoretical researchers, which, I hope, will be a hatchery for novel pictures of neuronal information processing. Here I introduce the basic features of the platform by showing my own research as an example. In this example, change in membrane potential of CA1 pyramidal neurons due to applied extra-cellular electric field was measured by use of fast voltage-imaging technique. Compartmental models with simple and realistic morphology were used to analyze the data.

## S1A-H4 Large scale simulations of cortical neuronal networks

M Diesmann, M_O Gewaltig, A Morrison, H E Plesser

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Higher brain functions emerge from large and complex cortical networks and their interactions. However, the large number of neurons combined with the high connectivity of the biological network and the heterogeneity in neuron and synapse types impose severe constraints on the explorable system size which have previously been hard to overcome. Furthermore, the continuous reorganization processes in the brain by different types of plasticity require simulations on the biological time-scale of minutes with sub-millisecond resolution. In this contribution we review three recent advances that enable us to investigate large-scale networks exploiting modern hybrid multi-node/multi-core computer architectures: (1) efficient communication in distributed simulation, (2) use of continuous spike times in a time-driven simulation scheme, and (3) a multi-threaded message-passing simulation kernel. The technical advances are illustrated by a study of spike timing dependent plasticity (STDP) in a model of a cubic millimeter of cortical tissue containing some 1,000,000,000 synapses.
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## S1A-J1 Neurobehavioral influences by early weaning stress

Takefumi Kikusui

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In mammalian species, pups highly depend on their mother, not only for nutrition but also physical interaction. Therefore disruption of mother-pup interaction changes physiology and behavior of the pups. We have reported that early-weaning resulted in persistent increase in anxiety as well as aggression in mice adulthood. These early-weaned mice also showed higher HPA activity in response to novelty stress. Neurochemically, precocious myelination in the amygdala, and a decrease of brain derived neurotrophic factor (BDNF) protein levels in the hippocampus and prefrontal cortex were observed in early-weaned male mice. In addition, the early-weaned males, but not females, showed reduced BrdU immunoreactivity in the dentate gyrus. These results suggest that the deprivation of mother-infant interaction during the late lactating period augments the anxiety in the adulthood by decreasing the level of BDNF in the pre-limbic system, and that these stress responses are sexually dimorphic, i.e., male is more vulnerable to early weaning stress.

KAKENHI 14760187

## S1A-J2 Intracellular signaling mechanism regulating mouse parental behavior in the medial preoptic area

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Parenting is essential for the survival and development of mammalian offspring. In laboratory mice, not only mothers but also fathers and virgins care for pups. During parenting, neurons in the medial preoptic area (MPOA) are activated and express transcription factors such as c-Fos and FosB. FosB-knockout mice are defective in parental care. Here we investigated gene expression profiles in the MPOA of parental versus nonparental mice, and identified upregulation of three genes (NGFI-B, SPRY1, and Rad) in parental mice, together with c-Fos and FosB. A common inducer of these genes, the extracellular signal regulated kinase (ERK) was shown to be phosphorylated in MPOA neurons upon pup exposure. Blockade of ERK phosphorylation inhibited the initiation of pup retrieving in virgin female mice, but did not affect the established parental care in parous females. Furthermore, FosB-knockout mice showed decreased induction of SPRY1 and Rad. These results suggest the pivotal role of ERK-FosB signaling in parental care.

KAKENHI17790832, RIKEN DRI Grant, HFSP LTF

## S1A-J3 Roles of parent-offspring relationship in the development of emotional and social behaviors in mice

Sonoko Ogawa[1], Mumeko Tsuda[1], Kazuya Tomihara[2]

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We have been studying the roles played by estrogen and estrogen receptors (ER) in the regulation of various reproductive behaviors such as sexual, aggressive and parental, as well as an array of emotional and anxiety-related behaviors. Using knockout (KO) mouse models, we have found that ER-$\alpha$ or ER-$\beta$ gene disruptions greatly affect a number of behavioral phenotypes of both sexes, including the expression of parental behavior itself. Recently, we have been focusing to determine possible roles of parent-offspring relationship in behavioral alterations found in ER-$\alpha$ or ER-$\beta$ KO mice. These include studies on 1) the effects of litter composition on the development of emotional and intra-sex social behaviors in ER-$\alpha$ KO mice, and 2) the effects of disruption of parental care on peri-pubertal expression of aggressive and social anxiety behaviors in ER-$\beta$ KO mice. In this talk, we will overview the outcome of these studies and discuss possible roles of parent-offspring relationship in normal development of emotional and socio-sexual behaviors.

KAKENHI17330151;17052001

## S1A-J4 Origins of and evolutionary modifications in the mother-infant interaction in humans

Hideko Takeshita

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Active sharing of attention by mothers with their infants precedes the emergence of combinatorial object manipulation behaviors in infants and might cultivate infants' behaviors to present their mother with objects or to give them objects, i.e., the behavior of sharing objects. Homologous action structures comprise the behaviors of combinatorial object manipulation, presenting objects, pointing toward objects, and word utterances. I argue that these structures were originally generated as a result of mother-infant interactions in the course of hominization. Chimpanzees, the closest relative to humans, exhibit mutual gaze and exchange play face (social smiling); such face-to-face interactions between mothers and infants increase from the time when the infants become one or two months old. Mothers' active sharing of food or objects with infants, however, has been observed only occasionally, which suggests the evolutionary foundation of the triadic mother-infant interaction in the common ancestors of both species.

KAKENHI(16203034)

## S1A-K1 Oscillatory versus persistent Hes1 expression promotes proliferation and differentiation of neural progenitors

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*Institute for Virus Research, Kyoto University, Kyoto, Japan*

The developing central nervous system is partitioned into compartments by boundaries. It has been shown that compartment and boundary cells display different characteristics in proliferation and differentiation: compartmental neural progenitors proliferate extensively and give rise to many neurons, whereas boundary cells proliferate slowly and do not give rise to neurons. The bHLH gene *Hes1*, a Notch effector, is expressed by both compartment and boundary cells, but the expression mode is different between them: variable in compartmental cells and persistent and high in boundary cells. Forced *Hes1* expression at persistent and high levels in compartmental progenitors retard cell cycle progression and inhibit neurogenesis, as observed in boundary cells. We here show by using the real-time imaging method that *Hes1* expression dynamically oscillates in compartmental progenitors. Our results indicate that oscillatory versus persistent Hes1 expression promotes proliferation and differentiation of neural progenitors.

KAKENHI (17024027)

## S1A-K2 Differential expression of the Notch ligand Dll1 among neural precursors is a critical determinant of neurogenesis in the developing mouse telencephalon

Yukiko Gotoh, Daichi Kawaguchi
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In the neurogenic phase of developing mammalian brain, only a subset of neural precursor cells (NPCs) differentiate into neurons, but the mechanisms underlying the selection of differentiating cells remain unknown. Here, we show that the Notch-Delta pathway plays an important role in this selection. We found that the expression patterns of Delta-like 1 (Dll1) and activated Notch1 were mutually-exclusive and segregated in distinct cells in the ventricular zone of developing mouse telencephalon. Dll1 expression resulted in neuronal differentiation when Dll1 was expressed in a small proportion of NPCs, but not in a large proportion of NPCs. This Dll1-induced neuronal differentiation was not observed when the cell density was reduced. Moreover, we found that ectopic expression of Dll1 and conditional deletion of Dll1 gene in a small number of NPCs promoted and reduced neuronal differentiation, respectively, in vivo. These results suggest that the lateral inhibition of the Notch-Delta pathway contributes to select differentiating cells.

## S1A-K3 Differential Notch/CBF1 signaling in telencephalic neural stem cells and neuroblasts

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Prior work has suggested that the telencephalic ventricular zone (VZ) contains both multipotent neural stem cells (NSCs) and more restricted neuroblast progenitors. Here we show that Notch signaling, through the canonical effector C-promoter binding factor (CBF1), is differentially utilized by these cell types. While NSCs contain high levels of Notch/CBF1 signaling, neuroblasts contain a heritable block to CBF1 activation, and shRNA-mediated knockdown of CBF1 promotes the conversion of NSCs into proliferatively active neuroblasts. Furthermore, even with CBF1 knockdown, neurogenesis is inhibited by Notch activation, suggesting that CBF1-independent Notch signaling promotes neuroblast maintenance. Finally, using a transgenic reporter mouse line, together with in vivo and in vitro differentiation assays, we show that NSCs and neuroblasts can be prospectively separated based on Notch/CBF1 activity.

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## S1A-K4* The regulation of cortical neuronal migration by RBP-J

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All the neurons and glial cells in the central nervous system are derived from precursor cells in the ventricular zone (VZ). Notch/RBP-J signaling is known to inhibit neuronal differentiation to maintain undifferentiated neural precursor population. However, the importance of Notch/RBP-J signaling during a complex developmental progress of cortical laminar formation is not well understood. To investigate how Notch-RBP-J signaling regulates cerebral laminar formation, we crossed RBP-J-floxed mice with CaMKII-Cre transgenic mice to induce specific deletion of RBP-J in neuronal cells and not in undifferentiated VZ cells. RBP-J-deficiency in neuronal cells caused a migration defect and resulted in cortical malformations. These data showed that RBP-J play pivotal roles in differentiated neurons to form regular cortical structures.

KAKENHI ( 17689014)

## S1A-K5 DNER-Notch signaling in the postnatal CNS

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We previously identified Delta/Notch-like EGF-related Receptor (DNER) as a novel transmembrane ligand of Notch that was specifically expressed in dendrites of postmitotic CNS neurons. In the developing cerebellum, DNER-Notch signaling mediates neuron-glia interaction during cellular morphogenesis of Bergmann glia. To understand the specificity and generality of DNER-Notch signaling in various cell-cell interactions in the developing CNS, we investigated functions of DNER in other brain regions. In the developing retina, Notch signaling plays central roles in fate determination of neurons and glia from common precursor cells. We found that DNER was expressed in early-born neurons and modulated fate specification of later-born cells in the developing retina. In the developing neocortex, Notch signaling has been implicated in contact-dependent regulation of dendrite arborization in postmitotic neurons. In vivo and in vitro analyses revealed that DNER-deficiency promoted dendrite branching of neocortical pyramidal neurons. We will discuss the function of DNER-Notch signaling in the CNS during postnatal development.

## S1A-K6 Novel regulatory mechanism of Hes1 gene expression

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Accumulating evidence has demonstrated the involvement of Notch signaling molecules in the cell fate decision. Members of the HES protein family, which are essential effectors of Notch signaling, maintain NSCs via repression of neuronal gene expression. However, several studies have reported that there is also Notch-independent pathway concerning the expression of Hes1. Here we show that a transcription factor is involved in the regulation of the Hes1 gene. This factor is expressed in the ventricular zone (VZ) and directly regulates the Hes1 promoter without interacting with the RBP-J binding sites. Overexpression of this factor in the developing mouse forebrain inhibited neuronal differentiation, which is a phenotype similar to Hes1 overexpression. In contrast, expression of dominant-negative forms of this factor promoted migration of the electroporated cells from the VZ and expression of neuronal markers in them. These results indicate that this factor and Notch signaling may contribute in concert to neurogenesis in the CNS.

SORST, JST

## S2A-A1 Signal Integration at Synapses

Sumiko Mochida

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The most important phenomenon for the chemical synapse is signal integration at synapses. Strength of synaptic transmission can be changed by various presynaptic inputs of excitatory, inhibitory and retrograde signals. Molecular mechanisms for proteins interaction and receptor localization in inhibitory inputs are a new topic and retrograde signals, such as cannabinoid released from postsynaptic neurons, have been revealed to modulate synaptic transmission in most of synapses in the brain. Short-term and long-term plasticity have been studied extensively. The long-term plasticity in hippocampal neurons are reported to be induced by new protein synthesis in post synaptic neurons, while the short-term plasticity is dependent on the residual increase in $Ca^{2+}$ in presynaptic terminals. Interestingly, modulation of $Ca^{2+}$ channel activity by $Ca^{2+}$ entered into the presynaptic terminal accompanying action potentials determines neurotransmitter release. Recent new findings presented in this symposium reveal molecular mechanisms for signal integration at synapses regulated multiply by inputs of action potential firings to determine efficiency of synaptic transmission.

## S2A-A2 The neuropeptide nociceptin regulates hippocampal long-term potentiation (LTP) and memory formation

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The neuropeptide nociceptin is a recently identified endorphin that regulates the excitability of hippocampal pyramidal cells through activation of G protein-activated inward rectifier $K^+$ channels. The other endorphins except nociceptin have no direct effect on pyramidal cells, but nociceptin induces hyperpolarizing currents in the pyramidal cell. Thus, nociceptin is likely to regulate hippocampal synaptic plasticity dynamically. We found that nociceptin and the nociceptin receptor were abundantly expressed in the hippocampus and that synaptically released nociceptin from interneurons inhibited LTP induction in the hippocampal CA1 region. Furthermore, LTP in hippocampal slices from mutant mice lacking the nociceptin receptor and learning abilities of the mutant mice in hippocampus-dependent behavioral tests were enhanced. These results suggest that the nociceptin system in the hippocampus plays a significant role in synaptic plasticity and memory formation.

KAKENHI (17023011, 18100003) and RISTEX, JST

## S2A-A3 Mechanisms of endocannabinoid release and retrograde synaptic suppression in the CNS

Masanobu Kano[1], Yuki Hashimotodani[2], Takako Ohno-Shosaku[3], Takashi Maejima[4]

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Endocannabonoids (eCBs) serve as retrograde messengers at synapses in various regions of the CNS. They are released from postsynaptic neurons and cause transient and long-lasting reduction of neurotransmitter release through activation of presynaptic cannabonoid CB1 receptors. eCB release is triggered by either depolarization-induced $Ca^{2+}$ elevation or activation of $G_{q/11}$-coupled receptors in postsynaptic neurons. When these two stimuli coincide, eCB release is markedly enhanced, which we term $Ca^{2+}$-assisted receptor-driven eCB release (Ca-RER). We found that Ca-RER is attributed to the $Ca^{2+}$ dependency of phospholipase C $\beta$ that is activated by $G_{q/11}$-coupled receptors and leads to production of an eCB, 2-arachidonoylglycerol. Our results suggest that Ca-RER is a physiological mechanism of eCB release from neurons in the CNS.

KAKENHI 17023021, 17100004

## S2A-A4 Inhibition of neurotransmitter release by group III metabotropic glutamate receptors

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Group III metabotropic glutamate receptors (mGluRs 4. 6, 7, 8) play a central role in regulating presynaptic activity through G-protein effects on ion channels and signal transduction enzymes. The extended intracellular C-terminal domains of these receptors bind various interacting proteins that are thought to be important for the synaptic targeting and downstream signaling by these receptors. Here, potential roles of $Ca^{2+}$-calmodulin, PICK1, protein kinase C and MacMARCKS in the regulation of presynaptic calcium channel activity will be discussed for mGluR7. In addition, evidence will be presented that posttranslational modifications, including phosphorylation and sumoylation, may be important for regulating feed-back inhibition of transmitter release via group III mGluRs.

## S2A-A5 Calcium channels & synaptic plasticity

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P/Q-type Ca current through Cav2.1 channels initiate synaptic transmission and are regulated by calmodulin (CaM) at a site in the C-terminal containing an IQ-like motif and a CaM binding domain (CBD). Brief, local increases in Ca support high affinity binding of the C-terminal lobe of CaM to the IQ-like domain, which causes facilitation. Sustained increases in Ca support binding of the N-terminal lobe of CaM to the CBD, which causes inactivation. The neuronal calcium sensor protein CaBP1 binds to the Cav2.1 channel, displaces CaM, and enhances inactivation, whereas VILIP-2 slows Ca/CaM-dependent inactivation and enhances facilitation. To examine the role of this regulation in synaptic plasticity, we expressed Cav2.1 channels in sympathetic neurons. Synaptic transmission initiated by wild-type Cav2.1 channels showed facilitation, augmentation, and depression, but this short-term synaptic plasticity was lost in mutant channels lacking the IQ-like domain and CBD. Evidently, regulation of Cav2.1 channels through interaction with calcium sensor proteins is responsible for short-term synaptic plasticity at this model synapse.

## S2A-B1 Glutamate receptor activity regulates drebrin-dependent accumulation of spine resident proteins

Tomoaki Shirao

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Drebrin is a side-binding protein of actin filaments, which is highly concentrated in dendritic spines. We have previously shown that knockdown of drebrin-A expression in postnatal developing neurons suppresses the clustering of F-actin and PSD-95 and decreases the density of cluster-type filopodia, resulting in the inhibition of spine formation. This indicates that developmentally-regulated expression of drebrin-A governs spine formation via the accumulation of drebrin-actin complex at postsynaptic sites. However, premature expression of drebrin A itself does not induce spine formation and instead forms megapodia. Here we show our recent findings about the role of synaptic activity on spine formation through modulation of drebrin A dynamics, and discuss the differential roles of alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid-type receptors (AMPARs) and N-methyl-D-aspartate-type receptors (NMDARs) for the spine formation.

KAKENHI(17023008; 18021004)

## S2A-B2 Involvement of ERM family actin-binding proteins in telencephalin-induced dendritic filopodia formation

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Dendritic filopodia are F-actin-containing, long, thin, and dynamic protrusions abundantly found in the developing neurons. They are morphologically and functionally transformed into spines as development proceeds. We found that a dendritie-associated IgCAM, telencephalin (TLCN; ICAM-5), slows spine maturation by promoting the dendritic filopodia formation and negatively regulating the filopodia-to-spine transition. To gain insight into the intracellular molecular mechanism underlying the TLCN-induced filopodia formation, we performed yeast two-hybrid screening and identified ezrin, radixin, and moesin (ERM) as specific binding partners of TLCN cytoplasmic region. Constitutively active ezrin induced dendtitic filopodia, while siRNA-mediated knockdown of ERM proteins resulted in decreased filopodia and increased spines. These results suggest that TLCN regulates formation, maintenance, and/or dynamics of dendritic filopodia through the interaction with ERM proteins during the critical period of brain development.

KAKENHI 16044249, 17024063; CREST

## S2A-B3 Modulation of excitatory synapse maturation by assembly of a postsynaptic protein complex containing PSD-95 and neuroligins

Alaa El-Husseini

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Processing of neuronal information is governed by the integration of excitatory and inhibitory synaptic inputs. One mechanism that influences excitatory synapse maturation is the recruitment of specific adhesion, scaffolding and signaling molecules to excitatory neuronal contact sites. We revealed that excitatory synapse maturation is influenced by the recruitment of neuroligins, a family of adhesion molecules implicated in synapse maturation. Studies on cultured hippocampal neurons revealed that association of neuroligins with PSD-95 influences the balance between neuronal excitation and inhibition, indicating that aberrant expression of these proteins may influence synapse maturation and neuronal excitability in vivo. Indeed, altered expression of neuroligins in mice results in major behavioral and social defects and altered neuronal excitability. These findings are of particular interest since mutations in neuroligins have been associated with neurodevelopmental psychiatric disorders such as autism, one of the most genetically determined neurodevelopmental disorders.



## S2A-B4 Precorsor BDNF is a novel regulator of synapse degeneration

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Since it was reported that precursor form of nerve growth factor (NGF) promotes neuronal apoptosis (Lee et al., 2001), it has been tested how precursor form of neurotrophins modulates neuronal structure and function, In this symposium, we will present precursor form of brain-derived neurotrophic factor (proBDNF) elicits spine degeneration. We used primary culture of hippocampal neurons and slice culture of hippocampus. As reported previously, treatment with mature form of BDNF for 2 days led to a significant spine protrusion. Unexpectedly, however, cultured hippocampal neurons exposed to proBDNF for 2 days reduced the density of PSD95-positive synapse, but not synaptophysin-positive ones. This effect of proBDNF was replicated in slice culture of hippocampus. We have now investigated the effect of this morphological change on synaptic function using electrophysiological methods. ProBDNF might be a novel regulator of synapse degeneration in central nervous system.

KAKENHI (40344171)

## S2A-B5 Involvement of Shc-related phospho-tyrosine signal adaptors in the dendritic spine formation

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Accumulative evidence suggests that various signal transduction molecules are involved in the neuronal activity-dependent remodeling of actin networks in post-synaptic spines. We previously isolated a neuron-specific homologue of phospho-tyrosine adaptor molecule Shc, i.e., N-Shc. We found that in the N-Shc gene deficient mice the hippocampal cognitive function such as learning and memory was elevated as well as the hippocampal long-term potentiation was enhanced. This is related to the interaction of N-Shc protein with the NMDA-receptor, possibly through the tyrosine phosphorylation by Fyn or Src-related kinases. In view of the spine morphology on functional modification of the synapses, we further wondered whether N-Shc might also be involved in the process of dendritic spine formation. When N-Shc was overexpressed in primary cultured hippocampal neurons, spine number was significantly diminished. This indicates that N-Shc is certainly involved in the activity-dependent synaptic modifications in the hippocampal neurons.

Grant-in-Aid for Scientific Research on Priority Areas

## S2A-C1 A role of RNA binding proteins in neuronal differentiation

Hirotaka J. Okano

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Paraneoplastic neurologic disorders (PNDs) are neurologic diseases occurring in the setting of non-neuronal tumors that express neuronal antigens. Hu, a neuronal RNA-binding protein, has been identified as a target antigen in PND associated with small cell lung cancer. Hu proteins are able to induce a neuronal phenotype when overexpressed in immature neural progenitor cells. Neural stem/progenitor cells in HuD KO mice exhibit increased self-renewal capacity and decreased differentiation ability toward neurons. Previous studies indicate that Hu induces cell cycle arrest and neuronal differentiation by binding to UTR region of several target mRNAs including CDK inhibitor p21. Recently, we identified hnRNP K as one of Hu-associating proteins, which restores Hu-induced p21 upregulation by binding to CU-rich element in p21 mRNA UTR. Furthermore Hu-induced cell cycle arrest and neurite outgrowth in N1E-115 are also antagonized by hnRNP K. These results indicate that functionally antagonistic two RNA binding proteins, Hu and hnRNP K, control the timing of the switch from proliferation to neuronal differentiation in neural progenitor cells.

KAKENHI

## S2A-C2 Nonsense mediated mRNA decay as a modifier of neurologic disease traits and phenotypes

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Nonsense mediated decay (NMD) pathway is an mRNA surveillance system, which typically degrades transcripts containing premature termination codons (PTCs), thereby preventing translation of unnecessary or aberrant transcripts. Recent studies on the mechanism by which nonsense transcripts are recognized and targeted for decay have revealed that not all PTC can be subjected to NMD. PTCs in specific positions in a gene are protected from NMD, thereby mRNAs that contain PTCs located within these protected regions are stably translated into aberrant truncated proteins, which can potentially express toxic effects in cells. Approximately one-third of mutations that cause human genetic disorders are nonsense and frameshift mutations. The phenotypes of such PTC-associated genetic diseases are likely to be modulated by triggering or escaping NMD. Here, I review the roles for the NMD pathway in altering the inheritance pattern for a disease trait and in modifying the ultimate phenotype for selected human diseases.

KAKENHI17390102, H18-Kokoro-Ippan-015

## S2A-C3 Is RNA involved in autistic behaviors?

Toru Takumi
*Osaka Bioscience Institute*

Mental or cognitive functions are difficult to be approached by molecular biological techniques, because no appropriate assay systems have been established yet. We cannot quantify the pathophysiological phenomena of higher brain functions except learning and memory. Even if we could identify the candidate genes for psychiatric diseases and make the targeting mice, we cannot prove that they are real model mice for these disorders. On the other hand, genetics is a powerful tool that brings us a molecule from the in vivo phenomena. We should perform human genetics and deal with psychiatric diseases, because we can make a diagnosis of psychiatric disease only in humans. However, mental illnesses are heterogeneous and no objective examination methods for their diagnoses are clinically used at this moment. In this situation, including availability of the recent tremendous data for human and mouse genomes, one way we took is, using the cutting-edge ES technology, to make model mice with human biological abnormalities like chromosomal aberrations, based on the clinical data. The mouse will be a founder for forward genetics and a target for further integrative studies.

KAKENHI(18023036)

## S2A-C4 In vivo application of RNA interference

Takanori Yokota
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Small interfering RNA (siRNA) is the process of sequence-specific, post-transcriptional gene silencing, that is expected to be a powerful tool for gene therapy of human diseases. We recently proved the principle that siRNA-mediated gene silencing can prevent the development of autosomal dominant neurodegenerative diseases, such as familial amyotrophic lateral sclerosis (ALS) with SOD1 mutation, by crossing the anti-SOD1 siRNA transgenic mice with SOD1G93A transgenic mice, a model for ALS. In gene therapy of dominantly inherited diseases with siRNA, mutant allele-specific suppression may be necessary for diseases in which the defective gene normally has an important role. Then, we used a new RNAi strategy for selective suppression of mutant allele, which can be applied to any mutation. The immune responses to siRNA/cationic vector complex and short-hairpin RNA toxicity in viral vectors have been important problems for the delivery of siRNA in vivo. To overcome these problems, we engineered new non-viral cationic vectors and developed a new strategy with adeno-assosiated virus vector, and discuss their feasibility and problems for future gene therapy.

## S2A-C5 Connection between RNA silencing and Fragile X Syndrome

Haruhiko Siomi
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Fragile X syndrome is the most common form of heritable mental retardation caused by loss-of-function mutations in the FMR1 gene. The FMR1 gene encodes an RNA-binding protein that associates with translating ribosomes and acts as a negative translational regulator. Recent work in Drosophila melanogaster has shown that the fly homolog of FMR1 (dFMR1) plays an important role in regulating neuronal morphology and function, which may underlie the observed deficits in behaviors of dFMR1 mutant flies. Biochemical analysis has revealed that dFMR1 forms a complex with AGO2, an essential component of RNA interference (RNAi) pathway in Drosophila. AGO2 associates tightly with exogenous and probably endogenous siRNAs and forms the RNA-induced silencing complex (RISC) that mediates cleavage of target RNAs. These findings suggest that dFMR1 may function in an RNAi-related apparatus to regulate the expression of its target genes. To test this, we are currently engaged in identifying small RNAs present in dFMR1/AGO2-associated complexes. The sequence information of these small RNAs should help determine target genes of dFMR1/AGO2-associated complexes.

MEXT and JSPS.

## S2A-D1 Long term observation of fine structural plasticity of neurons and glias in damaged cerebral cortex of living mice

Hiroaki Wake, Tomomi Nemoto, Junichi Nabekura
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The remodeling of neuronal network has been proposed during the recovery after various neuronal damages. However, because of technical limitation, remodeling of neuronal network in the central nervous system is still remained to be unclear. Two photon microscopy combined with transgenic animals expressing various fluorescent proteins in specific brain cells leads to observe fine structures of labeled cells in an in vivo animal. In addition to the improvement of imaging of cells in the deep cortical layers (1mm), time lapse imaging carried out on a single animal over several weeks allows us to observe a remodeling of neuronal fine structures. Damage also introduced a rapid change of processes movement of microglias following the transposition of their cell bodies. We also introduce several applications with two photon laser excitation in an in vivo animals.

CREST, JST

## S2A-D2 Dendritic excitability maturates somatosensory receptive field.

Shoji Komai
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Sensory experience is necessary for normal cortical development. This has been shown by sensory deprivation and pharmacological perturbation of the cortex. Since these manipulations affect the cortical network as a whole, the role of postsynaptic cellular properties during experience-dependent development is unclear. Here we addressed the developmental role of somatodendritic excitability, which enables postsynaptic spike-timing dependent forms of plasticity, in rat somatosensory cortex. We used siRNA-based knockdown of $Na^+$ channels to suppress somatodendritic excitability of small numbers of layer 2/3 pyramidal neurons in the primary sensory cortex, without altering the ascending sensory pathway. *In vivo* recordings from siRNA-expressing cells revealed that this manipulation interfered with the normal developmental strengthening of sensory responses. The sensory responsiveness of neighboring cortical neurons was unchanged, indicating that the cortical network was unchanged. We conclude that somatodendritic excitability of the postsynaptic neuron is needed for the regulation of synaptic strength in the developing sensory cortex.

Max Planck Society

## S2A-D3 Functional mapping of glutamate receptors in dendrites of mouse neocortical neurons in vivo using two-photon microscope

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Our laboratory has previously developed a two-photon uncaging and mapping technique to examine functions of glutamate receptors in dendrite, and found that functional expression of synaptic AMPA receptors is proportional to the volume of dendritic spine in hippocampus CA1 pyramidal cell in slice preparations. We now apply this uncaging and mapping technique to neocortical neurons in vivo. The neurons are whole-cell perfused with a fluorescent probe, and glutamate-induced currents are elicited at visually identified spines by two-photon uncaging of caged-glutamate compound applied into the cortex. We will report the relationship between geometry and functions of dendritic spines in neocortical neurons.

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## S2A-D4 The functional microarchitecture of the mouse barrel cortex

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Cortical maps are a hallmark of the organization of the cerebral cortex. However, the microorganization of corticalmaps at the level of single neurons is not known, mainly because of the technical limitations. Here, we used bulk loading of Ca2+ indicators combined with two-photon microscopy to image the activity of multiple single neurons in layer 2/3 of the moue barrel cortex in vivo. We developed methods that reliably detect single action potentials in approximately half of the imaged neurons. This allowed us to measure the spiking probability following whisker deflection and thus map the whisker selectivity for multiple neurons with known spatial relationships. At the level of neuronal populations the whisker map varied smoothly across the surface of the cortex. However, the response properties of individual neurons recorded simultaneously differed greatly, even for nearest neighbors. Trial-to-trial correlations between pairs of neurons were high over long distances. Our data suggest that the response properties of individual neurons are shaped by highly specific subcolumnar circuits and the momentary intrinsic state of the neocortex.

## S2A-D5 Single-cell resolution functional mapping with two-photon calcium imaging in vivo

Kenichi Ohki
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We labeled thousands of neurons of the visual cortex with a calcium-sensitive indicator in vivo. We then imaged the activity of neuronal populations at single-cell resolution with two-photon microscopy up to a depth of 400 micron. In rat primary visual cortex, neurons had robust orientation selectivity but there was no discernible local structure; neighbouring neurons often responded to different orientations. In area 18 of cat visual cortex, functional maps were organized at a fine scale. Neurons with opposite preferences for stimulus direction were segregated with extraordinary spatial precision in three dimensions, with columnar borders one to two cells wide. These results indicate that cortical maps can be built with single-cell precision.



## S2A-E1 Calcium store and presynaptic plasticity in the hippocampus

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A buildup of $Ca^{2+}$ within the presynaptic terminals during repetitive action potentials causes use-dependent enhancement of transmitter release. In addition to this common mechanism of presynaptic plasticity, some types of the synapse use additional mechanisms to amplify presynaptic $Ca^{2+}$ dynamics in an activity-dependent manner. $Ca^{2+}$-induced $Ca^{2+}$ release (CICR) from intracellular stores is one such likely candidate, and it has been suggested that ryanodine receptor-mediated CICR regulate presynaptic $Ca^{2+}$ dynamics at the hippocampal mossy fiber synapse, in which robust presynaptic forms of plasticity take place. Using presynaptic Ca2+ measurements, we examined the mode of activation of $Ca^{2+}$ store at the mossy fiber terminals and found that ryanodine receptors amplify presynaptic $Ca^{2+}$ dynamics in an activity-dependent manner, optimally at the LTP inducing condition. Immunohistochemical studies showed that ryanodine receptors are localized away from the active zones. These results suggest the specific and important roles of presynaptic calcium store in presynaptic plasticity at the hippocampal mossy fiber synapses.

## S2A-E2 Regulation of neural circuit and behavior by hippocampal mossy fibers

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The dentate gyrus of the hippocampus has been attracting special interest owing to possible involvement of adult neurogenesis of dentate granule cells (GCs) in the pathogenesis and/or treatment of psychiatric disorders. Synapses made by mossy fibers (MFs), axons of GCs, on CA3 pyramidal cells have characteristic properties such as large presynaptic terminals and remarkable synaptic facilitation, and have been supposed to play critical roles in the CA3 network. In fact, activation of a single MF input in bursts evoked action potentials in postsynaptic CA3 pyramidal cells and provided effective activity for induction of long-term potentiation at converging CA3 recurrent synapses. To reveal a role of MFs in regulating behaviors, effects of environmental manipulations on MF synaptic transmission and behaviors of mice were examined. Enriched housing caused a reduction in open-field locomotor activity and in magnitude of paired-pulse facilitation at the MF synapse, suggesting an involvement of MFs in locomotor regulation. Effects of chronic antidepressant treatment on behaviors and MF synaptic transmission will be also presented.

## S2A-E3 Development of functional networks by hippocampal mossy fibers

Ryuta Koyama
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Hippocampal mossy fibers, axons of dentate granule cells, run through the dentate hilus toward the CA3 region and make synapses with hilar and CA3 neurons. In the hippocampus of temporal lobe epilepsy patients and its animal models, mossy fibers intensely branch in the hilus and project oppositely to the molecular layer, where they provide a large number of excitatory synaptic contacts with the dendrites of granule cells. This abnormal development of mossy fibers, termed the mossy fiber sprouting, likely exacerbate the disease by forming hyperexcitable recurrent loops in the dentate gyrus that cause epileptiform burst firings of granule cells. My colleagues and I revealed that brain-derived neurotrophic factor (BDNF) induces branching of mossy fibers and are now investigating the role of cyclic adenosine monophosphate (cAMP) in regulating the direction of mossy fiber projections. In this talk, I will review the axon guidance mechanism of mossy fibers and discuss how they are misguided to the opposite direction under epileptic conditions, referring to our recent findings.

KAKENHI(18890052)

## S2A-E4 Adult hippocampal neurogenesis

Tatsuhiro Hisatsune
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New neurons are continuously generated in the subgranular zone of the hippocampal dentate gyrus thought adulthood, and are involved in some hippocampal-dependent memory tasks. Adult hippocampal neurogenesis is enhanced in particular physiological conditions (enriched environments, learning, exercise, etc.) and pathological conditions (epilepsy, ischemic stroke, etc.). We focus on the activity-dependent neurogenesis, and discovered the activation of hippocampal stem/progenitor cells by a typical hippocampal network activity, theta oscillations. Biochemical mechanism responsible for this activity-dependent regulation will be notified in this symposium. Since, the synaptic plasticity of new granular neurons is higher than old granular neurons; new neurons perhaps contribute to the dynamic reorganization of hippocampal circuit.

KAKENHI 18022008

## S2A-E5 Characterization of dentate mossy cell-restricted NMDA receptor knockout mice

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The glutamatergic mossy cells in the dentate hilus provide massive recurrent connections to dentate granule cells and mossy cells along the longitudinal axis and may play a significant role in hippocampal memory formation. To understand the role of mossy cell-mediated dentate networks in vivo we have generated mossy cell-restricted NMDA receptor (NR) knockout mice using the Cre/loxP system. We subjected the mutants and their controls to a battery of behavioral tasks. The mutants were impaired in exploratory behavior, in particular, after the animals are acclimated to the environment. They were also impaired in discriminating contexts in regards to contextual fear memory acquisition. However, there was no impairment in home cage activity, habituation to novelty, or anxiety/depression-like behavior, suggesting a crucial role of mossy cell NRs in episodic-like memory encoding. Furthermore, the mutants were more susceptible to kainite-induced seizure, suggesting a neuroprotective role of mossy cell NRs against external perturbations.

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## S2A-E6 Vesicular dynamics and its plasticity in the hippocampal synapse

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The synaptic vesicle dynamics and its plasticity is physiologically quantified by two parameters; the number of releasable vesicles (Nv) and the exocytotic probability of a vesicle (pexo). Here, individual large mossy fiber boutons were identified in the acute slice of hippocampus and the activity-dependent changes of synaptopHluorin, one of optical probes measuring exo-endocytosis were measured using a TV-42 transgenic mouse line that expresses synaptopHluorin specifically in these boutons. The large Nv with relatively small pexo appears to be one of mechanisms underlying the wide dynamic range of transmitter release. Either Nv, pexo or both were increased by PKC but the preference was variable from bouton to bouton. These properties appear to be fundamental for the hippocampus-dependent memory. *All animal procedures were conducted in accordance with the guiding principles of Japan Neuroscience Society.*

KAKENHI(17659061), Novartis Foundation Japan, Suzuken Memorial Foundation

## S2A-F1* The monkey pulvinar neurons differentially response to emotional face

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Pulvinar nucleus has been shown to receive visual inputs from the superior colliculus and to project to the amygdala. This circuit seems to provide a fast subcortical route for processing emotionally relevant visual stimuli. Nevertheless, the role of pulvinar nucleus on the recognition of emotional faces remains obscure. To investigate whether this circuit codes emotion recognition, we recorded pulviner neuronal activity during discrimination task for emotional expression of human face. Of 35% neurons responded to the visual stimuli. Among these visually responsive neurons, 24% neurons differentially responded to the facial stimuli. All these neurons responded differentially to facial identity and/or expressions. Furthermore, in some of the neurons, response latencies to the visual stimuli were shorter than the onset of saccadic eye movements. Taken together, the results indicated differentially responding neurons in the pulvinar regarding distinct emotional expressions of human faces and that it may mediate unconscious fast visual processing.

CREST, JST

## S2A-F2 Neuronal responses to species-specific emotional expression in the monkey amygdala

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The amygdala has been implicated in the processing of emotional signals. Many researches have focused on how the amygdala responds differentially to different types of facial emotion. Whereas who makes the emotion is also a crucial issue to produce appropriate social behavior, how amygdala neurons are affected by facial identity has not yet been well studied. Furthermore, the face and voice can convey the same information about emotion. However, it is still unclear whether amygdala neurons respond to vocal emotion. To address these issues, we recorded neuronal activity in the monkey amygdala while the monkeys watching nine video clips of species-specific emotional expressions (3 monkeys 3 types of emotion) and examined the responsiveness of amygdala neurons. In two-thirds of neurons responding to emotional expressions, the facial identity significantly affected their responses. Furthermore, one-fifth of neurons tested responded to both facial and vocal emotions. These results will be discussed.

## S2A-F3 More than just a pretty face: the importance of expression, gaze, and identity in the temporal lobe of the macaque

Kari L. Hoffman

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Macaques, like humans, are sensitive to a wide spectrum of social information present in faces: kin and gender, identity, facial expressions and gaze direction. Likewise, both groups process conspecific faces holistically, are sensitive to configurations of facial features, and demonstrate shifts of attention in responses to seen gaze shifts (i.e. joint attention). Possible neural correlates of several of aspects of face processing were examined using fMRI and multi-electrode recordings. When awake monkeys viewed faces that independently varied in expression and gaze, BOLD responses were seen over a large expanse of TE, and differential responses appeared in amygdaloid nuclei. Multi-site recordings of neural ensembles sampling from much of the temporal lobe reveal a variety of responses to several markers of identity, including faces, as well as to facial expressions. These results will be discussed in the context of previously-suggested conceptual modules for processing the multiple dimensions of face stimuli.

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## S2A-F4* Primate prefrontal neuronal activity during a competitive video game

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*Tokyo Metropolitan Institute for Neuroscience*

In our daily life we sometimes have competitive situations such as sports, contest, business etc. Competitive situations often evoke emotions in us; winning experience makes us feel pleasant and losing experience makes us feel frustrated. Furthermore, we would feel happier when we get a reward by winning a race (competitive situation) than when we simply get it without competition (uncompetitive situation). To investigate neural mechanisms of competition, we trained two monkeys in a shooting game. The monkey could obtain a reward by defeating the other monkey while it obtained no reward when defeated. We also trained monkeys in an uncompetitive game. Behavioral indices such as reaction time indicated that monkeys responded differently depending on whether the game was competitive or not, and depending on whether they won or lost the preceding competitive game. We recorded neuronal activity from the orbital and lateral prefrontal cortices, and found that prefrontal neurons responded differently depending on whether the game was competitive or not.

17022050

## S2A-F5 Dynamic social adaptation of parietal neurons in primates

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The brain function used to adapt our behaviour to social context is called social brain function, but the mechanism is not well studied at single cell. To reveal the mechanism, we recorded neuronal activity in parietal cortex of two monkeys while monkeys were sharing the social space with/without interacting each other. We found that the activity of left parietal cortices recorded simultaneously from two monkeys that showed robust response preference to action of self right arm but almost no response to motion of other, if they did not socially interact each other. But the preference was broken if social conflict emerged between monkeys, thus parietal neurons started to show complex combinatorial responses to motion of self and other. In the response modulation, parietal cortex adapted response properties depending on social contexts by discarding and recruiting different sets of neural populations. The results suggest that parietal neurons may contribute in contextual adaptation of social brain network system to novel environment.

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## S2A-F6 Mirror neurons and their role in the macaque social cognition

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Mirror neurons belong to a class of neurons found in the premotor and parietal cortex of macaques which activated by both execution and observation of goal-related actions. These neurons would serve the recognition of others' actions by matching the visual description of an action onto the monkey's cortical motor representation of that action. This matching system it has been reported to occur in macaques at behavioral level as they are able to recognize when being imitated by a human an experimenter. It has also been proposed that mirror neurons could be partly responsible of the primate capacities to repeat observed actions (i.e response facilitation and imitation). More recently, we explored the development of such abilities by testing infant macaques and found that soon after birth infant macaques imitate facial gestures made by a human experimenter. These data suggest that macaques are provided at birth with a neurophysiological mechanism, probably underpinned by mirror neurons, that allow them to understand others' behaviors and intentions and to tune the own behavior with others in an interactive exchange.



## S2A-G1 Mitochondrial dysfunction and aging

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A mutation in a subunit, cytochrome *b* large subunit (SDHC) of complex II in electron transport system, results in increasing $O_2^-$ production and therefore lead to premature aging in *C. elegans*. It is known that one of several mutations in this gene is found in some patients with an inherited propensity for vascularized head and neck tumors (i.e., paragangliomas).We have also established a transgenic cell line with a point mutation in the SDHC gene. There were many apoptotic cells in this cell line, and some cells that escaped from apoptosis underwent transformation. Oxidative stress from mitochondria leads to pathology such as apoptosis resulting precocious aging and also transformation resulting tumorigenesis. Recently, we have constructed a transgenic mouse ubiquitously expressing the mutated SDHC. This mutation leads to premature aging with several phenotypes such as decreasing of muscular power and lost of eyesight. The SDHC mouse is anticipated as a model animal to understanding the mechanisms of aging and age-related disease by $O_2^-$ from mitochondria.

KAKENHI (17500291); Aging Research from the Ministry of Health, Labor and Welfare, Japan.

## S2A-G2 Mitochondrial dysfucntion in Parkinson's disease

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Parkinson's disease (PD) is the most second neurodegenerative disorder next to Alzheimer disease (AD). However, the primary cause for PD is still unknown, but oxidative stress and mitochondrial dysfunction have been implicated as important contributors to neuronal death in substantia nigra (SN) of PD. Neurons as a post-mitotic cell could have error-avoiding mechanism against oxidative DNA damage. Indeed, there has been several DNA repairing enzymes such as MTH1, OGG1, and MUTYH. All the three enzymes up-regulated in the SN of PD patients, suggesting these three enzymes cooperate in mitochondrial DNA repairing in PD brain. In addition, although most of PD patient are sporadic, familial PD (FPD) have been reported. Total seven genes have been identified so far. PINK1 (Park6) has a mitochondrial targeting sequence. PINK1 functions upstream of parkin (Park2). Moreover, DJ-1 (Park7) is also link to mitochondrial function and oxidative stress. Considering the similarities of parkinsonism in Park2, 6, and 7, the three molecules could share a common pathway including mitochondrial function.

## S2A-G3 Mitochondrial dysfunction inducing neuro-muscular disorders known as mitochondrial encephalomyopathies

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Mitochondrial impairment results in a host of devastating conditions known generally as respiratory chain diseases. Furthermore, mitochondrial dysfunction has been conclusively implicated in nearly every chronic human neurodegenerative disease, including mitochondrial encephalomyopathy, amyotrophic lateral sclerosis, Huntington's, Alzheimer's, and Parkinson's diseases. Primary mutations in several distinct mitochondrial genes, as well as nuclear genes that encode proteins localized to the mitochondria, are known to cause a variety of related and devastating encephalomyopathies. There are over 150 mutations in mitochondrial genomes and depletion of the genomes causing these related diseases. Because mitochondrial encephalomyopathy is the disease that is most extensively studied at various levels including DNA, cell, tissue, organ, individual and social levels. Current status of the knowledge of complex and distinct genotype-phenotype relationship of the disease will be introduced and discussed.

KAKENHI 16390309

## S2A-G4 Mitochondrial dysfunction in mental disorders

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Although bipolar disorder (BD) is a multifactorial disease, it is infrequently manifested by one of pleiotropic effects of a single gene mutation. Patients with chronic progressive external ophthalmoplegia (CPEO) sometimes have comorbid mood disorders. CPEO is a mitochondrial disease, which is accompanied by mitochondrial DNA (mtDNA) defects caused by mutations in nuclear genes such as *POLG* (mtDNA polymerase). Mitochondrial dysfunction in BD has also been suggested by magnetic resonance spectroscopy and pharmacological studies. We generated transgenic mice, in which mutant POLG was expressed in a neuron-specific manner. The mice showed forebrain-specific defects of mtDNA and exhibited mood disorder-like phenotypes. These abnormal behaviors were worsened by tricyclic antidepressant and improved by lithium. In addition, we screened mutations in *POLG* gene in 466 BD patients and 508 controls and identified 21 mutations. Among them, mutations located in functional domains were found more frequently in patients. These results suggest that *POLG* mutations and consequent mtDNA defects could be one of genetic risk factors for BD.

KAKENHI17790830

## S2A-H1 Central extended amygdala as a key component for pain-induced aversion

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The bed nucleus of the stria terminalis (BST) is a component of the extended amygdala, and play crucial role in the regulation of negative affective states such as anxiety and fear. We examined the role of the noradrenergic (NA) transmission within the BST in the negative affective component of pain in rats. NA release within the BST was significantly elevated by noxious stimuli. Intra-BST injection of a β-adrenoceptor antagonist timolol significantly suppressed noxious stimuli-induced CPA without affecting nociceptive behaviors. Intra-BST inejction of isoproterenol, a β-adrenoceptor agonist, dose-dependently produced CPA. In addition, we tested the effect of intra-BST isoproterenol on the anxiety-like behaviors using an elevated plus maze test. Intra-BST isoproterenol dose-dependently reduced the open arm entries and time spent in the open arms, indicating the enhancement of anxiety. These results suggest that noxious stimuli-induced NA release within the BST contributes to the negative affective, but not sensory, component of pain.

## S2A-H2 Emotional memory of chronic pain engraved on the central amygdala synapses

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Human patients with chronic pain often complain of obstinate affective problems including depression, anxiety and discomfort, even after apparent recuperation of the primary causes of the pain. The ascending pathway from the nociceptive neurons in the superficial layers of the spinal cord to the laterocapsular part of the central amygdala (CeA) via the lateral nucleus of the parabrachialis (NPB) plays essential role in the link between pain sensation and emotional responses (Gauriau & Bernard, 2002). We have recently demonstrated that excitatory synaptic transmission between the afferent fibers arising from the NPB and CeA neurons is potentiated in the animal model of chronic neuropathic pain (Ikeda et al., Pain, 2007). Based on the observation that such potentiation remains after the recovery from neuropathic pain. We propose that structural consolidation of this potentiation in the CeA synapse is the primary mechanism for the emotional memory of pain.

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## S2A-H3 A role of the NPW/NPB system in regulation of emotional responses

Takeshi Sakurai

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GPR7 (NPBWR-1) is a G protein-coupled receptor (GPCR), whose endogenous ligands were recently identified as neuropeptide B (NPB) and neuropeptide W (NPW). Despite the strong expression of GPR7 in the amygdala and BNST, areas known to be associated with anxiety, fear, learning and memory, the exact roles of GPR7 in these functions were totally unknown. Here, we investigated the potential physiological roles of the NPB/NPW-GPR7 system by studying GPR7 knockout mice using a battery of behavioral experiments. GPR7-/- mice showed increased anxiety-related behavior in light-dark exploration test. In resident-intruder test, GPR7-/- mice produced more fear behaviors than wild-type control mice, including persisted chasing, longer physical contact time and higher and sustained elevations of heart rates and blood pressure. We also found that GPR7-/- mice showed decrease in freezing behavior in contextual fear conditioning, while they respond normally in auditory cued fear conditioning. These data suggest that GPR7 plays important roles in regulation of behavioral and autonomic responses to threat and environmental dangers.

KAKENHI

## S2A-H4 Regulatory Mechanism of Anxiety by Oxytocin

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Oxytocin is a peptide hormone that causes uterine contraction and facilitates the delivery of the newborn, and stimulates the milk ejection during motherhood. Abundant oxytocin receptors also appear in central nervous system (CNS), especially in limbic system. Oxytocin is involved in the regulation of social behaviors, including maternal care, affiliation and social behaviors in CNS. Moreover, recent studies have shown that oxytocin has anti-anxietic effect during motherhood. However, the exact molecular mechanism is unclear. We identified a crucial molecule involved in the regulation of anxiety by oxytocin. Oxytocin induced the expression of Regulator of G-protein Signaling2 (RGS2), which is known as a modulator of anxiety, in the amygdala slices of female mice. RGS2 expression in the amygdala of lactating mice was significantly higher than that of virgin mice. Moreover, the expression of RGS2 in virgin mice undergone restraint stress was facilitated compared with that of control mice. We will further discuss the molecular mechanism of anxiety control by oxytocin in amygdala.

Scientific Research on Priority Areas

## S2A-J1 Mechanisms of polyglutamine-dependent neurodegeneration revealed by automated imaging and structural approaches

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Huntington's disease is a devastating neurodegenerative disorder caused by an abnormal polyglutamine stretch within the huntingtin protein. A major goal of our laboratory is to elucidate relevant molecular and cellular mechanisms of pathogenesis. We have recently developed automated imaging systems and analytical approaches that make it possible to monitor the process of neurodegeneration in individual neurons over time. In turn, these approaches make it possible to identify cellular and molecular changes that predict a particular fate, thereby providing novel insights into their role in pathogenesis. We have used this system to study the role of inclusion formation in live neurons and are currently using it to study the significance of other cellular process such as proteasome activity and autophagy. In parallel, we have developed conformation-specific antibodies and are using crystallography to explore potential structural bases for mechanisms of neurodegeneration. We have evidence that monomers of mutant huntingtin may adopt structures with pathogenic significance.

## S2A-J2* Polyglutamine protein oligomer formation in cells characterized by fluorescence correlation spectroscopy

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The polyglutamine (polyQ) diseases, including Huntington disease and spinocerebellar ataxias, are caused by abnormal expansions of the polyQ stretch within disease proteins, which triggers the proteins to oligomerize into amyloid fibrils. However, the oligomerization *in vivo* has not been well characterized despite its important role in the pathogenesis. We here employed fluorescence correlation spectroscopy (FCS) to explore oligomer formation. PolyQ-GFP proteins expressed in cultured cells showed a time- and polyQ-length-dependent increase in particle size and diffusion time, indicating oligomer formation. We further show that the polyQ binding peptide QBP1, which suppresses polyQ-induced neurodegeneration *in vivo*, prevents oligomer formation of polyQ-GFP proteins. We therefore conclude that FCS is a useful technique to monitor oligomerization of the polyQ protein as a therapeutic target for the polyQ diseases.

KAKENHI(17028035, 18023025)

## S2A-J3* Soluble polyglutamine oligomer formation precedes inclusion body formation and increases the cytotoxicity

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Expanded polyglutamine (polyQ) repeats cause neurodegenerative disorders. Here we show the oligomerization of polyQ repeats by fluorescence resonance energy transfer (FRET) microscopy in live cells. FRET signals were detected when donor and acceptor fluorescence proteins attached to the same side of polyQ proteins, supporting a parallel $\beta$-sheet or a head-to-tail cylindrical $\beta$-sheet model. These FRET signals disappeared in semi-intact cells, indicating that polyQ oligomers are soluble. PolyQ formed soluble oligomers in a length-dependent manner, followed by the inclusion body (IB) formation. Notably, analysis by survival assay for neuronally differentiated cells revealed that cells with soluble oligomers died faster than those with IBs or monomers. These results indicate that a length-dependent formation of oligomers is an essential mechanism underlying neurodegeneration in polyQ disease.

## S2A-J4 Proteome analysis of soluble nuclear proteins links genotoxic stress to the polyglutamine disease pathology

Hitoshi Okazawa

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Nuclear dysfunction is a key feature of the pathology of polyglutamine (polyQ) diseases. Mutant polyQ proteins are suggested to impair nuclear functions, whereas a systematic analysis of quantitative changes of soluble nuclear proteins in the polyQ pathology has not been performed. In this study, proteome analyses of soluble nuclear proteins prepared from neurons expressing huntingtin (Htt) or ataxin-1 (AT1) protein reveal that mutant AT1 and Htt similarly reduce high mobility group B1/2 (HMGB1/2) proteins. HMGBs interact with mutant AT1 and Htt in IP and pull-down assays. Immunohistochemistry shows that HMGBs reduce in the nucleus of affected neurons. Compensatory expression of HMGBs ameliorates polyQ-induced pathology in primary neurons and in Drosophila polyQ models. Furthermore, HMGBs repress genotoxic stress signals induced by mutant Htt or transcriptional repression. Collectively, our proteomics analysis reveals a new pathology that genotoxic stress is activated in polyglutamine disease through reduction of HMGB proteins.

KAKENHI(18390254, 18023014), JST (PRESTO)

## S2A-J5 A new autosomal dominant cerebellar ataxia linked to chromosome 16q22.1 (16q-ADCA)

Hidehiro Mizusawa

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In Japan nearly 30% of spinocerebellar ataxia cases are hereditary and most of them are autosomal dominant. Etiologic genes of many cases however remained unknown. We identified an autosomal dominant cerebellar ataxia linked to the chromosome 16q22.1 (16q-ADCA). The phenotype of 16q-ADCA is late-onset pure cerebellar ataxia. The neuropathology revealed a unique degeneration of Purkinje cells surrounded by halo-like amorphous material positive for synaptophysin. Some Purkinje cells show somatic sprouts. A heterozygous C to T single-nucleotide substitution in 5-UTR of a new gene puratrophin-1 was found in all the patients examined. The full-length puratrophin-1 mRNA had an open reading frame of 3,576 nt predicted to contain important domains including spectrin repeat and Rho-GEF domain. Cytoplasmic aggregates immunoreactive for puratrophin-1 were seen in Purkinje cells of 16q-ADCA cases. A 16q-ADCA patient without the substitution has recently been reported and a different gene might be causative. 16q-ADCA appears very common next to SCA6 in Japan and the elucidation of the pathomechanism is very important.

## S2A-J6 Reversible dysfunctioin in polyglutamine disease

Gen Sobue

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Spinal and bulbar muscular atrophy (SBMA) is a hereditary neurodegenerative disease caused by expansion of CAG repeat in the androgen receptor (AR) gene. Elucidation of pathogenesis leads to emergence of candidate drugs to treat this devastating disease: LHRH analog, HSP inducer, Hsp90 inhibitor, and histone deacetylase inhibitor. Fluoro-gold labeling and sciatic nerve ligation demonstrated an impaired retrograde axonal transport in the SBMA mice. The mRNA level of dynactin1, an axon motor for retrograde transport, was significantly reduced in the SBMA mice resulting from pathogenic AR-induced transcriptional dysregulation. These pathological events were observed prior to the onset of neurological symptoms, but were reversed by castration. Overexpression of dynactin1 mitigated neuronal toxicity of the pathogenic AR in a cell culture model of SBMA. These observations indicate that polyglutamine-dependent transcriptional dysregulation of dynactin1 plays a crucial role in the reversible neuronal dysfunction in the early stage of SBMA.

## S2A-K1 Integrative functional analysis of the "brain" of the nematode *C. elegans* for odor-avoidance

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Recently we found that the avoidance behavior of *C. elegans* to repellent odors was enhanced, rather than being reduced, after preceded exposure to the odor. We also found that the formation of "memory" of the preexposure did not require the presence or absence of food as an unconditioned stimulus, and that the formed "memory" was stored at least for 1 hr. In contrast to the reduction of sensory response such as adaptation, the enhancement of sensory response has been poorly studied, particularly at the molecular level, with exceptions of mammalian pain sensation and gill-withdrawal reflex in *Aplysia*. Therefore, analyzing the enhancement of the avoidance behavior of *C. elegans* may lead to the identification of a novel regulatory mechanism of the sensory response enhancement. I will describe our integrative approaches to elucidate regulatory mechanisms for such plastic behavior in a small neuronal network.

## S2A-K2 The molecular basis for social behavior and caste differentiation in termites.

Toru Miura

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Social or altruistic behavior seen in social animals is peculiar in comparison with other solitary animals. Especially the eusocial insects like bees, ants and termites, show the highest sociality with labor division among castes. So far, we have investigated the physiological and developmental mechanisms underlying the termite sociality, using Hodotermopsis sjostedti. In particular, we focused on soldier differentiation, because the soldier caste shows the specialized morphology and defense behavior, and the artificial soldier induction is easily accomplishable. We analyzed the soldier morphogenesis and the accompanying gene expression profiles. Furthermore, neural system is modified in the soldier differentiation. Indeed we identified soldier-specific giant neurons that control mandibular muscles. Although such ontogenic modifications are important, the feedback system controlling caste ratio should exist to regulate the entire social organization. We report the analysis on pheromonal communications, which are important for the social systems.

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## S2A-K3 Development and function of spinal locomotor circuits in zebrafish

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Recent molecular genetic studies have implicated a number of transcription factors in the specification of different interneurons. Investigating how interneurons develop and function in amniotes, however, is not trivial due to enormous complexity of their spinal circuits. In contrast, zebrafish spinal circuits are much simpler with less number of distinct classes of neurons, making it more feasible to address this issue. We have set to define the morphology and functional properties of spinal neurons that express a particular transcription factor. Methodologically, the core of our approach is to visualize transcription factor positive cells by making transgenic zebrafish that express fluorescent proteins in these cells. The transparency of embryos and larvae greatly facilitate morphological and physiological analyses of the labeled neurons. We have made a number of transgenic fish lines, including chx10 and nkx2.2. Neurons that express these genes are mainly excitatory, and appear to regulate locomotion in zebrafish. I will present our recent data on these neurons.

## S2A-K4 Cognitive basis of social behaviour in the crow: understanding 'who' is doing 'what'

Ei-Ichi Izawa

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Corvids such as crows and jays are known to have higher socio-cognitive skills at the similar level of primates. Cognitive similarity between phylogeneticaly distinct corvids and primates is suggested as evolutional convergence based on prerequisites of ecological and physiological counterparts in the two species. Jungle crows (JC) flexibly change the social form depending on the age, the time of the day, and the season. This fission-fusion life style provides many occasions of inter-individual interactions where socio-cognitive skills should work advantageously for survival. Especially, understanding who is doing what can bring benefits / cost reduction to individuals.In this symposium, I will talk about JCs' cognitive abilities to understand who is doing what by showing our findings of stable dominance formation, vocal individual recognition, and observational learning. Neuroanatomical data of their highly developed forebrain will also be discussed. This talk is an attempt to show a potential of neuroethological approach not only in the research of sensory system but also in that of higher cognitive system.

## S2A-K5 Dendritic design implements algorithm for extraction of sensory information in insect sensory interneuron

Hiroto Ogawa

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While sensory information is encoded by firing pattern of individual sensory neurons, it is also represented by spatio-temporal patterns of activity in population of neurons. For example, directions of air-current surrounding the cricket are represented by the spatial patterns in ensemble activities of mechanosensory afferents. Postsynaptic sensory interneurons must decode these population responses and extract the directional information reliably. We examined how identified sensory interneurons decode the directional information represented by population of sensory afferents, using simultaneous imaging of pre- and postsynaptic $Ca^{2+}$ signals. Two types of the sensory interneurons, which have different dendritic design in electrotonic distances from dendritic branches to a spike-initiating zone, displayed different behavior for extraction of directional tuning property from the sensory afferents. It is possible that the differences in distribution of synaptic weights due to the dendritic geometry could be related to 'decoding algorithm' of sensory information in the sensory interneurons.

KAKENHI16570068

## S3A-A1 Mechanisms of memory formation by transcription factors

Satoshi Kida

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Gene expression regulation plays crucial roles in memory formation. To understand the mechanisms of memory formation from the view of gene expression regulation, we are investigating roles of transcription factors in memory formation using conditional mutant mice. CREB is phosphorylated at Serine 133 by cAMP and $Ca^{2+}$-dependent kinases and activates CRE-mediated transcription through the interaction with coactivator CBP. Recent our studies using two transgenic mice expressing dominant active CREB mutant (CREB Y134F or CREB DIEDML) showed that facilitation of interaction of CREB-CBP enhances LTP and memory formation. Taken together with our previous observation that inhibition of this interaction blocks memory formation, we are concluding that CREB-CBP interaction functions as a molecular switch to form memory. To further understand the contribution of transcription factors for memory formation and molecular network among them, we generated conditional mutant mice of several transcription factors and are investigating the roles of them in the processes of learning and memory formation.

Scientific Research on Priority Areas-Molecular Brain Science-(18022038)

## S3A-A2 Electrophysiological and behavioral analysis of gene-targeted mice to understand mechanisms of learning and memory

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To understand molecular and cellular mechanisms of learning and memory, combinations of genetic, electrophysiological, histological and behavioral techniques have been used. We show here some examples of gene-targeted mice that exhibit learning abnormalities as well as electrophysiological and/or histological abnormalities in some areas of the limbic system such as hippocampus and amygdala. For example, we generated mice with a knockin mutation of the tyrosine (Tyr)-1472 site to phenylalanine (Y1472F) of the NR2B subunit of the NMDA receptor. The NR2B subunit is tyrosine-phosphorylated in the brain, with Tyr-1472 its major phosphorylation site. The knockin mice showed impaired fear-related learning and reduced amygdaloid long-term potentiation. We thus identify Tyr-1472 phosphorylation as a key mediator of fear learning and amygdaloid synaptic plasticity.

KAKENHI (17023011, 18100003) and RISTEX, JST

## S3A-A3 Reconfiguration of synaptic network and enhancement of memory by magnesium ion

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Learning and memory are the one of fundamental capability of brain. Yet little is known about the endogenous molecules regulating this capability. Here we show that increasing magnesium consumption in intact animal enhances learning and memory function of rats in different aging. At the level of neuronal connections, elevated magnesium triggers a redistribution of synaptic weights on dendritic tree from fewer numbers of strong synapses to larger number of weaker synapses. This synaptic reconfiguration enables a selective enhancement of excitatory synaptic transmission for correlated inputs during coincident detection, while keeping it constant for uncorrelated inputs. This, coupled with up-regulation of NR2B-containing NMDARs, enhances the capacity of the synaptic network for modification by correlated inputs. Our findings suggest that magnesium is a key regulator of a proper synaptic configuration essential for memory function.

## S3A-A4 Symptomatic treatment for Alzheimer's disease: enhancing cholinergic function.

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Acetyl cholinesterase inhibitors (AChEIs) represent the current gold standard for treatment of Alzheimer's disease (AD). Although they can provide significant benefit for patients with mild to moderate symptoms, we still need to identify treatments that will show greater efficacy and tolerability, and reach patients who do not respond to AChEIs. Validating novel targets requires the development of model systems that can predict efficacy and safety in humans. We therefore developed and/or refined protocols for testing cognitive enhancement in normal rats. Our aim was to identify tests that are sensitive to donepezil, AChEI and the leading treatment for AD. After establishing positive effects of donepezil in two tests, novel object recognition (NOR) and the Morris water maze, we tested more specific cholinergic agonists to determine whether they might also be efficacious in humans. The results indicate that specific muscarinic agonists and nicotinic a7 agonists are both capable of enhancing cognitive function in normal adult rats.

## S3A-A5 Molecular and cellular cognition: unraveling mechanisms of learning and memory

Alcino J Silva

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Our laboratory is studying the role of hippocampal and prefrontal function in recent and remote memory. We are interested in uncovering general rules for how these two brain regions account for their unique roles in recent and remote memory. We are also studying how molecular mechanisms modulate cellular responses that underlie key microcircuit properties implicated in learning and memory. To accomplish this we are using a myriad of techniques including region and temporal specific transgenic manipulations, viral vectors, pharmacology, in vivo and in vitro electrophysiology, in vivo two-photon scanning confocal microscopy, as well as a battery of behavioral tasks designed to probe hippocampal and prefrontal cortical function. Our laboratory is also interested in understanding the mechanisms underlying cognitive disorders such as those underlying learning disabilities associated with Neurofibromatosis Type 1 (NF1), Tuberous Sclerosis and schizophrenia.

## S3A-A6 Molecular mechanism underlying maintenance of fear memory

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Memory process consists of at least three distinct phases, acquisition, maintenance, and retrieval. To address functional role activin plays in memory formation process, we generated activin and follistatin (that antagonizes activin function) transgenic mice (BIA/tTA and BIF/tTA, respectively) in which the transgene expression was tightly regulated by DOX in a forebrain-specific manner (Tet OFF system). Transgene expression was turned OFF or ON within 3d by (+/-) DOX. Contextual fear conditioning with these mice revealed that activin function is required during maintenance phase of fear memory for one week-retention. Furthermore, activin overexpression during maintenance phase enhanced long-term memory. Thus, according to the activin level in the forebrain during maintenance phase, fear memory that was once acquired tightly could be erased or further-consolidated.

CREST, JST

## S3A-B1 Epigenetic mechanism regulating fate specification and plasticity of neural cells

Kinichi Nakashima[1], Eriko Takatsuka[1], Toru Yamashita[2,3], Jenny Hsieh[4], Fred H. Gage[5], Masakazu Namihira[1], Hideyuki Okano[2], Kazunobu Sawamoto[3,6], Jun Kohyama[1]

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Methyl-CpG binding protein transcriptional repressors (MBDs) are expressed predominantly in neurons in the central nervous system. We report here that oligodendrocytes, which are devoid of MBDs, can transdifferentiate into astrocytes after injury in response to extracellular stimuli activating the JAK/STAT pathway, suggesting that differentiation plasticity in neural cells is regulated by cell-intrinsic epigenetic mechanisms in collaboration with ambient cell-extrinsic cues. These findings imply that demyelination after injury is due in part to oligodendrocyte to astrocyte transdifferentiation.

## S3A-B2 Signals regulating the differentiation vs. maintenance of neural progenitor cells

Takehiko Sunabori, Hideyuki Okano

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Signals regulating the maintenance and commitment of the neural progenitor cell have been intensively studied. However, many of the evidence are based on *in vitro* studies and also little is known about the relationship between those well learned signaling cascades. Here we would like to discuss the more detailed timing of the mammalian neural progenitor cell to determine its cell fate by verifying the activation of each signal especially *in vivo*.

SORST

## S3A-B3 Regulatory mechanisms of neural differentiation from pluripotent cells

Yoshiki Sasai

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We previously reported an efficient method that induces selective neural differentiation from mouse ES cells using coculture with PA6 stromal cells. This method, named SDIA, can be used to generate a wide variety of neural tissues including midbrain dopaminergic neurons and motor neurons. To apply a similar principle to human application, we recently established a culture method of hES cells that induces neural differentiation using human amniotic membrane matrix (AMED). On the other hand, SDIA (or AMED)-treated ES cells rarely differentiate into telencephalic cells. We demonstrate directed differentiation of telencephalic precursors from mES cells using serum-free suspension culture (SFEB). Treatment with Wnt and Nodal antagonists during the first five days of SFEB culture causes selective neural differentiation in ES cells. SFEB induces efficient generation (35%) of Bf1+ telencephalic cells from both mouse and huma ES cells. Thus, floating aggregates of ES cells in default of caudalizing signals generate naive telencephalic precursors that acquire subregional identities by responding to extracellular patterning signals.

## S3A-B4 Molecular mechanism of sox2 expression in neural stem cells

Toru Kondo

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Oligodendrocyte precursor cells (OPC) can behave as multipotent neural stem cells (NSC) when the cells are cultured in a specific condition. We have shown that a number of NSC markers are activated when OPC revert to NSC-like cells (NSLC). Among them, we focused Sox2, which is an essential transcription factor in NSC, and found that Sox2 is involved in the proliferation and maintenance of NSLC. Moreover we found that a SWI/SNF chromatin remodeling complex is recruited to the sox2 enhancer and histone H3 around the enhancer are converted to the active form in the reversion. Because the SWI/SNF complex does not recognize any specific DNA sequences, it is speculated that an unknown DNA-binding factor in NSC binds and recruits the SWI/SNF complex to the enhancer. Recently we have identified a candidate factor that recognizes the sox2 enhancer and associates with Brm, which is one of essential factors in SWI/SNF complex, in NSC. Thus these findings suggest that the OPC reversion depends on the epigenetic modification and that OPC might be an alternative source of NSC for CNS therapy.

RIKEN internal fund

## S3A-B5 Generation of high quality iPS cells

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Human ES cells faces difficulties regarding use of human embryos and rejection following implantation. One way to circumvent these issues is to generate pluripotent cells directly from somatic cells. We have previously shown that pluripotent stem cells can be induced from mouse fibroblasts by four factors (Oct3/4, Sox2, c-Myc and Klf4), and by selection for *Fbx15* expression. These iPS (induced pluripotent stem) cells showed morphology and proliferation, and teratoma formation similar to ES cells. These data demonstrated that pluripotent cells can be generated from fibroblast culture with a few defined factors.
However, microarray analyses revealed significant difference in gene expression between ES cells and iPS cells. Furthermore, iPS cells failed to produce adult or germ-line chimeras when transplanted into blastocysts. We are now trying to improve the quality of iPS cells by using other selection markers or other cell sources.

CREST, JST, Japan

## S3A-C1 BDNF stimulates neuronal cholesterol biosynthesis and accumulates presynaptic proteins in lipid rafts

Shingo Suzuki[1,2,3], Kazuyuki Kiyosue[4], Shunsuke Hazama[1,5], Akihiko Ogura[5], Tomoko Hara[1], Hisatsugu Koshimizu[1], Masami Kojima[1,3]

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Brain-derived neurotrophic factor (BDNF) can control transcription and protein synthesis to exert multiple biological functions in the CNS. However it still remains unclear whether BDNF regulates lipid biosynthesis to regulate these functions. Here we show that BDNF exerts cholesterol biosynthesis in cultured primary neurons, and that the BDNF-mediated increase in cholesterol was restricted to lipid raft microdomains. Further analysis demonstrated that BDNF-induced cholesterol biosynthesis constitutes a crucial step in BDNF modulation of synaptic transmission by increasing the RRP of synaptic vesicles. These results indicate a novel role for BDNF in cholesterol metabolism and synapse development.

## S3A-C2 Regulation of dendritogenesis via a lipid raft-associated $Ca^{2+}$/calmodulin-dependent protein kinase CLICK-III/CaMKIγ

Sayaka Takemoto-Kimura, Natsumi Ishihara-Ageta, Mio Nonaka, Hiroyuki Okuno, Haruhiko Bito

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$Ca^{2+}$ signaling plays a central role in activity-dependent regulation of dendritic arborization during CNS formation, but key molecular mechanisms downstream of calcium elevation remain poorly understood. Here we show that the C-terminal region of $Ca^{2+}$/calmodulin-dependent protein kinase CLICK-III (CL3) /CaMKIγ was uniquely modified by two sequential lipidification steps: prenylation and palmitoylation. These modifications were critical for efficient CL3 membrane anchoring and targeting into detergent-resistant lipid microdomains (or rafts). CL3 is highly expressed in the cortical plate, and specifically promoted dendritogenesis of cortical neurons by acting upstream of RacGEF STEF and Rac, both present in lipid rafts. Furthermore, CL3 was required for BDNF-stimulated dendritic growth. Thus, CL3 may represent a key element in the $Ca^{2+}$-dependent and lipid raft-delineated switch that turns on extrinsic activity-regulated dendrite formation in developing cortical neurons.

KAKENHI (18790193,17023010)

## S3A-C3 Asymmetric $Ca^{2+}$ signaling and membrane dynamics mediate growth cone guidance

Hiroyuki Kamiguchi

*RIKEN Brain Science Institute*

Growth cones migrate along the correct paths, not only directed by guidance cues but also contacted by local environment via cell adhesion molecules (CAMs). Many guidance cues attract or repel the growth cone via asymmetric $Ca^{2+}$ signals. Its turning direction depends on $Ca^{2+}$-induced $Ca^{2+}$ release (CICR) through the ryanodine receptor type 3 (RyR3): $Ca^{2+}$ signals with CICR mediate attraction whereas $Ca^{2+}$ signals without CICR mediate repulsion. The activity of RyR3 is controlled by CAMs via cAMP and protein kinase A. In this way, axon-guiding and CAM-derived signals are integrated by RyR3. Attractive $Ca^{2+}$ signals facilitate intracellular membrane transport to the leading front and subsequent VAMP2-mediated exocytosis on the side with elevated $Ca^{2+}$. In contrast, repulsive $Ca^{2+}$ signals do not trigger such membrane dynamics. Growth cone attraction, but not repulsion, is prevented by inhibition of VAMP2-mediated exocytosis. The results indicate that growth cone attraction is driven by asymmetric membrane dynamics and that growth cone repulsion is driven by different mechanisms, not simply by changing the left/right polarity of the same molecular machinery.

## S3A-C4 Lipid rafts and Abeta production

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Amyloid beta-protein (Abeta), a major component of the senile plaque that is a pathological hallmark of the Alzheimer's disease (AD) brain, plays a pivotal role for the development of AD. Abeta is produced from beta-amyloid precursor protein (APP), a type I membrane-spanning protein, through successive cleavages by beta- and gamma-secretases. While beta-secretase is a pepsin-like aspartyl protease and cleaves APP in its luminal region, gamma-secretase is a multimeric membrane protein complex with its catalytic aspartic acids sitting within the membrane and cleaves the transmembrane domain of APP. One of the major locations for Abeta production within the cells is considered to be lipid rafts, cholesterol and sphingolipid enriched membrane microdomains. Endocytosis associated with the microdomain fusion triggers beta-cleavage. Active gamma-secretase, which is present abundantly in these domains, subsequently takes place gamma- and epsilon-cleavages. Here, how gamma-secretase cleaves APP within the membrane and whether membrane lipid components affect on gamma-secretase activity will be presented.

## S3A-C5 Apolipoprotein E-isoform-specific modulation of cholesterol metabolism in neurons

Makoto Michikawa

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Because the cholesterol metabolism in the central nervous system (CNS) is segregated from the systemic circulation by the blood-brain barrier, the profile of the lipid transport system in CNS is quite different from that in the systemic circulation. The lipoprotein found in CNS is the high-density lipoprotein (HDL), and apolipoprotein E (ApoE) is one of the major apolipoproteins regulating lipid transport in CNS. Ii is shown that ApoE4, one of the ApoE isoform, is a strong risk factor for Alzheimer's disease (AD); however how ApoE4 is involved in AD pathogenesis remains undetermined. We found that cholesterol release to generate HDL induced by lipid-free ApoE is modulated by two major factors: the presence or absence of inter-molecular dimer formation and the intra-molecular domain interaction. We also found that ApoE on lipid particles has ApoE-isoform-dependent effect on lipid-particle-mediated cholesterol exchange between lipid particles and cell membranes.

## S3A-C6 Raft-facilitated signal transduction in the plasma membrane

Akihiro Kusumi

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About 10-20% of the receptor molecules are present in the extracellular domain, only anchored in the outer leaflet of the plasma membrane by way of a phospholipid, called glycosylphosphatidylinositol (GPI). Previous studies pointed to the involvement of raft microdomains for the signal transduction of GPI-anchored receptors (GPI-ARs), without any detail. Our single-molecule tracking results suggested that the rafts in the absence of the external stimulus may consists of only several molecules, and that they can be stabilized in the signal-induced clusters of GPI-ARs, upon the engagement of these receptor molecules. Single-molecule tracking further revealed that a cluster raft entices intracellular raft-associating signaling molecules Gi and Lyn, due to both protein-protein and raft-based interactions, where Lyn is activated by Gi. Lyn activation leads to the temporary binding of the cluster raft to actin filaments, leading to STALL of the cluster raft. Exclusively during this STALLing period, PLC is transiently recruited to the cluster raft, generating IP3, leading to calcium mobilization.

## S3A-D1* Production and analyses of mutant DISC1 transgenic mice: an animal model of schizophrenia

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*Disrupted-in-schizophrenia-1 (DISC1)* is a strong susceptibility gene for schizophrenia. To evaluate the function of DISC1 *in vivo*, especially its possible role in the pathophysiology of schizophrenia, we generated transgenic mice with forebrain-restricted expression of truncated human DISC1 that may have dominant-negative effects. We found that expression of mutant DISC1 was associated with deficit in prepulse inhibition and mild hyperactivity, and with enlargement of the lateral ventricles as measured with MRI. Our results suggest that the DISC1 transgenic mice can serve as a novel rodent model for schizophrenia.

NIH, Stanley, S-R, NARSAD

## S3A-D2 Molecular pathogenesis of schizophrenia: genes, or pathways?

Kazuo Yamada

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The identification of genes in schizophrenia pathogenesis is a complex task. The inheritance of a single major gene does not serve as a vulnerability determinant. Instead, the accumulation of heterogeneous, multiple genetic abnormalities, each with a small effect, contribute to an increased risk of disease in carriers. We have tested for genetic association between a subset of calcineurin-related candidate genes and schizophrenia. The calcineurin signaling cascade plays a critical role in the regulation of a diverse array of neuronal functions relevant to the pathogenesis of schizophrenia. We obtained genetic and biological evidence that PPP3CC and EGR3, both constituents of the calcineurin signaling pathway, may independently confer a modestly increased risk for schizophrenia. These genes represent promising but not yet compelling candidates. However, they do provide useful clues to unraveling the complicated pathophysiology of schizophrenia. Hopefully, further exploration of calcineurin-related signal transduction will enable rapid progress in our understanding of this illness and the development of new therapies.

## S3A-D3 Molecular basis of bipolar disorder

Tadafumi Kato

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Role of genetic factors, neuroprotective effects of mood stabilizers, increased incidence of subcortical hyperintensity, and elevated calcium levels in peripheral blood cells, collectively suggest that neurons of patients with bipolar disorder have some vulnerability. Altered energy metabolism in the brains and comorbidity with hereditary chronic progressive external ophthalmoplegia (CPEO) caused by deletions of mitochondrial DNA (mtDNA) suggested that bipolar disorder is related to mitochondrial dysfunction. We generated mutant POLG (polymerase gamma) transgenic mice carrying neuron specific mtDNA deletions. The mice showed periodic acitivity change and altered ciurcadian rhythm, which were improved by lithium. The mitochondria isolated from the brains of the mice showed enhanced calcium uptake rate, which may be caused by decreased level of cyclophilin D. Gene expression analysis of monozygotic twins discordant for bipolar disorder showed altered endoplasmic reticulum stress pathway. These findings altogether suggested that bipolar disorder is associated with genetic vulnerability of the neurons to cellular stress.

## S3A-D4 Glucocorticoid and brain-derived neurotrophic factor in mood disorders

Hiroshi Kunugi, Tadahiro Numakawa

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Hypothalamic-pituitary-adrenal (HPA) axis plays a key role in stress response and in the pathogenesis of depression. We have shown that the combined dexamethasone (DEX) and CRH challenge test is a sensitive state-dependent marker to assess HPA axis abnormalities. Elevated glucocorticoid levels may give rise to neuronal damage and decreased neurogenesis. BDNF is another key molecule in the pathogenesis of depression and in the mechanism of action of antidepressants. Our molecular genetic studies have suggested that polymorphisms of the BDNF gene and its low affinity receptor p75 are associated with the development of mood disorders. We therefore examined interface between glucocorticoid and BDNF. Experiments using primary cultured neurons suggested that glucocorticoid has substantial effects on functions of BDNF, due at least in part to suppressing phosphorylation of PLC-$\beta$. Moreover, glucocorticoid may affect intracellular expression of p75. These results suggest that normalization of HPA axis is required for BDNF to exert its effects in the recovery process from depression.

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## S3A-D5 Molecular imaging of mental disorders

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An increasing body of evidence favors a crucial role of extrastriatal regions in the pathophysiology of positive symptoms of schizophrenia, and the extrastriatal D2 receptor is expected to be the common site of action of antipsychotics. Using PET with high affinity ligand C-11 FLB457, we found reduced D2 receptor binding in the anterior cingulate cortex of patients with schizophrenia, and a significant negative correlation was observed between D2 receptor binding and the positive symptom score. Subregions of interest were defined on the thalamus using individual magnetic resonance images. D2 receptor binding was also lower in the central medial and posterior subregions of the thalamus in patients with schizophrenia. Alterations in D2 receptor function in the extrastriatal region may underlie the positive symptoms of schizophrenia. On the other hand D1 receptor binding was found to be lower in the prefrontal cortex and a significant negative correlation was observed between D1 receptor binding and the negative symptom score. Abnormality of D1 receptor function would be at the bottom of the negative symptoms and cognitive impairment of schizophrenia.

## S3A-E1 Arc is required for the late phase of cerebellar long-term synaptic depression

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The late-phase of cerebellar long-term depression (LTD) requires postsynaptic protein synthesis. However, the specific newly synthesized proteins involved and their function is currently unknown. We have shown that the immediate early gene Arc is essential for the maintenance of cerebellar LTD, as Arc knock out (KO) Purkinje neurons exhibit attenuated late-phase LTD. Constitutive Arc over-expression depresses synaptic transmission, occluding further LTD and is thereby unable to rescue the Arc KO phenotype. However, expression of a Bacterial Artificial Chromosome (BAC) construct containing Arc and its genomic regulatory regions fully rescues late-phase LTD in Arc KO neurons.

NIH RO1 MH51106 and P50 MH068830

## S3A-E2 Decoding Purkinje cell simple spike trains: Patterns and pauses

Erik De Schutter
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I will present combined modelling and experimental studies of the temporal structure of Purkinje cell (PC) simple spike (SS) trains. Deep cerebellar nuclei (DCN) neurons are known to produce strong rebound (RB) spikes upon release from inhibition. Such release requires a synchronized pause in firing of afferent PCs, indeed 13% of pauses are synchronized among neighbouring PCs. Therefore the pauses may form a temporal code in SS trains. If evoking RB spikes is an important coding principle, PCs must be able to regulate them. SS firing contains long stretches of regular firing, mostly at faster rates. These regular patterns form a perfect rate code, where firing rate translates into steady state inhibition of DCN neurons. This rate code may control the amplitude of RB spikes evoked shortly afterwards. Another way to regulate RBs is by changing their duration. The proportion of pauses in SS trains is highly variable between PCs, which may be linked to an underlying variable excitability. Another cause may be synaptic plasticity. Indeed, induction of long-term depression of the parallel fibre synapse shortens the SS pause.

FWO, IUAP, UA, EC and HFSPO

## S3A-E3 Cerebellar somatosensory mossy fibre input - Synaptic integration and synaptic plasticity.

Carl-Fredrik Ekerot, Henrik Jorntell
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The present study has addressed two important issues for cerebellar function, the processing of mossy fibre (MF) information by granule cells and the role of synaptic plasticity in parallel fibre (PF) synapses. The experiments were performed on unanesthetized decerebrate cats. Whole cell recordings from granule cells (GCs) showed that mossy fibres converging on a GC were activated from the same modality/submodality and had similar peripheral receptive fields (RFs). These observations suggest that GCs are error correcting elements that filter out uncorrelated activity (i.e. spontaneous MF activity). Climbing fibre(CF) evoked LTD of PF synapses on Purkinje cells (PCs) was long considered to be responsible for cerebellar learning. However, experiments with burst stimulation of PFs showed that PF input to PCs and interneurones was controlled by bidirectional reciprocal plasticity. Stimulation of PFs alone resulted in LTP of their synapses on Purkinje cells and LTD of synapses on interneurones whereas conjunctive stimulation of CFs and PFs had the opposite effect.

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## S3A-E4 Two-stage learning in eyeblink conditioning in mice

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Delay eyeblink conditioning is one of the most extensively characterized paradigms for motor learning and memory. Cumulative evidence indicates an essential role for the cerebellum in this paradigm. The site and mechanisms underlying memory formation, however, are under debate. The anterior interpositus nucleus (AIN) is a proposed site for the association memory. To unravel the mechanisms underlying memory formation, we systematically examined transcripts that change during conditioning in mice. There were two groups of genes with distinct spatial and temporal characteristics. Representative EARLY gene expression peaked on 1 d of training in broad cerebellar areas in both the paired and unpaired conditioning groups. Representative LATE gene expression was selectively increased in the AIN of the 7-d paired group, but not the unpaired group. These data fit well with the two-stage learning theory, which proposes emotional and motor learning phases, and support the role for the AIN as a memory site. Using EARLY genes as molecular probes, we are now investigating the role of emotional learning in eyeblink conditioning.

KAKENHI18022049

## S3A-E5 Cerebellar plasticity at ultrastructural level

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Morphological substrates for the memory formation in vivo have not been well demonstrated. We used short- and long-term adaptation of horizontal optokinetic response (HOKR) to study the link between learning and underlying ultrastructual changes in synapses and synaptic AMPA-type glutamate receptors. We used SDS-digested freeze-fracture replica labeling, which can detect AMPA receptor channels with almost one-to-one detection sensitivity. Almost all PF-PC synapses were labeled for AMPA receptors with a large variability in number and density of particles. In short-term adaptation of HOKR induced with one hour training, we detected a significant reduction of AMPA receptors by 25% at PF-PC synapses in the flocculus. On the contrary, in long-term adaptation induced with the one hour training for five consecutive days, we detected no significant changes in AMPA receptor density but found a significant reduction of synapse density by 33% in the flocculus. These results suggest that memory traces for short-and long-term memory are differently formed in the cerebellar motor learning.

SORST, Japan Science and Technology Corporation

## S3A-F1 Action value in the striatum and reinforcement learning model of cortico-basal ganglia network

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When we face a choice, evaluating the consequences of the choice is important. It has been suggested that the basal ganglia play a crucial role in reward expectation and voluntary movement. But exactly how the basal ganglia contribute to the processing between reward expectation and decision making remains unclear. To address this issue, we recorded monkey striatal projection neurons during a free-choice task in which two monkeys turned a handle to either left or right direction. In the delay period before the choices, magnitude of discharge rate in majority of recorded neurons (53/142) was correlated with the values of one of the two actions, which were estimated by a reinforcement learning (RL) model to fit history of monkey's choices and outcomes. These results supported a RL model of basal ganglia, in which action values are represented in the striatum, compared between possible actions to select one, and updated by expectation errors between the selected action value and obtained reward.

KAKENHI(17022032, 1800383, 18019033)

## S3A-F2 Multiple pathways and algorithms for reinforcement leaning

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Reinforcement learning theory for action learning and choice has been successfully used for modeling animal and human choice behaviors and for analyzing neural recording and brain imaging data. The theory provides a number of alternatives for action learning and choice algorithms, such as actor-critic, Q-learning, and model-based planning, and experimental results in support of each of them have been reported. Also a critical issue in reinforcement learning is setting of the parameters of the algorithms, such as the temporal discounting rate. Thus the next big question is to clarify how the brain as a whole selects the right algorithm and the parameters based on the nature of the task and the experience of the learner. We present a model in which model-based planning is implemented in the prefrontal cortico-basal ganglia loop. We also propose that different parts of the cortico-basal ganglia loops are optimized for different temporal discounting rate, and that they are under differential regulation of the serotonergic system. We will report our brain imaging and neural recording experiments to test these hypotheses.

KAKENHI

## S3A-F3 The anterior cingulate cortex in learning and reward-guided decision making

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Signal changes in the human anterior cingulate cortex (ACC) can be recorded when people make choices and when the outcomes of the choices will be used to guide subsequent behaviour. The size of the ACC signal varies with the degree to which the choice is free and unconstrained and with the importance of the outcome as a determinant for future behaviour regardless of whether or not the outcome is positive or negative. A recent fMRI study suggests that ACC activity changes as the length of the reward history that is needed to determine the next choice changes. Such information is critical for setting the rate at which learning occurs. When lesions are made in the ACC, macaques are as sensitive as controls to a decrement in reinforcement but the influence of the extended reward history on choice is diminished.

MRC, Royal Society

## S3A-F4 Top-down attention to prediction errors of action values in the prefrontal cortex

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When we are not sure of appropriate action in a given situation, we attend to the outcome of tried action to know its appropriateness for the next time. To uncover neural processes underlying such top-down attention in action selection learning, we recorded the neuronal activity in the medial and lateral prefrontal cortex (PFC) in the monkeys performing a task in which learning of action selection was repeated with visual feedback signals. About 15% of neurons in both the medial and lateral PFC gradually increased the firing toward the visual feedback onset in early phases of learning, and the activity disappeared after the appropriate action became certain. More exactly, the probability-weighted average of absolute discrepancy between the predicted value and actual outcome of potential actions gave the best fit of the changes in magnitude of the neuronal activity along learning. In each trial, the activity appeared earlier in the medial PFC than the lateral PFC. These results suggest that the medial PFC is essential in the top-down attention in action selection learning.

## S3A-G1 Kiss of microglia - Is it sweet or bitter-

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The function of microglia is unclear during motor neuron regeneration, while the swift transformation of microglia is amazingly dramatic. In response to motor nerve injury, microglial cells are activated and migrate towards the cell bodies of nerve-injured motor neurons. Those migrated microglial cells are then attached to the motor neuron cell bodies and subsequently wrapped the surfaces. In rat this microglial adhesion is always observed for about two weeks, and phagocytic features are not observed. However a minor population of nerve-injured motor neurons in mouse appears not to be adhered by the microglial cells. This difference between rat and mouse appears to correlate well with the fate difference of nerve-injured motor neurons between mouse and rat. To reveal a possible consequence of the microglial adhesion after nerve injury, we have morphologically examined the interaction between nerve-injured motor neurons and microglial cells during the regeneration and degeneration processes of the mouse motor neurons. The electron microscopic study revealed striking morphological differences between the motor neurons with microglial adhesion and those without.

## S3A-G2 Microglial activation by TLR ligands: usual and unusual suspects

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To sense environmental signals, microglial cells are equipped with an array of receptors. They also express several Toll-like receptors (TLR) and mount diverse responses to their agonists. We focussed on a protein tyrosine kinase activity which is involved in the signalling of various TLR. The PTK assists the MyD88 route and controls the intracellular calcium, thereby affecting activation-associated functions. Moreover, TLR may serve the recognition of a broader variety of signals indicating homeostatic disturbance. We identified endogenous proteins which apparently activate microglia through a TLR4/MyD88-dependent mechanism. LPS, the prototypic microbial ligand of TLR4, can be ruled as a confounding factor. The ability of the ligands to trigger TLR4 signalling seems to depend on critical structural requirements. Interestingly, although the ligands use TLR4 for mandatory signalling, additional (receptor) components appear to fine-tune respective consequences. TLR4 may thus serve responses to different microglia-challenging stimuli but the induced responses distinguish between individual agonists.

Hertie Foundation

## S3A-G3 Molecular mechanisms of ATP-induced microglial chemotaxis

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Microglia rapidly undergo morphological change from a ramified form in response to pathological stimuli and migrate toward the lesioned sites. The microglial responses are important initial steps of amelioration of the damaged CNS. ATP is known to regulate physiological functions of microglia through ionotropic (P2XRs) or metabotropic (P2YRs) purinergic receptors. We have previously shown that ATP induced membrane ruffling and chemotaxis of microglia through P2Y12R. P2Y12R has been reported to be essential for chemotactic responses to local CNS injury in vivo. To understand the mechanisms underlying microglial chemotaxis, we have examined signaling pathways in microglia stimulated with ATP and recently found that P2X4R is also involved in the chemotaxis. We here show that both activation of phosphatidylinositol 3-kinase (PI3K) and phospholipase C pathways are required for the chemotaxis and suggest that intracellular $Ca^{2+}$signal modulates the P2Y12R-mediated PI3K pathways.

KAKENHI(15082213)

## S3A-G4 Microglial modulation of pain signalling

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Recently, accumulating evidences indicate the important role of ATP receptors of activated microglia in the neuropathic pain being often a consequence of nerve injury. The mechanism of this pain is still unclear. We reported that the expression of P2X4 receptor, a subtype of ionotropic ATP receptors is enhanced in spinal microglia after peripheral nerve injury model, and blocking pharmacologically and suppressing molecularly P2X4 receptors produce a reduction of the neuropathic pain (Tsuda et al. Nature 424, 778, 2003). More recently, we have reported that the stimulation of P2X4 evokes the release of brain-derived neurotrophic factor (BDNF) from activated microglia, which causes a depolarizing shift in the anion reversal potential in spinal lamina I neurons in neuropathic pain rats (Coull et al. Nature 438,1017, 2005). This indicates that inhibitory neurotransmitter GABA is able to play an excitatory role in pain sensation.

KAKENHI(18GS0319)

## S3A-H1 Molecular-biophysical correlates in the pathological sensory neuraxis after spinal cord injury

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Injury to the spinal cord produces long lasting pain-related behavioral conditions that are associated with changes in the electrophysiological features of nociceptive neurons within the spinal cord lumbar dorsal horn and thalamus. These include shifts in proportions of cells responding to noxious stimulation, irregularity in spontaneous background activity, increased evoked activity to (formerly) innocuous and noxious stimuli, and increases in afterdischarge activity. Functional plasticity is linked to aberrant expression of Nav1.3, a rapidly-repriming voltage-gated sodium channel. Nav1.3 rapidly recovers from inactivation, a feature that poises it to contribute to the generation of abnormal, high-frequency neuronal firing after injury. I will review recent results showing abnormal expression of the Nav1.3 Na+ channel within second-order spinal cord dorsal horn neurons and third-order thalamic neurons along the pain pathway after SCI, and suggest that this change makes these neurons hyperexcitable so that they act as pain amplifiers and generators.

Christopher Reeve Foundation, NIH

## S3A-H2 Molecular changes in DRG and spinal cord in the model of neuropathic pain

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The neuroplastic changes in DRG and spinal cord induced by peripheral nerve injury are believed to have important roles in the induction and maintenance of neuropathic pain. We have examined the detailed changes in gene expression of pain-related molecules, such as TRPV1, TRPA1, and neurotrophin receptor p75 in DRG neurons after neuropathic injury. We have also reported the alteration of intracellular signal molecule MAPKs, such ERK and p38 and their modulatory effects on gene expression and pain behaviors in the neuropathic pain models. Here, I will talk about changes in another intracellular signal molecule, ERK5, after peripheral nerve injury in spinal cord glial cells and DRG neurons, which may have significant role in the development of neuropathic pain. Second, I will talk about changes in gene expression in spinal glial cells, microglia and astrocyte, after nerve injury. These dynamic changes in glial cells might be involved in altered pain processing in spinal cord in neuropathic pain condition.

Open Research Center grant from MEXT

## S3A-H3 Chronic pain induces the changes in glial function associated with emotional dysfunction

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Two-way communication between neurons and glia is essential for synaptic transmission and information processing and thus is required for normal functioning of the nervous system. Here, we found that chronic pain induced astrocyte activation in the cingulate cortex (CG) with no change in the neural or glial differentiation, and the migration of neural stem cells from the subventricular zone (SVZ). In the behavioral assay, the thermal hyperalgesia observed on the ipsirateral side in nerve-ligated mice was significantly suppressed by a single pre-microinjection of a glial-modulating agent propentofylline into the CG. We also found that the microinjection of either activated astrocyte or astrocyte-conditioned medium into the CG of mice aggravated the expression of anxiety-like behaviors. These results suggest that chronic painful stimuli induce astrocyte activation in the CG. The astrocyte activation in the CG may, at least in part, contribute to the development of a chronic pain-like state and the changes in emotionality in rodents with chronic noxious stimuli.

## S3A-H4 Plastic modification of TRP channel activities in the spinal dorsal horn following peripheral nerve injury

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Nociceptive thermal information is carried by C afferent fibers to the spinal dorsal horn. Application of TRPV1 receptor agonist has been shown to enhance a release of glutamate presynaptically in the spinal cord. Similar to TRPV1, nociceptive cold receptor TRPA1 agonist exhibited an enhancemnt of transmitter release from primary afferents. These releases were further enhanced following peripheral injury,suggesting that not only peripheral but also TRP channel expressed at the central nerve terminal is plastically modified. These changes were examined by using a slice preparation of the spinal cord obtained from peripheral nerve injury model rats and also in vivo patch-clamp recordings were made from superficial and deep dorsal horn neurons as well.

KAKENHI

## S3A-J1 Prefrontal cortex manipulates reasoning process during Koh-Do performances

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One most important human cognitive processes is reasoning. We recorded activities of bilateral prefrontal cortex (PFC) by near-infrared spectroscopy (NIRS) from experts and beginners of "Koh-Do" (Japanese traditional art of incense appreciation), while performing abductive reasoning procedures for an incense discrimination task; that is, to sort complex odour stimuli into abstract categories based upon traditionally acquired common symbolic knowledge regarding various kinds of incense with subtle difference. In experts trained for more than ten years, the right PFC showed a consistent patterned response during discrimination, while the left PFC showed a phasic stimulus-selective response modulated to match the internal subjective state of the reasoning process. In contrast, beginners did not show the organized response pattern found in experts, indicating that their response patterns are disorganized perhaps because lacking knowledge as a reference for decision making based on abductive reasoning.

## S3A-J2 Aesthetics in gustation: insight from recent neuroimaging studies

Ippeita Dan

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Humans use various higher cognitive processes for taste, including memorization, recollection, and imagination, in our dietary lives. Among sensory modalities, the study on gustation has been relatively unexplored mainly because of the technical difficulty in applying neuroimaging techniques to gustation studies. However, gustation researchers have gradually overcome the technical obstacles, and made progress in understanding primary and secondary gustatory processing, and now even higher gustatory processing is within their reach. Here, I will overview recent progresses in neuroimaging studies on primary and secondary gustatory cortices with emphasis on the role of pleasure in gustation processing. Also, I will review higher cognitive gustatory studies with emphasis on our recent studies on gustatory memory encoding using functional Near Infrared Spectroscopy (fNIRS). Based on these studies, I will argue on the nature of gustatory information, and further on the importance of aesthetics in gustatory information processing.

## S3A-J3 Neural correlates of beauty and desire

Hideaki Kawabata

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The search for the source of beauty and desire, of whether it resides in the perceiving subject, has exercised the speculation of philosophers and writers throughout the ages. Both beauty and desire which are directed to the attainment or possession of some objects from which pleasure or satisfaction is expected. We have been investigating the source of beauty and desire for several years, as mechanisms in the brain using a fMRI. Our research is oriented neurobiologically, but is keeping attitude same with previous magnificent philosophers. Kantian question, which are what are the conditions implied by the existence of the phenomenon of beauty and what are the presuppositions that give validity to our esthetic judgments, is especially perspicacious even in attempting by inquiring into whether there are specific neural conditions implied by one or more brain structures. Our special interest is to investigate the general mechanism of beauty and/or desire, which are correlated regardless of stimulus categories, and may reside in only humankind. this talk address the mechanism of beauty and desire from the viewpoints of biology of esthetics and humanity.

## S3A-J4 Imaging beauty and pleasure: A case study of the hypersonic effect

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Neuroscientific approaches to beauty and pleasure are basically challenges against the Cartesian dualism and thus inevitably associated with essential difficulties in practice. As a successful instance of neuroscientific approaches to music, I will introduce the discovery of the "hypersonic effect": the phenomenon that the supra-perceptible high-frequency component of air vibrations above the upper limit of human audible range activates the reward-generating neuronal system in the brain and intensifies beauty and pleasure of the sound. Regarding this phenomenon, there had long been a serious disagreement between artists and researchers, that is, musicians and engineers empirically believed a positive effect of the high-frequency component whereas researchers could not prove its effect through "strictly controlled" psychological experiments. The discovery of the hypersonic effect was brought by sharing scientific ability and artistic sensibility in a single personality. It is noteworthy that such an activity has been intentionally developed in Asian traditional culture.

## S3A-K1 Molecular basis of the avian song system development

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Songbirds have a complex vocal repertoire by vocal learning in the juvenile stage. In the brain, there is a neural network called the song system, constituted by a series of specific nuclei. Based on many anatomical and physiological studies, it is plausible that the song system plays crucial roles for song learning and production. However, molecular basis to establish this system still remain unknown. Recently it has been shown that many secreted or membrane-bounded proteins, specifically expressed in some brain areas, play multiple roles in neural development. In this study, to explore the molecular mechanism to make the song system, we carried out in situ hybridization screening, particularly focused on neural circuit formation genes, in the brain of a songbird, Bengalese finch. We found various molecules are specifically expressed in some song nuclei and their expressions are developmentally regulated. Now we are carrying out functional analysis of these genes by virus overexpression system from the molecular to the behavioral level.

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## S3A-K2 Synaptic plasticity related to song learning and maintenance

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We have begun to characterize specific forms of plasticity in songbird forebrain motor control nucleus RA possibly related to both learning and maintenance of song. In young birds during the song learning period, synapses in RA can express a form of long term depression (LTD). This LTD was induced by pairing slight postsynaptic depolarization with presynaptic stimulation, required activation of postsynaptic NMDA receptors, and was expressed by reduction of transmitter release after activation of presynaptic cannabinoid receptors. This LTD was reduced in bird s over 65 days old with normal learning experience, but remained in such birds whose learning capability was delayed by social isolation. In contrast, another form of plasticity was restricted to birds who had completed their learning. Simultaneous strong stimulation of inputs from LMAN and HVC induced LTP at HVC inputs, that appeared dependent on activation of presynaptic NMDA receptors and subsequent enhancement of transmitter release. These results suggest that different types of plasticity may mediate learning to sing and maintaining stable song as an adult.

RIKEN BSI

## S3A-K3 Dynamic cascades of vocalizing-driven gene regulation

Kazuhiro Wada

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Learned vocalization is a rare trait observed in few species, including in humans and in songbirds. In songbirds, learned singing causes increased expression of several genes, such as the transcription factor ZENK, in vocal nuclei. However, knowledge of co-behaviorally regulated genes is limited. Here, we utilized a high-throughput resource, songbird 18K cDNA microarray, we developed to identify and characterize an initial cascade of vocalizing-driven genes. We found over 35 genes that were regulated by singing. These genes spanned a range of functional categories to include additional transcription factors, repressors, chaperones, structural molecules especially associated with actin complexes, enzymes, membrane proteins, and synaptically released proteins. Every gene had a unique singing-driven pattern, summarized in 4 main categories. Time course analysis revealed at least 4 temporal patterns. These results suggest learned vocalizing activates a dynamic motor-driven cascade of different signal transduction pathways in different vocal nuclei, and that these cascades are further modified in young animals learning how to sing.

Uehara Foundation

## S3A-K4 Neural mechanisms of birdsong memory

Johan J. Bolhuis

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Songbird males learn their songs from an adult male when they are young. The neural representation of song memory is localised outside the song system, notably in the NCM and CMM, regions within the avian equivalent of auditory association cortex. Neuronal activation in the NCM correlated with the number of song elements that a male had learned from its tutor, suggesting that NCM may be (part of) the neural substrate for stored tutor song. Female zebra finches do not sing, but nevertheless they can learn the characteristics of male song, and they develop a perceptual preference for the song of their father over novel song. Females that were re-exposed to the song of their father showed increased neuronal activation in the CMM only, compared to novel song. Thus, the CMM may be (part of) the neural substrate for the representation of the memory of the song of their father song. Lesions to the NCM in zebra finch males impaired recognition of tutor song, but did not affect production of the birds own song. Thus, in both humans and songbirds the cognitive systems of vocal production and auditory recognition memory are subserved by distinct brain regions.

## S3P-A1 Visualization and therapeutic regulation of brain amyloidosis and neuroinflammation in Alzheimer's disease

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Our recent efforts using positron emission tomography (PET) have enabled visualization of amyloid $\beta$ peptide (A$\beta$) plaques in living mouse models of Alzheimer's disease. This system also facilitates preclinical selection of PET imaging agents adequate to specific purposes, including early detection of amyloid, assessment of pathological progression and evaluation of therapeutic effects. Moreover, proof-of-concept studies on anti-amyloid treatments, as exemplified by A$\beta$ immunization, are performable by utilizing PET imaging of activated microglia parallel to in-vivo amyloid quantification. Notably, our observations indicate that excessive microglial activation can be triggered by A$\beta$ immunization and may accelerate tau-induced neurodegeneration, leading to the notion that microglial activity along the course of anti-amyloid treatments should be stringently regulated by longitudinal monitoring. Direct modulation of microglial functions toward enhancement of amyloid-eliminating capacity and minimization of neurotoxicity is also being attempted by us in search of optimal therapeutic approaches.

KAKENHI (18023040)

## S3P-A2 Exploration of plasma biomarkers for Alzheimer's disease in coupling with biochemical and cognitive-psucological approach

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Alzheimer's disease (AD) is distinctive for its latent pathological condition, which is followed by overt disease. This has hampered to establish control population for differential analysis. To avoid latent AD in control, we employed examination of visuospatial working memory using computer assisted touch-panel test (TouchM). TouchM detected early cognitive changes in healthy middle-ages, suggesting this system might be able to exclude AD at a pathological condition. The A$\beta$ dependent neurotoxicity involves perturbation of $Ca^{2+}$ homeostasis of neuronal cells. As an example, elevation of annexin A5 (A5), a calcium chelater, was detected in patient plasma. Comparison of A5 level between patients and controls as assigned by TouchM showed 85 % sensitivity at 1.5 ng/ml as cutoff. Collectively, biochemical approach with evaluation of visuospatial working memory would provide an appropriate control population and fascilitate biomarker discovery for this grave condition.

KAKENHI(99999999)

## S3P-A3 Searching tau aggregation intermediates

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Abnormal accumulation of tau as filamentous structures is a neuropathological hallmark of tauopathies. The mechanism underlying the conversion of tau protein from a soluble state to one of insoluble aggregates remains unclear. To identify intermediates of tau filaments, we tracked structural changes of in vitro tau assembly and brain extract by atomic force microscopy. As results, we showed that tau form granular and fibrillar aggregates. The amount of granular tau aggregates was elevated in brains of Braak stage I cases compared to that of Braak stage 0 cases suggesting that granular tau aggregation precedes PHF formation. Moreover, we demonstrated that in vitro tau assembly involves two distinct dimers that differ in resistance to reduction. Assembly of distinct types of dimer was also observed in brain extracts from JNPL3 mice. Temporal change of tau protein led us to determine the precise status of tau aggregation intermediates as a pathological species. Reducing the level of pathological tau species may be a promising therapy for tauopathies.

Grant-in-Aid for Scientific Research on Priority Areas from MEXT, Japan

## S3P-A4 C. elegans model of Tauopathy

Tomohiro Miyasaka

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Tauopathy, a category of age-dependent neurodegenerative diseases including Alzheimer's disease (AD) and frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17), is featured by progressive cognitive decline, severe neuronal loss, and formation of tau-inclusions. To elucidate the molecular mechanisms and pathways involved in tauopathy, we established C. elegans model of tauopathy. These lines of C. elegans were generated to express wild-type or FTDP-17 mutant tau in the mechanosensory (mec) neurons which govern the gentle touch response and can be readily viewed using specific antibodies. In comparison with wild-type tau transgenic worms, the worms expressing FTDP-17 mutant tau showed much greater impairment in the touch response. At the same time, they accompanied marked neuritic abnormalities of the mec neurons and finally microtubule loss, but still maintained intact nuclei. Tortuous neurites probably representing initial phase of neurodegeneration were not necessarily associated with abnormal tau accumulation. Thus, tau, especially mutant one, induces neuronal dysfunction in the early stage of tauopathy without formation of intracellular tau-aggregates.

## S3P-A5 Structure and function of γ-secretase complex

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Genetic and biological studies provide evidence that the production and deposition of amyloid-β peptides (Aβ) contribute to the etiology of Alzheimer's disease. β- and γ-secretases, that are responsible for the generation of Aβ peptides, are plausible molecular targets for Alzheimer's disease treatment. γ-Secretase is an unusual aspartic protease that cleaves the scissile bond within the transmembrane domain. This unusual enzyme is composed of a high molecular weight membrane protein complex containing presenilin, nicastrin, Aph-1 and Pen-2. Drugs that regulate the production of Aβ by inhibiting or modulating γ-secretase activity could provide a disease-modifying effect on AD, although recent studies suggest that γ-secretase plays important roles in cellular signaling including Notch pathway. I will review our studies on the γ-secretase complex, and envision the direction for developing effective and selective γ-secretase inhibitors as therapeutics for AD.

KAKENHI (17390015)

## S3P-B1 Effects of "invisible" stimuli on plasticity

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Consciously invisible stimulus influences brain activity and behavioral performance (Watanabe, Nanez & Sasaki, *Nature*, 2001; Seitz & Watanabe, *Nature*, 2003). However, it is not clear how subthreshold stimuli are processed in the brain. We found that a task-irrelevant subthreshold coherent motion led to a stronger disturbance in task performance than did suprathreshold motion. With the subthreshold motion, activity in the visual cortex measured by functional magnetic resonance imaging was higher, but activity in the lateral prefrontal cortex was lower, than with suprathreshold motion. These results suggest that subthreshold irrelevant signals are not subject to effective inhibitory control (Tsushima, Sasaki & Watanabe, *Science*, 2006).

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## S3P-B2 What happens when motor prediction is betrayed?- Tugged hand illusion and stopped escalator effect -

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When controlling body movements, the brain would predict sensory consequences by using the 'efference copy' signal with causal (forward) model of the body dynamics. This prediction is automatically and subconsciously processed for sufficiently trained movements. We therefore are only aware of the movement mistakes or external actions, both of which might be derived from the mismatches between the predictions and consequences. Those sensations are usually reliable due to the well-trained internal models, but motor predictions are sometimes betrayed by subconscious process. We will introduce two intriguing examples: one is a misattribution of self-generated arm action to external action called as 'tugged hand illusion', and the other is an odd sensation perceived when riding on the stopped escalator. Experimental data suggest that these different perceptual phenomena can be explained by a common key factor, 'insufficient predictions' processed in the 'conscious' brain which cannot handle the unexpected subconscious sensorimotor processes.

## S3P-B3 Implicit brain responses during insight

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Despite many popular accounts of Aha! and insight, the underlying brain mechanisms are not known. Here we presented the results of two experiments on insight while EEG signals were recorded from human participants. In experiment-1, we chose verbal puzzles which are solved via insight. Greater gamma band (30-70 Hz) power was found in right fronto-central and frontal regions for (a) correct solutions, (b) successful hint utilization, and (c) sudden pop-out solutions. In experiment-2, we chose cRAT problems (verbal creativity test) and investigated the characteristic features of insight - mental impasse, restructuring, deeper understanding and suddenness. Stronger gamma band responses at parieto-occipital regions were found for trials with sudden solutions, whereas alpha band power in right prefrontal region was decreased for trials with restructuring. We conclude that insight is not a single event, rather consisted of multiple, both conscious and unconscious, cognitive events.

JST.ERATO Shimojo Implicit Brain Function Project, OENB-Jubilaumsfondproject Nr. 11497

## S3P-B4 Modulation of reward seeking behavior and the role of dopamine

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*Shimojo Implicit Brain Function Project, JST ERATO, Atsugi, Japan*

Reward seeking behavior, which is essential for our survival, sometimes becomes compulsive and loses its control. Drug addiction is a typical example of such compulsive reward seeking. Using rats as the model subjects, we are attempting to analyze neural mechanisms that regulate unusual, addictive reward seeking. After experiencing several parings of cocaine and a particular environment, e.g., a black box with meshed floor, rats begin to show preference for the environment. We have shown that the dopamine (DA) receptor protein increased in the hippocampus of these rats. Interestingly, DA receptor protein in the nucleus accumbens, which is important for reward seeking, increased in the control animals which received the same amount of cocaine but not paired with the environment. We also have shown that DA receptor in the basolateral amygdala regulates the expression of preference, despite no clear change in the protein level. Obviously, the dopaminergic function of various subcortical brain areas modulates the drug-related addictive reward seeking.

## S3P-B5 Consumer neuroscience: bringing neuroscience to the "real world "

Christian Scheier

*Decode Marketingberatung GmbH, Hamburg, Germany*

Despite billions of dollars spent every year for marketing and researching brands, products and commercials, 70% of product innovations fail every year. Consumers are overloaded with messages and brands. This talk will present a new approach to understand purchasing behaviour and real world decision-making: consumer neuroscience. We first review of recent fMRI findings of cortical and subcortical brain structures involved in processing brands and discuss psychophysical and behavioural measures of implicit cognitive processes relevant for marketing research (e.g. implicit memory and implicit preference measures). We then present empirical case studies of " applied brand psychophysics " where psychophysical and behavioural measures were used to analyze and predict real world learning and purchasing behaviour. The key insight is that most marketing signals are processed implicitely by consumers. Limits of this approach and future research are discussed

ERATO

## S3P-C1 Function and adaptive plasticity of tonic GABA(A) receptor mediated inhibition in the hippocampus

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GABA(A) receptor mediated signalling can be divided into fast synaptic (phasic) transmission and tonic GABA(A) receptor signalling in which extrasynaptic receptors are activated by ambient, extracellular GABA. The functional role of tonic currents is still unclear. We have recently found that tonic GABA(A) receptor currents in pyramidal cells are outwardly rectifying. This has an important effect on the function of tonic inhibition. In contrast to studies in the cerebellum, hippocampal tonic currents alter the offset rather than the gain of pyramidal cell input-output functions. The magnitude of tonic inhibition depends upon the concentration of extracellular GABA and the expression of extrasynaptic receptors. We have demonstrated that tonic currents show adaptive plasticity in epilepsy. This may compensate for increased network excitability at the expense of cognitive function. Thus tonic GABA(A) receptor mediated currents in the hippocampus show adaptive plasticity and play an integral role in regulating neuronal and network excitability.

Medical Research Council UK and Epilepsy Research Foundation

## S3P-C2 Modal shift of the tonic $GABA_A$ receptor-mediated conductance during neocortical development

Atsuo Fukuda

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Tonic inhibition or hyperpolarization by the continuous activation of extrasynaptic $GABA_A$ receptors (-R) has been reported in adult hippocampus and cerebellum. It has also been reported that continuous activation of the $GABA_A$-R contributes to a tonic depolarization in the embryonic cortex with a depolarized $E_{Cl}$. However, details including development are not known. In adult, the tonic inhibition in pyramidal neurons were due to the persistent activation of $\alpha 1$ and $\alpha 5$ $GABA_A$ and $GABA_C$-R subunits by GAT-sensitive spilt-over GABA. Thus, the subunits involved in neocortical tonic GABA conductance are rather unique. In contrast to adult, $GABA_A$-R mediated tonic currents in embryonic cortical plate cells were not GAT-sensitive, which could be recorded even in a deficiency of ambient GABA. Taurine transporter inhibitor induced inward shift of holding currents, which was reversed by a $GABA_A$-R antagonist. The results indicate taurine may be an endogenous co-agonist for embryonic tonic $GABA_A$-R in the neocortex, that is taken over by spilt-over GABA later in life. The physiological significance of tonic conductance would change.

## S3P-C3 Regulation of adult neurogenesis by GABA

Hongjun Song, Shaoyu Ge, Eyleen Goh, Kurt Sailor, Yasuji Kitabatake, Guo-li Ming

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Adult neurogenesis, a process of generating functional neurons from adult neural stem cells, represents a striking form of activity-dependent structural plasticity in the adult brain. Using a retrovirus-based strategy for birth-dating and genetic marking of proliferating neural progenitors and their progeny in vivo, we have characterized the sequential events of adult hippocampal neurogenesis. Interestingly, new neurons are tonically activated by ambient GABA before receiving any synaptic inputs. Tonic GABA activation depolarizes newborn neurons and the amount of activation is regulated activities of local interneurons. Furthermore, newborn neurons lack of GABA-induced depolarization exhibit significant defects in their dendritic development and formation of GABAergic and glutamatergic synaptic inputs in vivo. These studies suggest a novel activity-dependent mechanism for regulation of adult neurogenesis where new neurons, before receiving any synaptic innervations, may sense neuronal network activities through local ambient GABA levels.

NIH

## S3P-C4 Shunting tonic conductance synchronizes neuronal networks

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Gamma oscillations provide a temporal structure for information processing in the brain. They contribute to cognitive functions, and are disturbed in some psychiatric disorders. Networks of GABAergic neurons are key elements in the generation of gamma oscillations. Wang and Buzsaki (1996) have proposed a model of interconnected inhibitory neurons in which cells can synchronize their firing. This model may explain generation of gamma rhythms in the brain. Authors assumed that interneurons receive tonic excitation which can homogenize firing rates. In fact replacing tonic excitation for synaptic excitatory input in the model significantly reduced the ability of the network to synchronize. We introduced inhibitory tonic current to the interneurons receiving synaptic excitation. This tonic current significantly increased the synchronization of the network in magnitude and reverse potential dependant manner. We speculate that tonic current mediated by extrasynaptic $GABA_A$ receptors in hippocampal interneurons is essential for regulation of gamma rhythms in the brain.

## S3P-D1 Phosphoinositide-sensitive LL5 $\beta$ enhances lamellipodium formation through Filamin A, inhibits FILIP activity and is essential for radial migration

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Filamin A is critical for controlling migrating cell behavior as well as migration start of radial migration in the developing neocortex. FILIP negatively regulates radial migration by enhancing the degradation of Filamin A. Here, we report that LL5 $\beta$, whose subcellular localization is directed by membrane phosphatidylinositol 3,4,5-triphosphates, binds to Filamin A and enhances lamellipodium formation and cell motility in COS-7 cells. In the developing neocortex, almost all radially migrating LL5 $\beta$-knocked down cells, as well as cells with LL5 $\beta$-lacking its pleckstrin homology domain, are arrested in the intermediate zone 3-4 days after transfection. Filamin decreases in LL5 $\beta$-deficient cells and the migration arrest of these cells is reversed by restoration of Filamin A. Furthermore, over-expressed LL5 $\beta$ abrogates FILIP activity. Therefore, LL5 $\beta$ is a significant regulator of radial migration through Filamin A, in a manner dependent on membrane phosphoinositides.

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## S3P-D2 Regulation of neuronal migration and layer formation in the developing cerebral cortex

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Mammalian cerebral cortex has a six-layered structure, where the neurons are aligned depending on their birth-date. Since the migrating neurons invariably stop migrating just beneath the marginal zone (MZ), the cortical plate (CP) is formed in an inside-out manner. These well-organized developmental events are orchestrated by Reelin, which is secreted by Cajal-Retzius neurons in the MZ. Using reaggregation culture system of dissociated migrating neurons, we found that the cells acquired a birth-date-dependent segregation property (or the fate to acquire this property) in a Reelin-independent manner before their somas reach beneath the MZ. Ectopic overexpression of Reelin on the way of migration revealed that Reelin affected the cell aggregation patterns of the migrating neurons *in vivo*. These results suggest that the appropriate layer formation in the cortex might be accomplished through the interplay between the cell-intrinsic mechanism such as segregation properties and the extracellular signals including Reelin.

KAKENHI

## S3P-D3 CRMP family proteins that regulate axon guidance, synapse maturation and cell migration

Yoshio Goshima

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In the developing nervous system, post mitotic neurons migrate and extend their neurites and form precise patterns of connections that emerge through the interaction between the growth cone and attractive or repulsive guidance molecules. Semaphorin3A (*sema3A*$^{-/-}$) is the prototyptical repulsive axon guidance molecule that potently induces growth cone collapse stalling neurite extention. Collapsin response mediator protein (CRMP2) was identified as a signaling molecule of *sema3A*$^{-/-}$. CRMPs are now known to be composed of five homologous cytosolic proteins CRMP1-5. By analyzing *crmp1*-deficient mouse phenotypes, we obtained several evidence for the involvement of *crmp1*$^{-/-}$ in *Reelin*$^{rl/rl}$ signaling. I'll discuss with some molecular links between cell migration, axon guidance and synapse maturation.

## S3P-D4 Nkx2.1 specifies cortical interneuron fate by directly activating Lhx6

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In the mammalian forebrain progress in uncovering the genetic basis of cell fate determination has been hindered by the complexity of cell types and by the protracted maturation of forebrain neurons. Expression of Nkx2.1 in the medial ganglionic eminence is required for the specification of two major subgroups of cortical interneurons, those that express parvalbumin (PV) or somatostatin (SST), but cortical interneuron fate determinants downstream of Nkx2.1 remain to be uncovered. We find that electroporation of Nkx2.1 cDNA into the ventral telencephalon of slice cultures from Nkx2.1-/- embryos, followed by transplantation to permit postnatal analysis of their fate, rescues the loss of PV and SST-expressing cells. This paradigm also rescues expression of Lhx6, which appears to be a direct target of NKX2.1. Lhx6 induction is required for the rescue of PV and SST expression, and is itself sufficient to rescue these phenotypes in cells transplanted from Nkx2.1-/- slices. The slice transfection/transplantation methods employed here are beginning to uncover embryonic mechanisms for specifying neuronal fates that mature postnatally.

## S3P-E1 Autism: Clinical medicine to neuroscience

Masumi Inagaki, Makiko Kaga

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Autism spectrum disorder (ASD) is characterized by deficits in social and communicative skills, such as imitation, pragmatic language, theory of mind and empathy. We investigated topographical mapping of N400 event related potential (ERP) with a semantic category decision task in control children and ASD. In the task, the first word was always one of the two category names (living things and non living things) and the second word was a member of the 2 categories. Topographical mapping of ASD was different from that of control. It was characteristic that ASD patients showed more congruent errors than incongruent ones. We also studied event related gamma activity (>30 Hz of frequency band) of the paradigm in ASD using multiple band frequency analysis (MBFA). Age matched control children showed increment of gamma spectral power in vertex area. However there was little change of the gamma power of semantic decision task in ASD group. These results implicate the importance of high-frequency EEG oscillations with respect to the differential diagnosis of developmental disorder.

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## S3P-E2 Neuroimaging in autism spectrum disorders

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Autism is diagnosed on the basis of behavioral and developmental features such as impairment of reciprocal social interaction, communication and imagination, and the presence of repetitive and ritualistic behavior. It has been recently pointed out that scientific merits from studies using a comprehensive model assuming a single explanation for autism may be limited, since various behavioral features of autism are largely independent in terms of genetic background as shown by twin cohort studies. Thus, a bottom-up type approach which explores abnormal or preserved functions in relatively fundamental cognitive processes based on simple cognitive models may be still important in the autism research strategy. In this presentation, we will report neurophysiological and neuroimaging investigations into brain regions subserving executive function, attention, and social cognition in autism spectrum disorders, using near-infrared spectroscopy (NIRS), event-related potentials (ERPs), and magnetoencephalography (MEG), and magnetic resonance imaging (MRI).

H17-kokoro-ippan-004, Ministry of Health, Labour and Welfare

## S3P-E3 Clinical syndromes as models of autism

Toshiaki Hashimoto

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Autism is a cognitive and behavioral developmental disorder in three domains as following: social relatedness, communication and imagination. Cause of autism is unknown, but genetic origin is mostly assumed. Sometimes in addition to autistic syndromes, another known medical syndrome is complicated, so-called double syndromes. Case like this becomes a model of autism. Kluver-Bucy syndrome is one of famous model of autism. In this syndrome, in which medial temporal lobe structures are bilaterally destroyed, there is severe disorder of recognizing the emotional significance of objects, stereotyped behavior and social relationship. In tuberous sclerosis many tubers develop in various brain regions. In case with autism a lot of tubers is in the temporal lobe compared with cases without autism. West syndrome is a hardly treatable age dependent epilepsy. When compared between West syndrome with autism and without autism, amygdala volume was smaller in the former than the later. In this symposium the author will describe about clinical syndromes as models of autism and mechanism of development of autistic symptoms.

## S3P-E4 Clinical medicine to molecular biology

Eiji Nanba

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Autism is considered to be strongly genetically influenced by the family and twin studies. The inheritance is complex and epigenetic mechanism will be important role. Although the huge genetic studies have been reported, no contributing gene has yet been identified definitely. I here reported initially the molecular studies for the related single gene disorders with fragile X syndrome, Rett syndrome and Tuberous sclerosis. The Secondly, the unique polyhistidine variations of HOXA1 gene were found both in normal and autism. The ectopic expression of expanded variants led to enhanced intranuclear aggregation and accelerated cell death. Moreover, the deleted and expanded variants inhibited neurite outgrowth in retinoic acid-induced neuronal differentiation. Finally I analyzed more than 50 genes localized on chromosome 2, 7 and 15 autism susceptibility loci by bisulfite direct sequencing for elucidate the epigenetic mechanism.

## S3P-E5 Experimental animals as models of autism

Masaaki Narita
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The difficulties in creating an animal model of autism lie in reproduction of heterogeneic social and communicative defects of human autism in animals. We have recently established an autism model rat by exposing embryonic day (E) 9 rat embryo to either thalidomide (THAL) or valproic acid (VPA). THAL or VPA was orally administered to pregnant female Wister rats, and offspring were evaluated biochemically, morphologically, and behaviorally. (1) Both THAL- and VPA-exposed rats exhibited hyperserotonemia, and significant increase of serotonin concentration in the hippocampus.(2) Both THAL- and VPA-exposed rats commonly showed morphological changes in the dorsal raphe nuclei, the origin of serotonergic neurons, suggesting a migration disorder of serotonergic cells in early developing stage. (3) They showed impairment of the learning task achievement and increased non-exploratory movement in the radial maze test, some of which are relevant to human autism. These results indicate that two potential autism inducing teratogens, THAL and VPA, may disrupt early serotonergic neuronal development in the embryo, and may cause behavioral changes observed in autism.

## S3P-F1 Critical role of electrical coupling in generating population oscillations in neocortex, at frequencies from <1 Hz to >100 Hz

Roger D. Traub
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Neocortex generates oscillations at many different frequencies, whose pattern correlates in vivo with the sleep/wake cycle, with sensory stimulation and cognitive tasks, and with the initiation of epileptic seizures. Many of these oscillations can be replicated in brain slices, from rodents and humans, with a remarkable similarity, at the cellular level, to in vivo oscillations. Detailed network simulations have advanced to the level where cellular oscillation patterns can be replicated and specific experimentally testable predictions offered - in some cases, verified. Most oscillation types in neocortex and hippocampus depend on electrical coupling between pyramidal neurons, and such coupling appears to exist between axons. I shall review morphological data on this coupling, and the phenomenology and mechanisms of gamma (30 - 80 Hz), beta2 (20 - 30 Hz) and very fast (>80 Hz) oscillations; and I shall outline how large-scale modeling of a cortical column has contributed to our understanding.

## S3P-F2 Coding by action potential waveform in the cortex

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Until recently, it was generally assumed that an action potential (AP) in a cortical neuron signals an all-or-nothing event to its postsynaptic cells. However, we recently showed that in regular-spiking pyramidal neurons, fluctuations in input conductance reliably translate into variations of the AP waveform, allowing a high rate of information transfer between synaptic input and AP shape (de Polavieja et al,, 2005, J. Neurosci. 25:5657). Subsequently, it was demonstrated that such modulations of AP shape reach the presynaptic terminals and lead to variations in synaptic transmission (Shu et al., 2006, Nature 441:761, Alle & Geiger, 2006, Science 311:1290). Different shapes of back-propagating APs will also interact differentially with coincident synaptic input. Thus, AP waveform encoding is able to transmit the dynamical state of neurons in the local cortical circuit much faster, and more reliably, than rate or spike-time coding alone.

## S3P-F3 Is electric field a way for neuronal communication? Steep decrease of dendritc membrane resistivity controls neuronal responses to extracellular electric fields

Masashi Inoue[1], Tohru Aonishi[2]
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Nonsynaptic interactions between neurons have been eclipsed by our increasingly detailed understanding of chemical synapses, but they do play significant roles in the nervous system. Field effect interactions are mediated by large extracellular currents and potential fields generated by the hippocampus and other cortical structures. Both endogenous and applied electric fields alter neuronal excitability at field strengths over a few millivolts per millimeter. We investigated the effect of DC extracellular electric fields on neuronal membrane polarization in rat hippocampal slices using patch-clamp and optical recordings. The CA1 pyramidal neurons showed unique responses to applied electric fields. The soma polarized with slow time constant, the polarization was biphasic in the apical dendrites and there was a time-dependent shift in the polarity reversal site. By numerical simulations, we showed that the unique response to electric fields is possibly due to the steep decrease of membrane resistivity at the apical dendrites.

## S3P-F4 Is electric field a way for neuronal communication? Steep decrease of dendritc membrane resistivity controls neuronal responses to extracellular electric fields -Theoretical aspect-

Tohru Aonishi
*Tokyo Inst of Tech*

Nonsynaptic interactions via extracellular electric field provide a new idea for the global interaction of neural populations. Bikson et al. (2004) reported that response of hippocampal CA1 pyramidal neurons to DC extracellular electric field was different between distal and proximal dendrites; a biphasic response appears in distal dendrite whereas a monophasic response appears in proximal dendrite. The unique response suggests that the distal dendrite has dynamical properties to enhance and detect temporal variations of extracellular electric field. To understand the essential factor inducing the biphasic responses, with numerical simulations we tried to reproduce optical imaging data recorded by Bikson et al. It was found that the biphasic and monophasic responses were consistently reproduced in the distal and proximal dendrites, respectively, if we assumed a steep decrease of dendritc membrane resistivity. Therefore, the steep decrease of membrane resistivity possibly contributes to the global neural interaction via extracellular electric fields.

## S3P-F5 An attempt of modeling signal transduction system in neurons: Computer simulation of PKC translocation wave along dendrites of cerebellar Purkinje cells

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Protein kinase C (PKC) plays important role in controlling neuronal excitability. Classical PKC including γ-PKC is translocated from the cytosol to the membrane fractions in response to Gq-coupled metabotropic receptors associated with enzymatic activity. Recently, translocation of γ-PKC in living cerebellar Purkinje cell dendrites have directly observed using a fusion protein of γ-PKC and GFP of transgenic mice. The translocation of γ-PKC is induced by electric stimulation of parallel fibers. This event slowly propagates along dendrites. Calcium imaging studies have shown that the parallel fiber stimulation also indices slowly propagating calcium wave, which could lead the PKC translocation wave, in dendrites at the same velocity. To understand those physiological mechanisms and significance, we constructed a compartmental model of Purkinje cell dendrites. Computer simulations suggest interplay between γ-PKC, calcium channels and potassium channels on the plasma membrane of dendrites.

## S3P-G1 Functional role of GPCRs expressed in astrocytes

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We recently developed a novel strategy to screen GPCRs that are highly or selectively expressed in particular cells of the brain. Since recent literature suggests causative roles of glial cell dysfunction in many neuropsychiatric disorders, we first characterized GPCRs expressed in cultured astrocytes and neural progenitor cells (NPCs) using the method. We found that type 2 neurotensin receptor (Ntsr2) was abundantly expressed in mouse cultured astrocytes and brain. Mice lacking Ntsr2 showed altered emotional behaviors. In NPCs, PACAP receptor (PAC1) was identified as one of the highly expressed GPCRs. We observed that PACAP and PAC1 were co-localized in NPCs of the mouse embryonic cortex and that activation of PAC1 potentiated growth factor-induced proliferation of the glial progenitors. Furthermore, VPAC2, a structurally related GPCR to PAC1, was detected in reactive astrocytes in vivo. These observations suggest potential roles of Ntsr2, PAC1 and VPAC2 in the development, expression and maintenance of the brain function.

## S3P-G2 Regulation of glial functions by protein phosphorylation

Masami Takahashi, Makoto Itakura, Keiichi Yasuda, Tsukiko Sugaya

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Recent studies revealed that glial cells release various bioactive substances by an exocytosis, and these activities play important roles in neuronal survival, brain inflammatory response, and regulation of neuronal networks. Different sets of SNARE proteins play crucial roles in exocytosis in neurons and glial cells. In neurons, neurotransmitter release is positively regulated by protein kinase C (PKC) and the phosphorylation of SNAP-25 at $Ser^{187}$is likely to be involved in this regulation. We studied the PKC-dependent regulatory mechanisms of exocytosis in cultured rat brain astrocyte and in clonal rat glioma C6 cells. Phorbol 12-myristate 13-acetate (PMA), an activator of protein kinase C (PKC), suppressed the $Ca^{2+}$-evoked peptide release from astrocyte and C6 cells, and this suppression was reversed by a PKC inhibitor, bisindolylmaleimide I (BIS). PMA-treatment caused a mobility shift of SNAP-23 in SDS-PAGE, and this change was also abolished by BIS. These results suggested that PKC regulate exocytotic release of transmitters in opposite directions in neurons and glial cells.

KAKENHI(15082206)

## S3P-G3 Disruption of amino acid metabolism in astrocyte and neurological disorders

Yasushi Enokido

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The link between cystathionin $\beta$-synthase (CBS) deficiency and homocystinuria (MIM236200) was first described over 40 years ago, and mental retardation was the first clinical feature of the disease to be classified. Recent studies have shown that abnormal homocysteine metabolism associates with various neurological disorders, such as depression, schizophrenia and Alzheimer's disease. However, very little is known about their pathogenesis and the role of genes associated with homocysteine metabolism in such diseases. In the present study, we showed for the first time that CBS is preferentially expressed in the radial glia/astrocyte lineage in the developing and adult mouse CNS, and found that CBS expression is remarkably up-regulated in some neuropathological condition. Our present results support the idea that a deficiency of CBS causes various neurodevelopmental defects which may result in complex neuropathological features associated with abnormal homocysteine metabolism, and also suggest that radial glia/astrocyte lineage cells might be a new therapeutic target for preventing and treating them.

KAKENHI(16047232)

## S3P-G4 A dual role for reactive astrocytes after spinal cord injury

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In the injured central nervous system, reactive astrocytes form a glial scar and are considered to be detrimental for axonal regeneration, but their function remains elusive. Here we demonstrated that reactive astrocytes play a crucial role in wound healing and functional recovery by using mice with a selective deletion of Stat3 under the control of Nestin gene promoter/enhancer. Reactive astrocytes in Stat3 conditional knockout mice showed limited migration and resulted in markedly widespread infiltration of inflammatory cells, neural disruption and demyelination with severe motor deficits after contusive spinal cord injury. These results suggest that Stat3 is a key regulator of reactive astrocytes in the healing process after SCI, providing a potential target for intervention in the treatment of CNS injury.

## S3P-G5* BMP receptor signaling differentially regulates astrocytic proliferation.

Masahisa Yamada[1], Haruo Kishida[1], Naohito Kitamura[1], Yuji Mishina[2], Runa Araya[1]

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BMP signaling contributes to gliogenesis. Several BMP ligands and BMP type I receptors are expressed in astrocytes. BMP ligands are highly expressed during development but less in the adult brain. When tissues or cells in the CNS incur damage, glial cells proliferate, migrate, and differentiate to generate glial scarring, one of the major impediments to axonal regeneration. Precise roles of BMP signaling in glial sccaring is largely unknown. We show here that depletion of BMPRIA or ALK2 by a small interference RNA inhibits astrocytic proliferate actively. By contrast, we demonstrate that depletion of BMPRIB by a small interference RNA promotes astrocytic proliferation. These findings suggest that the ratio of BMP type I receptors expressed in glial cells may have unique function on control of growth, which associate to initiate their differentiation.

KAKENHI 18053024

## S3P-G6 The two-photon microscopic analysis of cortical spreading depression

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Cortical spreading depression (CSD) is a self-propagating wave of large depolarization that has been implicated in migraine and progressive neuronal injury following stroke and head trauma. Using two-photon microscopy in live mouse cortex, the propagation of CSD was visualized as a progressive wave of transient calcium flux in astrocytes. Tissue swelling was not due to astrocytic, but the neuronal swelling, with severe disfiguration of dendrites. Metabolic state monitored by NADH intrinsic fluorescence revealed that the tissue consists of two distinct areas of decrease and increase of the NADH. This distinctive NADH pattern followed neither the cellular organization nor the cell types, but outlined the vasculature. Thus the perivascular tissue increased the rate of oxidative metabolism, causing the rest of the tissue hypoxic, even with the transient vasodilation. Our results suggest that processes of astrocytes and neurons form a metabolic unit that shares the redox state rather than having independent controls.

NIH NS30007 & NS38073

## S3P-H1 Analysis of pain cognition by neuroimaging study

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Electroencephalography (EEG) and magnetoencephalography (MEG) were recorded following both A-delta (first pain) and C fiber (second pain) stimulation. EEG and MEG findings following C fiber stimulation were similar to those following A-delta fiber stimulation except for a longer latency. At first, primary somatosensory cortex (SI) contralateral to the stimulation is activated and them secondary somatosensory cortex (SII), insula, amygdala and anterior cingulate cortex (ACC) in the bilateral hemispheres are activated sequentially. Event-related functional magnetic resonance imaging (fMRI) was also recorded following A-delta and C fibers stimulation. The stimulation of both C and A-delta nociceptors activated the bilateral thalamus, SII, middle insula, and Brodmann's area (BA) 24/32. However, activity in the right BA32/8/6, including ACC and pre-supplementary motor area (pre-SMA), and the bilateral anterior insula was significantly stronger following the C fiber stimulation than A-delta fiber stimulation. These findings were probably due to the differences in the emotional and motivational aspects of either pain.

## S3P-H2 Analysis of cortical reorganization (CR) of the patients with phantom-limb pain and the mechanism of motor cortex stimulation

Youichi Saitoh, Takufumi Yanagisawa, Satoru Oshino, Masayuki Hirata, Tetsu Goto, Koichi Hosomi, Naoki Tani, Toshiki Yoshimine

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We have treated 12 patients with phantom-limb pain with motor cortex stimulation (MCS). Six of 12 patients showed pain reduction with MCS. The detailed mechanism of pain reduction with MCS has been unknown. Prof. Flor suggests that phantom-limb pain was attributable to the cortical reorganization (CR). We analysed CR in phantom-limb pain with MEG (magnetic encephalography), cortical mapping by navigation-guided TMS (transcranial magnetic stimulation). In the operated patients with cortical electrodes, SEP recording and motor cortical mapping were performed. And EEG was measured during various imaginal movement of phantom-limb. One patient showed remarkable CR with MEG and TMS cortical mapping, and remarkable pain reduction with MCS. Other patient could not imagine the movement of phantom-limb, and showed poor pain reduction with MCS. We would like to discuss the analysis of CR and the mechanism of MCS in the phantom-limb pain.

## S3P-H3 Thalamic sensory relay nucleus stimulation and phantom limb pain

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The primary somatosensory cortex undergoes reorganization of receptive field representation in patients with phantom limb pain (PP). The extent of cortical reorganization and the degree of PP have been reported to be correlated. Similar reorganization has been shown within the thalamic sensory relay nucleus (nuclei ventrocaudales: VC). VC stimulation sometimes dramatically relieves chronic PP, while stimulation rarely alters phantom limb sensation itself in the same patients. This finding suggests that PP may be controlled by VC stimulation through restoration of the original cortical organization in response to artificial activation of non-functioning somatosensory pathway. It has been suggested that conscious sensation occurs only when peripheral sensory stimulus activates wide spread neural networks, including the limbic cortex, beyond the somatosensory pathway. In contrast to patients with intact somatosensory pathways, the patients with PP report various vivid somatosensory and/or somatomotor sensations. This finding indicates that perceptual level of neural networks involved in conscious sensation is heightened in these patients.

## S3P-H4 Cerebral reorganization by adaptable robot hand

Hiroshi Yokoi[1], Ryu Katoh[1], Alejandro Arieta Hernandez[1], Tamaki Miyamoto[2], Katsunori Ikoma[2], Takashi Onishi[3], Wenwei Yu[4], Tamio Arai[1]

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The prosthetic care for the handicapped persons requires new and reliable robotics technology. This study investigates the reactions of our brain to the adaptable robot hand. The adaptable robot hand is structured EMG controlled robot hand with learning function for EMG pattern recognition for Transradial (below elbow) prostheses. The mutual adaptation among man and adaptable robot hand is analyzed by using f-MRI in order to clarify the plasticity of motor and sensory area of cortex according to the change of prosthesis. The robot hand with 13 DOF is developed that has 3 motors on thumb finger and two motors on each rest of 4 fingers, and two motors for wrist. The tactile feedback is applied by using electric stimulus. The f-MRI data shows the illusions and adaptation process of replacement from a phantom limb image to the robot hand image.

KAKENHI (16360118)

## S3P-J1 Impact of brain-behavior phenotypying of genetically-engineered mice on research of neuropsychiatric disorders

Tsuyoshi Miyakawa

*Frontier Technology Center, Kyoto*

Although all the genome sequences have been determined, we do not yet know the functions of most of these genes. To reveal the functions of genes expressed in the brain, and to accelerate the progress of neuroscience, we are proposing to develop a large-scale mouse phenotyping center and/or consortium in Japan (Aiba et al., 2007). In this project, phenotype data would be obtained systematically with reasonably standardized methods, and the data obtained in such projects should be included in a public database. So far, in collaboration with 50 laboratories, we have conducted comprehensive behavioral phenotyping of about 60 strains of genetically-engineered mice. Surprisingly, more than 90 % of those strains of mutant mice showed at least 1 behavioral phenotype, indicating that many of the genes expressed in the brain could have functions that affect behavior. The purpose and the current status of the project will be introduced and discussed, particularly focusing on its significance in understanding human neuropsychiatric disorders.

KAKENHI( 16680015, 18016012, 18023022, 18053015), JST BIRD, NIBIO

## S3P-J2 Systematic generation of conditional knockout mice using C57BL/6 ES cell RENKA

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Generation of conditional targeting mice has greatly contributed to the understanding of brain functions, although problems of their genetic background and time- and cost-consuming work remain to be solved. To make up for the problems in 129 strain mice, for which they are not suitable for behavioral analysis of brain higher functions, we established a new ES cell line RENKA derived from C57BL/6N mouse blastocyst. We then improved the culture condition of RENKA and chimera formation parameters etc. to establish our original protocol, where germline transmission was as efficient as in the 129 strain. We also developed universal vectors and a unique recombination system using BAC clones to shorten the time for targeting vector construction. With the additional efforts made to increase homologous recombination ratio in RENKA leading to reduced screening colony number and the cost, we are entering a new era of systematic generation of the conditional targeting mouse having pure C57BL/6 genetic background.

SORST JST, KAKENHI 17300099

## S3P-J3 Standardized mouse phenotyping platform and integrative database system

Hiroshi Masuya

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Following whole mouse genome sequencing, collaborative programs of knockout (KO) mouse production of every mouse genes, such as KOMP and EuCOMM, have just started in USA and Europe. Beside, production of phenotype data of the expanded mouse mutants by a comprehensive phenotyping platform and standard operation procedures (SOPs) has also launched in Europe.
In RIKEN, we established a comprehensive platform consisting of 14 packages of phenotyping assays, which was developed in a large-scale ENU mutagenesis program. Subsequenlty, we developed a new website, "PhenoSITE" (http://www.gsc.riken.go.jp/Mouse/), which includes databases of terminology, operation procedures and baseline data. Furthermore, now we propse a new concept of format to describe procedures, which provides framework of procedure descriptions and enables direct comparison of parameters of the procedures. This is referred as "Standardized Description of Operating Procedures (SDOP)". It will assist on-line publication and better sharing of not only the standardized procedures but also the local procedures used in multiple laboratories.

National Bioresources Project

## S3P-J4 The genes to cognition programme

Noboru H. Komiyama, Seth_GN Grant

*Wellcome Trust Sanger Institute*

The Genes to Cognition programme (G2C) was established to provide a framework for studying genes, brain and behaviour in order to link basic molecular research from genomes and experimental genetic organisms with human clinical studies of cognition. The G2C programme will collect and integrate data in the areas of psychiatry, human and mouse psychology, cellular neurophysiology, proteomics and biochemistry, molecular biology, human and mouse genetics and genomics. Bioinformatics will be used to create new tools for presenting this diverse data in novel relationships that will facilitate mining of information across existing boundaries in neuroscience. G2C will create, and make publicly available, novel databases for basic and clinical neuroscience; and establish a general model for tackling diseases and brain function. G2C supports a consortium of scientists and offers research, training and collaborative opportunities and aims to become a network for neuroscientists. For more detailed information on G2C is available at the web site: http://www.genes2cognition.org/.

Functional Genomics Development Initiative of the Wellcome Trust

## S3P-J5 Genetic and proteomic approaches to mGluR1-signaling

Atsu Aiba

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We previously generated mGluR1 KO and mGluR1a- and mGluR1b-rescue mice to study the role of mGluR1 in cerebellar Purkinje cells (PCs). Using these mice, we showed that mGluR1 in PCs is required for motor coordination, motor learning, long-term depression and regression of multiple climbing fiber innervation. We recently established mGluR1 conditional KO (cKO) mice by using the tetracycline-controlled gene expression system. The mGluR1 cKO mice express mGluR1 only in PCs and its expression can be turned off with doxycycline administration. We found that suppression of the mGluR1 expression in the adult mGluR1 cKO mice led to impaired motor coordination, indicating that mGluR1 is essential for cerebellar function not only during postnatal development but also in adulthood. To identify mGluR1-signaling molecules responsible for these phenotypes, we analyzed mGluR1-complexes isolated from wild-type, KO, mGluR1a- and mGluR1b-rescue cerebella by mass spectrometry. We found mGluR1 complexes include more than 100 proteins and some proteins interact with specific mGluR1 splicing variants, suggesting divergent signaling of mGluR1 variants in PCs.

KAKENHI(17024038)

## S3P-J6 The organizing principles of real-time memory encoding: Neural clique assemblies and universal neural codes

Joe Z Tsien

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Recent identification of network-level coding units, termed neural cliques, in the hippocampus has allowed real-time patterns of memory traces to be mathematically described, directly visualized, and dynamically deciphered. Those neural clique assemblies are invariantly organized in a hierarchical and categorical manner, forming various *'general-to-specific feature-encoding pyramids'*. The *hierarchical organization of memory coding units* allows not only for encoding nearly infinite amount of episodic events, but also for converting daily experiences into generalized knowledge and concepts in the brain. This general-to-specific encoding mechanism is further supported by the recent discovery of hippocampal cells which appear to be involved in the encoding of abstract concepts from episodic experiences. Moreover, mathematical conversion of activation patterns of neural cliques into binary codes provides a potential framework for universal categorizations of various experiences across individuals and animal species.

Keck Foundation, NIMH, and NIA

## S3P-K1 Central and peripheral theories of emotion ~flow of emotional process~

Etsuro Hori[1,2], Toru Tazumi[2,3], Taketoshi Ono[1,2], Hisao Nishijo[1,2]

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Relationships between emotion and bodily responses have been investigated for many years; central theory by Cannon and Bard (1929) suggests that we feel fear when we see a snake and then run away, while peripheral theory by James and Lange (1922) suggest that we feel fear since we run away from snake. The amygdala has been suggested to be one of the core systems for emotional processing. However, relations between neuronal activity in the amygdala and bodily responses remain unclear. In the present study, to investigate this issue, neuronal activity in the monkey amygdala and pupil radius (autonomic output) were recorded during a delayed non-matching to sample task using facial expression and gaze direction. Our preliminary results based on cross-correlation between neuronal activity and autonomic responses suggest that both theories seem to fit the case.

CREST, JST

## S3P-K2 Dynamical properties of whole-head EEG synchronization: spontaneous and evoked activity

Cees van Leeuwen

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We studied spontaneous human EEG activity recorded at the scalp in the resting state, in the intervals between stimulation, and evoked activity. Resting-state activity is characterized by patterns of synchronization that show considerable variability. Nevertheless, it is possible to detect lawfulness in the way these patterns change over time. We analyze data and present evidence for such lawfulness, and simulate the results by a theoretical model. In-between tasks activity is predictive of intrinsic biases in the way perceptual information is organized. Evoked activity shows patterns of synchronized activity that reflect task demands and perceptual organization. The results support a hypothesis that suggests that the role of spontaneous and evoked synchrony is that of communication between brain areas.

## S3P-K3 A hypothesis of oscillation networks with multiple frequency synchronization for cognitive control

Yoko Yamaguchi[1], Keiichi Kitajo[1], Hiroaki Mizuhara[1,2], Naoyuki Sato[1], Takashi Ozaki[1]

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Brain activities are traditionally characterized by frequencies of oscillations observed by electroencephalogram (EEG) in human and animal. In spite of their complexity, synchronization of oscillations universally ensures a trick for coordination of complex systems. Here we try to propose a fundamental hypothesis on the brain dynamics for cognitive functions by focusing on temporal relations among elementary activities. We have developed simultaneous EEG/fMRI analysis to identify the brain network associated with scalp EEG oscillation index. Furthermore, we analyzed phase synchronization in multiple frequency bands locked to perceptual switching. Our results support the hypothesis that cognitive control is manage by an interlay between the central executive function network emerging by slow oscillation synchronization and modality specific modules emerging by fast oscillation synchronization.

## S3P-K4 Coordination of oscillatory and non-oscillatory modes of brain activity during active cognition in humans

Scott Makeig

*University of California San Diego*

Simply playing a scalp EEG recording through computer speakers (suitably sped up and amplified) is disappointing. Instead of hearing brain rhythms as short tones in a quiet background, one typically hears only colored noise. The terms 'synchronization' and'desynchronization' were used by early neurophysiologists to describe changes in cortical recordings from large-amplitude, low-frequency periodic activity to 'low-voltage fast' noise. Modern spectral analysis of brain electrical activity, however, typically usesfrequency-by-frequency statistics, as if power at each frequency varied independently from power at other frequencies. To test the adequacy of this assumption, Julie Onton and I decomposed log spectrograms of independent EEG source signals, likely generated in spatially compact cortical domains, into independent modulator processes. Two main types emerged: changes in periodic alpha or narrow-band beta activities, and truly broad-band modulations across the source activity spectra. I will discuss interrelationships between time courses of these modulator processes and their relations to subject experience and behavior.

# 抄録 Abstracts

## 一般口演
## Oral Session Abstracts

## O1P-A01 A role for glycogen synthase kinase 3 β and β-catenin in coordinating proliferation and differentiation of telencephalic neural precursor cells

Takeshi Shimizu[1], Tetsushi Kagawa[1], Toshihiro Inoue[1], Shinji Takada[2], Tetsuya Taga[1]

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Proliferation of neural precursor cells and their neuronal differentiation are mutually exclusive, suggesting (an) unidentified molecular link(s) between proliferation and differentiation. Through misexpression studies with β-catenin and dominant-active GSK3 β we recently proposed a model in which nuclear β-catenin concurrently coactivated LEF/TCF transcription factors for inducing proliferation and Notch/RBPJκ transcription factors for inhibiting neuronal differentiation. In this study, we have performed loss-of-function analysis of them. Pharmacological inhibition of GSK3 β activity increased BrdU-positive proliferating cells and decreased Tuj1-positive neurons. Knockdown of β-catenin expression by siRNA reduced FGF2-induced promoter activation of hes1, a major Notch signaling target. Taken together, inactivation of GSK3 β and subsequent accumulation of β-catenin manage two distinct transcriptional machineries involved in proliferation and differentiation of neural precursor cells.

KAKENHI18700365

## O1P-A02 Properties of mammalian neural progenitor cells as revealed by genome-wide single cell gene expression profiles

Ayano Kawaguchi[1], Tomoko Ikawa[1], Takeya Kasukawa[2], Hiroki Ueda[2], Kazuki Kurimoto[3], Mitinori Saitou[3], Fumio Matsuzaki[1,4]

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Information at single cell resolutions is important to elucidate molecular mechanisms underlying neurogenesis. We generated 102 single cell cDNAs from mouse brain at E14, and applied 70 of them for DNA microarrays (GeneChip) to make the global gene expression profiles. The obtained data were then subjected to the clustering analysis, allowing us to categorize neural progenitor cells into 3 distinct populations. We also examined the spatial expression pattern of the genes that characterize each population by *in situ* hybridization. Combining these data, we were able to identify the progenitor-type specific "markers" and relationships among progenitor types. Further analyses of the "cell to cell" variations in gene expression within a cell type lead us to new insight into the nature of the progenitor cells.

KAKENHI (18680030)

## O1P-A03 Nuclear translocation of histone deacetylase SIRT1 affects neuronal differentiation

Shin Hisahara[1,2], Susumu Chiba[2], Hiroyuki Matsumoto[2], Makoto Sato[3], Shun Shimohma[2], Yoshiyuki Horio[1]

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In neurosphere assay, SIRT1 is prominently expressed in the cytoplasm of neural stem cells (NSCs). We found that transfected SIRT1-EGFP showed that nuclear translocation of SIRT1 occurred after the transfer of NSCs into differentiation medium with time-lapse imaging. Overexpression of *SIRT1-siRNA* and *dominant negative SIRT1* reduced Tuj1$^+$ cells in neurospheres upon differentiation. Additionally, the overexpression of SIRT1mtNLS, which stays in the cytoplasm, did not increase the number of neuron-containing spheres after differentiation. *In utero* electroporation showed that *SIRT1-siRNA* affected neuronal differentiation. SIRT1 and N-CoR, a transcriptional repressor interacting with SIRT1, synergistically induced neuronal differentiation and inhibited Hes1 transcription. These results suggest that nuclear translocation of SIRT1 plays a pivotal role in the differentiation of NSCs.

## O1P-A04 Tailless maintains neural stem cell renewal by activating cell-cycle genes and repressing Prospero in the *Drosophila* brain

Mitsuhiko Kurusu[1], Yasushi Maruyama[2], Masataka Okabe[3], Emiko Suzuki[1], Katsuo Furukubo-Tokunaga[2]

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Regulated proliferation of neural stem cells is crucial for the generation of the diverse yet precise number of post-mitotic cells in the brain. However, the underlying mechanisms that determine the temporal window and thus the number of times each stem cells divide are poorly understood. Here we show that Tailless, a conserved orphan nuclear receptor, has a crucial function in the proliferation of the *Drosophila* mushroom body neuroblasts and ganglion mother cells through activation of cell cycle regulators. Intriguingly, in these progenitor cells, Tailless represses Prospero, which arrests cell division to promote terminal differentiation. We propose an evolutionary conserved choice mechanism of neural stem cell proliferation and differentiation, in which Tailless/Tlx and Prospero/Prox counteract to determine the precise temporal window of progenitor proliferation in both the *Drosophila* and vertebrate brains.

## O1P-A05 *In vivo* and *in vitro* analysis of Musashi family in mammalian CNS and PNS development and regeneration

Shinsuke Shibata[1], Shin-ichi Sakakibara[2], Hirotaka J. Okano[1], Hideyuki Okano[1,3]

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Musashi(Msi) is an evolutionarily conserved RNA-binding protein. *Drosophila*-Msi regulates asymmetric cell division in neural development. In mammalian CNS, Msi1 and Msi2, are co-expressed in neural precursor cells including neural stem cells, suggesting Msi family proteins regulate the immaturity of CNS stem cells. To reveal the roles of Msi *in vivo*, we generated Msi1 and Msi2 deficient mice. Msi1 -/- mice frequently died of obstructive hydrocephalus with aberrant cell proliferation of cerebral aqueduct. Msi2 -/- mice survive to adult show poor weight gain, with motor and sensory dysfunction with the malformation of PNS. Msi1 -/- Msi2 -/- mice die in a few hours after birth with severe defects in the brain, including malformation of hippocampal dentate gyrus. *In vitro* and *in vivo* analysis show Msi proteins control the expression of several downstream target molecules, and play important roles cooperatively in mammalian CNS and PNS development and regeneration.

KAKENHI18800047

## O1P-A06 Differentiated horizontal interneurons can proliferate after synaptogenesis

Itsuki Ajioka[1], Rodrigo Martins[1], Ildar Bayazitov[1], Dianna Johnson[2], Stacy Donovan[1], Stanislav Zakharenko[1], Michael Dyer[1,2]

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During neurogenesis, the progression from a progenitor cell to a differentiated neuron is believed to be unidirectional and irreversible. Last year, we reported that p107-single (*Chx10-Cre;Rb$^{Lox/Lox}$;p107$^{+/-}$;p130$^{-/-}$*) horizontal cells (HC) re-entered the cell cycle after arriving at their final position, clonally expanded and formed metastatic retinoblastoma. In this study, to address the morphological feature of the proliferating HC, we performed electron microscopic (EM) and 2-photon time-lapse analysis.

EM and time-lapse analysis demonstrated that both the proliferating HC and the retinoblastoma cells maintain their morphological features including neuronal processes and synapses.

This is the first demonstration that differentiated neurons can proliferate and form cancer while maintaining their differentiated state.

PEW 180144010

## O1P-A07 Exploring the glial progenitor cells in the intact adult spinal cord

Masaaki Kitada, Yutaka Itokazu, Chizuka Ide, Mari Dezawa

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Progenitor or stem cell populations have been reported in the central nervous system. In the adult spinal cord, the existence of oligodendrocyte progenitor cells was reported in 1989 but no other type of progenitor cells has been yet identified. Here, we show the direct evidence that the ependymal cell is the second type of progenitor cells giving rise to glial cells in the adult spinal cord. BrdU labeling presented that the proliferated ependymal cells gradually moved to the outer layer of the bilayer of the ependymal cells. Specific labeling of the ependymal cells showed that the ependymal cells migrated out to the parenchyma to differentiate into astrocytes in the intact adult spinal cord. In addition, the ependymal cells were collected by FACS with the ependymal cell-specific antibody and cultured. After induction of the differentiation, GFAP-positive cells were given rise to. These findings clearly showed that the ependymal cells play a role as astrocyte progenitor cells in the intact adult spinal cord.

## O1P-A08 Paradoxial neuron differentiation in the hindbrain: Olig2+ cells also differentiate into branchiomotor/visceromotor neurons.

Katsuhiko Ono[1], Kazuyo Ikeda[2], Hirohide Takebayashi[1], Keisuke Watanabe[1], Kazuhiro Ikenaka[1]

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Neural cells in the ventricular zone of embryonic neural tube differentiate into specific cell types depending on their position within the dorsoventral and anterior-posterior axes. It has been reported that, in the caudal hindbrain, cells in the most ventral Nkx2.2 domain differentiate into branchiomotor and visceromotor neurons (BMN/VMN) as well as serotonergic (5HT) neurons, whereas those in the Olig2 domain differentiate into somatomotor neurons (SMN). Here, we examined in vivo differentiation property of Olig2+ cells in this region using Olig2-CreER knock-in mice. After tamoxifen injection at E9.5, recombined Olig2 lineage cells give rise not only to SMN in the hypoglossal nucleus, but also to BMN/VMN in the dorsal motor nucleus of vagus and 5HT neurons. Olig2 deficiency results in total loss of SMN and a partial loss of BMN/VMN. These results clearly indicate that Olig2 plays an important role not only in SMN development, but also in that of BMN/VMN.

KAKENHI(18300110)

## O1P-B01 REM sleep enhancement in CNS-specific CRH-overexpressing mice

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Corticotropin-releasing hormone (CRH), known as the initial modulator that activates the hypothalamic-pituitary-adrenal axis, affects sleep. However, its mechanism is not fully defined because of a difficulty to distinguish its effects from those of other stress hormones on sleep. Recently, we generated CNS-specific CRH overexpressing (CRH-OE*Nes*) mice. Using this conditional mutant, a solo effect of central CRH on sleep-wake regulation was studied. CRH-OE*Nes* mice were subjected to EEG/EMG recordings, and sleep patterns were compared among 3 genotypes. During baseline, homozygous (hom) and heterozygous (het) CRH-OE*Nes* mice showed suppressed nonREM sleep in the light period, whereas CRH-OE$^{hom}$ mice markedly increased REM sleep throughout the day. After 6-h sleep deprivation, elevated levels of REM sleep became more evident in both CRH-OE$^{het\&hom}$ mice, which was reversed with a CRH receptor type 1 antagonist. Plasma levels of corticosterone were not different across genotypes in all conditions. Thus, constantly elevated brain CRH is likely responsible for upregulated REM sleep, as seen with depression.

## O1P-B02 Cell cycle in proliferating embryonic stem cell in relation to circadian rhythm

Yoshiaki Isobe[1], Hiroyuki Tsuda[2]

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Self-oscillating systems are running in vertebrate organ that are circadian rhythm and cell cycle. Circadian rhythms are established by transcriptional feedback loops. The cell cycle machinery is integrated by external signals with basic oscillator. How the circadian rhythm signal transmitted to each stage of cell cycle is interesting to investigate. We obtained proliferating neurosphere from the dispersed cell culture of preoptic neuroepitherium in E14 rat fetus brain. After the second passage, cells were divided to four wells. The cells were collected 6 hr intervals at 9:00, 15:00, 21:00 and 03:00. Each sample was analyzed by FACS and measured clock related gene products mRNAs. Proportion of $G_1$ cells at 9:00 was smaller than that at 21:00. Per2 and p27 mRNAs showed similar circadian rhythms, which peaked at 21:00. Rev-erb $\alpha$ mRNA rhythm peaked at 3:00. These results were compared with the data obtained from the cells derived in pallietal neuroepitherium of fetus brain.

## O1P-B03 Synchronization of theta activity in hippocampus and amygdala during REM sleep

Akihiro Karashima, Mio Hayashi, Motohiko Shimazaki, Norihiro Katayama, Mitsuyuki Nakao

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Theta wave in the hippocampus and amygdala and pontine phasic potential (P wave) which is known to reflect the activity of cholinergic neurons in the pons was recorded during REM sleep. Three-way correlations between hippocampal and amygdaloid theta wave dynamics and the densities of P wave were analyzed. The analyses showed that i) the hippocampal theta wave was recorded throughout REM sleep, but amygdaloid theta wave appeared intermittently, ii) the frequency of theta wave in the two regions increased associated with increase in the P wave density, iii) theta synchronization between the two areas increased with the P wave density. Because cholinergic neurons in the pons send projections to the amygdala and hippocampus, the results suggest that the cholinergic neurons accelerate theta activity and increase the theta synchronization. Theta synchronization during waking was considered to be functionally related to the memory retrieval. Therefore, the synchronization during REM sleep may be related to memory function.

KAKENHI 17605001

## O1P-B04 A temporal sequence of changes in firing among the wake- and sleep-promoting neurons at the transition between sleep and wakefulness in mice

Kazumi Takahashi[1], Yoshimasa Koyama[2], Yukihiko Kayama[1], Jian-Sheng Lin[3], Kazuya Sakai[3]

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Cholinergic (ACh), noradrenargic (NA) and histaminergic (HA) neurons in the brain play important roles in generation of wakefulness (W). The preoptic neuron that shows a higher discharge rate during sleep (S-neuron) is considered to be GABArgic and sleep-promoting one. We recorded single unit activity from these neuronal groups, and compared the time of the onset and termination of firing at the transitions across sleep-wake cycles. At the transition from slow-wave sleep (SWS) to W, S-neuron stopped firing and ACh and NA activity elevated before EEG desynchronization, while HA neuron started to discharge after the EEG change. At the transition from W to SWS, cessation of HA neuron and discharge of S-neuron were observed before EEG synchronization, while ACh and NA activity reduced after the EEG change. These results suggest that ACh and NA activity may underlie HA activity during W.

KAKENHI(18603006)

## O1P-B05 Modulation of spinal unit responses evoked from muscle afferents in a monkey performing an instructed delay task.

kazuhiko seki[1,2], Tomohiko Takei[1,3]

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To elucidate the function of muscle afferent input for generating voluntary movements, we trained a monkey to perform wrist flexion-extension movement in an instructed delay task. Mild Electric stimuli were applied repetitively (2Hz) to the muscle nerve innervating wrist extensors (deep radial nerve; DR) through a nerve cuff electrode, and activity of neurons showing monosynaptic response (latency <1 ms; n=13) was recorded from cervical spinal cord through an implanted chamber. Firing rate was increased during both flexion and extension movement but their responsiveness to DR stimuli was suppressed only during flextion movement. Spike- and stimulus triggered averaging revealed that 84 % (11/13) of recording site produced an output effect to at least one wrist muscle. Results suggested that presynaptic inhibition on Group I muscle afferents suppresses the reflex action which is antagonistic to an ongoing movement.

Kakenhi18500315,18047027,18020030

## O1P-B06 Spatial distribution of cortico-muscular coupling during precision grip in monkeys

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Distribution of cortico-muscular coherence was investigated between local field potential of cortex with and EMG of various forearm muscles in a macaque monkey during performance of the precision grip task. We have found that Beta band coherent activity with intrinsic hand muscles found in primary motor cortex (M1), sensory cortex (S1) and ventral premotor cortex. High coherency ware observed not only on digit and wrist area but also arm area of M1. In the case of S1, the coherency ware observed only on digit and wrist area. Interestingly, arm area of M1 showed no coherence with arm muscles even task related EMG exist in arm muscles, but showed clear coherence with intrinsic hand muscles. The post-stimulus effect could be observed in intrinsic hand muscles from these sites when we increased the stimulus intensity up to 40 μA and applied repetitive stimulus (20 pulses at 333 Hz). We conclude that coherent activity in M1 areas of both hand and arm area may engage networks of neurons controlling the intrinsic hand muscles.

CREST, JST and KAKENHI(17021041)



## O1P-B07 Activity in posterior parietal cortex mediates the visual dominance over kinesthesia

Nobuhiro Hagura[1,2], Tomohiko Takei[1], Satoshi Hirose[1,3], Yu Aramaki[3,4,5], Michikazu Matumura[1], Norihiro Sadato[4], Eiichi Naito[1,3,5]

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We investigated how visual and kinesthetic information of hand is processed when humans perceive the spatial location of a hand.We used tendon vibration to induce an illusory movement of an immobile hand, while the participants viewed a live image of either the vibrated or nonvibrated static hand through an on-line video camera.The intensity of illusory movement was significantly attenuated only when the participants viewed the static image of the vibrated hand. The fMRI study showed that the posterior parietal cortex (PPC) is specifically involved in the attenuation of illusory movement, and that the activity of the PPC reflected the degree of attenuation.These indicate that PPC is involved in the multisensory processing where vision overrules simultaneously available kinesthesia in order to estimate the spatial location of a limb (visual dominance over kinesthesia).

JSPS, the 21st Century COE Program

## O1P-B08 Motor control-learning model for arm's reaching movements without desired trajectory

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In this paper, we propose a motor control model that generates arm's reaching movements without desired trajectory, and that can describes process of learning. Using the model, we simulated several kinds of reaching movements in the sagittal plane. In the simulation, we used an arm model with two-joints and six-muscles. To control the arm model without desired trajectory, we designed the control model that consists of feedback controller(FBC), inverse statics model(ISM) and forward dynamics model(FDM). Actor-Critic method, Feedback-Error-Learning scheme and the supervised learning are used to train FBC, ISM and FDM, respectively. After training trials, we simulated human subjects' reaching movements using our model. The hand paths and speed profiles of the simulation were qualitatively in good agreement with those of subjects. We also verified that past experimental result thought to be unable to explain without assuming the existence of desired trajectory can be reconstructed without desired trajectory by our model.

## O1P-B09 Cortico-nuclear topographic organization in terms of morphology of single Purkinje cell axons and aldolase C expression in the rat cerebellum

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The cerebellar cortex is compartmentalized into more than 20 longitudinal stripes by the aldolase C (=zebrin) expression pattern, which is tightly correlated with the topography of the olivocortical climbing fiber projection. Here we labeled Purkinje cells in different aldolase C stripes of the cerebellar cortex with biotinylated dextran amine and analyzed their axonal trajectories to clarify the compartmentalization of the cerebellar nuclei in the rat. Each Purkinje cell axon had a relatively small termination area in the cerebellar nucleus, which were often elongated from the surface toward the hilus of the nucleus. The axonal projection seemed to be organized according to fine cortico-nuclear topography. Purkinje cells in the aldolase C-positive and negative stripes in the cortex projected to the caudoventral and rostrodorsal parts of the nuclei, respectively. These results supported that the aldolase C expression is tightly related with the functional cortico-nuclear organization.

KAKENHI (18019013, 18500309)

## O1P-B10 Basal ganglia modulation of fictive locomotion in decerebrate cats

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The midbrain locomotor region (MLR) is located in the lateral part of the mesopontine tegmentum, which receives GABAergic projections from the substantia nigra pars reticulata (SNr), one of the basal ganglia output nuclei. Here we examined how the SNr-MLR projection modulates locomotion. In acute decerebrate cats (n=11), repetitive electrical stimulation (30-50 μA, 50 Hz) applied to the MLR generated rhythmic membrane potential oscillations in extensor and flexor motoneurons (n=47) (fictive locomotion). Although stimulation of the SNr (20-60 μA, 50-100 Hz) alone did not alter the excitability of motoneurons, the stimuli attenuated and finally suppressed the MLR-induced fictive locomotion by decreasing the amplitude of the hyperpolarizing phase of the oscillations, resulting in tonic membrane depolarization of motoneurons. We conclude that SNr stimulation evokes co-contraction of extensor and flexor muscles so that the MLR-induced locomotion can be terminated.

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## O1P-B11 Single cell tracing of thalamostriatal projection neurons with reference to patch and matrix compartments of rat striatum

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Striatum consists of the patch compartment immunopositive for mu-opioid receptor (MOR) and matrix compartment negative for MOR, and receives glutamatergic inputs from cerebral cortex and thalamus. Thalamic intralaminar nuclei are known to project preferentially to the matrix compartment. However, it has not been clarified which thalamic nuclei project to the patch compartment. In the present study, we combined single cell tracing with immunohistochemistry for MOR, and revealed the distribution of thalamostriatal axon terminals in relation to striatal compartments. About a half of thalamostriatal terminals from single neurons in the thalamic midline nuclei are distributed in the patch compartment. Since the patch compartments occupy about 10% of the total volume in the striatum, these single neurons are considered to project preferentially to the patch compartment. In contrast, terminals from the thalamic intralaminar nuclei distributed preferentially to the matrix compartment, or equally to both the compartments.

## O1P-B12 Single-axon tracing study of thalamocortical projections arising from the motor nuclei of the rat

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The motor cortex receives inputs from the basal ganglia and cerebellum via the motor thalamic nuclei, ventral anterior and ventral lateral complex (VA/VL), in the rat. It was found that immunoreactivities for glutamic acid decarboxylase 67 and calbindin D28K were intense in the rostral part of the VA/VL, but weak in the caudal part. In contrast, the immunoreactivity for vesicular glutamate transporter 2 was dominant in the caudal part. Thus, in the present study, the VA/VL was divided into two compartments, rostral part (VA/VLr) and caudal part (VA/VLc). Single neurons in the VA/VL were infected with Sindbis virus vector expressing membrane-targeted green fluorescent protein, visualized by the immunoperoxidase method, and reconstructed. In most cases, VA/VLc neurons formed axonal arborization densely in layer III of the primary motor cortex, while VA/VLr neurons formed arborization in layer I and III of the primary and secondary motor cortex, and a dense plexus in the neostriatum.

## O1P-B13 Neural correlates of dynamic visuomotor matching for meaningless gesture imitation

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Previous studies have indicated that responses of the human mirror neuron system (MNS) are dependent on the action goal, indicating that the MNS plays roles in imitating or understanding actions based on the motor repertoire of imitators. In contrast, humans can imitate novel actions that cannot be directly mapped onto motor representations. We hypothesized that action meanings (goal or outcome) are essential for imitation of known actions, whereas dynamic spatiotemporal motion information of observed action plays essential roles in imitating novel meaningless actions. This study identified brain regions related to such dynamic visuomotor matching. We employed a 2*2 factorial design, with factors of stimulus presentation type (dynamic or static) and task (imitation or observation). Results showed a significant interaction of activation with higher response during dynamic imitation in the right parietal operculum and bilateral extrastriate body area, indicating that these areas are involved in dynamic visuomotor matching between observed action and self-movement.

## O1P-B14 MEG activity during a delayed visuomotor reaching task

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In reaching, spatial visuomotor transformations take place in our brain. We also have an ability to accomplish the transformation, even when a gain for the relationship is changed, for example, in a microsurgery. To understand the brain mechanisms, we recorded neural activity of humans using MEG during a delayed reaching task, and analyzed the activity during various stages of information processing. In each trial of the task, a visuospatial target was instructed during a delay period on a screen, while the gain between hand position monitored by a computer mouse and its location on the screen was systematically changed. During the delay, the subjects were asked to maintain their eye and hand movements at the central hold zone, and were required to initiate a reaching movement to the target when the central hold zone was extinguished. The MEG, behavioral, and EOG data were analyzed and compared between conditions. We found activity representing information flows in the cerebral cortex, possibly contributing to transformation of coordinates in reference frames for reaching.

KAKENHI (18020002)

## O1P-B15 Premotor cortex (PM) is involved in converting a concept for action into a motor plan

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This study examined the involvement of PM in converting a concept for action into an actual motor plan. We recorded neuronal activity from PM while monkeys were performing a behavioral task that required the determination of an action concept, followed by the specification of an actual movement. Initially, a visual object was presented as an instruction cue, indicating 'reach left' or 'reach right'. Subsequently, a choice cue consisting of a pair of squares appeared at various locations on the monitor. Because the instruction cue signaled only the action concept, the animals could prepare an actual movement for the first time after the appearance of the choice cue. If the monkey reached to a square located at the instructed side, a reward was given. We found that the neuronal activity in PM initially reflected the action concept, subsequently taken over by activity reflecting the actual direction of planned movement. These results suggest that PM is involved in the conversion of an action concept into a motor plan specifying an actual movement.

KAKENHI (18680035)

## O1P-B16 Inhibition of human motor cortex excitability during stop signal task

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Inhibitory control is an essential part of human behavior. To know whether motor inhibition is a stop of movement flow or an active process suppressing primary motor cortex (M1), we investigated M1 excitability by using Transcranial Magnetic Stimulation (TMS) during stop signal task in which subjects were asked to press a button using right or left thumb following Go cue. Stop cue follows Go cue in 20% of trials, where subjects were instructed not to press. TMS was given at left M1 at variable time delay from Go cue. To estimate M1 excitability, Motor Evoked Potential (MEP)was recorded from both hands. On delivering TMS 400 ms after Go cue, we observed significant MEP reduction at right hand in successful stop trials, which was lost in failed stop trials. These findings suggest that voluntary inhibition of movement in this task is an active process.

KAKENHI(18019020, 18020014, 18500239)

## O1P-C01 Human vision can predict semi-random but linear process.

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It is advantageous for animals and humans to predict changes in the environment.Note that the change of the environment is not only monotonous, but also much more dynamic. To uncover mechanisms for such a prediction is one of the most important topics in brain science. The aim of this study is to investigate a predictive ability of humans on visuomotor system, especially saccadic system. In our behavioral experiment, subjects were required to fixate a small target that was presented on monitor, predict the next target position, and gaze to the predicted position. We used a 1st-order or 2nd-order AR process as the target time-series in order to make the ime-series aperiodic, and thus the periodic-pattern-based (memory-based) approach was unsuccessful. he acquired results suggest that good predictive performance of all subjects, and that the subjects employed the similar dynamics to the one which was used to generated the presented time-series for each task.

## O1P-C02 Neural control of saccadic decision-making

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Decision making(DM) is a hallmark of voluntary control. Psychological models of DM and neural recordings from awake behaving monkeys have shown evolving decision as information accumulation process with time-course tied to saccadic eye movements. But the time when the decision is committed relative to the eye movement is still unclear. Is it before saccade execution, providing a distinct point of no return in saccadic DM, or is it during execution. Subjects performed multiple gaze shifts to single target due to induced dysmetria. A 2nd target appeared on random catch trials instructing subjects to cancel the partially prepared response to the 1st target. Subjects could inhibit the wrong primary saccade(PS);on failing they could inhibit the secondary saccade(SS) to 1st target. So we could observe inhibitory control at multiple instances in time. Reaction times(RT) of erroneous PS could be predicted by a racemodel framework but RT of SS were underestimated. We observed that PS could be interrupted even during execution. We dint find evidence of inhibitory control on SS. These findings suggest that control can be effected during the execution of PS but beyond that we reach a ballistic stage in DM.

DST, INDIA



## O1P-C03 Ocular following responses elicited by binocular first-order motion in primates

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Previous studies indicate that the ocular following responses (OFRs) elicited by moving images are mediated by cortical neurons tuned to both motion and binocular disparity. However, it is not known if the OFR can be elicited by pure binocular motion signals in the absence of any monocular component. Here, we developed a new binocular motion stimulus that lacked any monocular motion component. In this stimulus, motion emerges only after the summation of inputs from both eyes. We found that this pure binocular motion elicited the OFR, but with latencies longer than those elicited by monocular motion. We further manipulated the intensities and directions of the monocular and binocular motion components independently and observed enhanced responses due to a facilitative interaction between the monocular and binocular motion mechanisms. This finding is in contrast to the results of additional experiments showing that other types of motion stimuli, which cannot be detected by the linear motion detectors, have only modest effects on the OFR while evoking clear motion sensation.

JSPS16GS0312

## O1P-C04 Neural correlate of residual vision in the animal model of blindsight

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Blindsight patients can discriminate the target positions while they deny awareness of the targets. To examine whether monkeys have blindsight, two macaque monkeys with unilateral V1 lesion were tested with two saccade tasks; a forced-choice (FC) task, in which the monkeys have to make saccade to one of the two possible positions, and a yes-no (YN) task, in which the monkeys have to maintain fixation when the target is absent. The monkeys had a better performance in the FC task that requires discrimination than in the YN task that requires detection. This suggests that the monkeys exhibit behavior that parallels to the human blindsight. Then we recorded from the superior colliculus of the monkey performing a delayed saccade type of the two tasks and found that the response to the visual target was larger when the monkey successfully discriminate or detect the target than when it failed. We conclude that the target-related activity comprises a part of neural correlates of residual vision.

KAKENHI 13854029, 16700343 and CREST

## O1P-C05 Quick learning of stimulus-reward association by neurons in the monkey inferior temporal areas

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To study the role of the area TE and perirhinal cortex in forming association between visual stimulus and its reward value, we recorded spike activities from neurons in the two areas while monkeys were performing a visually cued GO/NOGO task with differential reward conditions. The visual cue indicated both GO/NOGO and reward condition. Correct (GO and NOGO) responses were rewarded with a liquid reward or just acknowledged by a sound, depending on the cue. We previously reported that perirhinal and TE cells represented the associated reward condition in their responses to the 80 object images that had been consistently used in the task for several months. In addition to the 80 familiar stimuli, we used 8 novel object images that were exchanged every day. Cells in both areas responded to the novel stimuli, and the magnitude of the responses started to reflect the associated reward condition within a few trials. These results suggest that the area TE and perirhinal cortex are involved in learning stimulus-reward contingency, even in its very early phase.

## O1P-C06 Axonal arborizations of single thalamocortical neurons in the rat visual cortex

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Although axonal arborizations of single LGd neurons in primary visual cortex (V1) have been investigated, little is known about those of LP neurons in secondary visual cortex (V2). In this study, we analyzed projections of single LP and LGd neurons, using Sindbis virus which expresses membrane-targeted green fluorescence protein. One of the observed LP neurons projected mainly to medial area of V2 (V2M), sending axon collaterals to lateral area of V2, thalamic reticular nucleus (Rt), and neostriatum. In the V2M, the axons were observed mediolaterally over 0.8 mm, and anteroposteriorly 1.2 mm. Its terminal boutons were distributed dominantly in layers III (22.5% of total boutons) and IV (33.8%). On the other hands, one of the observed LGd neurons projected to V1 and Rt. In the V1, the axons extended mediolaterally 0.5 mm and anteroposteriorly 1.6 mm, and the boutons were found mostly in layers I (15.4%), III (28.7%) and IV (32.3%) of V1. These results suggest that the Sindbis virus enables us to reveal different projection patterns of LP and LGd single neurons.

## O1P-C07 Form representation via center-surround organizations of V1 neurons revealed by high-order receptive field analysis.

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It is well known that stimuli presented outside the classical receptive field (CRF) of a visual cortical neuron suppress its activities. This effect has traditionally been considered as providing auxiliary modulations to the properties determined by the CRF. However, its roles and the spatial organization of this suppressive effect have not been clear. We have recently developed a new method for reconstructing the details of the CRF (center) and surround structures simultaneously. Based on the two-dimensional maps obtained by this method, we tested an idea that these structures constitute next-stage processing units suitable for encoding high-order contours such as contrast or texture boundaries. The majority of the center-surround maps showed a variety of oriented organizations, and cell responses were selective for orientation of high-order contours. These results indicate that a population of neurons with surround suppression is able to represent higher-order forms defined by contrast or texture borders.

KAKENHI(18020017,13308048,15700258), Uehara Foundataion

## O1P-C08 Role of striatal visual pathway in facilitatory and inhibitory effect on visual attention

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Peripheral visual stimulus captures the subject's attention for a short time after the stimulus presentation (100-200ms; attention capture) but then inhibits reaction to that location afterwards (>200ms after the stimulus; inhibition of return, IOR). These two effects on attention can be measured by the change in the saccadic reaction time (SRT) to that location. The attention capture shortens and IOR prolongs the SRT. Previous studies revealed that superior colliculus (SC) is important for IOR. However, the neural mechanism of IOR has been still unclear. To address this question, we made a lesion in unilateral primary visual cortex (V1) in the macaque monkey and observed its effect on visual attention. It was found that the attention capture remained but IOR disappeared after the V1 lesion. The results suggest that attention capture and IOR are mediated by different neural systems: attention capture by retino-tectal visual pathway and IOR by striatal visual pathway.

CREST, JST and KAKENHI (18700384)

## O1P-C09 Responses of MT neurons during task switching: II. Correlation between neuronal responses and perceptual judgments.

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Neurons in area MT are crucial for motion direction and depth discrimination when performed independently. However, it is unknown how MT neurons are used when both tasks are interleaved. Here we determined how MT signals are read out in this situation. A monkey performed task switching (see previous abstract), while we recorded from 62 isolated MT neurons. Neurons were classified into two groups, "Congruent" and "Incongruent", where the preferred direction and depth corresponds to the same (e.g. UP/FAR) or different (e.g. UP/NEAR) behavioral responses, respectively. To assess the functional coupling between MT responses and perceptual decisions, we computed choice probabilities (CPs). CPs for congruent neurons were large for both direction and depth discrimination tasks ($R = 0.39$, $p = 0.035$). However, CPs for incongruent neurons were large for either task ($R = -0.44$, $p = 0.010$). The results suggest that congruent neurons contribute to both direction and depth discrimination tasks, whereas incongruent neurons only contribute to either task.

HFSP

## O1P-C10 Responses of MT neurons during task switching: I. Psychophysical and neuronal switch ratio.

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One can attend to different visual features and select different action even when presented with the same sensory information. Here we investigated how the same sensory signal is converted into different movement signals. A Japanese monkey was trained to switch attention to either motion direction or depth. On each trial, the color of the fixation point indicated which dimension the monkey had to attend to, and the monkey discriminated either direction (UP or DOWN) or depth (FAR or NEAR) in a moving random dot stereogram using saccades (UP or DOWN). We recorded from 62 isolated MT neurons while the monkey performed task switching. We quantified how well the monkey and neurons switched between direction and depth discrimination by calculating a "Switch Ratio (SR)". If the monkey and neurons could perfectly switch tasks, then SR = 1: if not at all, then SR = 0. Behavioral SR was $0.67 \pm 0.19$, whereas neuronal SR was $-0.09 \pm 0.58$ ($n=62$). The data suggests that, although the monkey successfully switched attention between direction and depth, neural activity in area MT does not depend on task.

HFSP

## O1P-C11 Neural responses selective for the orientation of the luminance gradient in the inferior temporal cortex of the monkey

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Shading generated by luminance gradient is an important visual stimulus for perception of the depth structure and surface quality of objects, but little is known about how shading is represented in the brain. We recorded neuron activities from the lateral surface of the posterior inferior temporal cortex (PIT) of awake monkey performing a visual fixation task and found that many neurons sensitive to shading stimuli were concentrated in a small region in this area. A shading stimulus had a linear luminance gradient within a circular region and in one of 8 directions separated by 45 deg. Many neurons in this sub-region of PIT exhibited strong selectivity to the direction of luminance gradient, and the sharpness of selectivity varied from cell to cell. Many of them showed position invariance in direction selectivity; when the position of the stimulus changed within the RF, the preferred direction was largely maintained. We suggest that PIT cortex plays an important role in the coding of shading stimuli.

## O1P-C12 Multiple column activity in macaque area TE can encode object identity across view angles.

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Past studies show that complex object images are represented with combinations of activities from multiple functional columns. In this study, we asked whether the combination of activities from functional columns, rather than the activity from a single column, could help to encode object identity in a more view-invariant manner. Using a densely arranged grid-type multiple-electrode array system, and treating the data from a single electrode as the putative columnar activity, we found that evoked response elicited by different views of the same object can be better discriminated from the response of other objects with combination of multiple columnar activity than with single columnar activity.

## O1P-C13 NMDA receptor blockade during monocular deprivation prevents the maintenance of ocular dominance shift produced by the deprivation

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We investigated the effect of the NR2B-containing NMDA receptor (NR2B-NMDAR) antagonist Ro 256981 on ocular dominance shift produced by monocular deprivation (MD). We recorded visual evoked potentials (VEPs) from left visual cortex of pigmented rats that underwent MD of right eye from postnatal 26 to 31 days, and assessed the ocular dominance shift using the ratio of VEP amplitude for right eye to that for left eye. When the antagonist (3 mM) was infused into left visual cortex during MD, ocular dominance, assessed soon after MD, shifted toward the non-deprived eye (0.70±0.038, n=7), as was seen in control MD (0.70±0.048, n=7). However, the ratio returned with time after MD (3 day, 1.4±0.18, n=9; 1 month, 1.75±0.11, n=8) to the normal level seen in rats with no MD (2.0±0.08, n=9). This recovery was not found in rats without the infusion. These results suggest that NMDAR activation during MD is necessary to maintain the modified ocular dominance.

## O1P-C14 Regulation of extracellular signal-regulated kinase activity and subcellular localization by visual input in rat visual cortex

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Monocular deprivation (MD) induces significant plasticity in the primary visual cortex (V1) in the early postnatal life. Although the activity of extracellular signal-regulated kinase (ERK) has been reported to be necessary for the ocular dominance plasticity, it remains to be elucidated how the ERK activity is modulated by MD. Therefore, we examined effects of MD on ERK activity in rat V1. We found a significant decrease in the amount of pERK in V1 receiving deprived eye inputs in both young and adult MD rats. As to the subcellular localization, we found a significant increase in the nuclear pERK after MD only in young rats. While MD for 12 hours did not increased nuclear pERK, the strongest increase was observed after 24-hr MD. The increase has declined in 3-day MD rats and was not observed after 1-week MD. These results suggest that nuclear signaling of ERK transiently upregulated following MD in youg rats.

KAKENHI(176959)

## O1P-C15 Cellular components in electrical synapses between retinal amacrine cells contributing to lateral interactions

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We measured fraction of open channels in gap junctions in ganglion (J Neurosci, 2004) and amacrine cells (J Intgra Neurosci, 2005). The open fraction in cells expressing connexins was on the order of 0.1 to 1. The open fraction is small, when we consider whether electrotonic transmission acts to synchronize spikes and extends receptive filed size beyond dendritic field size (space length constant $\lambda > 1.9$ mm). Our study showed that similar electrical synapses are present in cells expressing the specific connexin. Next I investigated cellular components in several amacrine cells performing lateral interactions, because different types of cells make differential inhibitory synapses. Electrical current spread through connections of specific cell types is expected to synapse certain inhibitions. I measured junction conductance between cells by dual patch-clamp. In high-voltage electron microscopy (Hitachi 1250M, NIPS, 2006HVEM-13), the size and dendritic location of gap junctions were measured. A total size of gap junctions between the cells was correlated with functional electrical interactions within the networks.

21st COE

## O1P-C16 Comparison of electrophysiological properties of layer III pyramidal neurons between cortical areas V1 and TE of the macaque

Ayako Ishikawa[1], Atsuko Maruyama[2], Daisuke Hosoyoma[1], Yumiko Yoshimura[2,3], Hiroshi Tamura[1,2], Hiromichi Sato[4], Ichiro Fujita[1,2]

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Electrophysiological properties of layer III pyramidal neurons were compared between areas V1 and TE, the two ends of the ventral visual cortical pathway in macaque monkeys. We applied whole-cell patch-clamp techniques to brain slices taken from adult animals. Neurons in the two areas markedly differed in several electrophysiological properties; 1) the membrane time constant was smaller and the input resistance was higher in V1 than in TE, 2) the rate of spike firing evoked by a long-duration (500 ms) depolarizing current pulse was more rapidly and strongly attenuated in V1 than in TE, and 3) the rise time of miniature excitatory postsynaptic currents in V1 was shorter than that in TE. The results suggest that V1 neurons are suited for processing information with higher temporal resolution, while the properties of TE neurons have an advantage in integrating information over a longer time range.

CREST

## O1P-D01 Molecular logics to generate the olfactory sensory map

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While it has been thought that odorant receptor (OR) molecules play an instructive role in forming the glomerular map, it has remained entirely unclear how this occurs at the molecular level. Our recent data indicated that ORs may not directly act as guidance receptors or adhesion molecules, but instead establish the neuronal identity by a set of cell recognition molecules expressed at axon termini. After axons are guided to approximate destinations in the olfactory bulb, the axon termini are sorted based on the expressed OR species. Our results demonstrated that the levels of transcription of a set of cell adhesive and repulsive molecules are uniquely correlated with the expressed OR species and are regulated in an activity-dependent manner. We propose that a unique combination of cell-recognition/axon-guidance molecules, whose expression levels are determined by OR molecules, constitute the neuronal identity code for individual OSNs.

CREST

## O1P-D02 Odorant receptor-dependent transcriptional regulation for the olfactory glomerular mapping

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In the mouse olfactory system, odorant receptors (ORs) direct the axons of olfactory sensory neurons (OSNs) toward targets in the olfactory bulb. We have recently shown that genetic manipulation of ORs, Gs, protein kinase A, and CREB shifted the axonal projection sites along the anterior-posterior axis in the olfactory bulb (Science, 314, 657- (2006)). Thus, it is the OR-derived cAMP signals, rather than direct action of OR molecules, that determine the target destinations of OSNs. To uncover the target genes of OR/cAMP signaling in axonal projection, we developed an improved single-cell microarray method. The genes identified included axon guidance molecules and synaptic proteins. One of the cAMP-regulated axon guidance receptor, neuropilin-1, showed an anterior-low/posterior-high gradient at the OSN axon termini. Genetic manipulation to increase the neuropilin-1 expression caused a posterior shift of the axonal projection. The cAMP-regulated genes identified here provide molecular bases for the OR-specific glomerular mapping in the olfactory bulb.

## O1P-D03 Repulsive effect of Sema3F on lot cells, the guidepost neurons in the lateral olfactory tract

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The accurate neuronal migration within the developing brain is a critical process for formation of the functional nervous system. In this study, we examined migratory mechanisms of a unique neuronal population, lot cells. These neurons differentiate from the neocortex region and migrate long distances on the surface of the neocortex ventrally and tangentially. After the migration, they align at the presumptive lateral olfactory tract region. Such behavior of lot cells is quite unique, but molecular regulation of the lot cell migration still remains unknown. To identify guidance factors for the lot cell migration, we conducted an candidate screening and found that Sema3F repels the migration of lot cells in culture. Sema3F receptor, Neuropilin-2 (Nrp2), was expressed in lot cells, and Sema3F was expressed in the medial region of the developing brain. In sema3F and nrp2 mutant mice, lot cells were ectopically distributed in the medial region of the brain. These results suggest that Sema3F-Nrp2 signaling regulates the migration of lot cells by its repulsive activity.

## O1P-D04 Development of the mouse amygdala as revealed by labeling with EGFP gene transfer and molecular markers

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Amygdala is composed of several nuclei and is involved in the control of emotion. The mechanisms of amygdalar development are still poorly understood. In the present study, we have investigated cell migration and nuclear formation in the mouse embryonic amygdala. We applied the EGFP gene transfer into the caudal ganglionic eminence (CGE) and diencephalon to trace neuronal migration and identified the nuclear formation by molecular markers. By labeling CGE during E11-12, basolateral complex expressing Tbr1 originated from the ventral pallium and corticostriatal angle. By labeling the neuroepithelium of the third ventricle (3V), medial nucleus expressing Lhx9 originated at least in part from the neuroepithelium of the 3V. These findings suggest that each amygdalar subdivision originates from the temporally- and spatially-restricted neuroepithelium of the telencephalon and diencephalon to form a mosaic of amygdalar complex.

## O1P-D05 Cdk5 is required for multipolar-to-bipolar transition during radial neuronal migration in the cerebral cortex

Toshio Ohshima[1,2], Motoyuki Hirasawa[3], Hidenori Tabata[4], Takuji Iwasato[5], Shigeyoshi Itohara[5], Kazunori Nakajima[4], Ashok Kulkarni[3], Katsuhiko Mikoshiba[1]

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The mammalian cerebral cortex is generated via coordinated neuronal migration. Neurons transform from multipolar to bipolar shape during their radial migration. To study the role of Cdk5 in this process, we visualized migrating Cdk5-/- neurons as well as neurons expressing the Cdk5-DN. We found that Cdk5 deficiency caused specific impairment of multipolar-to-bipolar transition. Dendrite morphology of pyramidal neurons was impaired in postnatal cortex of Cdk5 conditional knockout mice. These results indicate that Cdk5 is required for multipolar-to-bipolar transition of migrating neurons and its deficiency results in an abnormal dendritic morphology of pyramidal neurons in the postnatal cerebral cortex.

KAKENHI18022050

## O1P-D06 Mouse Dab1, an essential component in layer formation of cerebral cortex, shuttles between the nucleus and the cytoplasm

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Disabled1 (Dab1) is an intracellular mediator of the Reelin-signaling pathway and essential for correct neuronal positioning during brain development. So far, Dab1 has been considered a cytoplasmic protein. Here, we show that Dab1 is subject to nucleocytoplasmic shuttling. In its steady state, Dab1 is mainly located in the cytoplasm. However, treatment with leptomycine B, an inhibitor of the chromosomal region maintenance 1 (CRM1)/RanGTP-dependent nuclear export, resulted in nuclear accumulation of Dab1. By using deletion or substitutional mutants of Dab1 fused with green fluorescent protein, we have mapped a nuclear localization signal (NLS) and two nuclear export signals (NESs). These targeting signals were functional in both Neuro2a cells and primary cerebral cortical neurons. Using purified recombinant proteins, we have shown that CRM1 binds to Dab1 directly in a RanGTP-dependent manner. These results suggest that Dab1 is a nucleocytoplasmic shuttling protein, and raise the possibility that Dab1 plays a role in the nucleus as well as in the cytoplasm.

## O1P-D07 CXCR4-dependent "wandering period" of migrating cortical interneurons

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Functioning of the cerebral cortex requires coordinated migration of projection neurons and interneuron (IN)s. While radial migration of the former is well documented, cellular and molecular mechanisms controlling migration of INs within the cortex remain largely unknown. Here we show that migrating INs in mice embryo stay in the marginal zone (MZ) for a long period of time. Analysis of the laminar distribution and migratory behavior of INs was done after labeling by *in vivo* electroporation. We find INs transiently accumulate in the MZ and stay there for >one day. These INs are either stationary or "wandering" within the plane of MZ. The accumulation of INs in the MZ was diminished after IN-specific removal of chemokine receptor CXCR4. Our results reveal a novel behavior of migrating neurons, "wandering", which may lead them to move in step with the development of surrounding tissues.

## O1P-D08 Molecular basis of NMDA receptor-mediated neuronal migration in the early stage of mouse cortical development

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Time-lapse imaging of immature neurons visualized by EGFP has shown that NMDA receptor antagonist caused abnormal direction in the neuronal migration in the developing mouse cortex. To elucidate the molecular basis in neuronal migration mediated by NMDA receptor, we initially investigated the phosphorylation of several signalling molecules regarding neuronal migration. We found that Try-861 phosphorylation of focal adhesion kinase (FAK) was dramatically decreased in the NMDA receptor antagonist-treated mouse. Then, we constructed FAK mutant replaced of Tyr by Phe (FAK-Y861F). Further study by time-lapse imaging using FAK-Y861F suggested that FAK was involved in the NMDA receptor-mediated neuronal migration.

KAKENHI18591176

## O1P-D09 Wnt4 is as a local repulsive cue that determines synaptic target specificity

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How synaptic specificity is molecularly coded in target cells is a long-standing question in neuroscience. We conducted single-cell microarray analysis of two neighboring muscles (M12 and M13) in Drosophila innervated by distinct motor neurons. We identified a number of potential target cues that are differentially expressed between the two muscles, including M13-enriched Wnt4. When the functions of Wnt4, or its putative receptors, were inhibited, motor neurons that normally innervate M12 (MN12s) formed smaller synapses on M12 but instead, formed ectopic nerve endings on M13. Conversely, ectopic expression of Wnt4 in M12 inhibits synapse formation by MN12s. These results suggest that Wnt4 determines target specificity by preventing synapse formation on a non-target muscle. Ectopic expression of five other M13-enriched genes also inhibits synapse formation by MN12s. These results demonstrate an important role for local repulsion in generating cell-to-cell target specificity.

## O1P-D10 The kinase signaling network regulating establishment and maintenance of dendritic fields

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Little is know about how neurons establish and maintain their unique dendritic fields in development. Drosophila da sensory neurons can be classified into 4 subtypes (I-IV) based on their dendritic morphology, and the dendritic field of class IV da neurons is shaped in part through a like-repels-like tiling behavior of dendrite terminals. We have identified the protein kinases Tricornered (Trc) and Hippo (Hpo) as essential regulators of dendritic tiling in class IV da neurons (Cell 119, 245, 2004; Nature 443, 210, 2006). Hpo physically associates with Trc in neurons and can phosphorylate Trc in vitro, consistent with a model in which Hpo regulates Trc activity. In addition, Hpo-mediated regulation of dendrite morphogenesis requires functions of the Warts/Lats (Wts) kinase, which is known to act with Hpo to promote cell cycle arrest and apoptosis. Taken together, we propose that Hpo regulates multiple aspects of dendrite development in maturing class IV da neurons through distinct downstream signaling pathways: the Trc kinase pathway for dendrite tiling and the Wts kinase pathway for dendrite maintenance.



## O1P-D11 Filopodia of nerve growth cones autonomously rotate in the direction of the right-handed screw

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It has been believed that neurites grow straight in the absence of directional guidance cues. This notion, however, is not exactly correct, since neurites turn rightward on poly-lysine substrate (Heacock and Agranoff, Science 198, 64, 1977). Based on our recent findings that neurites turn rightward on two-dimensional but not in three-dimensional substrates, we hypothesized that growth cones autonomously rotate in the direction of the right screw. This hypothesis was tested by time-lapse analysis of growth cone motility. Although the growth cone shaft did not appear to rotate, each filopodium rotated in the right screw direction. We further found that expression of dominant-negative myosin V markedly reduced filopodial motility including rotational movement. These results demonstrate a novel mode of filopodial motility that is driven by actomyosin interactions.

KAKENHI(15650060,18500255)

## O1P-D12 Analysis of entorhino-hippocampal projection in PTPδ mutant mice

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Although the role of LAR class protein tyrosine phosphatases in the axon guidance has been shown, the relation to specific guidance cues has been poorly understood. We found that PTPδ, one of the LAR class members, forms a complex with Neuropilin-1 and participates in Semaphorin-3A signaling. To clarify in vivo function of this interaction, we analyzed PTPδ mutant mice. PTPδ mRNA predominantly expressed in cortical plate of late embryonic stages. The expression was partially overlapped with Neuropilin-1. Semaphorin-3A-induced repulsion of entorhinal axons was reduced in PTPδ mutant mice. Entorhino-hippocampal projection was also aberrant in the mutant mice. These results suggest that PTPδ mediates Semaphorin-3A signaling in the specific region of the central nervous system.

15590250

## O1P-D13 CRMP-2, Slp1 and Rab27B mediate anterograde transport of TrkB

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The most dramatic membrane recruitment to the tips of neurites occurs during neuronal development, which allows neurite to increase their total membranous components and the cell surface proteins, such as tyrosine kinase receptor B (TrkB). However, the mechanism of this recruitment to the growing axon is largely unknown. Here we report that collapsin response mediator protein-2 (CRMP-2), synaptotagmin-like protein 1 (Slp1/JFC1/Exophilin7), and Rab27B form a complex, and mediate the anterograde transport of TrkB. CRMP-2 directly interacts with light chain of Kinesin-1 (KLC), and Slp1 associates with Kinesin-1 via CRMP-2. The velocity of TrkB-mCherry is relatively high in growing neurites, and its anterograde transport is decreased by knockdown of Slp1, CRMP-2, or KLCs. This interaction might be a regulatory point required for linking motor and cargos, and enable the selective recruitment of TrkB, which is an essential survival signal moving back from axonal terminal.

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## O1P-D14 MARCKS participates in neurite initiation and outgrowth through the association with lipid rafts

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Myristoylated alanin-rich C kinase substrate (MARCKS) is abundantly expressed in central nervous system and some portions seem to be associated with cholesterol-enriched membrane microdomains (lipid rafts). Although we previously reported that MARCKS plays an important role in neurite initiation and outgrowth induced by insulin-like growth factor-I (IGF-I) in SH-SY5Y neuroblastoma cells, the precise mechanism remains unknown. In this study, we detected that MARCKS is translocated to lipid rafts by the treatment with IGF-I. The amount of MARCKS in 1% Brij-35 -insoluble lipid raft fractions was increased 30 min and 24 hr after the treatment, which was almost unphosphorylated form. The knockdown of endogenous MARCKS by RNA interference significantly attenuate the neurite outgrowth induced by IGF-I. These results suggest a novel role for MARCKS in neurite initiation and outgrowth by IGF-I through the association with lipid rafts.

## O1P-D15 UEV-3, a ubiquitin conjugating enzyme E2 variant, functions in the p38 MAP kinase pathway for synaptogenesis

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Synapses commonly contain a single electron-dense region at its presynaptic terminal, which is surrounded by a cluster of synaptic vesicles. Loss-of-function mutations in the *C. elegans rpm-1* gene, a putative RING finger/E3 ubiquitin ligase, cause disorganized presynaptic cytoarchitectures. The gene product RPM-1 negatively regulates a p38 MAP kinase pathway that is composed of DLK-1 (MAPKKK), MKK-4 (MAPKK), and PMK-3 (p38 MAPK) (Ref. 1). Genetic suppressor analysis has further revealed that UEV-3 functions in the RPM-1-DLK-1-p38 MAP kinase pathway and acts downstream of MKK-4. Expression analysis has uncovered that UEV-3 locates in nuclei and cytoplasm of motor neurons, similar to PMK-3 expression patterns. We have also found UEV-3 physically interacts with PMK-3. These results suggest that UEV-3 may promote the PMK-3 activity by acting as a scaffolding protein or chaperone in synaptogenesis. (1) Nakata, K. et al. Cell, 120: 407-420, 2005

## O1P-D16 Regulation of dendritic development by E3 ubiquitin ligase Nedd4

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Regulation of protein degradation by polyubiquitination and proteasome is critical for the basic neuronal and synaptic function. Ubiquitination of target proteins are regulated by 3 enzymes, E1 ubiquitin activating enzyme, E2 ubiquitin conjugating enzymes, and E3 ubiquitin ligase. Specificities of targets are determined by E3 ligases. In order to show the function of certain specific ubiquitination, we generated an E3 ubiquitin ligase Nedd4 knockout mouse. In my presentation, I would like to show the regulation of dendritic development by Nedd4.

## O1P-E01 Redistribution of Kv4.2 potassium channels in the hippocampus induced by acute neuronal excitation *in vivo*

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The Kv4 subfamily of voltage-gated potassium channels in the brain are involved in regulation of subthreshold membrane potentials, and thus reception and integration of synaptic signals. We recently reported targeted clustering of Kv4.2 channels at postsynaptic sites of GABAergic synapses in addition to the localization at extrasynaptic sites. However, their functional significance is still unclear. To get better insight into the role of Kv4.2 channels, here we analyzed the activity-dependent changes in subcellular localization of Kv4.2 channels *in vivo*. After neuronal excitation induced by electrical stimulation, we encountered acute clustering of Kv4.2 channels in the somata of CA3 pyramidal cells. We also found increased expression of phosphorylated Kv4.2 channels in granule cells. Our results indicate a possible involvement of Kv4.2 channels in synaptic plasticity in the hippocampal region.

UEHARA MEMORIAL FOUNDATION

## O1P-E02 Nav1.1 predominantly localizes to axons of parvalbumin-positive inhibitory interneurons: a circuit basis for epileptic seizures in Nav1.1-deficient mice

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We have recently generated a Nav1.1-deficient mouse line and shown that these mutant mice developed epileptic seizures. We here report Nav1.1 localization in the developing neocortex and hippocampus. In neocortex, Nav1.1 is predominantly found at the axon initial segments (AISs) of parvalbumin-positive (PV) interneurons. Moreover, in mutant mice, trains of evoked action potentials in these PV, fast-spiking cells exhibited pronounced spike amplitude decrement late in the burst. In the hippocampus, Nav1.1 is predominantly distributed within AISs and somata of PV interneurons. Our data indicate that Nav1.1 plays critical roles in the spike output from PV interneurons and further, that the specifically altered function of these inhibitory circuits may contribute to epileptic seizures in the mice.

KAKENHI 17790736

## O1P-E03 High-threshold voltage-gated $Ca^{2+}$ channels may mediate capacitive $Ca^{2+}$ entry in central neurons

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In central neurons, $Ca^{2+}$ derived from the intracellular stores plays important roles such as induction of synaptic plasticity. The stores running out of $Ca^{2+}$ are refilled with $Ca^{2+}$ entering through cation channels on the plasma membrane. Using fura-2 fluorometry, we characterized $Ca^{2+}$ influx following thapsigargin-induced depletion of the intracellular stores in cultured mouse cerebellar Purkinje cells. Agatoxin IVA (agaIVA), a P/Q-type high-threshold voltage-gated $Ca^{2+}$ channel (hVGCC) blocker greatly reduced this $Ca^{2+}$ influx. Store depletion might also activate cation channels whose auxiliary $Na^+$ conductance could possibly cause depolarization and activation of hVGCCs. However, this cannot explain the agaIVA-sensitive $Ca^{2+}$ influx because it persisted in $Na^+$-free saline. Moreover, the agaIVA-sensitive $Ca^{2+}$ influx was susceptible to xestospongin C, an inositol trisphosphate receptor antagonist. These results suggest that P/Q-type hVGCCs may contribute to intracellular store refilling in central neurons.

KAKENHI 18019022, 17023021, and 17100004

## O1P-E04 Why does the neuronal firing rate increase with a greater injected current?

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Neuronal firing rate increases as a greater current injected into the neuron. It is broadly believed that the speeded increase in membrane potential caused by a larger current shortens the time for exceeding the threshold level, and thus reduces the periods. However, this understanding is surprisingly ungrounded. The present study addresses the mechanism determining the relationship between the input current and firing rate using a simplified Hodgkin-Huxley model with two state variables, whose behavior can be readily analyzed on a phase plane. Our analysis revealed that the increase in the firing rate stemmed from the elevated level of interspike membrane potential and the reduction in the amplitude of oscillation of $K^+$ conductance, and that the latter factor originated from the increase in the minimum level of $K^+$ conductance activation. In other words, the reduction of the path length on the phase plane for each cycle of spike generation is the essential cause for shortening the period. This suggests a close relationship between the neuronal firing rate and the spike amplitude.

## O1P-E05 Brall, brain-specific link protein, is essential for stabilizing extracellular matrix at the node of Ranvier in the CNS.

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Myelinated axons in the nervous system are organized into distinct domains with glial cell. The node of Ranvier ensures rapid propagation of the action potential along myelinated fibers. The hyaluronan binding chondroitin sulfate proteoglycans, called lecticans are the abundant ECM molecules in the brain. Brain link protein-1 (Brall) is colocalized with versican V2, a lectican member, at the node of Ranvier in the CNS. In this study, we have generated Brall-deficient mice via homologous recombination in embryonic stem cell. The formation and structure of myelin appeared normal. However, conduction velocity of the optic nerves of mutant mice showed about 20% decrease as compared with that of controls. Moreover, versican V2 and other ECM molecules showed aberrant localization in Brall-deficient mice. Our results suggest that Brall plays an essential role of stabilizing extracellular matricies at the node of Ranvier and ECM complex is partially concerned with conduction velocity in the CNS.

KAKENHI (17046012, 18791206)

## O1P-E06 Balanced inputs "clamps" firing irregularity decoupled from rate fluctuations on any timescale

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Cortical neurons exhibit highly irregular spike patterns. Since interspike intervals fluctuate greatly, irregular firing makes it difficult to estimate instantaneous firing rates accurately. If, however, the spiking irregularity is decoupled from rate modulations, the estimate can be improved. Here, we introduce a novel coding scheme to make the firing irregularity orthogonal to the firing rate in information representation. The scheme is valid if an interspike interval distribution can be well fitted by the gamma distribution. We investigated in computational models and whole-cell patch-clamp recordings whether fluctuating current input generate gamma process-like spike outputs, and whether the two quantities are actually decoupled. The spiking irregularity remained approximately constant irrespective of large rate fluctuations when we injected balanced inputs where excitatory and inhibitory synapses are activated concurrently. These results indicate that the balanced input may improve coding efficiency by clamping spiking irregularity.

KAKENHI(18020007, 18079003, 1810171)

## O1P-E07 Non-uniformity of membrane property improves dendritic signal transfer in hippocampal CA1 pyramidal neuron

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It is controversial whether somatic firing can be induced just with synaptic inputs to distal dendrite. Recent computational and experimental studies (Omori et al., 2006) revealed a spatial profile of dendritic membrane property in hippocampal CA1 pyramidal neurons; there is a steep decrease of membrane resistance between distal and proximal dendrites and distal dendrite is leakier than proximal one. To clarify effect of the non-uniformity on signal transfer from distal dendrite to soma, we analyzed cable equation and performed simulations of compartment model assuming non-uniform membrane property. Results by simulations show that anterograde signal transfer is enhanced due to the non-uniformity; somatic firing is induced by distal inputs in non-uniform model whereas no somatic firing is induced with the same inputs in uniform model. Our results suggest that non-uniform membrane property has functional role to improve dendritic signal transfer and to promote somatic firing just with inputs to distal dendrite.

KAKENHI(06J06774,18020007,18079003,18700299)

## O1P-E08 A simple model can predict the firing times of a cortical neuron

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Since Hodgkin and Huxley described the ionic flux across the neuronal membrane with nonlinear differential equations, continuous efforts have been made to construct realistic models by including more ionic channels in the model. These models can reproduce some qualitative aspect of electrophysiological phenomena. However, those models have rarely been applied to quantitative reproduction as well as prediction of spike times. The difficulty of quantitative reproduction would be due to the complexity of the model accompanied with a large number of undetermined free parameters. In this contribution, we propose a simple model that efficiently predicts spike times of biological neurons. We also categorize neurons in terms of the model parameters that determine the spiking characteristics.

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## O1P-E09 Trans-endocytosis of CD47 and SHPS-1 and its molecular mechanism

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CD47 and SHPS-1 are transmembrane proteins, both of which are highly expressed in the brain. The extracellular region of CD47 interacts with that of SHPS-1 and these two proteins constitute a cell-cell communication system, CD47-SHPS-1 system. We have here found that the trans-interaction of CD47 with SHPS-1, which occurred upon a contact of CD47-expressing cells with SHPS-1-expressing cells, resulted in endocytosis of such ligand-receptor complex into either types of cells (trans-endocytosis). The trans-endocytosis of CD47 with SHPS-1 was mediated by clathrin as well as dynamin. The trans-endocytosis is also regulated by either Rac or Cdc42 through actin cytoskeleton reorganization. The trans-endocytosis of CD47 indeed occurred from cultured hippocampal neurons to neighboring SHPS-1-expressing cells. The trans-endocytosis of CD47 and SHPS-1 may participate in removal of both protein complex from neuronal cell surface and hence regulates the function of CD47-SHPS-1 system in the brain.

KAKENHI16500236

## O1P-E10 Importin-$\alpha$ and $\beta$ expression in the neonatal and adult mouse brain

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Importin-$\alpha$ and $\beta$ mediate nucleocytoplasmic translocation of macromolecules across the nuclear pore complex. While the functional differences of importins in the neural cells remain unclear, the expression features should be quite an important issue to elucidate these functions. Recent reports showed importin-$\alpha$ and $\beta$ except for importin-$\alpha$2 were expressed in the brain, although the detail expression profile of each gene has not yet been investigated. We performed *in situ* hybridization to clarify the distributions of mouse importin-$\alpha$s and $\beta$ in the central nervous system of neonatal and adult mice. Importins mRNAs were expressed throughout the brain and spinal cord, whereas imporitn-$\alpha$2 mRNA was expressed only in olfactory bulbs, locus ceruleus and cranial nerve nuclei. In addition, importin-$\alpha$2 and $\beta$ mRNAs were expressed in the external granule layer of neonatal cerebellar cortex. Ubiquitous expression of importins suggests their housekeeping properties, while discrete expression of importin-$\alpha$2 implies a particular function in the central nervous system.

## O1P-E11 The functional analysis of TRPM1 in retinal bipolar cells

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Retinal bipolar cells convey neural signals from photoreceptors to amacrine and ganglion cells. TRPM1, one of the transient receptor potential (TRP) family members, was identified as a prognostic marker for metastasis of localized melanoma. However, its channel activity and biological function have been poorly understood. We cloned a TRPM1 long form containing an ion transporter domain, and we detected TRPM1 expression specifically in retinal bipolar cells. Interestingly, TRPM1 KO mice lacked electrophysiological activities under both photopic and scotopic conditions, although they retained intact photoreceptor activities under the same conditions. Our data indicate a pivotal role of TRPM1 in electrophysiological function of retinal bipolar cells.

18390092, 18022044, 18590090

## O1P-E12 Lateral mobility of voltage sensor domain of bacterial sodium channels

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Proximity between voltage sensor and pore domains of bacterial Na channels (NaChBac) was examined by introducing paired cysteines on various residues in extracellular part of S4 and S5 segments. *E. coli* expression test showed that T110C/M164C mutant most efficiently makes intersubunit disulfide bonds. When expressed in HEK cells, the mutant was electrophysiologically inactive but resumed normal activity upon bath application of DTT. Single mutation of M164C (S5) showed neither a disulfide bond nor current reduction. To our surprise, unlike those of double mutants, the current recordings however showed that T110C (S4) single mutant was likely to make disulfide bonds together in the channel tetramer. These results were also confirmed by another strategy using $Zn^{2+}$ chelation between histidine residues introduced at the same site as above. Our results suggest that the voltage sensor domains of NaChBac should be much more mobile laterally than that has been suggested in other voltage-gated ion channels.

## O1P-E13 Synchronization of excitatory neurons with strongly heterogeneous phase response

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Synchronous firing of cortical neurons is considered to play an active role in cognitive functions of the brain. Synchronizing dynamics depends not only on the neuronal network structures but on the cellular intrinsic properties. Phase response curve (PRC) is an intrinsic property of neurons, and it describes how a timing shift in the next output spike depends on the timing of the input stimulus and has crucial information about synchronization. Our in vitro study demonstrated that the PRC shapes of cortical neurons are heterogeneous. Little is known about the dynamics of the neurons with heterogeneous PRC. We investigated the dynamics of such populations through the globally coupling with the excitatory synaptic interaction. We found that in hard-to-synchronizing neurons, small heterogeneity of PRC can destroy the frequency synchronization, while in the easy-to-synchronization neurons, heterogeneity drives the perfectly synchronizing state into the partially synchronizing state.

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## O1P-E14 Nefiracetam potentiates NMDA receptor function via protein kinase C activation

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Nicotinic acetylcholine receptors and NMDA receptors are known to be down-regulated in the brain of Alzheimer's disease patients. We have previously demonstrated that the nootropic drug nefiracetam potentiates the activity of both nicotinic acetylcholine and NMDA receptors and that nefiracetam modulates the glycine binding site of the NMDA receptor. We studied their roles in nefiracetam action on the NMDA receptor by the whole-cell patch-clamp technique and immunoblotting analysis using rat cortical or hippocampal neurons in primary culture. The nefiracetam potentiation of NMDA currents was inhibited by the PKC inhibitor chelerythrine, but not by the PKA inhibitor H-89. In immunoblotting analysis, nefiracetam treatment increased the PKCalpha activity with a bell-shaped dose-response relationship peaking at 10 nM, thereby increasing phosphorylation of PKC substrate and NMDA receptor. Such an increase in PKCalpha-mediated phosphorylation was prevented by chelerythine.

## O1P-E15 Computational studies of activity propagations through feed-forward networks of burst neurons

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Feed-forward networks of neurons, synfire-chains, are a well-studied circuit to propagate spikes. Recent experiments showed neurons in the high vocal centre of songbirds exhibited sequences of bursts while the bird was singing. The result indicates the neural circuit of the bird has a feed-forward network that propagates spike bursts. While the propagation of singlet spikes has been studied, that of bursts remains unknown. Here, we studied the propagation of bursts in a feed-forward network of intrinsic bursting neurons and found that the burst propagation has novel properties that was not present in the single-spike propagation: (i) bursts propagate with a speed that depends on the bursting mode, i.e., the number of spikes in each burst (ii) temporal structures of spikes within a burst show complex evolution during propagation. Our results indicate a possibility that intrinsic properties of cells may play an active role to increase computational abilities of sequence generation in the brain.

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## O1P-E16 Adenosine uptake *via* P2X7 receptors

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We reported that P2X7 receptors (P2X7Rs) were activated in mouse astrocytes under not only ligand-stimulated but also resting conditions. It was reported that $NAD^+$ can prevent PARP-1 activation-mediated cell death by adding extracellularly, but its molecular mechanism has not been understood yet. Since it was suggested that $NAD^+$ can get into cells as an intact form, we postulated a possibility that P2X7R-forming pore might act as its pathway. Uptake of adenine ring [$^{14}$C]-labeled, but not nicotinamide [$^3$H]-labeled, $NAD^+$ was time- and temperature-dependent, and enhanced by apyrase and decreased by ecto-nucleotidase inhibitors and P2X7R down-regulation, indicating that $NAD^+$ was metabolized extracellularly and then taken up into the cells *via* P2X7Rs. In fact, genetic and pharmacological P2X7R inhibition resulted in decrease of [$^3$H]adenosine uptake. Collectively, it was suggested that exogenous $NAD^+$ was extensively metabolized and adenosine as its metabolite was incorporated into the astrocytes *via* P2X7Rs. This is the first evidence that P2X7R is the pathway for adenosine uptake.

## O1P-F01 Kspot, a novel ubiquitin ligase, regulates synaptic activity

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We identified a novel ubiquitin ligase (E3), Kspot as a target molecule of a high titer autoantibody produced in the serum of a patient with stomach cancer, associated with psychiatric symptoms. Kspot is highly conserved throughout species and expressed in the mammalian nervous system. In this study we demonstrated that Kspot formed an SCF complex and mediated ubiquitination of cellular proteins. Furthermore, overexpression of Kspot in primary cultured rat hippocampal neurons resulted in a decrease in the miniature excitatory postsynaptic current frequency. We propose that Kspot plays an important role in the regulation of the neurotransmitter release at the presynaptic site.

## O1P-F02 SUMOylation regulates kainate receptor-mediated synaptic transmission

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The small ubiquitin-like modifier protein (SUMO) regulates various cellular events inside the nucleus. The identification of SUMO substrates outside the nucleus is progressing but little is yet known about the neuronal role of protein SUMOylation. Here we report that in neurons multiple SUMOylation targets are present at synapses and we show that the kainate receptor subunit GluR6 exhibits low levels of SUMOylation under resting conditions and is rapidly SUMOylated in response to a kainate treatment. Reducing GluR6 SUMOylation using the SUMO-specific isopeptidase prevents kainate-evoked endocytosis of the kainate receptor. Furthermore, a mutated non-SUMOylatable form of GluR6 is not endocytosed in response to kainate. Consistent with this, recordings in hippocampal slices demonstrate that kainate receptor mediated EPSCs are diminished by SUMOylation and augmented by deSUMOylation. These data reveal a previously unsuspected role for SUMO in the regulation of synaptic function.

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## O1P-F03 IQ-ArfGEF/BRAG1 is a guanine nucleotide exchange factor for Arf6 that localizes and interacts with PSD-95 at excitatory synapses.

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ADP ribosylation factor 6 (Arf6) is a small GTPase that regulates dendritic and spine formation. In this study, we characterized a novel guanine nucleotide exchange factor for Arf6, termed IQ-ArfGEF/BRAG1. *In situ* hybrization analysis showed its predominant expression in the forebrain and cerebellar granule cells. Interestingly, IQ-ArfGEF/BRAG1 mRNA exhibited the somatodendritic localization in the hippocampus. Immunohistochemical analysis showed its colocalization with PSD-95 at excitatory synapses. Furthermore, immunoprecipitation revealed its coprecipitaion with PSD-95 family proteins and NMDA receptors. All these findings indicate that IQ-ArfGEF/BRAG1 is a GEF for Arf6 that interacts and localizes with PSD-95 at excitatory synapses.

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## O1P-F04 Cloning and functional analysis of Pikachurin: a novel Agrin/Perlecan-related protein localized at the active zone of retinal photoreceptor ribbon synapse

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Mechanisms of active zone formation have been poorly understood. Retinal photoreceptors have active zone structure, known as "synaptic ribbon". We newly cloned a novel gene, "*pikachurin*", encoding an extracellular matrix-like protein which shows homology with Agrin & Perlecan. Pikachurin was detected at the active zone of photoreceptor ribbon synapse by immunostaining. EM analysis on *Pikachurin* KO mice revealed that the number of invaginated dendrites of rod bipolar cells in mutant mice was significantly reduced. Furthermore, electroretinogram (ERG) showed that the implicit time of b-wave was significantly prolonged. Our results revealed that *pikachurin* plays an important role in retinal photoreceptor ribbon synapse formation.

KAKENHI(18390092, 18022044)

## O1P-F05 Developmental change in the calcium channel subtypes involved in the excitatory transmission and D1-like receptor-mediated presynaptic inhibition in the rat basal forebrain cholinergic neurons

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A whole-cell patch-clamp study was carried out using brain slice preparations of the rats (P21-38) to elucidate the developmental changes in calcium channel subtypes involved in non-NMDA glutamatergic transmission onto cholinergic neurons in the basal forebrain (BF) and D1-like receptor-mediated presynaptic inhibition. Non-NMDA EPSCs were evoked by focal stimulation. The EPSC fraction reduced by ω-CgTX (3 μM), an N-type calcium channel blocker, decreased with age, whereas that reduced by ω-Aga-TK (200 nM), a P/Q-type channel blocker, increased. Inhibition of EPSCs by SKF 81297, a D1-like receptor agonist, also increased with age in parallel with the increase in ω-Aga-TK-induced inhibition. SKF 81297 showed no further inhibition of EPSCs after the effect of ω-Aga-TK had reached steady state throughout development. These results suggest the functional link between presynaptic D1-like receptors and P/Q type channels for the glutamate release throughout development.

KAKENHI (17500281)

## O1P-F06 Presynaptic and postsynaptic inhibition by $GABA_B$ receptor in fusiform cells in mice dorsal cochlear nucleus

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Fusiform cells (FCs) in mammalian dorsal cochlear nucleus (DCN) integrate auditory and somatosensory information. FCs receive excitatory inputs from parallel fibers (PFs), which convey the latter information and inhibitory inputs from the glycinergic and GABAergic interneurons innervated by PFs. However, the significance of the difference in the inhibitory transmission is still unclear. Therefore, to investigate the role of GABA, we tested the effect of baclofen, a $GABA_B$ receptor agonist, on FCs using patch clamp recording. We found that baclofen induced membrane hyperpolarization accompanied by reduction of input resistance, which was due to activation of GIRK current. Baclofen also reduced the EPSC amplitude evoked by PFs stimulation and increased the paired pulse ratio. These results indicate that $GABA_B$ receptor contribute to presynaptic and postsynaptic inhibition. These results suggest that GABA in the DCN has distinct role in the regulation of the neural excitability, whereas glycine acts only on postsynaptic glycine receptors, contributing fast inhibitory synaptic transmission.

17023027

## O1P-F07 Development of pyramidal cell inhibition in visual cortex

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Inhibition of layer 2/3 pyramidal cells is driven by two distinct pathways, via an integration of input signals in the somatodendritic domain of inhibitory cells (conventional inhibition) and via direct activation of inhibitory terminals by output signals of nearby pyramidal cells (inter-pyramidal inhibition). We studied development of these inhibitions using dual whole-cell recordings from nearby layer 2/3 cells of rat visual cortical slices. Inter-pyramidal IPSCs were detected less frequently at postnatal 14-17 (early) than 21-25 days (late stage), although there was no difference in amplitude. IPSCs from fast-spiking (FS) to pyramidal cells were detected more frequently in the early than late stages. Initially, FS-pyramidal IPSCs were similarly small in both connections with and without return excitatory inputs. Later, the inhibitory connection remained with an increased strength only in reciprocally connected pairs. These distinct developmental changes suggest that each inhibition contributes to different aspects of cortical functions and their development.

KAKENHI and CREST

## O1P-F08 A role for laminin in postsynaptic differentiation at the neuromuscular junction

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Laminin $\alpha 4$, $\alpha 5$, $\beta 2$ subunits are specifically concentrated at the neuromuscular junction. $\beta 2$ laminins are muscle-derived organizers of presynaptic differentiation that act by binding to calcium channels in nerve terminals (Nishimune, Nature, 2004). To assess roles of laminin $\alpha$ subunits at the synaptic cleft, we analyzed mice with a null mutation of the $\alpha 4$ gene (Patton, Nat Neurosci., 2001), conditional mutants selectively lacking $\alpha 5$ from muscle, and double mutants (DKOs). Topological maturation of the acetylcholine receptor clusters (AChR) from plaque- to pretzel-shaped was delayed in $\alpha 5$ mutants and arrested in DKOs. Wild type myotubes cultured aneurally show topological maturation of AChR, but myotubes from DKO did not. Levels of dystroglycan immunoreactivity in the postsynaptic membrane were markedly reduced in DKOs. Our results suggest that postsynaptic differentiation of NMJ involve autocrine signalling of laminin $\alpha 5$ to dystroglycan.

INBRE, NINDS/NIH

## O1P-F09 Analysis of synaptic transmissions by UV and IR laser photolysis

Hiroshi Kojima[1], Tomohiko Maruo[2], Shiori Katsumata[3], Satoshi Tojoh[1], Youko Nakazato[1], Minoru Tsukada[1], Shigeo Okabe[2], Ko Sakai[3]

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Photolysis by laser beam focused on neurons were performed to stimulate multiple locations of cell dendrites. The details of the photolysis system have been already reported. Stimulations were delivered to the dendrites of cerebellar Purkinje cells of Rat slice preparations and mouse cultured pyramidal cells. Confocal images were taken by 548 Green-HeNe laser and electrical signals were recorded from the cellbodies by pipettes filled with the solution containing Alexa 568. Inward currents responses obtained from the cell somata by laser photolysis were comparable with the mEPSCs. Fine areas which correspond to spines were activated by photolysis and the functional and morphological changes upon these stimulations were investigated. Furthermore, we tried to find possible parameters, which would induce synaptic plasticity, of a kinetic model of AMPAR channels by combining the present results with a computer analysis.

## O1P-F10 Presynaptic NMDA receptors regulates synaptic transmission by inhibiting voltage-gated calcium channel at a central glutamatergic synapse

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At the calyx of Held, excitatory transmitter L-glutamate inhibits transmitter release. Both mGluR (Takahashi *et al*, 1996) and AMPAR (Takago *et al*, 2005) in the nerve terminal activate heterotrimeric G proteins, thereby inhibiting presynaptic $Ca^{2+}$ currents ($I_{pCa}$). Here we report that inhibition of $I_{pCa}$ by glutamate can additionally be mediated by NMDARs. NMDA inhibited $I_{pCa}$ and the NMDAR blocker D-AP5 or 7-ClK attenuated this effect. Further, intraterminal loading of MK-801 abolished the NMDA-induced $I_{pCa}$ inhibition. As a result, NMDA decreased AMPAR-mediated EPSCs. Intraterminal GTPγS did not affect the inhibitory effect of NMDA on $I_{pCa}$. Consistently, in the presence of intraterminal GTPγS, L-glutamate inhibited $I_{pCa}$, with its effect being attenuated by D-AP5 and 7-ClK. These results suggest that caliceal nerve terminal provides both G protein-dependent and -independent mechanisms for $I_{pCa}$ inhibition, and that the latter is coupled with presynaptic NMDARs.

## O1P-F11 Developmental changes in calmodulin-dependent presynaptic calcium current inactivation at a calyx-type synapse

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At the calyx of Held of immature rats, $Ca^{2+}$/calmodulin (CaM)-dependent inactivation of presynaptic $Ca^{2+}$ currents (IpCa) is proposed to be a major cause of synaptic depression during low frequency stimulation. We examined whether this mechanism might operate throughout postnatal development. At postnatal days (P) 7-9, IpCa showed paired-pulse inactivation (PPI) peaked at 0.5 s interval, which was attenuated by CaM inhibitors. A CaM inhibitor also attenuated synaptic depression at 0.5-5 Hz. However, at P13-15, the CaM-dependent PPI of IpCa became undetectable, and the CaM inhibitor had no effect on synaptic depression. At P7-8, shrinkage of $Ca^{2+}$ domain by intra-terminal application of 10 mM EGTA abolished the PPI of IpCa. At P14-15, CaM-immunoreactivity was similar to that at P8, and a high-frequency stimulation could still induce CaM-dependent IpCa inactivation. Thus the CaM-dependent IpCa inactivation and its contribution to synaptic depression decline with development, as a result of presynaptic $Ca^{2+}$ domain reformation at the calyx of Held.

## O1P-F12 The role of Arcadlin isoforms in synaptic remodeling

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We previously reported that homophilic interaction of rat Arcadlin (Acad), an orthologue of human and mouse protocadherin8, activates p38 MAPK via a splicing form of TAO2 (TAO2 $\beta$), and reduces spine numbers. *arcadlin* comprises three coding exons, and lacks a 294-base pair exon encoding the cytoplasmic region. We henceforth describe a splicing variant that has this exon as Acad-L, which has a 98 aa-longer cytoplasmic domain than that of Acad. Cells expressing Acad-L demonstrated a stronger trans-adhesiveness than Acad. While both Acad isoforms biochemically interacted with TAO2 $\beta$, FRET studies indicated that only Acad interacted with the TAO2 $\beta$. Homophilic interaction of Acad triggered endocytosis of Acad with TAO2 $\beta$, whereas homotypic association of Acad-L did not induce any internalization. As *arcadlin* knockout increased spine numbers, we are testing which isoform could rescue this phenotype. Thus, Arcadlin isoforms may differentially regulate synaptic remodeling by causing distinct cellular adhesiveness.

## O1P-F13 Regulation of Arc localization at synapses via interaction with CaMKII β

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The neuronal immediate early gene *Arc* is dynamically regulated by synaptic activity and tightly linked to information processing in neuronal circuits. Recent studies have indicated that *Arc* plays roles in synaptic plasticity/homeostasis and long-term memory formation. Arc is present in the postsynaptic density of activated neurons. However, how activity induces Arc and how such activity can also target Arc to the synapse have remained unclear. Here, we found an unexpected role of CaMKII in regulating Arc localization at synapses. The β subunit of CaMKII (CaMKII β) was identified as an Arc-interacting protein. Direct binding was confirmed by an *in vitro* assay using purified recombinant proteins. Consistently, the level of Arc accumulation in spines was decreased by RNAi knockdown of CaMKII β, but not of CaMKII α, and this effect was rescued by an RNAi-resistant CaMKII β. These results indicate that CaMKII β may function as an anchoring protein for Arc in dendritic spines.

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## O1P-F14 Characterization of Cbln1 binding to the PSD fraction of the cerebellum: implication for specific binding proteins for Cbln1 in Purkinje cells

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Cbln1 is a member of the C1q family to which adiponectin and tumor necrosis factor α belongs. Cbln1 is secreted from cerebellar granule cells (GC) and controls synaptic structure and plasticity of GC-Purkinje cell (PC) synapses. Recently, we have shown that recombinant Cbln1 proteins prepared from transfected mammalian cells form a hexamer, which preferentially binds to the PC spines in both primary culture and acute slice preparations. Here, we show that recombinant Cbln1 was also capable of binding to the synaptosomal fraction prepared from adult cerebellum *in vitro*. Similar specific binding was observed in postsynaptic density (PSD) fraction. In contrast, mutant Cbln1 that did not form a hexamer did not bind to these fractions. These data indicate that specific binding proteins for Cbln1 are present in the PSD fraction of Purkinje cells that recognize hexameric Cbln1 proteins.

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## O1P-F15 Lrfn2/SALM1, a synaptic leucine-rich repeat-containing transmembrane protein regulates synapse formation and function

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We have been investigating the role of a neuronal LRR transmembrane family, Lrfn1-5, in vertebrate CNS. Here we focused on the synaptic function of Lrfn2/SALM1. Immunofluorescence staining and subcellular fractionation studies indicated that Lrfn2 is an excitatory postsynaptic protein. Lrfn2 co-precipitated PSD-95 and NR1 subunit of NMDA receptor in an immunoprecipitation assay using mouse brain lysates. Co-expression of Lrfn2 and PSD-95 in cultured hippocampal neurons resulted in the activation of the synaptic activity, while an Lrfn2 mutant lacking PSD-95-binding ability did not show such activation in the same assay. To explore the role of Lrfn2 in synaptic function in vivo, we generated Lrfn2 knockout mice. Hippocampal neurons from Lrfn2 KO mice showed a reduced clustering of both PSD-95 and NR1, suggesting that Lrfn2 is required for the proper formation of the excitatory synapses.

## O1P-F16 Neural mechanisms for calculation of the interaural intensity difference in the chicken auditory brainstem

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Each sensory nervous system transduces signal of specific modality, then processes and integrates them to extract several features in the course of transmission to the higher centers. Localization of sound source is made by utilizing two specific auditory cues; the interaural time difference (ITD) cue and the interaural intensity difference (IID) cue. I investigated how the IID was processed in the auditory brainstem utilizing anesthetized chicken. Intensity feature of sound was first extracted in the nucleus angularis (NA) and then the interaural difference is calculated in the posterior part of the dorsal lateral lemniscal nucleus (LLDp). However, the extraction of intensity feature in NA was not a simple process, because of the bilateral interaction generated by the interaural canal, through which bilateral middle ears are connected. I found that the effects of the interaural canal were dependent on the sound pressure level and the phase of bilateral sound signal. This bilateral interaction was found to enhance the sensitivity of IID calculation in LLDp.

KAKENHI(17.2283)

## O1P-G01 Imaging of AMPA receptor dynamics in behaviorally activated neurons

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Synaptic trafficking of AMPA-type glutamate receptors (AMPARs) has been demonstrated to play a key role in synaptic plasticity. However, many questions about AMPARs dynamics in vivo still remain unanswered. c-fos and other immediate-early genes are rapidly and transiently induced in response to neuronal activity and have been widely used as activity markers for mapping neurons involved in a variety of animal behaviors including learning and memory. In order to visualize and study the AMPAR dynamics in neurons activated by in vivo experiences, we have developed transgenic mice expressing GFP-GluR1 under the control of c-fos promoter and tetracycline inducible system. In the transgenic mice, activity-induced tetracycline regulated transactivator (tTA) initiates transcription of GFP-GluR1 in the absence of doxycycline. Using the transgenic mice, we examined the GFP-GluR1 dynamics in neurons that have been activated by fear conditioning to determine whether specific limited subsets of synapses are preferentially labeled and whether learning is accompanied by post-synaptic structural changes that are specific to activated neurons.

NIH

## O1P-G02 Hippocampal model for spatial learning by using reward related cells

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Significance of a place is often changed by the animal's own actions even in the identical environment. Therefore, the cognitive map should flexibly change according to such situations. In the rat hippocampus, in addition to the cells purely representing places, the cells representing places' significance are recently revealed. Here, we propose a hippocampal model for spatial learning which can quickly change the place's significance using these two cell-types. In our model, when the rat gets a reward, the place cells interact with nearest neighbors and the reward related cells fire various durations. Supposing a Hebb like rule between these two cell-types, the reward related cells are bound to the place cells and consequently have various sizes of place fields. The rat can arrive the rewarded site from any place by heading in the direction that makes the total reward related cells' activities larger. This model enables one-shot place learning and works well even if in the intricately shaped environment.

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## O1P-G03 A learning model of head-direction cells and grid cells by VQ layers connected via anti-Hebbian synapses

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In this paper, we proposed three layered self-organizing model to extract head direction and position of a moving object separetely.The model consists of three layers, each of which is a self-organizing vector quantization (VQ) model.The second layer receives inhibitory input from the first,and the thrid layer receives inhibitory input from the first and the second.The first layer is to detect head direction and the second and the third are to detect position.The information representation in the second and the third layers is shown to be multi-ary expression and the units in the third layer develop receptive field with grid structure as was observed in entorhinal cortex of a rat.

## O1P-G04 Dentate Gyrus theta phase precession learned by enthorhinal grid cells

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Space perception in rat is tightly related to the presence of hippocampal place cells firing at selective location in the environment; the so-called "place cells". Moreover, the dynamics of activity of these cells shows precise phase precession with the theta rhythm. Previous papers have already discussed the important role played by this mechanism in memory formation. Still, it was an open question to know if this characteristic neural correlate of behavioral activity was created inside the hippocampus or if it was already present in previous stage of the brain information processing.Recently, it was discovered that cells in the medial entorhinal cortex (MEC), having similar theta phase precession, fire when the rat is located at any of the vertices of grid fields covering the environment. We propose here a plausible computational model which demonstrates that hippocampal phase precession and place field firing rate can be learned by EC phase precession and grid fields. It enhances the important role played by EC phase precession to maintain temporal relationships between the various hippocampal areas.

## O1P-G05 Population coding of song element sequence in the songbird brain nucleus HVC

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Birdsong is a complex learned vocalization composed of various song elements organized according to sequential rules. Because of the importance of auditory feedback in song learning, numerous studies have investigated the auditory neural representation. In this study, we recorded the auditory responses to all possible element pair stimuli in the Bengalese finch brain nucleus HVC. Our results show that each neuron has broad but differential sequential response distribution properties. We calculated the time course of population activity vectors and mutual information between neural activities and stimuli. We found that the clusters of population vectors responding to second elements had a large overlap. Confounded information that represents the relationship between first and second song elements significantly increased at the same time. Thus, sequential information causes an overlap in the population response. These results indicate that the song element sequence is encoded in a neural ensemble in HVC via population coding.

KAKENHI(18700303)

## O1P-G06 Impact of dendritic branches on the function of an entire neuronal network

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Plenty of experimental and modeling studies have suggested that various kind of nonlinear operations in a dendrite of a single neuron could perform lots of computations. However, many studies about dendritic computations deal with only a single neuron, without considering its role in an entire neuronal network. We propose a computational function of arborescent dendrites of neurons in a network model of cortical short-term memory. Specifically, we show that branch-localized shunting inhibition provided by dendrite-targeting inhibitory neurons could naturally endow the network with several features such as input-intensity-invariant responses, which are more difficult to be realized in conventional models considering only somatic inhibition. We construct a model, in which network architecture and dendritic arborization of single neurons are described at once, and analyze it taking advantage of a kind of mean-field approximation under the assumption that each neuron has many dendritic branches.

## O1P-G07 Dynamical model of working memory system

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Temporary storage of information in working memory is one of the most important functions of the prefrontal cortex. In recent years both neurophysiologists and physicists are interested in its studying and modeling. There appeared an interesting approach based on the oscillatory origin of the working memory. Working memory is supposed to be stored in a network of pyramidal neurons with afterdepolarization. The network is stimulated by subthreshold sinusoidal signal, and its elements interact with the inhibitory feedback. Storing the memory in such the network is associated with its spatio-temporal activity. In the current work we develop a dynamical model of the oscillatory short-term memory system. We study collective dynamics of the network of neurons with afterdepolarization, sinusoidal stimulation and inhibitory feedback. We have shown that several images can be stored in this network in the form of clusters of periodical neural activity. These clusters fire in every cycle of the sinusoidal signal, and the inhibitory feedback provides temporal division of them, so that they fire sequentially one after another.

## O1P-G08 Spatial information of hippocampal pyramidal cells along the dendrosomatic axis of behaving rats

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The soma and dendrite are substrates for neuronal computation. However, the difference between the neuronal codes by the soma and dendrite in the brain of behaving animals remains unknown. Here, using a spike-sorting with an extension of independent component analysis (ICSort) together with 12-site wire-electrodes (dodecatrodes), we show that in the hippocampal CA1 of behaving rats, the soma and dendrite of pyramidal cells compute distinct spatial information. We examined spatial information coded by signals predominantly originating from the soma and dendrite of pyramidal cells during forging in a familiar open field. The results suggest that the somatic units showed lower firing rates, higher spatial specificity and lower sparsity than the dendritic units. Therefore, we conclude that a hippocampal pyramidal cell can sharpen redundant spatial information received from presynaptic neurons via the dendrite into more place-specific information along the dendrosomatic axis.

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## O1P-G09 Medial prefrontal cortex is involved in extinction learning for recent but not remote contextual fear

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Extinction learning for fear memory is thought to be a process underlying cognitive behavioral therapy for anxiety disorders such as post-traumatic stress disorder. The time-window of the treatment is one of the important factors in this therapy. It is proven that the medial prefrontal cortex (mPFC) plays a critical role in the extinction that is induced immediately after acquisition of fear memory. However, the participation of the mPFC in the extinction for 'remote' fear memory has not been reported. Fear-conditioned mice were repeatedly exposed to the context without unconditioned stimulus (electrical footshock), and the time they show a freezing response was measured. The freezing time was reduced by the repetitive exposure both in the case that the exposure was started from 1 (recent memory test) or 21 day (remote memory test) after conditioning. This reduction was prevented by intra-mPFC-injection of lidocaine, a local anesthetic, in the recent but not the remote memory test. These results suggest that the mPFC is scarcely involved in the extinction for remote contextual fear memory.

## O1P-G10 Perceptual ambiguity by luminance contrast and reward predictive activity in the brain

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To investigate neural mechanisms of reward prediction, previous studies have used perceptually distinct stimuli. In the present study, we examined influence of perceptual ambiguity on reward-predicting brain activity by manipulating luminance contrast of Gabor Patch stimulus with using fMRI. After a conditioning of left or right orientation of the Gabor Patch with juice reward, participants were presented with easy and difficult discrimination stimuli to predict the reward. We applied Rescorla-Wagner model with or without influence of perceptual ambiguity, and found significant correlation of activity in reward-related regions such as midbrain and putamen with the model influenced by perceptual ambiguity. Our results suggest that reward-related activity in dopaminergic areas is modulated by perceptual ambiguity.



## O1P-G11 Phasic responses of the pedunculopontine tegmental nucleus in primates

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The cholinergic pedunculopontine tegmental nucleus (PPTN) in the brainstem is thought to associated with reward-related behaviors by integrating polymodal signals related to the motivational state of animal, sensory information, motor control and reward. Since PPTN is one of the strongest excitatory input sources to the midbrain dopamine neurons, we hypothesized that PPTN may play an important role in reinforcement learning by relaying salient event and reward-related signals to dopamine neurons. we recorded various phasic responses of PPTN neurons in primates during visually guided saccade tasks, and examined the context dependency of these activities. About half of visual and movement-related activities were modulated by the behavioral and reward context. On the other hand, most of reward responses were independent of behavioral contexts and depended on actual reward volume.

KAKENHI(18020019)

## O1P-G12 Different groups of pedunculopontine tegmental nucleus neurons signal the expected and actual task reward

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Evidence has accumulated suggesting that midbrain dopamine neurons encode "reward prediction error" and that the basal ganglia use this signal to perform reinforcement learning. However, the question of how this reward prediction error is computed remains elusive. The pedunculopontine tegmental nucleus (PPTN) of the brain stem supplies strong excitatory inputs to the dopamine neurons, and we were interested in the possibility that PPTN supplies the information for this computation.This hypothesis was tested by studying the activity of PPTN neurons in monkeys performing a visually guided saccade task for a juice reward. We found two groups of PPTN neurons that were selectively responsive to the reward-cue and the reward delivery, respectively, the former encoding the magnitude of the expected reward and the latter that of the actual reward, which are the two essential pieces of information required to compute the reward prediction error.

KAKENHI(18020019)

## O1P-G13 Processing of infant-directed speech in parents: An fMRI study

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Adults address infants with typical modification of prosody and lexicon. Although these linguistic components are independent and considered to be lateralized in opposite hemispheres of the brain, both are functionally connected in terms of infant-directed speech (IDS). Using fMRI, we showed that two types of IDS shared neural processing during perception in gender-/experience-dependent manners. Unlike fathers and non-parents, mothers exhibited left-lateralized activity in language areas elicited by either IDS when compared with adult-directed speech. Further, perception of prosodic IDS activated speech-related motor areas as a function of personal extraversion scores in mothers, which suggests individual variation in mother's mirror-neuron system.

KAKENHI(18730478)

## O1P-G14 Effects of regularity, familiarity and congruity of stimuli on auditory and visually evoked magnetic fields

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Effects of Regularity, Familiarity, and Congruity of Stimuli were investigated on Auditory and Visually Evoked Magnetic Fields. Regularity was controlled by exponent n on a fluctuation of $1/f^n$, Familiarity was controlled by the score of word familiarity in a database of Lexical Properties of Japanese. Congruity was controlled by the semantic distance of the components of the constructed stimulus. Decrement of these three factors of stimuli enhanced the amplitude and prolonged the latency of the AEF activity of the N100m component of primary auditory aria. The Increment of the three factors of visual stimuli enhanced the VEF of the 150ms and 220ms components. The three factors of stimuli play similar roles for activity enhancement of the auditory and visual cortex, and provoked expectation of auditory stimuli and figure ground segmentation for visual stimuli. The activities of the cortex reflected the basic function of the conventionalization of the pattern of a repetitive experience unit in the process of the emergence of linguistic structure.

## O1P-G15 Selective impairment of syntactic processing in patients with brain tumor

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In spite of previous aphasic studies, the precise identification of agrammatism and responsible cortical regions remains unclear. In the present study using fMRI and picture-sentence matching tasks, we examined the performance of 14 right-handed patients with brain tumor in the left dorsal inferior frontal gyrus (L. dIFG), the right ventral IFG (R. vIFG), or other regions. Although all patients have no apparent deficits in communication, we found significant main effects of tumor location (L. dIFG & R. vIFG vs. others) and sentence type (scrambled vs. active & passive) in the performance, as well as their interaction. Task-related activations in the L. IFG were also observed among all patients with a R. vIFG tumor. These results suggest that selective deficits in syntactic processing are induced by the dysfunction of the L. dIFG or R. vIFG.

CREST, JST

## O1P-G16 Prefrontal activations predict future performance improvement in learning phonics rules of English orthography and phonology

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To clarify the neural basis of individual differences in rule learning associated with second language acquisition, we investigated brain activations in non-native English speakers (n = 48) using fMRI. Phonics rules were presented just before each of eight scanning sessions (training period), but not during subsequent eight sessions (testing period). In each session, a phonics task was performed to match a visually presented word with auditorily presented pronunciation of that word. Activations in the left dorsal inferior frontal gyrus (dF3t) during the training period were positively correlated with the performance improvement between these periods, but not with the performance during the training period. The high tertile, determined according to the signal changes of the left dF3t during the training period, indeed exhibited significantly higher performance than did the low tertile during the testing period. These results suggest that activation in the left dF3t is a good indicator of the degree of future performance improvement in learning phonics rules.

CREST, JST

## O1P-H01 Involvement of caspase activity in the pathfinding of olfactory sensory neurons

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Caspase was identified as cell death regulator. However, it is largely unknown where or when caspase is activated. Its significance is still unclear. We are studying caspase activity in the olfactory development. Axons of olfactory sensory neurons (OSN) expressing a given odorant receptor converge to a few glomeruli in the olfactory bulb (OB). This specificity arises during early development. We first examined the patterns of caspase activation at this stage. The anti-active caspase-3 antibody labeled a part of OSNs, especially in the anterior region of the epithelium. Caspase-3 was highly activated in the OSN axons that project to the anteromedial region of the OB. For the functional analysis of caspase activation, we next applied *apaf-1* deficient mice. The projection patterns of OSNs were examined by introducing *OR-IRES-tauLacZ* mutations into the *apaf-1* (-/-) background. The loss of caspase activity resulted in the disturbed patterns of OSN axon guidance to the bulb, suggesting that caspase affects the pathfinding of OSNs in vivo.

## O1P-H02 Involvement of oxidative stress and MAP kinases in NO-induced apoptosis in cultured astrocytes

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NO induces apoptosis-like cell death in astrocytes that is Bax-dependent, but caspase-independent. The present study characterized further the mechanism of NO-induced cytotoxicity in cultured astrocytes. The NO donor SNP caused the stimulation of MAP kinases (ERK, p38 MAP kinase, JNK) and the production of ROS. SNP-induced cell death was attenuated by MAP kinase inhibitors and the radical scavenger edaravone. SNP-induced ROS production was inhibited by edaravone and the ERK inhibitor U0126, but not the p38 MAP kinase inhibitor SB203580 and the JNK inhibitor SP600125. SNP-induced phosphorylation of three MAP kinases was inhibited by edaravone and U0126. These results suggest that only ERK may be involved partly in NO-induced ROS production, and that ROS play a key role in NO-induced MAP kinase signals which are responsible for apoptosis in cultured astrocytes.

## O1P-H03 Critical role of the active site cysteine in oxidative stress-induced aggregation of GAPDH

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Glyceraldehydes-3-phosphate dehydrogenase (GAPDH) is involved in neuronal cell death triggered by oxidative stress, and is locally deposited at lesions in various neurodegenerative diseases. Here we show that both in vitro and in vivo oxidative stresses induce aggregation of GAPDH through aberrant disulfide bonds between the active site cysteine. Under non-reducing conditions, oxidants induced the aggregation of rabbit GAPDH through formation of intermolecular disulfide bonds in vitro. To identify the specific cysteine, we generated four cysteine-substituted mutants, C149S (active site), C153S, C244A, and C281S. Treatment of C149S with NOR3 could not induce the aggregation. The aggregates had some characteristics of amyloid. Treatment of GAPDH-overexpressing HeLa cells to oxidants also produced disulfide bonding, amyloid-like aggregates of GAPDH in the particulate fractions, concomitant with cell death. Thus, the aggregation of GAPDH formed via the active site cysteine might play a significant role in the pathology of neurodegeneration.

## O1P-H04 Real time imaging study on injury-induced microglial activation in the hippocampal slice culture

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Mechanisms for microglial activation in two distinct brain injury models were studied by using a real time imaging technique in the organotypic hippocampal slice cultures prepared from Iba I-EGFP mice. When the slice cultures were injured by laser ablation, rapid extension of microglial processes to the injured site was observed. This extension was suppressed by an ATP-degrading enzyme apyrase or P2 purinergic receptor antagonists, indicating the critical role of endogenous ATP in this response. In another injury model, accumulation of microglial cells in the pyramidal cell layer was observed 4 days after NMDA-induced neuronal cell death. This accumulation was not abrogated by apyrase, suggesting the unessentialness of endogenous ATP.

## O1P-H05 Parkinsonism-causing toxins enhance gap junction in astrocytes

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It has been reported that altered gap junction (GJ) might result in compromised homeostasis and exacerbated pathologies of various neural disfunctions in the microenvironment of the central nervous system (CNS). Astrocytes represent a major non-neuronal cell population in the CNS and are strongly coupled through GJ. It has recently been shown that astrocytes modulate synaptic transmission. We then hypothesized that GJ status of astrocytes may exert serious influences on the integrated function of neuron network. The purpose of this study is to investigate whether complex I inhibitors influence GJ in cultured astrocytes, focusing on the role of GJ as a potent downstream target of complex I inhibitors as a parkinsonism-causing toxins. We found that complex I inhibitors treatment enhanced GJ being accompanied with increased total Cx43 protein levels in treatment for 48hr.

## O1P-H06 Neuronal injury induces the expression of multiple chemokines in astrocytes and microglia in organotypic cortico-striatal slice cultures

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Glial cells are rapidly activated under the pathological conditions such as cerebral ischemia and traumatic brain injury to produce various chemokines. Little is known about the mechanisms for regulating the chemokine production in the injured brain. We previously reported that NMDA-induced neuronal injury induced astrocytic MCP-1 production via the activation of a MEK/ERK pathway in the cortico-striatal slice culture. In this study, we investigated the effects of NMDA-induced neuronal injury on the expression of several chemokines. In addition to MCP-1 mRNA, NMDA induced MIP-1 $\alpha$ and CINC-1 mRNAs. Further experiments revealed that MIP-1 $\alpha$ was dominantly produced by microglia, and its production was partially inhibited by a JNK inhibitor, but not by a MEK inhibitor. These results suggest that NMDA-induced neuronal injury activates both astrocytes and microglia, but productions of a different set of chemokines are induced in each cell type via the distinct intracellular signaling pathway.



## O1P-H07 Zinc induces microglial activation

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Excitotoxic neuronal injury after ischemia can be induced by excess accumulation of free zinc after release from presynaptic glutamatergic terminals. Excitotoxic neuronal injury is also associated with inflammatory responses, driven by microglial activation. Poly(ADP-ribose) polymerase-1 (PARP-1) has been known to regulate microglial activation. Here we showed that, in primary microglial cultures and mouse brains, zinc induced apparent morphological changes, release of nitric oxide and poly(ADP-ribose) (PAR) formation. Zinc-induced microglial morphological changes and PAR formation were blocked by the genetic and pharmacologic PARP-1 or NADPH oxidase inhibition. Furthermore, microglial activation following transient global ischemia was prevented by intracerebroventricular injection of an extracellular zinc chelator, CaEDTA. These results suggest that zinc can induce microglial activation, mediated by PARP-1 activation through NADPH oxidase pathway, which may cause excitotoxic neuronal injury after ischemia.

## O1P-H08 Implication that TRPV4 is involved in the communication between astrocytes and neurons

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We have reported that a themosensitive-TRP channel, TRPV4 (activated by heat, >34 °C) is expressed in hippocampal neurons, and enhances the neural activities through its activation at body temperature (Shibasaki et al., J. Neurosci. 27, 2007). TRPV4 was also observed in only subsets of astrocytes, indicating that we can classify the characteristics of astrocytes depending on TRPV4 expression. Functional-TRPV4 expression was confirmed in cultured astrocytes by a $Ca^{2+}$-imaging. Following the increased calcium level only in the subsets of astrocytes, significant calcium oscillations were observed in among most of the astrocytes, indicating that communications among astrocytes were initiated through the TRPV4 activations. We hypothesized that the calcium oscillations among astrocytes might regulate the neural activities. Therefore, we examined how astrocytes communicate each other and how the astrocytes affect neural excitation by a biosensor method to detect the neurotransmitter released from astrocytes.

The Uehara Memorial Foundation

## O1P-H09 The regulatory mechanism of neuronal damage by endothelial PGE2 via astrocytes

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Prostaglandin (PG) $E_2$ has been implicated in the $Ca^{2+}$-dependent glutamate release in astrocytes. We found $PGE_2$ synthase, mPGES-1 was induced in endothelial cells with $PGE_2$ elevation by kainate (KA) seizure. We investigated the effect of mPGES-1 on astrocytic $Ca^{2+}$ levels, and neuronal damage by KA. Following the KA injection to hippocampus, $PGE_2$ level was increased and CA3 neurons were completely lost. The $PGE_2$ elevation was blocked and neuronal loss was decreased in the *mPGES-1* knockout mice (*mPGES-1(-/-)*). Astrocytic $Ca^{2+}$ imaging of hippocampal slice cultures revealed that the $Ca^{2+}$ levels after KA treatment were decreased, and neuronal damage was improved in mPGES-1 (-/-) slice. It suggests that mPGES-1 accelerates hippocampal neuron damage by $Ca^{2+}$-dependent glutamate release from astrocyte.

## O1P-H10 Transport characteristics for nicotinamide in astrocytes

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DNA damage after oxidative stress induces activation of poly(ADP-ribose)polymerase-1 (PARP-1), which contributes to cell death including astrocytes through $NAD^+$ depletion as well as genomic integrity. Interestingly, nicotinamide, an essential precursor of $NAD^+$ and also a PARP inhibitor, prevents PARP-1-mediated cell death, but the mechanisms by which nicotinamide is taken up into cells are obscured. Therefore, in this study, we examined characteristics of nicotinamide uptake into primary cultured mouse astrocytes. Nicotinamide uptake showed time-, concentration- and pH dependency, and was inhibited and stimulated by co-administration and preload, respectively, of N-methylnicotinamide, a substrate of organic cation transporter, but not by other substrate/inhibitors. Furthermore, representative PARP inhibitors had no effect on the uptake. These results demonstrate that nicotinamide uptake is mediated by a $H^+$-organic cation exchanger, which is different from well-known organic cation transporters, and the system dose not accept other PARP-inhibitors as substrates.

## O1P-H11 Restoration of FcRγ/Fyn signaling repairs central nervous system demyelination

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Our previous finding indicating the critical involvement of the γ chain of immunogloblin Fc receptors (FcRγ) and Fyn signaling in oligodendrocyte differentiaion and myelination demands a fundamental revision of the strategies used for multiple sclerosis therapy, because antigen-antibody complexes may induce the direct dysregulation of myelination process as well as the inflammatory destruction of myelin sheath. Here we show that the FcRγ/Fyn signaling cascade is crucially involved in cuprizone-induced demyelination/ remyelination. The critical importance of the phosphorylated myelin basic proteins (pMBPs), especially the 21.5 kDa isoform, was shown by the analysis of the aged, cuprizone-treated, and FcRγ/Fyn double knockout mice. The herbal medicine Ninjin-yoeito repaired the aging- and cuprizone-induced demyelination by restoring the FcRγ/Fyn-Rho(Rac1)-MAPK(P38MAPK)-pMBPs signaling cascade which represents a new approach for the treatment of demyelinating diseases.

## O1P-H12 A novel function of leukemia inhibitory factor in myelination

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We have previously demonstrated that leukemia inhibitory factor (LIF) promotes myelination via an activity dependent mechanism from axons using an in vitro culture system. In order to better understand the role of LIF in myelination, we examined myelin formation during postnatal development in the optic nerve of LIF knockout (ko) mice. Using immunohistochemistry, we found that LIF-ko mice had a delay in myelination. MBP-positive myelin was shown throughout the optic nerve in wild-type mice at postnatal day 10, however, it was observed only at the chiasmal region in LIF-ko mice of the same age. Interestingly, the composition of glial cells is dramatically altered in LIF-ko mice. The number of Olig2-positive cells decreased compared with wild-type mice and these cells were observed only at the chiasmal region in LIF-ko mice. These results suggest that LIF has a crucial role in myelination in the optic nerve during development.

## O1P-H13 UDP, a novel mediator of microglial phagocytosis

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Microglia engage in the clearance of dead cells, which is a crucial event in maintaining brain functions. We previously showed that ATP triggers a dynamic change in the motility of microglia to cause chemotaxis, while microglial phagocytosis has received only limited attention. Here we show that microglia express the metabotropic $P2Y_6$ receptor, the activation of which by endogenous agonist UDP triggers microglial phagocytosis. UDP facilitated the uptake of microspheres in a $P2Y_6$ receptor-dependent fashion, which was mimicked by endogenous UDP that was leaked when hippocampal neurons were damaged by kainic acid. The $P2Y_6$ receptor would function as a sensor for phagocytosis by sensing diffusible UDP signals, which is a novel pathophysiological function of P2 receptors in microglia.

KAKENHI, The Uehara Memorial Foundation

## O1P-H14 Inhibition of synaptic transmission precedes synaptic stripping in vagal motoneurons after axotomy

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Following axotomy, microglia adhere to injured motoneurons, and spread on their surface to disconnect synapses. This phenomenon is known as synaptic stripping. We examined whether changes in synaptic inputs after axotomy are correlated with synaptic stripping in the dorsal motoneurons of vagas (DMV). At 2 days after axotomy, there was a significant decrease in frequencies of both spontaneous EPSCs and IPSCs recorded from injured DMV neurons of rats. At this stage, synaptic stripping was not evident and high-$K^+$ stimulation restored frequencies. These results suggest that the inhibition of synaptic transmission precedes synaptic stripping. ATP was found to be potential candidates that inhibit the glutamate release, because PPADS, a P2 receptor antagonist, restored spontaneous EPSCs frequency recorded from injured DMV neurons.

## O1P-J01 Gene transfer of hepatocyte growth factor (HGF) attenuates behavioral and neuropathological abnormalities in a transgenic mouse model of Huntington's disease

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Huntington's disease (HD) is a neurodegenerative disorder caused by expansion of abnormal polyglutamine repeats in huntingtin. Transgenic R6/2 mice, which overexpress exon1 of the human mutant huntingtin gene, progress behavioral and neuropathological abnormalities that resemble phenotypes of HD patients. Here, we show that gene transfer of HGF by injecting viral vector that carries rodent HGF into the striatum at 4 weeks of age reduced neuronal loss in the striatum of R6/2 mice compared to the control vector that carries LacZ. Importantly, HGF improved motor function and neuropathological abnormalities associated with HD. HGF prevented the activation of caspase-1, -3 and -9 in the striatum of R6/2 mice. Our findings raise the possibility of the therapeutic intervention of HGF in the patients with HD and related disorders.

## O1P-J02 High-throughput RNAi screening for modifiers of mutant Huntingtin aggregation in *Drosophila melanogaster*

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The fruitfly *Drosophila melanogaster* is well established as a model system in the study of human neurodegenerative diseases. Utilising RNAi, we have carried out a high-throughput screen for modifiers of aggregate formation in stable *Drosophila* cells expressing N-terminal truncated huntingtin (tNhtt) fused to EGFP with an expanded (62Q) polyglutamine repeat . 7200 genes, around 50% of the *Drosophila* genome, were screened, resulting in the identification of 514 candidates that either suppress or enhance aggregation. Our list of candidates was intelligently pruned to remove candidates with many off-target effects (OTEs) or those that may non-specifically affect transgene expression. We have established *in vivo* models of HD by expressing polyQ-EGFP in the *Drosophila* compound eye and nervous system. We carried out a secondary screen *in vivo*. Most candidates identified have mammalian orthologues, validating the use of *Drosophila* to screen for genes relevant to human disease.

JSPS Post-doctoral Fellowship

## O1P-J03 The Rab5 activator ALS2/alsin acts as a novel Rac1 effector via Rac1-activated macropinocytosis

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Mutations in the *ALS2* gene account for a number of recessive motor neuron diseases (MNDs). ALS2 is a guanine nucleotide exchange factor for Rab5 (Rab5GEF) and involved in endosome dynamics. However, the spatio-temporal regulation of the ALS2-mediated Rab5 activation remains unknown. Here we identified an upstream activator for ALS2, and showed a functional significance of the ALS2 activation in endosome dynamics. ALS2 preferentially interacts with GTP-bound activated Rac1. In the cells, activated Rac1 recruits cytoplasmic ALS2 to membrane ruffles, and subsequently to nascent macropinosomes via macropinocytosis. Macropinosomal ALS2 augments fusion of the ALS2-localized macropinosomes with the transferrin-positive endosomes by its Rab5GEF activity. Thus, ALS2 is a novel Rac1 effector and involved in an unfamiliar endocytic mechanism called macropinocytosis. All together, it is conceivable that a perturbation of macropinocytosis and/or the following membrane trafficking caused by loss of ALS2 underlie neuronal dysfunction in the ALS2-linked MNDs.

## O1P-J04 Mutant ALADIN responsible for triple A syndrome impairs nuclear import of aprataxin, a DNA repair protein causative for ataxia.

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Triple A syndrome is an autosomal recessive neuroendocrinological disease, caused by mutations in the ALADIN gene. ALADIN is a component of nuclear pore complex (NPC). We here report a patient who had adult-onset motor neuron disease-like phenotype with a novel mutation in this gene (I482S). I482S failed to target NPC. In fibroblasts (I482Sf) from this patient, nuclear accumulations of aprataxin (APTX), a DNA repair protein causative for ataxia, and of DNA ligase I, another repair protein, were impaired. Cell survival assay showed hypersensitivity of I482Sf to a glutathione depleting agent that creates oxidative stress. This agent increased DNA damage in I482Sf. I482Sf transfected with both APTX and ligase I had increased resistance to oxidative stress. We propose that oxidative stress damaged DNA beyond the limited capacity of decreased nuclear DNA repair proteins, triggering cell death.



## O1P-J05 Analysis of Slitrk1- and Slitrk2-deficient mice

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The Slitrk family consists of six type I transmembrane proteins, which are predominantly expressed in the nervous system. In the extracellular region, they share conserved leucine-rich repeat domains with marked homology to the secreted axonal guidance molecule Slit. Each Slitrk protein except Slitrk1 also shows homology to Ntrk neurotrophin receptors in their intracellular domains, sharing a conserved tyrosine residue. In cultured neuronal cells, overexpression of Slitrk1 induces unipolar neurites, whereas overexpression of Slitrk2 inhibits neurite outgrowth. Mutations in Slitrk1 gene are found in the patients of Tourette's syndrome and Trichotillomania. We developed Slitrk1 and Slitrk2 knockout mice and analyzed their phenotypes. In behavioral analysis, Slitrk1 null mice show hypoactivity and increased anxiety-like behavior, whereas Slitrk2 null mice show hyperactivity. The linkage between molecular properties of Slitrks and behavioral abnormalities of their knockout mice will be beneficial for further understanding of the neuronal circuit development.

KAKENHI(18700330)

## O1P-J06 Altered expression of proteins in the brain of mouse model for Down syndrome

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Down syndrome (DS) is the most common genetic disorder associated with mental retardation and is caused by trisomy for human chromosome 21. Although some pathological changes have been shown in the brains of patients and animal models with DS, biochemical changes of specific proteins are poorly understood. Therefore, we utilized a proteomics approach to identify proteins that are differentially expressed in the brain of Ts[Rb(12.1716)]2Cje (Rb2Cje), a mouse model for DS. The proteins extracted from the whole brain of Rb2Cje (n=5) and wild type (n=4) mice at 3-month-age were analyzed by two-dimensional differential in-gel electrophoresis coupled with ESI-LC-MS/MS analysis, and we identified five proteins differentially expressed in Rb2Cje. These five proteins are known to play important roles in oxidative stress or cell death. These results support earlier reports that oxidative damage might play an important role in the abnormal phenotypes observed in DS.

## O1P-J07 Proteome analysis of Wig rat brain: a model for attention-deficit hyperactivity disorder (ADHD) research

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Wiggling-Wistar King Aptekman/Hokkaido rats (Wig) were designated from a strain of the Long-Evans Cinnamon as a new rat model of attention-deficit hyperactivity disorder (ADHD), an extremely common brain developmental disorder. To elucidate the mechanism of behavioral abnormalities in Wig rats, we investigated proteomes of the frontal cortex, striatum, and midbrain by two-dimensional (2-D) gel electrophoresis and mass spectrometry (MS). On 2-D gels, 42 silver nitrate stained protein spots were differentially expressed, out of which 24 non-redundant proteins were identified by MS. Changed proteins were functionally categorized into energy metabolism, cell communication and signal transduction, cellular transport, etc. Interestingly, some proteins were previously reported to be involved in neurodegenerative disorders, which will be discussed in relation to ADHD.

## O1P-J08 Genetic and epigenetic analyses in monozygotic twins with Rett syndrome with extreme differences in early development

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Rett syndrome (RTT) is an X-linked disorder with mental retardation in females caused by MECP2 mutations. We report 3.5-year-old, monozygotic twin girls with RTT and noticeable differences in development. To know the etiology of the clinical differences, we performed several genetic and epigenomic analyses. The twins shared a frame-shift mutation (1-bp deletion) in MECP2. No difference of the X-chromosome inactivation patterns was found. Since MeCP2-binding sites may alter the ability of MeCP2 to repress transcription, resulting in the clinical differences, we studied methylation status in the putative MeCP2-binding sites in BDNF and DLX6 loci. However, no remarkable differences were found between the twins. Since existence of epigenomic differences has recently been reported in monozygotic twins, differential methylation may exist in unidentified MeCP2-binding sites.

## O1P-J09 Glomerular map formation and odor perception in the zone1- and class II-depleted mutant mice

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The glomerular map in the olfactory bulb (OB) is divided into functionally distinct domains based on the nature of odorous ligands. We generated a transgenic mouse, in which the OSNs expressing the class II ORs are specifically tagged with a GFP reporter, and found that class I OSNs (GFP-negative) project their axons to the most antero-dorsal domain in the OB. Using intrinsic signal imaging, we found that the glomerular cluster for class I OSNs corresponds to the functional domain that responds to the aliphatic acids and aliphatic aldehydes. We also generated mutant mice in which OSNs, either of zone1 or those expressing class II ORs, are specifically depleted from the OE. In the zone1-depleted mouse, the dorsal domain in the OB was devoid of targeting OSN axons and devoid of glomeruli. In contrast, in the class II-depleted mouse, glomeruli were detected only in the most antero-dorsal region of the OB, where class I glomeruli are normally found in the wild-type mouse.

## O1P-J10 An ENU-induced mouse mutant with ADHD-related behavioral abnormality carries a missense mutation in the intracellular C0 domain of NR1

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We isolated an ENU-induced mouse mutant (M-174) with ADHD related behavioral abnormalities. Linkage analysis and direct sequencing of positional candidate genes revealed that M-174 has a missense mutation in intracellular C0 domain of NR1. [$^3$H]MK-801 saturation binding assays revealed slight reduction of NMDAR binding and affinity in M-174. In primary culture of cortical neuron, maximum calcium influx and duration at half-maximal (T1/2max) after 10 μM NMDA stimulation were significantly enhanced and prolonged in M-174. M-174 also showed hypersensitivity to MK-801. Normal regional histology was observed in Nissl stain of the major brain regions obtained from M-174. These results suggest the missense mutation in NR1 altered NMDAR function and affected the behavior.

## O1P-J11 Impaired pattern separation of hippocampal place representation in the dentate gyrus-specific NMDA receptor mutant mice

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NMDAR has high permeability to Ca2+ but is blocked by Mg2+ in a voltage-dependent manner. This property is a molecular basis of NMDAR-dependent LTP, which is thought to play a central role in learning and memory. We have generated the genetically engineered mice in which mutated NMDAR defecting in Mg2+ binding ability are expressed specifically in the dentate granule cells. The mutation decreased LTP threshold at the PP-GC synapse. To elucidate the effect of the mutation on the information processing in the hippocampus, we recorded the place cell activity from CA1 cells. Robust place-specific discharge is found in mutant CA1 cells, and no significant difference in firing propertiies between mutant and control mice. However, in the mutant mice, the receptive fields are relatively insensitive to environmental differences. The result suggests that the mutant have difficulty in pattern separation, which is proposed as a function of the dentate gyrus.

## O1P-J12 Autistic-like phenotypes in CADPS2/CAPS2 knockout mice and aberrant CADPS2 splicing in autistic patients

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CADPS2/CAPS2 mediates the exocytosis of dense-core vesicles, and the human CADPS2 is located within the autism susceptibility locus 1 on chromosome 7q. Here, we show that CADPS2 knockout mice not only have impaired BDNF release but also show autistic-like cellular and behavioral phenotypes. Moreover, we found an aberrant alternatively spliced CADPS2 mRNA that lacks exon 3 in some autistic patients. Exon 3 was shown to encode the dynactin 1-binding domain and affect axonal CADPS2 protein distribution. Our results suggest that a disturbance in CADPS2-mediated neurotrophin release contributes to autism susceptibility.

RIKEN, KAKENHI(17700322)

## O1P-J13 Serum phospholipids and glucose level in rats with tolerance to heat stress

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It has been reported that serum triglyceride level is decreased by acute heat stress. The inbred FOK rat, with congenital tolerance to heat stress, also shows remarkably low triglyceride level even at normal ambient temperature. In the present study, serum phospholipids (PL) and glucose level were determined in male FOK rats kept at an ambient temperature of 25-26°C in comparison with control strain, the WKAH rat. FOK rats showed normal glucose level at an age of 3 and 18 months. FOK rats at age of 18 months were rather fat than the both strains at age of 3 months. Serum PL in FOK rats at an age of 3 months was significantly higher than those in WKAH rats. Fatty acid composition in serum PL in FOK rats showed higher stearic acid and docosahexanenoic acid (DHA), and lower linoleic acid than those in the WKAH rats. The increased polyunsaturated fatty acid (n-3) may have a role in resistance to heat stress.

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## O1P-J14 PPAR $\alpha$ is a potential therapeutic drug target of circadian rhythm sleep disorders

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The endogenous clock in the suprachiasmatic nucleus (SCN) controls various physiological and behavioral circadian rhythms in mammals. Here we demonstrate that bezafibrate, a hypolipidemic drug, advances the phase of circadian behavior of mice independently of the SCN. The circadian expression of clock genes was also phase advanced in tissues (cortex, liver, and fat) without affecting the SCN. Bezafibrate also phase advanced the *Clock* gene-mutated delayed sleep phase syndrome (DSPS) model mice. Our results indicated that PPAR $\alpha$ is involved in circadian clock control independently of the SCN and that PPAR $\alpha$ could be a potent target of drugs to treat circadian rhythm sleep disorders including DSPS.

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## O1P-K01 Ras activation of Rac1 exchange factor Tiam1 mediates neurotrophin-3-induced Schwann cell migration

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Endogenous neurotrophins positively and negatively regulate migration of premyelinating Schwann cells before the initiation of myelination. Neurotrophinp-3 (NT3) acting through the TrkC receptor tyrosine kinase stimulates Schwann cell migration through the Rho GTPases Rac1 and Cdc42. We previously demonstrated that the TrkC kinase phosphorylates and activates Dbs, the guanine-nucleotide exchange factor (GEF) for Cdc42, to mediate Schwann cell migration. Here we identify T-lymphoma invasion and metastasis (Tiam) 1 as the Rac1-specific GEF involved in NT3-induced Schwann cell migration. Furthermore, the interaction between a small GTPase Ras and Tiam1 plays an essential role in the activation of Rac1. Taken together, these results suggest that NT3 activation of TrkC stimulates Schwann cell migration through the two parallel signaling units, Ras/Tiam1/Rac1 and Dbs/Cdc42, and that the former is uniquely regulated by two different types of small GTPases.

## O1P-K02 Tyrosine-phosphorylation of Tiam1 mediates BDNF-induced neurite outgrowth in cortical neurons

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Small GTPases of the Rho family play key roles in the formation of neuronal axons and dendrites by transducing signals from guidance cues, such as neurotrophins, to the actin cytoskeleton. However, there is little insight into the mechanism by which neurotrophins regulate Rho GTPases. Here we show the crucial role of the ubiquitous Rac1-specific guanine-nucleotide exchange factor (GEF), Tiam1, in transducing neurotrophin-mediated neurite outgrowth. We demonstrated that the brain-derived neurotrophic factor (BDNF), acting through the TrkB receptor tyrosine kinase, directly binds and specifically activates Tiam1 by phosphorylating Tyr-829, leading to Rac1 activation and neurite outgrowth of cortical neurons. A point mutation in Tiam1, Tyr-829-to-Phe-829, blocked BDNF-induced changes in number of neurites of cortical neurons. The findings are evidence of a novel mechanism for the activation of Tiam1 and of a role for this effector in neurotrophin-mediated signal transduction leading to neurite outgrowth.



## O1P-K03 Regulation by CD47-SHPS-1 system of neurite development

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Both CD47 and SHPS-1 are transmembrane proteins as well as members of immunoglobulin superfamily proteins, and are highly expressed in the brain. The extracellular region of CD47 interacts with that of SHPS-1 and these two proteins constitute a cell-cell communication system, CD47-SHPS-1 system. We have now investigated the role of CD47-SHPS-1 system with hippocampal neurons. CD47-deficient neurons manifested impaired development of dendrites and axons, whereas overexpression of CD47 promoted such development. Interaction of SHPS-1 with CD47 also induced the formation of dendritic filopodia and spines. These effects of CD47 were prevented by inhibition of small GTPases of the Rho family, Cdc42 or Rac, or Src family protein tyrosine kinases. Our present results suggest that CD47, together with SHPS-1, promotes development of dendrites and axons in hippocampal neurons in a manner dependent, at least in part, on activation of Cdc42 and Rac mediated by Src.

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## O1P-K04 Activation of STAT3 by pituitary adenylate cyclase-activating polypeptide(PACAP)during PACAP-promoted neuritogenesis of PC12 cells

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PACAP is a 38-amino-acid neuropeptide that was first isolated from ovine hypothalamic extracts on the basis of its ability to stimulate cAMP formation in anterior pituitary cells. Addition of PACAP into the cultured PC12 cells promoted neurite outgrowth of the cells. Using DNA macroarray techniques, we have characterized the PC12 cell transcriptome, revealing that several genes were regulated by PACAP. Among many, we focused to investigate whether STAT3 molecule might be involved in PACAP signaling. PACAP increased gene expression of STAT3, at least up to 24 h, being maximum 9.5 fold with 3-h treatment of 1 nM PACAP. ELISA revealed that PACAP increased the amount of STAT3 proteins about 30%. Phospho-STAT3 Tyr705 was increased in the nuclei by the neuropeptide. During the activation of STAT3, IL-6 was also secreted by PACAP in dose dependent manner. Treatment with IL-6-neutralizing antibody significantly diminished PACAP-activated STAT3 promoter activity. Together, our findings have demonstrated that STAT3 is involved in PACAP signaling and function.

## O1P-K05 Synaptic ubiquitin-specific protease (synUSP) is transported to nucleus and regulates p65/RelA stability

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It is well known that the ubiquitin-mediated proteolysis of Ikappa B regulates nuclear factor-kappaB (NF-kappaB) signaling. Recently, presence of another, but a similar regulation mechanism has been suggested by the report in which the ubiquitination of p65/RelA, one of the NF-kappaB subunit, was mediated by E2 ubiquitin-conjugating enzyme, ubcH5alpha, and E3 ubiquitin ligase, SOSC-1 (Ryo et al., Mol. Cell, 12:1413-1426). However, the detailed mechanisms of the regulation through the p65/RelA ubiquitination remain unknown. Here we propose a novel mechanism of regulation of NF-kappaB signaling through the action of synaptic ubiquitin-specific protease (synUSP). The synUSP, also known as USP48 or USP31, is a de-ubiquitinating enzyme which we found in 2003 (Tian et al., J. Neurochem., 87: 665-675). We demonstrated that synUSP was transported to the nucleus and increased the stability of p65/RelA. Nuclear translocation of USP48 is blocked by deletion of its nuclear localization signal (NLS), which suggested a role of classic nuclear transport pathway.

## O1P-K06 Involvement of ERK cascades in the anterior cingulate cortex in pain-related negative emotion in rat

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Activation of ERK in dorsal horn neurons of the spinal cord by peripheral noxious stimulation contributes to both short-term and long-term pain hypersensitivity. Mounting evidence indicates that the ACC is involved in pain emotion. Our study is to investigate the activation of ERK in the ACC and its contribution to pain-related negative emotion using formalin-induced conditioned place avoidance animal model. Intraplantar injection of 5% formalin induced a rapid and sustained ERK activation in ACC neurons. Intra-ACC microinjection of MEK inhibitor PD098059 or U0126 could significantly block the formation of F-CPA. Incubation of ACC slices with NMDA , Forskolin or Sp-cAMP produced significant activation of ERK, which was suppressed by preincubation with their respective antagonists APV , SQ22536 and Rp-cAMP. Similarly, F-CPA was blocked by premicroinjection of SQ22536 or Rp-cAMP. Taken together, NMDAR-dependent AC-PKA-ERK signal transduction may play an important role in pain-related negative emotion.

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## O1P-K07 Fyn protein tyrosine kinase as a target for antipsychotic drugs

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Antipsychotics exert their effect through the blockade of dopamine D2 receptor (D2-R), but the signal cascade triggered by D2-R blockade remains unclear. Recently, we have reported Fyn tyrosine kinase mediates that cascade. In the control mice, D2-R blockers induced (1) a behavioral response, catalepsy, (2) Fyn activation and enhanced tyrosine phosphorylation of a NMDA-R subunit in the striatum, and (3) increased NMDA-R channel activity of striatal neurons, but these responses were significantly reduced in Fyn-deficient mice. Thus, Fyn mediates intracellular signaling between D2-R and NMDA-R in the striatum which mediates a behavioral response. Further proteomics analysis on antipsychotics-induced tyrosine phosphorylation and spatiotemporal analysis of Fyn activation are now in progress. Our present findings strongly suggest that Fyn links the signaling downstream of D2-R, and can be a new target for antipsychotic drugs.

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## O1P-K08 Transcriptomics of unilateral irradiated and contralateral rat striata after gamma knife surgery

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Cobalt 60 sourced gamma irradiation (60 Gy) was targeted to unilateral striatum, which was examined at the genomic level using oligo DNA microarray (rat whole genome 44K, Agilent) along with unirradiated contralateral striata and controls, 16 h post irradiation. A total of 56/19 genes were up-/down-regulated in unilateral striata, compared to 163/51 up-/down-regulated genes in contralateral striata. In the irradiated striata, interaction with the environment and metabolism categories were highly-induced, whereas signal transduction and transcription were highly induced in the contralaterla striatum. Results were validated by RT-PCR and provide a snapshot of gene expression changes caused by gamma irradiation, and reveal bilateral effects caused by unilateral irradiation to the striatum.

## O1P-K09 Dual phase prostanoid production during excitotoxic hippocampal injury: A comprehensive lipidomics analysis

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Kainate (KA)-induced seizure involves eicosanoid production, but their mechanism and functions are poorly understood. We profiled the eicosanoid production by a comprehensive lipidomics analysis. Systemic KA administration caused biphasic production, which consisted of an initial burst in the first 30 min and a sustained late phase production. The initial phase was specific to the hippocampus and was blocked by intracerebroventricular administration of KA receptor antagonists. A selective cyclooxygenase (COX)-2 inhibitor, NS398, completely inhibited the initial phase productions, except for prostaglandin (PG)D2 and thromboxane B2 whose productions were also dependent on COX-1. These results suggest that KA signals directly stimulate the arachidonic acid cascade in the initial phase, and that COX-1 and COX-2, both constitutively expressed at low levels, differentially contribute to PG productions.

## O1P-K10 Involvement of prostaglandin $E_2$ EP3 receptors in stroke-reperfusion injury

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We have recently demonstrated that microsomal prostaglandin E synthase-1 (mPGES-1) is a critical factor of stroke-reperfusion injury. In this study, we tried to identify the downstream effectors of mPGES-1 neurotoxicity using in vitro and in vivo models. Excitotoxicity induced by glutamate in CA1 neurons of cultured hippocampal slice was significantly attenuated and augmented by EP3 receptor antagonist and agonist, respectively. Antagonists or agonists of EP1, 2 and 4 receptor had no effect on glutamate toxicity. EP3 antagonist also reduced not only brain infarction, neuronal apoptosis and edema but also the behavioral neurological dysfunctions induced by 2-hr of middle cerebral artery occlusion and 24-hr of reperfusion in wild-type mice, but not in mPGES-1 knockout mice. EP3 antagonist had no effect on mean arterial pressure and changes of cerebral blood flow. These results suggest that EP3 receptor inhibition may be a valuable therapeutic option in ischemic stroke.

## O1P-K11 Subclinical alteration of motor related magnetic field in patients with cerebral vascular occlusive disease.

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Cerebral vascular occlusive disease is an established risk factor for ischemic stroke. However, little is known about its affection on brain function in those patients without stroke. In Using synthetic aperture magnetometry, we evaluated spatial distribution and intensity of event-related desynchronization of beta band ( $\beta$-ERD) during hand motor task in 27 patients with cerebral vascular occlusive disease due to atherosclerosis (AS group; n=19) and other etiologies (n=8), and that of 8 age-matched control subjects. Contrary to the normal activation, enhanced ipsilateral $\beta$-ERD was detected significantly frequent in AS patients during grasping task using the contralesional hand. This abnormal ipsilateral activation showed significant relations to higher grade of periventricular hyperintensity or sulcal atrophy, etiology, and age (over 65). Ipsilateral activation would reflect the subclinical functional alteration due to atherosclerotic vascular occlusive disease.

## O2P-A01 FGF8-induced semaphorin 3F regulates growth polarity of midbrain dopaminergic axons

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Organizing centers governing the dorsoventral (DV) polarization of the neural tube, the roof plate and floor plate, play crucial roles in axon guidance along the DV axis. However, the role of organizing centers governing the rostrocaudal (RC) polarization in axon guidance along the RC axis remains unknown. Here, we show that rostrally directed growth of axons from midbrain dopaminergic neurons (mDANs) is under the control of fibroblast growth factor (FGF) 8, an organizing molecule expressed in the midbrain/hindbrain boundary. FGF8-coated beads placed in the diencephalons repelled mDAN axons and induced a chemorepellent, *semaphorin 3F* (*sema3F*). Moreover, outgrowth of mDAN axons was inhibited by Sema3F. mDAN axons expressed the Sema3F receptor, Neuropilin-2 (Npn-2), and the removal of *npn-2* by gene targeting caused caudal growth of mDAN axons. These results indicate that FGF8 induces Sema3F that, in turn, regulates growth polarity of mDAN axons.

## O2P-A02 U-shaped development of corticospinal innervation

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We showed that the early elimination of once established corticospinal (CS) innervation is followed by "second wave" of reinnervation. In this study we examined development of CS innervation by single axon tracing *in vivo* and optical imaging of synaptic activity in *in vitro* acute slices of the spinal cord. Single neuron was anterogradely labeled with biotin dextran. Spatial distribution of EPSP evoked by stimulation of dorsal funiculus was optically imaged with voltage-sensitive dye (optEPSP). The total number of branches of single neurons in the spinal gray decreased from P7 to P10, but again increased at P18, where not only higher but also lower order branch number increased. CS axons did not branch into more spinal segments at P18 than at P10. The optEPSP in the ventral area peaked at P6-7 once reduced at P10 but increased again at P15. The optEPSP in the dorsal area unchanged from P6 to P10 but increased at P15. Our results suggest U-shaped time course in development of CS innervation and that its later part, "second wave" is mainly due to branching of CS axons within the spinal gray, which is accompanied by synapse formation.

## O2P-A03 GluRε2(2B) dependent synapse elimination of corticospinal tract in mice

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To explore the mechanisms of NMDA-dependent synapse elimination of corticospinal (CS) synapses in vitro, which we showed in rat, we studied it using C57 BL/6 mice. CS synapses were formed diffusely throughout the spinal gray matter at 7-8 DIV; but until 13 DIV those in the ventral side were eliminated, which was blocked by a GluRε2-specific blocker, ifenprodil. To test whether the responsible site for GluRε2 dependence is the cortex (Cx) or spinal cord (SC), two types of heterotypic co-cultures using the slices from GluRε2 knockout (KO) and wild type mice (WT) were prepared. fEPSP amplitudes and optically-imaged EPSP with voltage-sensitive dye reduced in the ventral side at 13 DIV in Cx-KO/ SC-WT pairs but not in Cx-WT/ SC-KO pairs. Anterograde labeling of axon terminals with biocytin or the single neuron imaging with EYFP showed that the axon terminals in the ventral area were eliminated until 13 DIV in Cx-KO/ SC-WT pairs but not in Cx-WT/ SC-KO pairs. These result suggest that the GluRε2 in the SC is responsible for the synapse elimination.



## O2P-A04 A role of Disable-1 as a transcription factor in Reelin signaling

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Reelin controls neuronal migration and positioning in the developing brain by inducing tyrosine phosphorylation of an intracellular adaptor protein, Disabled-1 (Dab1). We clarified that Dab1 tyrosine phosphorylation initiated the internalization of Reelin, and that Dab1 dissociated from Reelin receptors after their internalization. Furthermore, we found that Reelin induced the nuclear import of tyrosine-phosphorylated Dab1. In the nucleus, Dab1 bound to DNA and regulated gene expression in cooperation with a transcriptional co-activator to control neuronal migration. These results suggest that Reelin regulates correct neuronal migration and positioning by inducing the nuclear import of tyrosine-phosphorylated Dab1.

KAKENNHI (18780222)

## O2P-A05 The somatodendritic targeting of DNER requires transcytosis

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Establishment of neuronal polarity depends on targeting of proteins into different plasma membrane domains, axons and dendrites. It has been shown that many dendritic proteins reach the somatodendritic membrane based on selective sorting and transport of carrier vesicles. Using cultured hippocampal nerurons, we examined the trafficking pathways of DNER, a transmembrane protein specifically expressed in CNS dendrites. Mutations of DNER that increased surface expression disrupted its somatodendritic localization. Inhibition of endocytosis resulted in disruption of polarized distribution of DNER, indicating that somatodendritic targeting of DNER was dependent on endocytosis. DNER binds to a clathrin adaptor AP-2 via multiple sites. However, clathrin-dependent endocytosis only partly mediated the somatdendritic targeting of DNER. Instead, a clathrin independent endocytosis was also responsible for its somatodendritic distribution. Thus, selective retention mediated by clathrin-dependent- and independent- endocytosis is critical for polarized targeting of a somatodendritic protein.

## O2P-A06 Microtubule dynamics regulating migration of cerebellar granule cells

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During neuronal migration in the developing brain, it is thought that the centrosome precedes the nucleus and provides a cue for nuclear migration along the microtubules. In time-lapse imaging studies of radially migrating granule cells in mouse cerebellar slices, we observed that the movements of the nucleus and centrosome appeared to occur independently of each other. The nucleus often migrated ahead of the centrosome during its saltatory movement, negating the supposed role of the centrosome in pulling the nucleus. We observed the perinuclear structure consisting of dynamic and stable microtubules, neither of which was converged at the centrosome. In addition, stable microtubules showed the polarized distribution in contrast with the overall distribution of dynamic microtubules. Disruption or excess formation of stable microtubules attenuated nuclear migration, suggesting that polarized arrangement of microtubules is prerequisite for nuclear migration.

## O2P-A07 Functional analysis of mouse Mahya genes

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We identified one of the Urbilaterian genes, *Mahya*, which is specifically conserved between Hymenoptera and Deuterostome. It has been implicated in the higher order of brain functions enabling social behavior and cognitive ability. *Mahya* encodes a secretory protein with a follistatin-like domain, two immunoglobulin domains, and a C-terminal novel domain. Mouse *Mahya* genes (*mMahya-1* and *mMahya-2*) are expressed in the olfactory bulb, hippocampus (CA3 region and dentate gyrus), and cerebellum (granule cells and Purkinje cells) of the adult brain. To understand the functions of *mMahya* genes in the brain functions we have generated KO mice for *mMahya-1* and *mMahya-2* in collaboration with the RIKEN CDB group. Both of homozygous KO mice are viable, and thus we are analyzing the structural changes in the brain areas and behaviors of these KO mice.

## O2P-B01 The signaling pathways involved in dendritic spine formation via Shc, phospho-tyrosine adaptor molecule

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Shc binds to phospho-tyrosine residues of the activated receptors. In central nervous system, N-Shc (neural Shc) is predominantly expressed and transmits neurotrophin signals from the TrkB receptor to Ras/MAPK pathway. Studies on the N-Shc knock out mice reveals a novel function of N-shc; N-Shc is also involved in the regulation of NMDA receptor functions and hippocampal synaptic plasticity. Since N-Shc also interacts with key regulators for actin-cytoskeletal rearrangements such as Crk and Grit (RICS), we then tested whether N-Shc is involved the formation of synaptic architecture, dendritic spines. Following to exogenously overexpression of N-Shc in primary cultured hippocampal neurons, the spine number was significantly reduced, and the effect was more severe when N-Shc/Crk cascade was disturbed. However, additive inhibition of N-Shc/Ras/MAPK cascade rescued the spine formation. Those results indicate that both N-Shc/Crk and N-Shc/Ras/MAPK pathways cooperatively regulate dendritic spine formation.

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## O2P-B02 Roles of monoacylglycerol lipase in the regulation of endocannabinoid signaling

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Endogenous cannabinoids (eCBs) are released from postsynaptic neurons and retrogradely suppress synaptic transmission by acting on presynaptic CB1 cannabinoid receptors. 2-Arachidonoylglycerol (2-AG), a major molecule of eCBs, is degraded by monoacylglycerol lipase (MGL), which is expressed at presynaptic terminals. In this study, we investigated how MGL regulates eCB signaling at hippocampal inhibitory synapses. Inhibition of MGL resulted in a gradual suppression of basal IPSCs. This suppression was blocked by a CB1 antagonist and attenuated by inhibiting 2-AG synthesis, suggesting that 2-AG is constitutively released and degraded by MGL. MGL inhibition also prolonged the recovery of IPSCs from the suppression by exogenous 2-AG application or by eCB released by depolarization. These results indicate that presynaptic MGL plays an important role in both regulating basal eCB tone and terminating the retrograde eCB signal.

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## O2P-B03 Strength of GABAergic transmission influences climbing fiber synapse elimination during cerebellar development

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We studied whether GABAergic tone influences developmental elimination of redundant climbing fiber (CF) to Purkinje cell (PC) synapses in the cerebellum. CF synapse elimination after postnatal day 11 (P11) was impaired in glutamic acid decarboxylase 67 (GAD67)-GFP knock-in mice that are heterozygous for the GAD67 mutation (GAD67$^{+/GFP}$). Local application of a GAD inhibitor to the wild-type cerebellum by means of Elvax caused similar impairment when the Elvax was implanted on P10. Normal CF innervation was restored in GAD67$^{+/GFP}$ mice by enhancing $GABA_A$ receptor sensitivity with intraperioneal injection of diazepam from P10 to P15. These results suggest that strength of GABAergic transmission to PCs greatly influences CF synapse elimination during the second postnatal week.

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## O2P-B04 Increase in GAD2 expression in the periolivary region of the mouse following cochlear ablation

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Periolivary neurons are located around the superior olivary complex and are implicated in the sound localization and signal detection in noisy condition. Here we conducted in situ hybridization with digoxigenin-labeled probes in the adult mouse and examined the changes of GAD1, GAD2 and glycine transporter type 2 (GlyT2) expression in auditory brainstem regions following unilateral cochlear ablation. We found a significant increase in GAD2 expression in the ipsilateral periolivary neurons 7 days following cochlear ablation. This is in marked contrast to GAD1 and GlyT2 in these neurons that were expressed at normal levels. We noted a decrease of more than one inhibitory transmitter following cochlear ablation as follows: (1) a decrease in GAD1 and GAD2 expression in the contralateral inferior colliculus and (2) a decrease in GAD1 and GlyT2 expression in the contralateral ventral nucleus of the lateral lemniscus. These decreases may reflect a unilateral reduction of excitatory auditory inputs into the brainstem regions.

## O2P-B05 The δ2 glutamate receptor does not function as an ion channel to regulate cerebellar long-term depression

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The δ2 glutamate receptor (GluRδ2) is crucial for cerebellar long-term depression (LTD), a synaptic plasticity model responsible for motor learning. Although a mutation occurring in *lurcher* mice causes constitutive channel activity of GluRδ2 (GluRδ2$^{Lc}$), which is similar to that of $Ca^{2+}$-permeable AMPA/kainate receptors, whether wild-type GluRδ2 functions as a channel has remained elusive. To address this issue, we introduced a mutant *GluRδ2*, in which the putative channel pore was disrupted, into *GluRδ2*-null Purkinje cells by using Sindbis virus vector. Surprisingly, a mutant GluRδ2, as well as a wild-type GluRδ2, rescued abrogated LTD in *GluRδ2*-null mice. Furthermore, 1-naphtylacetylspermine, a potent blocker of GluRδ2$^{Lc}$ and $Ca^{2+}$-permeable AMPA/kainate receptors, failed to block LTD in wild-type cerebellar slices. These results indicate that GluRδ2, a member of the ionotropic glutamate receptor family, does not serve as an ion channel to regulate LTD induction.

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## O2P-B06 Ultrastructural analysis of Purkinje cell spines reveals a highly asymmetrical organization of the PSD and ER, having profound influence on molecular signal processing

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The role of the synaptic spine as a semi-secluded bioreactor has long been established. Yet, spines are often modeled as simple homogenous "reaction vessel", with the PSD located at the apex. We show here that both assumptions are invalid. We reconstructed . Membranes of >250 Purkinje cell spines, shafts and organelles therein were traced from serial 90nm EM sections. Selected spines were imaged using EM tomography to analyze the sER/PSD interaction. The PSD was systematically located laterally on the spine head rather than at the apex, irrespective of the spine shape and incidence angle of the parallel fiber. The functional consequences of this specific cytoarchitecture was evaluated using realistic reaction-diffusion simulation of $IP_3$ signaling, in reconstructed and synthetic spine morphologies. We show that the relative position of sER and PSD drastically influence the efficacy of molecular signaling in the spine.

## O2P-B07 Regulation of actin cytoskeleton is involved in activity-dependent traffickings of AMPA-receptors in cerebellar Purkinje cell

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We examined roles of actin dynamics in constitutive and activity-dependent traffickings of AMPA-type glutamate receptors (AMPA-Rs) in dendritic spine synapses between parallel fibers (PF) and Purkinje cells. Using whole-cell recording methods, we found that jasplakinolide (Jas, 1 μM), a blocker of actin-depolymerization, suppressed, while latrunculin A (Lat, 10 μM), a blocker of actin-polymerization, enhanced long-term depression (LTD) of PF-EPSC in rat cerebellar slices. Image-analysis of cultured Purkinje cell demonstrated internalization of GluR2, caused by glutamate (40μM) and KCl (40mM) stimulation, which was blocked by Jas but enhanced by Lat. Also, toxin B, a blocker of monomeric GTP binding proteins, Rho, Rac and Cdc42, inhibited LTD, whereas exoenzyme C3, a specific Rho blocker, had no such effect. We suggest that Rac and/or Cdc42 is required for regulation of actin dynamics underlying LTD induction in Purkinje cell.

## O2P-B08 Molecular mechanism of inhibitory synaptic plasticity in the cerebellum

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At the GABAergic synapses between inhibitory interneurons and a Purkinje neuron in the cerebellum, postsynaptic depolarization induces long-term potentiation of transmission efficacy mediated by $GABA_A$ receptors (rebound potentiation: RP). Here we show that $GABA_A$ receptor-associated protein (GABARAP), which binds both to $GABA_A$ receptor γ2 subunits and to tubulin, plays a critical role in RP. Disturbance of GABARAP association with $GABA_A$ receptor γ2 subunits suppressed the induction and maintenance of RP. Expression of mutant GABARAP lacking tubulin binding region inhibited the RP induction. Further, fluorescence resonance energy transfer (FRET) analysis revealed that GABARAP undergoes sustained conformation change depending on CaMKII activity. The susceptability to undergo sustained conformation change was abolished by single amino acid replacement of GABARAP, which resulted in impairment of the RP induction. Taken together, binding of $GABA_A$ receptor γ2 subunit with structually altered GABARAP underlies the expression of RP.

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## O2P-B09 Further evidence for the distinction between LTP and RISE, a long-lasting synaptic plasticity accompanied by synaptogenesis.

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Using stable cultures of rat hippocampal slices, we previously reported that repeated inductions, but not a single induction, of LTP produced a long-lasting (≥3w) enhancement of synaptic strength accompanied by a new synapse formation. We call this new plastic phenomenon RISE (Repetitive-LTP Induced Synaptic Enhancement) to discriminate it from conventional single LTP. In the present study, we add further evidence that RISE is distinct from LTP itself using the recording of miniature EPSC (mEPSC) from CA1 pyramidal neurons. The cultures undergoing RISE showed more frequent occurrence of mEPSC than the control cultures. However, the amplitude of mEPSC was indifferent. This situation was distinct from that of LTP, in which both the frequency and amplitude of mEPSC were increased. Together with previously provided evidence (slow development after extinction of LTP, etc.), we confirm that RISE is not a mere extension of LTP.

## O2P-B10 The characteristics of TEA-induced plasticity at hippocampal CA3 synapses

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$K^+$ channel blockade by tetraethylammonium (TEA) induces synaptic plasticity at CA1 and dentate gyrus synapses, but the effects of TEA application on CA3 synaptic responses remain unclear. To examine the effects of TEA and GABAergic modulation on plasticity, field EPSPs were recorded from mossy fiber (MF)-CA3 or associational fiber (AF)-CA3 synapses of rat hippocampal slices. At AF-CA3 synapses, TEA induced long-term potentiation (LTP) regardless of GABAergic inputs, and also evoked LTP at MF-CA3 synapses in the presence of GABAergic inputs. However, there was no plasticity at MF-CA3 synapses induced by TEA under blockade of $GABA_A$ receptors using picrotoxin or gabazine. To clarify the mechanisms of GABAergic excitatory effects on MF-CA3 plasticity, TEA was applied under a blockade of $Na^+$-$K^+$-$Cl^-$ cotransporters (NKCCs) by 1 μM bumetanide, which reduces $Cl^-$ uptake at the mossy fiber terminals. The blockade of NKCCs resulted in the inhibition of TEA-induced LTP, suggesting that GABAergic inputs for MF-CA3 synapses facilitate TEA-induced LTP due to the higher equilibrium potential of $Cl^-$ at mossy fiber terminals.

## O2P-B11 The LTP regulation system by $D_2$ receptor and neurotensin receptor type-1 in the basolateral amygdala

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Neurotensin (NT) receptor type-1 (NTR1) is suggested to modulate the action of dopamine (DA) neurons. DA is involved in the induction of long-term potentiation (LTP) in the amygdala. The DA-NT double positive afferent fibers projects to the basolateral amygdala (BLA) from the ventral tegmental area. Thus, NTR1 may have regulatory effects via DA systems on the induction of LTP in the BLA (BLA-LTP). To examine it, we demonstrated whole cell patch clamp recordings in the BLA pyramidal neuron and compared the BLA-LTP elicited by the electrical stimulation of the lateral amygdala in the wild and NTR1-knockout (KO) mice. NTR1-KO mice showed the facilitated BLA-LTP, which was abolished by a $D_2$ receptor antagonist, an NMDA receptor antagonist and the administration of GDP-β-S into recorded cells. These data suggest that NTR1 suppresses the BLA-LTP through the regulation of post-synaptic $D_2$ receptor.

## O2P-B12 Laminar variation of Homer1a/Vesl-1S effects on long-term depression in the rat visual cortex

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The scaffold protein family Homer/Vesl is known to link mGluRs to IP3Rs. Homer1a/Vesl-1S is regarded also to regulate such coupling in an activity-dependent manner. The present experiments examined whether Homer 1a takes part in induction of long-term depression (LTD) in the rat visual cortex. In layer VI, pyramidal cell LTD was suppressed with Homer 1a injected. LTD in layer VI was dependent on mGluRs, but not on NMDARs. In layer II/III and layer V, on the other hand, Homer 1a injection was unable to affect LTD which is dependent on NMDARs. Induction of endogenous Homer 1a was attempted by electroconvulsive shock (ECS). Homer 1a thereby induced, as well as Homer 1a injected, was significantly able to reduce the magnitude of LTD in layer VI. In other layers, however, LTD was still induced even after ECS. These results suggest that mGluR dependence of LTD may relate to its layer-specific sensitivity to endogenous and exogenous Homer 1a.

## O2P-B13 Local synchrony formed by spike-timing-dependent plasticity working on asynchrony-favoring neurons

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Computational studies of the spike-timing-dependent plasticity (STDP) have been mainly performed using the leaky integrate-and-fire (LIF) model whose synchrony tendency does not reflect the real neurons. Neurons in the brain are categorized into two types quantified by a phase response curve(PRC): those that favor synchronous firing and those that favor asynchronous firing. We show that STDP working on neurons that favor asynchrony converts an asynchronous firing population into three or more clusters of neurons that fire synchronously within a cluster. The neurons can fire synchronously within a cluster despite their intrinsic preference to asynchrony because STDP has disrupted intra-cluster but not inter-cluster connections. This interesting local synchronization never happens when STDP works on the population of neurons that favor synchrony. They simply synchronize globally. We show that not only PRC determines the way STDP works, but also STDP determines the way how PRC governs the synchrony tendency. Therefore, STDP and PRC can work synergetically to carve functional circuits in the brain.

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## O2P-B14 Two coincidence detectors model can reproduce synaptic plasticity induced by various protocols

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Recently, it has been suggested that the coincidence detector through NMDA receptors is involved in synaptic potentiation, and that the coincidence detector through metabotropic glutamate receptors is involved in synaptic depression. Based on the suggestion, Karmarkar and Buonomano (2002) proposed a model incorporating the two coincidence detectors, and showed that the model can reproduce synaptic plasticity experimentally induced by a simple pairing stimulation protocol. However, recent experimental studies using various timing-dependent stimulation protocols, including triplet stimulation protocols, suggest that synaptic plastcity can not be reproduced by any combination of those by the simple pairing protocols, nor by the Karmarkar and Buonomano's model. Here, we proposed a different model based on the Karmarkar and Buonomano's idea and showed that the model can reproduce the synaptic plasticity observed in the various protocols.

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## O2P-B15 Actin-based metaplasticity in cocaine addiction

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In addiction, psychostimulant-evoked glutamate-dependent memory/learning-like plasticity is translated to automated habit-like compulsive drug-seeking. Glumatamergic and dopaminergic input to the nucleus accumbens (NAc) is, in part, responsible for this transition. We previously found that actin cycling in the NAc was enhanced by repeated cocaine administration. To examine the effect of actin cycling on the cocaine-induced kinetics of postsynaptic density (PSD) proteins, we performed a time-course dependent PSD protein profiling. In control animals, a cocaine challenge recruited many PSD proteins relative slowly in PSD in 6hr, whereas in chronic cocaine animals, this process was augmented in 2hr, and it returned to the basal level in 6hrs. Enhanced actin cycling by TAT-cofilin peptide in combination with cocaine initially promoted PSD kinetics, but by 6hr there was a marked decreases in the PSD. These data suggest that enhanced actin cycling induces oscillation-like PSD motility and is responsible for the augmented PSD motility in chronic cocaine animals.

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## O2P-B16 A kinetic model for molecular mechanisms of cortico-striatal synaptic plasticity

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The plasticity of cortico-striatal synapses is regulated by dopamine input from the substantia nigra (SN). While glutamate input induced by cortical stimulation alone results in long-term depression (LTD), SN stimulation combined with cortico-striatal stimulation switches LTD to long-term potentiation (LTP). LTP is also induced by cortico-striatal stimulation when membrane potential of medium spiny neurons in striatum is high.To examine the dynamic mechanism of the plasticity, we constructed a signaling pathway model in medium spiny neuron of the striatum.Our model reproduced bidirectional synaptic plasticity and revealed molecular mechanisms of striatal synaptic plasticity. We conclude that PKA-PP2A-DARPP32 positive feedback loop is essential to cortico-striatal synaptic plasticity.Especially, PKA and DARPP-32 are essential to dopamine dependent cortico-striatal synaptic plasticity and CaMKII plays an important role in membrane potential dependent synaptic plasticity.

## O2P-C01 Neural mechanism for extracting object features critical for visual categorization task

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The ability to group visual stimuli into meaningful categories is a fundamental cognitive process. Some experiments are made to investigate the neural mechanism of visual categorization. Although experimental evidence is known that prefrontal cortex (PFC) and inferiortemporal cortex (ITC) neurons sensitively respond in categorization task, little is known about the functional role of interaction between PFC and ITC in categorization task To address this issue, we made a functional model of visual system, and investigated the neural mechanism for the categorization task of line drawings of faces. We show here that PFC generates a working memory state, in which only the information of face features relevant to the categorization task are sustained. We also show that the feedback connections from PFC to ITC enhance the sensitivity of ITC neurons responsive to diagnostic features of face images in the categorization task.

## O2P-C02 Functional model characterization for neurons in the early visual cortex

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Early visual cortical neurons are classically divided into simple and complex cells, based on selectivity to contrast polarity. However, this classification is not necessarily dichotomous between the two groups. Is there a better single unified framework that can capture variations between the extremes? Responses to stimuli with different contrast polarities were measured for both groups of cells with dynamic noise stimuli, and were analyzed in terms of summed outputs of multiple linear subunits followed by nonlinearity. This functional model provided a good characterization for both groups of cells. The number of subunits tended to be larger for complex cells than simple cells. Contrary to standard models, however, some simple cells required more than one subunit whereas many complex cells required less than four subunits. These results suggest that the conventional classification scheme is inadequate for capturing the variety of intrinsic structures of neurons in the early visual cortex. The new unified framework offers a superior alternative.

MEXT(18020017), JSPS(13308048), COE21

## O2P-C03 Effective resolution of visual filters applied to natural images

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Recent studies modeling the selectivity of visual cells have been interested in the relation between natural image statistics and the properties of receptive fields. A recurring component of these models is the use of the correlation operator as a first step in the computation of responses to visual stimuli: models of V1 cells involve the non-linear combination of one or more correlation filter outputs. Here, we present a computational study that identifies the resolution of detail, represented in visual features, that can effectively be captured by the correlation operator. For this, we measure the responses of filters over image ensembles and examine the evolution of the filter response properties as the resolution is progressively reduced. Our simulations and theoretical analysis show that in natural images, filter response properties are not affected by resolution reduction up to the limit of about 15 pixels. In particular, the results suggests that complex shapes can be effectively encoded by combinations of features of the limit resolution and have consequences for the modeling of inferotemporal neurons function.

## O2P-C04 Relative weights on conscious and non-conscious visual processing in human retinotopic areas

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Robust responses to visible and invisible stimuli have been reported in both early and higher human visual areas, denying simple dichotomy between conscious and non-conscious visual processing subserved by each area. We thus investigated the relative weights on conscious and non-conscious visual processing in nine retinotopic areas. To this end, fMRI activity was measured while subjects viewed a rotating stimulus which was sometimes visible and sometimes invisible due to interocular suppression. We found that, when the stimulus became invisible, mid- and high- level areas (V3, V4, V7, V8, LOc) showed much reduced responses, while early and dorsal visual areas (V1, V2, V3A, MT) showed less reduced responses. This result gives strong evidence that conscious visual processing is tightly coupled with anatomical and functional organization of human visual cortex.

COE21, JSPS



## O2P-C05 Effectiveness of dithering to destroy spike coincidences

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Correlation analysis of neuronal spiking activity relies on theavailability of distributions for assessing significance. At present, these distributions can only be created by surrogate data. A widely used surrogate, termed dithering, adds a small random offset to all spikes times. Here, we analyze the effectiveness of dithering and its bounds of destroying spike coincidences by using simulations and closed form expressions. We show that the effectiveness of dithering depends also on the method chosen for coincidence detection. Application of dithering as a control will be illustrated on data of monkey visual cortex in free viewing condition.

## O2P-C06 Contribution of the GABAergic pathway to the correlated activities of chicken retinal ganglion cells

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In the present study, the spatiotemporal pattern of chicken retinal ganglion cells' firing activity in response to full-field white stimuli was investigated. Cross-correlation analysis showed that nearby ganglion cells of sustained subtype fired in precise synchrony (delay lag within 2 ms, narrow correlation); the activities of neighboring ganglion cells of transient subtype were correlated with distributed time lags (within 20 ms, medium correlation). Further pharmacological studies demonstrated that the intensity of the medium correlations could be strengthened when exogenous GABA was applied and attenuated when picrotoxin was used. Meanwhile, application of GABA or picrotoxin had no significant influence on the narrow correlations. These results suggest that in the chicken retina, GABAergic pathway should be involved in the formation of medium correlations between ganglion cells. Neurons might fire at a lower rate but with stronger synchronization to improve the efficiency of information transmission, with the mechanism involving the GABAergic inhibitory input.

## O2P-C07 Precisely timed inhibition can modulate the internal structure of bursts in salamander retina

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Under visual stimulation, the output neurons of the vertebrate retina generate spike bursts. We have reported that the ISIs of these bursts have distinct combinations depending on the stimulus, and code multiple visual features. To understand the neural mechanisms of such burst coding, we constructed a network model of the retina. We used the standard leaky integrate-and -fire neurons to model the output neurons. These neurons had three inputs: stimulus-dependent excitatory and inhibitory currents from interneurons and a noise current. The simulation showed that in the case of three-spike bursts, the excitatory input dominate the total burst duration and usually modulate the two ISIs coherently. However, the inhibitory input, if precisely timed and modulated in the amplitude, regulate the two ISIs without causing any change in total burst duration. These results suggest that inhibitory interneurons, presumably amacrine cells, may shape the internal structure of bursts and contribute to the burst coding.

## O2P-C08 Non-stationary dynamics of synchronous oscillatory spike events in the LGN - Bootstrap significance test -

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Precisely synchronized oscillatory spike events have been commonly observed in the visual pathway. In most models, synchrony was assumed to be a stationary process lasting throughout the stimulus presentation. To test this hypothesis, we applied the unitary event analysis to unit pairs recorded in the LGN of the anesthetized cats. We extended the original method for application to highly oscillatory spike trains. Firstly, we found that the significance test based on the bootstrap samplings of trial shuffled data worked superior to the test based on the Poisson distribution. Secondly, for the significance test of non-stationarity in synchrony, we developed other bootstrap samplings that kept the same mean synchrony strength as the test data. The global statistics suggests that 48% of the samples (N=840) have significant synchrony and 47% of the significant synchrony show significant non-stationary modulation that could not be predicted by the change in firing rates. We conclude that implicit assumption of stationary synchrony is a misleading concept. Ito, H. *Statistics in Medicine*, 2007 (to be published).

KAKENHI17021036

## O2P-C09 Strength of surround suppression is proportional to the time course of response magnitude in cat V1

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In the primary visual cortex (V1), a neuronal response to stimulation of the classical receptive field (CRF) is suppressed by the stimulus presented at the receptive field surround (SRF). This surround suppression is supposed to play a role in controlling neuronal responsiveness (gain) according to the spatiotemporal context of stimulation. In order to know how response gain is modulated by surround stimuli, we investigated the temporal dynamics of surround suppression in V1 cells of anesthetized cats, using stationary flashes (500 ms) of sinusoidal grating with optimal parameters and varying radii as stimuli. We found that the strength of the suppression dramatically changed in time in proportion to that of excitatory response to CRF stimulation, suggesting a divisive gain reduction of the neuronal response. Thus, the surround suppression is scaled according to visual response in cat V1 neurons.

## O2P-C10 Disparity- and size-selective neurons in macaque area V4 as a neural basis for size constancy

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The perceived size of an object remains relatively stable despite changes in the size of retinal images accompanying changes in distance from the observer. In this perceptual phenomenon (size constancy), the brain uses distance information for calibrating the perceived size. Binocular disparity provides a clue about distance. To examine interaction between disparity and stimulus size information, we recorded neuronal responses in macaque V4, a visual cortex known to be involved in stereopsis and size discrimination. Random dot stereograms (RDSs) were used for visual stimuli. A majority of neurons were selective for both binocular disparity and cyclopean image size. In some cells, the tunings for binocular disparity and size showed significant interaction; the preferred size shifted to larger for an RDS presented nearer. This behavior of V4 cells was consistent with psychophysical performance of human observers tested with the same stimuli. The results suggest that V4 cells contribute to size constancy.

MEXT(17022025), CREST and Takeda Science Foundation

## O2P-C11 Biased competition based on hierarchical grouping representations in the human visual cortex

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The present study tested the nature of stimulus-driven attentional control in visual grouping by using event-related potentials (ERPs). Ten participants paid their attention to one hemifield during rapid presentations of bilateral stimuli, which were connected by a thin or thick line, or unconnected. Spatial attention was reflected by a larger amplitude of posterior ERPs at the hemisphere contralateral, rather than ipsilateral, to the attended hemifield. The amplitude of the lateralized potential decreased according to the extent of grouping in an early phase (150-210 ms post-stimulus), while that in the unconnected condition was larger than those in the connected conditions (i.e., the bilateral stimuli were perceived as an object) in a later phase (330-390 ms). The results suggest that hierarchical representations in visual grouping bias competitive interactions in the visual cortex in a moment-to-moment fashion.

KAKENHI (16700246)

## O2P-C12 Reconstruction of visual images from fMRI signals by combination of multi-resolution local image decoders

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Recent studies have shown that visual features (e.g., orientation) can be predicted from fMRI activity patterns. They focused on a single dimension of visual features, and thus did not reconstruct a visual image itself. Here we present a method to reconstruct a subject's percept on an image basis given fMRI signals evoked by arbitrary visual images. Using fMRI signals evoked by a sequence of random pattern stimuli, local image decoders were trained to predict the contrast of local image segments of multiple spatial scales. The trained decoders were optimally combined so as to minimize the estimation error for reconstructing the random patterns. The combined decoders were then applied to fMRI signals evoked by a new stimulus set of geometric patterns. The results showed fine reconstruction performance for all presented patterns. Our approach provides a procedure to reconstruct complex perceptual experience by combinatorial representation of perceptual elements.

NSF, NiCT-KARC

## O2P-C13 The diffusion tensor image and tractography of the patients with post-stroke pain

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In tractography based on diffusion tensor image (DTI), the direction and the connection of nerve bundle in the white matter can be examined. In this study, tractography was done on 23 patients with post-stroke pain, who obtained some pain reduction with repetitive transcranial magnetic stimulation (rTMS) of primary motor cortex (M1). We examined the correlation of the results of tractography with the effect of rTMS.Cerebral peduncle and the motor cortex or the sensory cortex was set to region of interest and tractography was done. 12 of 23 were able to describe in this method. Their lesion were putamen for six, thalamus for five, and brainstem for one.Sectional areas of tractography were measured. The pain reduction with rTMS was not correlated with the size of motor tract with tractography but with that of sensory tract significantly ($p=0.013$). This result suggests that sensory tract may play more important role in pain reduction with rTMS of M1.

## O2P-C14 Blockade of glycine transporters ameliorated mechanical allodynia developed in neuropathic pain model in mice

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Neuropathic pain is refractory against conventional analgesics and thus novel medicaments are desired for the treatment of the pain. Glycinergic neurons play an important role for regulation of pain signal transduction in spinal cord. In the present study, the effects of glycine transporter (GlyT) inhibitors on mechanical allodynia were examined in several neuropathic pain models in mice. Either intrathecal or intravenous injection of GlyT1 inhibitors, ORG25935 and sarcosine, and GlyT2 inhibitors, ORG25543 and ALX1393 ameliorated mechanical allodynia in neuropathic pain models. The antiallodynic effect induced by intravenous injection of these inhibitors was blocked by intrathecal administered strychnine and siRNA of glycine receptor $\alpha 3$ (GlyR $\alpha 3$). These results demonstrated that GlyT inhibitors by enforcing glycinergic inhibitory neurotransmission in spinal cord produced potent anti-allodynic effect and may be novel candidate for medicament of neuropathic pain.

## O2P-C15 Neural correlates associated with perceptual difference between itching and pain: A human fMRI study

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Although the perceptive discrimination between itching and pain is clear for us, it is still unclear how our brains produce these different sensations. Thus, we investigated the neural substrates of perceptual differences between itching and pain by fMRI. The anterior cingulate cortex, the anterior insula, the basal ganglia and the pre-supplementary motor area were commonly activated by itching and pain. Neural activity in the posterior cingulate cortex (PCC) and the posterior insula associated with itching was significantly higher than that associated with pain and significantly proportional to itching sensation. Pain, but not itching, induced an activation of the thalamus for several min, and neural activity of this brain region significantly correlated to pain sensation. These differences in brain activity would be responsible for the perceptual difference between itching and pain.

## O2P-C16 Minocycline attenuates spinal microglia activation and tactile allodynia caused by interferon-γ

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Pro-inflammatory cytokines have now received much attention as important players in modulating pain signaling not only in the periphery but also in the CNS. Interferon-γ (IFN-γ), a pro-inflammatory cytokine, has been reported to be upregulated in the spinal cord of animal models of neuropathic pain, raising the possibility that IFN-γ has a role in modulating pain processing. In the present study, we found that intrathecal administration of IFN-γ produced tactile allodynia (abnormal hypersensitivity to innocuous stimulus) and microglia activation (hypertrophy and proliferation) in the spinal cord. Minocycline which can inhibit microglia activation attenuated both tactile allodynia and changes in spinal microglia. Our present findings indicate that IFN-γ causes long-lasting tactile allodynia via activating spinal microglia.



## O2P-D01 Wnt3a promotes mouse hippcampal progenitor cell proliferation by shortening cell cycle

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Activation of Wnt canonical pathway is known to promote proliferation of neural progenitor cells. In marked contrast, several reports demonstrated that Wnt signaling rather induced differentiation of postmitotic neurons. To understand the discrepant roles of Wnt signaling, we applied Wnt3a on the primary culture prepared from E15 mouse hippocampus. Cells were maintained in a chemically defined medium and at a low cell density to reduce cell-cell interactions. Wnt3a increased the number of total cells as well as Tuj1+ neurons with a comparable ratio. Wnt3a did not affect cell survival or asymmetric cell division but rather accelerated cell cycle progression by shortening the cell cycle, which could result in increases of both proliferative cells and differentiated neurons. Our data suggests that Wnt3a promotes cell proliferation by shortening the cell cycle but does not particularly affect the fate switch from progenitors into neurons.

KAKENHI17500255, KAKENHI18053018

## O2P-D02 Wnt signaling promotes retinal regeneration in adult mammals

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We have demonstrated that in the adult mammalian retina, Müller glia de-differentiate and produce retinal neurons after injury in vivo. However, the number of new neurons is limited. Here we report that Wnt signaling promotes proliferation of Müller glia-derived retinal progenitors and neural regeneration after damage or during degeneration. Wnt3a increases proliferation of de-differentiated Müller glia in the photoreceptor damaged retina. Supplementation with retinoic acid or valproic acid induces differentiation of these cells into photoreceptors. Injury induces nuclear accumulation of β-catenin, cyclin D1 upregulation, and Wnt/β-catenin reporter activity. Activation of Wnt signaling by glycogen synthase kinase-3β inhibitors promotes retinal regeneration, and conversely inhibition of the signaling attenuates regeneration. This Wnt3a-mediated regeneration also occurs in rd mice, a model of retinal degeneration. These results provide evidence that Wnt/β-catenin signaling contributes to CNS regeneration in adult mammals.

## O2P-D03 Tsukushi inhibits proliferation of retinal stem cells by Wnt signalling inhibition

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Wnt signalling orchestrates multiple aspects of central nervous system development. Wnt2b is expressed in the marginal-most tip of the embryonic chick retina. It was proposed that Wnt2b plays a role in the maintenance and the proliferation of retinal progenitor cells without cell differentiation.
We have identified a BMP antagonist, Tsukushi (TSK), which is expressed in the primitive streak and Hensen's node during chick gastrulation. When Wnt2b was over-expressed into chick optic vesicle, Wnt2b induced prolonged proliferation of retinal progenitor cells. However, when Wnt2b was co-over-expressed with TSK, the proliferative activity of Wnt2b was almost inhibited. We now show that TSK inhibits the retinal progenitor cells proliferation by functioning as Wnt signalling inhibitor.

KAKENHI

## O2P-D04 Hes5-expressing supporting cells in the mammalian cochlea possess a high proliferative potential

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During development, cochlear supporting cells possess potentials to proliferate and differentiate into hair cell phenotypes. However, after maturation, the supporting cells loss the potentials. In this study, we examined a proliferative potential of Hes5-expressing (EGFP+) supporting cells in the mammalian organ of Corti by using pHes5-d2EGFP transgenic mouse in which EGFP transcript was regulated by *Hes5* promoter activity. By FACS, the EGFP+ cell population was collected from the cochleae of the postnatal day 2 to 5 mice. Results of a colony-forming assay showed that EGFP+ cell population possessed a high potential to form colonies, while EGFP-negative population did not form any colonies. The results of this study indicate that Hes5 is a marker of high proliferative potential progenitors in the mammalian organ of Corti. Hes5 expression may be required for maintenance of the high palsticity of the cochlear progenitors.

KAKENHI17591785

## O2P-D05 Continuous deep brain stimulation has a regenrative effect on the ischemic rat model in the chronic stage

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We examined the effects of Continuous deep brain stimulation (DBS) on the rat middle cerebral artery occlusion model in the chronic stage. One month after lesion, the stimulation electrode was implanted into the ischemic border zone, and stimulation was delivered to the electrode for a week. The rats with DBS showed significantly better behavioral recovery in the spontaneous activity or stroke test than those without DBS at two months after the lesion. The infarct volume significantly reduced with DBS compared to that of pre-DBS or without DBS. The proliferated cells accumulated around the DBS electrode through the concentration of neurotrophic factors, and the newly neurons derived from the subventricular zone and a number of vessels were found around the ischemic border zone in the DBS group. These results suggest that DBS has a regenerative effect by enhancement of neurogenesis and angiogenesis in the ischemic brain.

## O2P-D06 Effects of the monomeric, oligomeric and fibrillar A $\beta$ 42 peptideson the proliferation and differentiation of adult neural stemcells from SVZ

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The incidence of amyloid plaques, composed mainly of $\beta$-amyloid peptides (A $\beta$),does not correlate well with the severity of neurodegeneration in patients withAlzheimer's disease (AD). The effects of A $\beta$ 42 on neurons or neural stem cells (NSCs)in terms of the aggregated form remain controversial. We prepared three forms ofoligomeric, fibrillar and monomeric A $\beta$ 42 peptides and investigated their effects on theproliferation and neural differentiation of adult NSCs, according to the degree ofaggregation or concentration. A low micromolar concentration (1microM) of oligomeric A $\beta$ 42increased the proliferation of adult NSCs remarkably in a neurosphere assay. It alsoenhanced the neuronal differentiation of adult NSCs and their ability to migrate. Theseresults provide us with valuable information regarding the effects of A $\beta$ 42 on NSCs inthe brains of patients with AD.

## O2P-D07 Enhanced survival of neural progenitor cells derived TH positive neurons under surveillance of olfactory ensheating cells on exposure to 6-hydoxydopamine

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Transplantation of Neural progenitor cells (NPCs) is a promising therapeutic strategy for neurodegenerative disease like Parkinsons. Posttransplantation recovery depends on % survival /functional capacity of grafted cells. Considerable death of which occurs due to lack of neurotrophic factor(NTF)support which were found essential for survival of dopaminergic neurons.Prolong exposure to NTF can also be used to enhance the post transplant survival of NPCs. This was explored by in vitro exposure of 6-hydroxy dopamine (6-OHDA) to NPCs co-cultured with Olfactory Ensheating Cells (OECs) rich in multiple NTF. A concentration dependent decrease in viability of NPCs on exposure to 6-OHDA ($10^{-7}$ to $10^{-4}$ M) less was prominent in NPCs co-cultured with OEC was observed. Co-cultured NPCs further showed higher expression of tyrosine hydroxylase, Neu N , GDNF, Bcl-2 and GAP-43. Our result suggest that NTF support through OEC is capable of protecting the NPC from 6-OHDA toxicity and also help in their better differentiation to neuronal phenotype

DST

## O2P-D08 DSCAM in the hippocampal neurogenesis of ischemic monkeys

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Molecular signals regulating adult neurogenesis in primates are largely unknown. Here differential display was done to analyze gene expression changes that occur in dentate gyrus of adult monkeys after transient cerebral ischemia. Among 14 genes upregulated, we focused on Down syndrome cell adhesion molecule (DSCAM), and characterized its expression pattern at the protein level. In contrast with upregulation of Dscam gene on days 5 and 7, DSCAM showed a decrease after ischemia until day 9 followed by recovery on day 15. In the control, DSCAM was detected in DG granule cells and CA4 neurons, but their immunoreactivity decreased in the post-ischemic monkeys. However, in the subgranular zone, ischemia provoked a marked increase of DSCAM-positive cells on days 9 and 15. DSCAM upregulation was seen in two cell types: one is immature neurons positive for PSA-NCAM or $\beta$ III-tubulin, while another is astrocytes positive for S100 $\beta$. Young astrocytes were in intimate contact with newly-generated neurons. These data suggest implication of DSCAM in adult neurogenesis.

KAKENHI18390392

## O2P-D09 Polyunsaturated fatty acids promote proliferation of neural progenitor cells in the hippocampal dentate gyrus

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Fatty acid binding protein (FABP) is postulated to serve as a lipid shuttle, solubilizing hydrophobic fatty acids and delivering them to the appropriate metabolic system. Among the FABP family proteins, FABP7 (B-FABP, BLBP) and FABP5 (E-FABP, S-FABP, K-FABP, DA11) are expressed in the ventricular zone in embryonic stages. Especially, FABP7 is widely used as a marker for radial glial cells and regulates neuroepithelial cell proliferation during cortical development (Arai et al., 2005). However, their specific role in the postnatal neurogenesis is not yet fully established. In this study, we found that FABP7 was mostly expressed in the GFAP+ early progenitor cells and FABP5 in the PSA-NCAM+ late progenitors. Moreover, hippocampal neural progenitor cells dramatically increased in wild type rats fed with polyunsaturated fatty acids (PUFA). Taking all together, it is concluded that FABPs and PUFA are essential for proliferation of neural stem/progenitor cells in the dentate gyrus.

CREST

## O2P-D10 Functional significance of adult neurogenesis in the mouse brain

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New neurons in the adult brain continually replace older ones within mature neural circuits. However, the in vivo rate and functional significance of neuronal replacement in adult mammals remain poorly understood. Here, we generated transgenic mice that express CreERT2 in the germinal zone of the adult brain under the control of the nestin promoter and enhancer. In the presence of Tamoxifen, Cre-mediated recombination occurred efficiently in adult neural progenitor cells of the SVZ/LV and SGZ/DG. Crossing with ROSA26-reporter lines, we estimated the rate of neuronal replacement of granule neurons at the olfactory bulb (OB) and the dentate gyrus. In the case of OB, most granule neurons were replaced within one year period. To investigate the functional impact of neuronal replacement, newly generated neurons and neuroblasts were genetically ablated by crossing with NSE-DTA mice. We are now analyzing the effect of ablating adult neurogensis on the neural circuit maitenance and the higher brain functions.

CREST

## O2P-D11 Feeding-related time window of fate decision of newborn granule cells in the adult mouse olfactory bulb

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Granule cells (GCs) in the olfactory bulb (OB) are continuously generated and incorporated into the preexisting neuronal circuit even in adult. Survival and death of newborn GCs are determined with sensory experience-dependent manner. However, it is not known whether their fate decision occurs in specific time windows of a day. Here, we show that fate decision of newborn GCs occurs extensively during feeding time. Under the ad libitum feeding condition, a small number of apoptotic cell death (with caspase-3 activation) of newly-generated GCs occurred across whole circadian time points. When food was supplied only during a specific time window in a day, the number of caspase-3-activated newborn GCs increased by 3-4 folds in entire OB specifically during the feeding time. We also show that sensory input greatly affects the fate decision of newly-generated GCs during the feeding period. These results indicate the existence of feeding-related time window of fate decision of newly-generated GCs in the OB.

KAKENHI (18022009)

## O2P-D12 Molecular mechanisms for the neuropoietic actions of CNTF on cultured adult rat dorsal root ganglion neurons

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Ciliary neurotrophic factor (CNTF), a member of the interleukin-6 cytokine family, promotes neuronal survival and axonal regeneration after peripheral nerve injury, but molecular mechanisms how it acts on neurons remain to be elucidated. In this study, recombinant rat CNTF applied to culture medium (5, 50 or 500 ng/ml) significantly enhanced survival and neurite outgrowth of cultured adult rat dorsal root ganglion (DRG) neurons. By Western blot analysis, CNTF induced phosphorylation of STAT3, Akt and ERK in the neurons. The CNTF-induced neurite outgrowth was totally abolished by co-treatment with 5 μM of JAK2 inhibitor AG490, 25 μM of PI3K inhibitor LY294002, or 50 μM of MAPK kinase (MEK) inhibitor PD98059. The enhancement of survival was also affected by AG490 and LY294002, but not by PD98059. These findings suggest that JAK2-STAT3, PI3K-Akt and MEK/MAPK signaling pathways are involved in the CNTF-induced neurite outgrowth in mature DRG neurons, while the former two pathways may play major roles in mediating the survival response of neurons to CNTF.

KAKENHI (17500267)



## O2P-D13 Autocrine FGF signaling in ectoderm is involved in neural induction and patterning in *Xenopus* development

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We explored possible involvement of autocrine FGF signaling in ectoderm in neural induction and patterning by employing morpholino-antisense oligonucleotides (MOs) against the five members of FGF family, which are present in ectoderm (FGF-2, 3, 4, 8 and 9). When each of these FGFMOs was injected into a dorso-animal blastomere of 8-cell-stage embryos, which is destined to ectodermal and subsequently neural region, only FGF-2MO and FGF-8MO caused severe head defects on the injected side. The defects were largely rescued by co-injection of cognate FGF-2 or -8 that was mismatched to respective MO. We then examined the expression of position-specific neural markers along the A-P axis (*otx2*, *XHR1*, *fkh5* and *HoxD1*) as well as an early pan-neural marker (*Sox2*) in FGF-2- or -8-depleted embryos. The in situ analysis showed that the expression of all the five markers was suppressed in the FGF-2- or -8-injected region that was marked by co-injected lineage-tracing GFP mRNA. We propose a novel model for neural induction and patterning in *Xenopus*, which includes autocrine FGF signaling in ectoderm.

## O2P-D14 Hepatocyte growth factor (HGF) as a trophic factor for oligodendrocyte progenitor cells (OPCs) during postnatal development in the rat

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Trophic effects of HGF on neurons, such as neurite extension and survival promoting activities, are well characterized, while those on OPCs are poorly understood. Here, we provide the first evidence that c-Met/HGF receptor is expressed and physiologically tyrosine-phosphorylated (activated) in OPCs in postnatal developing rat. Intrastriatal injections of recombinant human HGF and subsequent BrdU+/NG2+ and BrdU+/MBP+ staining revealed that OPC proliferation numbers increased, while new oligodendrocyte (OL) numbers decreased. Western blot and electron microscopic analyses revealed that HGF decreased MBP protein levels and myelination. Anti-HGF IgG injections reversed reduction of new OLs by HGF. Therefore HGF promotes proliferation of OPCs, while attenuates differentiation into OLs. Our findings suggest that HGF/c-Met signaling plays an important role in developmental oligodendrogenesis and suggest the implication of HGF in increasing reservoir cells (OPCs) of OLs in demyelinating diseases.

## O2P-D15 Neurotrophic effects of EGF / ErbB1 signals on developing midbrain dopaminergic neurons

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Epidermal growth factor (EGF) receptors (ErbB1) are enriched in the developing brain and distributed in the midbrain and neocortex. However, their biological functions in the dopaminergic system are not fully understood. To characterize their actions on dopaminergic neurons, we determined the neurotrophic features of EGF. EGF increased tyrosine hydroxylase level and dopamine uptake. Subchronic treatment of rat neonates with ErbB1 inhibitors attenuated the development of dopaminergic neurons in substantia nigra, but not those in ventral tegmental area. Levels of tyrosine hydroxylase and dopamine transporter are reduced in the striatum but not in the frontal cortex of these animals, as well as a result from the knockout mice. Although the trophic support for the dopaminergic neurons are redundant, ErbB1 ligands including EGF, have a specific role in promoting and maintaining the development of dopaminergic neurons in substantia nigra.

## O2P-E01 Sept4, a component of presynaptic scaffold and Lewy bodies,is required for the suppression of α-synuclein neurotoxicity

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In Parkinson disease (PD), α-synuclein aggregates (Lewy bodies) often sequester Sept4, a GTP-binding scaffold protein. We show the physiological association of Sept4 with α-synuclein, the dopamine transporter, and other presynaptic proteins in dopaminergic neurons; mice lacking *Sept4* exhibit diminished dopaminergic neurotransmission due to scarcity of these presynaptic proteins. In transgenic mice that express human α-synuclein$^{A53T}$, loss of *Sept4* significantly enhances neuropathology and locomotor deterioration. In this PD model, insoluble deposits of phosphorylated α-synuclein$^{A53T}$ are negatively correlated with the dosage of Sept4. In vitro, direct association with Sept4 protects α-synuclein against self-aggregation and phosphorylation. Thus, Sept4 may be involved in PD as a dual susceptibility factor, as its insufficiency can diminish dopaminergic neurotransmission and enhance α-synuclein neurotoxicity.

## O2P-E02 1BnTIQ, a Parkinson Disease-related neurotoxin binds to tubulin

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1BnTIQ, an endogenous neurotoxin, has been proposed as one of the etiological factors for Parkinson Disease(PD). The levels of 1BnTIQ in cerebrospinal fluid of patients with PD were reported to be three times higher than those in control. We tried to identify 1BnTIQ binding proteins. Rat brain homogenate was incubated with a photoaffinity labeled 1BnTIQ analog, GIF-0732-R-S1, and tentative 1BnTIQ binding proteins were made fluorescent. We performed 2D electrophoresis and identified the proteins by MALDI-TOF mass. One of the 1BnTIQ proteins was beta-tubulin. Polyubiquitinated tubulin in human neuroblastoma SH-SY5Y cells was reduced in the presence of 10uM 1BnTIQ for 48 h or 50nM for 1 month. It is suggested that 1BnTIQ causes the same inability as that of tubulin ubiquitination by Parkin mutant in familial PD.

## O2P-E03 Molecular mechanism of neurodegeneration caused by familial mutaions (P123H and V70M) of β-synuclein

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Recently, two missense mutations (P123H andV70M) of β-synuclein (β-syn) were identified in dementia with Lewy body (DLB). To determine if mutations of β-syn were causative for neurodegeneration, both mutants were stably transfected to B103 neuroblastoma cells. Distinct from other cell types, mutant β-syn overexpressing cells exhibited eosinophilic cytoplasmic inclusion bodies which were immnopositive for mutant β-syn as well as lysosomal markers. Notably, formation of inclusions was greatly stimulated by co-expression of α-syn, which was dependent on the S129-phosphorylation and was efficient by A53T familial mutant compared to wild type α-syn. Further analyses demonstrated that formation of lysosomal inclusions was protective. Taken together, these observations support a contention that missense mutations of β-syn linked to DLB may represent pathogenic mutations.

KAKENHI (17300115)

## O2P-E04 Long-term recovery in a primate model of Parkinson disease after gene transfer of dopamine-synthesizing enzymes

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Adeno-associated viral (AAV) vectors can transduce neurons efficiently. We report here long-term behavioral recovery in a primate model of Parkinson disease following AAV vector-mediated gene transfer of dopamine-synthesizing enzymes. AAV vectors expressing the enzymes were injected into the left putamen in three MPTP-treated cynomolgus macaques. The monkeys were followed up for more than six, four, and three years, respectively. In all three monkeys, manual dexterity of the right forearm had improved remarkably after gene delivery and the behavioral improvement persisted throughout the follow-up. A PET scan showed increased [$^{11}$C]L-dopa uptake even 6 years after the vector injection. AAV vector-mediated gene transfer is effective and safe over the long term.

KAKENHI (18050401)

## O2P-E05 L-DOPA persuades enhanced GABA release in the medial globus pallidus in a rat model of L-DOPA-induced dyskinesia

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L-DOPA is the most effective agent used for symptomatic relief in patients with Parkinson disease (PD) but long term L-DOPA treatment often accompanies problematic L-DOPA-induced dyskinesia (LID). We have shown that a rat model of LID (the 6-OHDA-lesioned rat, the PD model, repeatedly treated with L-DOPA) has enlarged axon terminals of the GABAergic projection from the striatum to the medial globus pallidus (MGP) (the direct pathway) and increased density of synaptic vesicles in the enlarged terminals. These findings suggest that amplified GABAergic neurotransmission in the MGP is involved in the genesis of LID. The present study, using in vivo microdialysis, evaluated the effect of a L-DOPA injection on GABA release in the MGP of the PD and LID models. L-DOPA-induced increase in extracellular GABA levels in the MGP was significantly higher in the LID model than in the PD model. This result strongly indicates that enhanced GABA release in the MGP is related to the occurrence of LID.

## O2P-E06 Involvement of endogenous dopamine in dopaminergic neuronal death induced by glutamate

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It has been proposed that endogenous dopamine could serve as a susceptibility factor in dopaminergic neurodegeneration in Parkinson disease. Although hyperactivation of glutamatergic transmission may be involved in the degenerative process, a role of dopamine in glutamate excitotoxicity has not yet been elucidated. We demonstrated that dopaminergic neurons were more vulnerable to glutamate toxicity than non-dopaminergic neurons in primary mesencephalic cultures. Depletion of intracellular dopamine by α-methyl-*p*-tyrosine, an inhibitor of tyrosine hydroxylase (TH), suppressed dopaminergic neuronal death induced by glutamate. Exposure to glutamate caused phosphorylation of TH which facilitates dopamine synthesis. PD98059, an inhibitor of MEK, suppressed phosphorylation of TH and protected dopaminergic neurons from glutamate toxicity. These results suggest that the vulnerability of dopaminergic neurons to glutamate toxicity is dependent on endogenous dopamine.

## O2P-E07 Effects of zonisamide on experimental tremors in rats

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Recent clinical studies have suggested that zonisamide, an antiepileptic, may be effective for the treatment of Parkinson disease and essential tremor. The present study aimed to study the effect of zonisamide on experimental tremors in rats.
Methods. Three representative rodent tremor models were used. Harmaline- or oxotremorine-induced tremors, and cholinomimetic-induced tremulous jaw movements (TJMs), which have been proposed as a rodent model of pharmacologically induced parkinsonian tremor. Results. Zonisamide significantly suppressed both harmaline- and oxotremorine-induced tremors dose-dependently. Zonisamide also significantly suppressed tacrine-induced TJMs, and this effect was not lost under conditions of monoamine-depletion by reserpine or dopaminergic blockade by a cataleptic dose of haloperidol.
Conclusions. Although zonisamide is knowns as a dopamine release enhancer, the anti-tremor effects of zonisamide may be achieved by a non-dopaminergic mechanism. Zonisamide may be a non-specific, anti-tremor drug.

KAKENHI: 17590889

## O2P-E08 A reevaluation of the network via patch and matrix compartments of rat neostriatum

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The current model of basal ganglia rests on the idea that the striofugal system is composed of two separate (direct and indirect)pathways originating from distinct cell populations in the striatum. The striatum itself is divided into two major compartments, the patch and matrix , which differ by their neurochemical characters and input/output connections. Here, the differences in the synaptic organization of these striatopetal afferents between the striatal compartments were examined with two kinds of vesicular glutamate transporters. Furthermore, the labeled axons from single neurons located in either patch or matrix were entirely reconstructed. Our findings are at odds with the previous concept of a dual striatofugal system: direct/indirect and patch/matrix, and call for a reevaluation of the organization striatofugal projection system.

KAKENHI 16500217, 1600025, 17022020, 17650100, 17022024

## O2P-E09 Molecular basis for impulsive and self-controlled behaviors in delay-discounting task

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When two alternatives are available in instrumental behavior, animal' behavior is biased toward responding on one lever with which each behavioral response results in delayed but large reward, and against responding on the other lever with which each response results in immediate but small reward. This has been used as an index of impulsive behavior and is known to be sensitive to lesions of the nucleus accumbens (NAC). Pharmacological studies with systemic administration of antagonists have shown that dopaminergic transmission is involved in controlling choice behavior in a delay-discounting task. Thus, a major question arises as to whether dopamine in the NAC is really involved in controlling behaviors in the task. To this end, we implanted cannulas into the NAC to identify neuronal substances in controlling behavior. We confirmed dopamine as the chemical substance in the NAC that controls choice behaviors in the task. Dopamine transmission is converted to second messengers through dopamine receptors. We are identifying which of them are responsible for choice behaviors in the the task.



## O2P-E10 Dynamical modes in a computational model of the subthalamo-pallidal circuit

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Recent findings of the basal ganglia, especially from the pathological studies on the circuit, have suggested that temporal properties of the neural activity such as rhythmicity and synchronicity are involved in the functions. Therefore, understanding of dynamical properties of the circuit is increasing in importance. We investigated stability of synchronous spike activity of subthalamic nucleus (STN) and the external segment of global pallidus (GPe) applying the phase analysis to their model neurons. The result showed interconnected STN neurons tended to synchronize whereas GPe neurons essentially desynchronized within the range of experimentally observed in vivo firing rates. In addition, we conducted numerical simulations of mutually connected STN-GPe network changing some parameters, which suggested pallido-subthalamic synapses are one of the crucial factors to control dynamical modes of the nuclei.

KAKENHI(18019036)

## O2P-E11 Role of the primate lateral habenula in negative motivational control of behavior

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Animal behaviors are facilitated by expectation of big reward and suppressed by expectation of small or no reward. Midbrain dopamine (DA) neurons are thought to be involved in such reward-based control of motor behavior. However, it is unclear which parts of the brain provide DA neurons with signals necessary for the action. We tested the hypothesis that projections from the lateral habenula (LHb) transmit reward-related signals to DA neurons. First, we recorded the activity of LHb and DA neurons while monkeys were performing a visually guided saccade task with positional reward bias. LHb neurons were predominantly excited by a noreward-predicting target and inhibited by a reward-predicting target. In contrast, DA neurons were excited and inhibited by reward and noreward-predicting targets, respectively. The LHb excitation started earlier than the DA inhibition. Second, we examined the effect of electrical stimulation of the LHb on the activity of DA neurons. The stimulation elicited strong inhibitions in DA neurons. Our results suggest that the LHb suppresses less rewarding motor behavior by inhibiting DA neurons.

## O2P-E12 The medial prefrontal cortex controls dopamine-induced motor behavior via the subthalamic nucleus activation

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Microinjection of GABAA receptor agonist muscimol into the medial prefrontal cortex (mPFC) suppresses both hyperlocomotion and activation of c-fos gene in the subthalamic nucleus (STN) induced by dopaminergic stimulation. These reactions are mediated by dopamine D1-like and D2-like receptors in the mPFC. The present study examined the role of the mPFC-STN pathway in dopamine-induced hyperlocomotion. When a retrograde tracer FluoroGold (FG) was injected into the STN, the labeled pyramidal cells were found in the mPFC. Intra-mPFC injection of biotinylated dextran amine gave anterogradely labeled fibers in the STN. Systemic injection of methamphetamine (METH) elicited c-fos expression in glutamatergic, but not GABAergic, mPFC neurons. Some of c-fos-positive mPFC neurons were also labeled by FG injected into the STN, indicating that some METH-activated mPFC projection neurons monosynaptically innervate the STN. These results suggest that the mPFC neurons facilitates dopamine-induced hyperlocomotion via the corticosubthalamic pathway.

KAKENHI (18500244)

## O2P-E13 Abnormal oscillatory discharges of basal ganglia neurons in parkinsonian monkeys

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Classical models concerning the pathophysiology suggest that Parkinson's disease is induced by the changes in the firing rates of basal ganglia neurons following loss of dopaminergic neurons from the substantia nigra. However, recent emphasis has been placed on the abnormal firing patterns of basal ganglia neurons, such as oscillation and bursting. In our primate model of Parkinson's disease, abnormal oscillatory discharges were so often observed in the globus pallidus and the subthalamic nucleus, as compared to normal controls. Muscimol inactivation of the subthalamic nucleus eliminated the abnormal oscillations from the globus pallidus, and vice versa. These results indicate that the abnormal oscillatory discharges of basal ganglia neurons seen in Parkinson's disease may be facilitated by the neuronal interactions between the globus pallidus and the subthalamic nucleus.

KAKENHI (18300135) and JAPAN INTRACTABLE DISEASES RESEARCH FOUNDATION

## O2P-F01 Network formation by theta and gamma-band synchronization in visual perception

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To investigate the relationship between the dynamics of large-scale neural synchronization and perception, we analyzed endogenous changes in EEG synchrony in multiple frequency bands during Necker cube perception in sixteen human observers. We also investigated changes in neural synchrony during top-down modulation of Necker cube perception. We measured 62ch EEG and calculated response-locked phase-locking values (PLV) to measure large-scale phase synchronization between pairs of EEG signals. We found top-down biasing effects on the distribution of perceptual lengths and long-lasting large-scale theta synchrony (3-7 Hz) among the frontal, parietal and occipital areas, suggesting that neural communication occurs across the distant brain regions. Transient gamma synchronization (>26Hz) was also observed associated with perceptual switching. Our findings suggest that cognitive control is implemented by cooperative dynamics of theta and gamma oscillatory networks which are associated with perceptual monitoring and the emergence of a conscious percept, respectively.

## O2P-F02 EEG phase synchronizaion during Attentional Blink

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A target presented in a rapid stimulus sequence (~10 items/sec) usually is detected easily. However, when two targets (T1 and T2) are presented in sequence, T2 is often missed when the two are 200-500 ms apart. This phenomenon is called attentional blink. Brain activities that correlate with attentional blink (AB) phenomenon were reported before, around, and after the targets. Before T1 onset, EEG phase synchronization in the $\gamma$ band increases when T2 is detected. Around T1 and T2, $\beta$ band MEG and $\gamma$ band EEG phase synchronization increased when T2 is detected. After T2, studies using ERP technique suggest that synchrony in lower bands, such as $\theta$ to $\delta$ bands. In the current study, we analyzed EEG during AB task using dynamic cross-lag phase synchronization index from $\delta$, to $\gamma$ bands. In all bands, epochs of large-scale synchronization is observed when T2 was detected in AB condition. Timing of the synchronization varied; before T1 onset, the synchronization was observed in all bands. Around T1, it was observed in $\beta$ band. After T2 onset, a large scale synchronization in $\theta$ band proceeded that in $\delta$ band.

## O2P-F03 Interaction between noise and pattern in vision

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When two eyes see radically different images simultaneously, the images compete in visual perception. The homogeneous field scarcely rivals a highly patterned image and the vision under "one" eyelid (i.e. a closed eye) is therefore not usually perceived, or at most is perceived as intrinsic dynamic visual noise only noticeable with careful inspection. However, recent studies revealed that activity of the primary visual cortex with both eyes closed is actually patterned as if an oriented stimulus were presented. Then, what happens between ongoing activities and those induced by oriented stimulus? Here we show a novel visual phenomenon, Diagonal Mesh Pattern (DMP), in which visual noise rivals an oriented stimulus but at the same time is spatio-temporally highly modulated by them. The main characteristic of DMP is a noisy mesh pattern diagonally intersecting each point of the repetitive pattern. DMP shows that the figural information of the suppressed stimulus surprisingly survives rivalry, to the extent that it conflicts with the very definition of BR. We "see" interocular interaction in the early visual process through DMP. We also propose a simple explanatory model.

## O2P-F04 Mental rotation of hands and feet involves somatotopically organized brain regions

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Some cognitive functions are implied to rely on motor strategies, but previous studies have produced conflicting results as for the roles of the motor cortex in the mental rotation of body parts. We readdressed this issue by investigating brain activity during mental rotation of body parts along with various motor control tasks. Sixteen healthy volunteers were scanned with a 3-T MRI after a 30-min training session. Typical effects of rotation angles on reaction time were evident. During fMRI scanning, they perfomed mental rotation of hands and feet presented visually. In coparison with the foot rotation task, the hand rotation task activated lateral premotor cortex, occupying the rostral part of the activity for the hand movement task. Conversely, the foot rotation task involved supplementary motor areas and bilateral medial central zones at the border of primary motor cortex and somatosensory cortex. The present results support the involvement of somatotopically organized brain areas for mental rotation of body parts.

KAKENHI(17500210)

## O2P-F05 Effects of prefrontal and anterior cingulate lesions in monkeys on a Wisconsin Card Sorting Task (WCST) analogue

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Macaques were trained on a WCST analogue with two dimensions (color and shape). In each trial animals chose one of three stimuli that matched the sample according to the currently reinforced matching dimension. No cues were given as to the currently correct matching rule. Whenever performance reached a criterion, the rule was switched unannounced. After animals learned the task, we made bilateral lesions to orbitofrontal (OFC), principal sulcus (PS), superior-dorsolateral parts of the prefrontal cortex, or to the anterior cingulate sulcus (ACS). None of the lesions impaired control matching tasks. However, OFC, PS and ACS groups achieved significantly fewer rule-shifts per day in the WCST task. Further, different cognitive components of the task were impaired after these three lesions including more fragile rule memory in PS group, reduced sensitivity to positive feedback in OFC group, and quick immature responses in ACS group. Thus a distributed network supports flexible task performance.

## O2P-F06 Mnemonic function of lateral prefrontal cortex in conflict-induced behavioral adjustment

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To determine the role of anterior cingulate cortex (ACC) and dorsolateral prefrontal cortex (DLPFC) in conflict detection/resolution process, we conducted complementary lesion and single-cell recording studies with monkeys. The monkeys performed a matching task in which they had to resolve a conflict between two abstract rules (matching by shape and matching by color). Trials with conflict were intermingled with those without conflict. We found that the monkeys selected the target faster after they experienced a conflict in the previous trial. This conflict-induced behavioral adjustment persisted after bilateral lesion within ACC, but disappeared following lesions within DLPFC. A definite proportion of neurons in DLPFC showed different firing rates during the inter-trial period depending on the presence/absence of the conflict in the previous trial. These results show that DLPFC, but not ACC, is essential for the conflict-induced behavioral adjustment and suggest that maintenance of information of experienced conflict across trials is an important process mediated by DLPFC.

## O2P-F07 Neural correlates of human cognitive bias resulting in illogical inference

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Humans tend to make illogically inferred decisions, that is, after 'if A then B (AB pair)' relation was learned, human subjects readily, though not logically true, select 'A' when presented with 'B' (BA pair), whereas animals rarely exhibit this illogicality. Such a cognitive bias called 'symmetry', a constituent of 'stimulus equivalence', may be fundamental to human cognitive functions. BOLD signal was measured while the subject performed an inference task with a logical pair (AC) and three illogical pairs (BA, CB, and CA), after trained with AB and BC pairs outside the fMRI scanner. We found that the right prefrontal cortex was activated when the subject decided that the presented stimuli were in the abstract category consisting of A, B and C. The activation of bilateral intraparietal area was detected upon the abstract category formation in which illogically symmetrical inference was made and relational orders were ignored.

## O2P-F08 Corticotropin releasing factor increases attentional function in rats

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Several studies have shown that stress affects attentional function and corticotropin releasing factor (CRF) is closely related to stress responses. In the present study, we examined the relationship between CRF and attentional function using 5-choice serial reaction time task (5-CSRTT). Male Listar hooded rats were used. In the 5-CSRTT, a light briefly flashes (0.5 sec) in the one of the five holes. Rats were trained to poke their noses into the one hole in which the light flashed. Each test session consisted of 100 trials. Accuracy rate was used as the measure of attention. Human/rat CRF was intracerebroventricularly injected (0, 0.01, 0.1, 1 μg / 5 μl saline) 20 min before the beginning of the test trial. As a result, 0.1μg of CRF increased the accuracy rate in the 5-CSRTT. However, CRF did not affect other parameters indicating impulsivity, motivation, perseverative tendency, and motor function. These results showed that CRF selectively increases attentional function. The present results suggest that CRF may be involved in the stress-related changes of attention.



## O2P-F09 Elevation of circulating level of annexin A5 in dementia patients (molecular marker)

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Alzheimer disease (AD) differs from other dementia in their strong relation to amyloid β (A β). The A β dependent neurotoxicity involves perturbation of Ca2+ homeostasis of neural cells and may be distinct from other dementia. We exploited this dissimilarity by identifying the specific markers in patient blood plasma by their $Ca^{2+}$ dependent binding to acidic phospholipid liposome as a biological probe. A molecule found specifically in AD was revealed to be annexin A5 which possesses type II $Ca^{2+}$ binding sites. The plasma level of annexin A5 was significantly higher in AD than in controls. These results suggest that a relationship of this molecule to A β -induced cell damage may be interpreted in AD, providing a strategy to explore pathobiological markers for diagnosis of the serious disease.

18591303

## O2P-F10 The mechanism of memory improvement by Kihi-to

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We aimed to clarify the mechanism of Kampo formula, Kihi-to for memory impairment using an Alzheimer's disease model. We focused on calpain as one of target molecules of Kihi-to. Kihi-to (100 mg/kg/day, p.o.) or MDL28170 (calpain inhibitor, 10 mg/kg/day i.v.) was administered for 3 days at 10 days after the i.c.v. injection of A β (25-35) (25 nmol). Kihi-to and MDL28170-treated groups significantly recovered the memory acquisition in a water maze test. However, only a Kihi-to-treated group significantly improved the memory retention. A β (25-35)-induced impairment of the object recognition memory was also recovered by Kihi-to treatment, but not MDL28170. Kihi-to treatment reduced the calpain expression, and increased the calpastatin expression almost similarly to MDL28170. By contrast, densities of axons and dendrites, synapses and myelins were markedly recovered more by Kihi-to than by MDL28170. Kihi-to treatment also increased the kinesin-1, especially in the perirhinal cortex. In conclusion, Kihi-to may improve memory impairment at least partly by calpain inhibitor-like action and by up-regulation of kinesin-1.

## O2P-F11 RB1CC1 insufficiency causes neuronal atrophy through mTOR signaling alteration and involved in pathology of Alzheimer's diseases

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To clarify whether RB1CC1 insufficiency is involved in neuronal atrophy and Alzheimer's pathology, we investigated modifications of RB1CC1 as a possible cause of atrophy or death through the disturbance of mTOR signaling in Neuro-2a cells. We also evaluated the correlation between RB1CC1 and mTOR signaling in a series of Alzheimer's tissues. Though RB1CC1 introduction enhanced neurite growth, RNAi-mediated knockdown of RB1CC1 or rapamycin treatment caused neurite atrophy and apoptosis due to mTOR signaling reduction in differentiated Neuro-2a cells. Both TSC1 and RB1CC1 were equally functional and maintained mTOR signaling in 69% of Alzheimer's (9/13 cases) and all of normal brains (6/6 cases). However, scanty RB1CC1 expression caused phospho-S6 disappearance in 31% of Alzheimer's tissues (4/13 cases). These findings suggest that RB1CC1 insufficiency results in mTOR signaling repression through unbalanced TSC1 abundance, induces neuronal atrophy, and has implications for the pathogenesis of Alzheimer's disease.

KAKENHI (18012022)

## O2P-F12 Estrogen prevents cerebrovascular deficits by the lack of M5 muscarinic acetylcholine receptor through MAPK activation

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M5 muscarinic receptor (M5R) plays a crucial role in mediating acetylcholine-dependent dilation of cerebral vessels. Previously, we reported that male M5R knockout mice (M5R-/-) displayed constriction of cerebral arteries and reduced cerebral blood flow (CBF). However, female M5R-/- mice did not show any impairment in CBF. Furthermore, ovariectomized female M5R-/- mice showed impaired CBF similar to male M5R-/- mice. Although hippocampus-dependent memory was impaired in male M5R-/- mice, female M5R-/- mice displayed normal neuronal structures and hippocampus-dependent memory. Therefore, we hypothesized that the observed gender differences in M5R-/- mice might be caused by estrogen regulating cerebrovascular and neuronal function. NO release and MAPK activation were induced by 17 β -estradiol (E2) in mouse cerebral vessel derived cells. Our results suggest that estrogen prevents cerebrovascular deficits in M5R-/- mice through MAPK and NO production.

KAKENHI16300116

## O2P-F13 Aberrant expression of eNOS is associated with accumulation of beta-amyloid in brain ischemia-induced microvascular injury

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In the present study, we found that immunoreactivity of β-amyloid significantly increased in the microvessels 12 weeks after microsphere embolism in aged male rats. Likewise, Western blotting analyses using extracts from brain microvessels demonstrated that peroxynitrite level elevated 6-24 hours after brain embolism in aged rats, and more pronounced increase was found 8 and 12 weeks after ischemia, which preceded accumulation of β-amyloid peptide in the brain microvessels. The β-amyloid peptide accumulation in microvessels following brain embolism was apparently in part due to increase in eNOS overexpression and subsequently protein tyrosine nitration in brain microvessels. Furthermore, in 8-12 weeks after ischemic injury, β-amyloid peptide accumulation also coincided with GSK3 β activation and tau hyperphosphorylation in neurons, along with CaMKII activity down-regulation. Taken together, mild ischemia-induced peroxynitrite formation via eNOS expression in the microvessels likely triggers the early events of AD.

## O2P-F14 Conformation and intermolecular interaction of amyloid β-peptide in association with GM1 micelles as revealed by NMR spectroscopy

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The conversion of monomeric amyloid β-peptide (Aβ) to its aggregated, toxic form is considered to be a crucial step in Alzheimer's disease. It has been reported that Aβ specifically binds GM1 and consequently initiates Aβ aggregation by acting as a seed. To elucidate the molecular mechanisms underlying this phenomenon, we conducted structural analyses of Aβ(1-40) in association with GM1 micelles by use of ultra-high field NMR spectroscopy.
GM1-bound Aβ assumes an α-helical structure followed by a flexible C-terminal segment at low Aβ/GM1 ratios. At the Aβ/GM1 ratio of 1:15, where a makimum thioflavin T (ThT) fluorescence intensity was observed, the C-terminal segment of Aβ exhibited a multiple conformational states reactive with ThT.
Based on these results, we propose that the GM1 clusters induce inter-molecular interactions between the C-terminal regions of the Aβ molecules and thereby trigger the conformational changes leading to Aβ fibrillogenesis.

## O2P-F15 Human chorionic gonadotropin increases exploratory behaviour and impairs episodic-like memory in PS1M146V mice

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Elevated Luteinizing hormone (LH) levels occurring at menopause have been associated with increased risk of Alzheimer's disease (AD) in women. Our laboratory has previously shown that LH and its more potent analogue, human chorionic gonadotropin (hCG), increase the production of the neurotoxic Alzheimer's beta amyloid (Aβ) peptide. This study aimed to determine if chronic administration of hCG would functionally impair learning and memory in the PS1M146V mouse model of AD. Mice treated with hCG displayed significantly increased exploratory behaviour and impaired performance on the repeated reversals Morris water maze task, a measure of episodic-like memory. However performance on the classic Morris water maze task was unaffected. We are currently characterizing the biochemical and neuropathological changes underlying these functional impairments.

## O2P-F16 Microglial activation precedes tau pathology and neurodegeneration in a P301S mutant tau transgenic mouse

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The discovery of tau mutations pathogenic for parkinsonism linked to chromosome 17 (FTDP-17) revealed that tau abnormalities are sufficient to cause neurodegeneration, but it is still unclear tau inclusions directly cause neuronal dysfunction and death. To investigate the early pathological events before abnormal tau accumulation, we generated a P301S tau transgenic mouse. In this mouse, filamentous tau accumulation developed at 6 months of age, and progressively accumulated in association with striking neuronal loss by 9-12 moths of age. Interestingly, microglial activation was observed at 3-4 months of age, and simultaneity, synaptic loss developed, preceding astrogliosis, tangle formation and neuronal loss. Thus, our observations indicate that microglial activation and synaptic pathology may be the earliest manifestation of neurodegenerative tauopathies.

## O2P-G01 The contribution of the dorsolateral prefrontal cortex to executive function

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To date, neuroimaging studies while providing a new opportunity for testing executive function directly in humans, still suffer from the limitation that they are able to provide only neuronal correlates of executive performances. They cannot determine whether a specific cerebral area is specifically required for a certain executive task or whether its involvement is only an epiphenomenal activity. Today, we are able to transiently inhibit the activity of a given cortical area in the human brain by using repetitive transcranial magnetic stimulation (rTMS). Here, we applied rTMS over the human dorsolateral prefrontal cortex (DL-PFC) during different stages of the wisconsin card sorting task (WCST). We showed that rTMS of the human DL-PFC specifically influenced monitoring of the information held in the working memory without interfering with other executive stages of the WCST.

CIHR, FRSQ, REPRIC

## O2P-G02 Correlation between human brain activities and the autonomic nervous system elicited by emotional video movies

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Recent advance in brain and neuroscience indicates that the brain is the center of regulating psychological states of the body. Here we investigated the relationship of brain activities and body states by simultaneously measuring fMRI BOLD activities, heart rates, the pupil size, and the body temperature when human subjects observed video movies that contained emotional scenes such as excited, funny, fearful, and anger-induced ones. They were compared with ones under a condition in which the subjects watched relaxed video scenes. We found significant BOLD activities increased in the amygdalate complex, the entorhinal and orbitofrontal cortices as well as the dorsolateral prefrontal cortex when the excited video movies were seen. They were accompanied by increased heart rates as well as increased pupil size. No change was found as to the body temperature. The results showed the correlated activities among the cortical, limbic, and the autonomic nervious systems during the feeling of excitement.

## O2P-G03 Brain activities related to inferring familiar and unfamiliar persons` decision-makings: An fMRI study

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Objective: We investigated brain areas involved in inferring familiar or unfamiliar persons` decision-makings. Methods: The stimuli were visually presented questions about decision-making in daily life. In the two F and L conditions, subjects were asked to choose a decision of which they thought their friends and lay persons, respectively, would prefer. Results: We found significant activation in the right fusiform gyrus by F versus L comparison, and in the left temporo-parietal junction by vise versa ($P<0.05$, corrected). Conclusion: This study revealed that, when mentalizing others in a decision-making situation, fusiform gyrus is used for familiar persons, and that the left temporo-parietal junction is used for unfamiliar persons.

## O2P-G04 Abnormalities in white matter structure in autism spectrum disorders detected by diffusion tensor imaging

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This study tests the hypothesis that autism spectrum disorders (ASD) are attributable to impaired connectivity between cortical areas responsible for social and language function. Diffusion tensor imaging was performed on 20 male youths (ASD=10, control=10), group-matched for age, handedness, and IQ. Fractional anisotropy (FA) was compared between groups, and volumes of interest (VOIs) were identified using predetermined probability and cluster thresholds. Tractography confirmed anatomic location of VOIs. Reduced FA values were observed along the corpus callosum, cingulum, superior & inferior longitudinal fasciculi, inferior fronto-occipital fasciculus, and bilaterally near the fusiform gyri. All findings survived after covarying for age, IQ, and brain volume. These results provide evidence for impaired connectivity which may contribute to social and language impairments characteristic of ASD.

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## O2P-G05 Respiratory neuronal activities of the frog brainstem visualized *in vitro*

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Common frogs have two ventilation modes, buccal and lung. In the lung ventilation, buccal dilation (Bd) inhales air into the buccal cavity, and buccal constriction (Bc) pumps air into the lung. We recorded optical signals from the ventral medullary surface of juvenile frogs *in vitro* using a voltage-sensitive dye to visualize the respiratory neuronal network. Respiratory-related areas were identified bilaterally as longitudinal columns extending from the level of trigeminal nerve rootlets to the level of hypoglossal nerve rootlets. The respiratory neuronal activity started at the area near 'the buccal area' essential for the buccal ventilation. During Bc, pixels rostral to the IX/X cranial nerves depolarized, whereas pixels caudal to them tended to hyperpolarize. These results indicate that 1) Bd is initiated in the area close to the buccal area, and 2) the Bc-associated neuronal activity is mainly localized in the area close to 'the lung area' essential for the lung ventilation.

## O2P-G06 Difference in response selectivity between inhibitory and excitatory neurons in visual cortex, as revealed by *in vivo* two-photon functional $Ca^{2+}$ imaging

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GABAergic inhibition is hypothesized to play a role in the formation of orientation selectivity of visual cortical neurons. This hypothesis implies that response properties of GABA neurons may be different from those of excitatory neurons. To test this, we applied *in vivo* two-photon functional $Ca^{2+}$ imaging to GAD67-GFP knock-in mice, in which GABA neurons express enhanced green fluorescent protein. Astroglia were stained by an astrocyte specific dye. Thus, the three types of cells in layer II/III of the cortex were differentially identified and their responses to moving visual stimuli at different orientations were measured with changes in a $Ca^{2+}$-sensitive dye. We found that almost all GABA neurons have orientation-insensitive responses, while most of excitatory neurons have orientation-selective responses. These results are consistent with the hypothesis that non-oriented or broadly tuned cortical inhibition raises the firing threshold of excitatory neurons to make them selective.

SORST, KAKENHI(18700329)

## O2P-G07 Optical dissection and reconstruction of neural networks in the mouse cortex using a photo-reversible fluorescent protein

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During live imaging, it is often impossible to distinguish between individual neurons. We have taken advantage of Dronpa, a green fluorescent protein whose fluorescence can be erased with strong blue light, and reversibly highlighted with violet or ultraviolet light, in zebrafish. We erased the fluorescence entirely; re-highlighted it in a single neuron; then repeated this procedure for other single neurons. After collecting images of several individual neurons, we then recombined them in multiple pseudo-colors to reconstruct the network. (Aramaki, Hatta. Dev Dyn 235:2192-2199 (2006); Hatta, Tsujii, Omura, Nature Protocols 1: 960-967 (2006)). Here we tested the technique in mouse. We introduced Dronpa cDNA in the cortex by in utero electroporation. After birth, the brain was observed in live either from the surface or in slices. We could highlight individual neurons or glia to reconstruct them in pseudo-colors, indicating that the method can be effectively applied also to mammals.

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## O2P-G08 Pitfall in the study of intravital fluorescence imaging during ischemia

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In the study of intravital imaging, ischemia may affect the fluorescence intensity (FI), since the hemoglobin absorbs excitation (Ex) and emission (Em) lights. To analyze the effects of ischemia on FI, the fluorescent microspheres (0.2-μm diameter, similar Ex and Em to those of dye) were microinjected into brain and images were taken upon ischemia. In anesthetized rats, 4-vessle occlusion (4vo) and imaging were performed under a fiber-coupled confocal microscope for hippocampus CA3 (Hx), and a confocal microscope for cortex (Cx). 4vo decreased blood flow (mean±SD%, n=6) to 20±11 in Hx and 29±7 in Cx. FI (%) of spheres increased up to 108±5 in Hx (n=4) and 127±9 in Cx (n=3) during ischemia. FI of fluo-3, a $Ca^{2+}$ indicator, in Hx, and of HPF, a hydroxyl radical indicator, in Cx were corrected based on the results of spheres. They showed no change during ischemia, whereas the raw data showed increase. We emphasize: 1) the effect of ischemia should be carefully analyzed; and 2) our method using microsphere is useful.

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## O2P-G09 Activity of monkey amygdalar face neurons sensitive to both gaze and head directions

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To investigate a role of the amygdala (AM) in social cognition, neuronal activity was recorded from the monkey AM in a DNMS task of gaze direction. The stimuli consisted of facial photos of 2 persons with 2 head and 3 gaze directions. Of the 1091 neurons recorded, 44 responded selectively to the facial stimuli. The ratio of the head orientation-differential neurons was significantly larger in the gaze direction-differential neurons (21/25) than in the gaze-nondifferential neurons (10/19). Of 21 gaze direction- and head orientation-differential neurons, 15 displayed significant interactions between gaze and head direction. Factor analysis suggests 2 factors correlated with 2 head directions. Furthermore, the overall mean response magnitudes to directed gaze was significantly stronger than those to averted gaze in each head direction. These results suggest information of both gaze and head direction is integrated in the AM, and the AM plays a role to detect directed gaze.

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## O2P-G10 Personality-dependent cortical network during understanding other persons' mental states

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The present study investigated cortical activity and connectivity, and their personality dependence during theory of mind. False belief paradigms were adapted to event-related fMRI design. Consistent with previous studies, significant activity was observed in the medial prefrontal cortex (MPFC), temporo-parietal junction (TPJ), and temporal pole (TP). Network analysis for event-related activity also revealed significant functional connectivity between MPFC and TPJ. Correlation analysis was then performed to test whether the activity and connectivity was dependent on the personality traits of the subjects. Significant correlation was observed between TP activation and a personality score for Neuroticism by NEO-PI-R. Furthermore, significant correlation was observed between the Neuroticism score and the TP-MPFC connectivity and between the Neuroticism score and the TP-TPJ connectivity. These results suggest neuroticism-dependent TP network for social cognition.

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## O2P-G11 Neural basis of emotional facial recognition between parents and children

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The relationship between parents and children is one of the most important social relationships. We hypothesized that the emotional facial recognition between parents and children would induce specific neural activities. Ten pairs of mothers and their own adulthood daughters saw and judged on their happy, neutral and anger facial expressions each other during fMRI. Seven pairs of female friends received same procedure. The recognition of familial faces induced brain activities in the anterior paracingulate cortex, posterior cingulate cortex and bilateral posterior superior temporal sulcus (pSTS) while the recognition of friend's faces induced brain activities in the right pSTS. When daughters saw their own mothers' anger faces, the subgenual prefrontal cortex (sgPFC) deactivated compared to neutral faces. This deactivation was observed neither in mothers nor in friends. These results suggest that the sgPFC of children play a specific role in the anger facial recognition of parents.

Meiji Yasuda Mental Health Foundation

## O2P-G12 Who is to marry or to be a friend?: an fMRI study of social decision making

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During a social decision-making, the effects of social context and given information on cortical activation were examined using a factorial design fMRI experiment. Normal subject was presented with a pair of two opposite-sex faces (Fa) or snapshots of different daily behaviors (Bh). Each subject decided which picture gave better impression as a person to marry (Mr) or to be a friend (Fr). A brightness judgment task served as a control task. The right temporoparietal junction and the polar part of the medial prefrontal cortex were activated during Bh conditions only, suggesting a role of these regions in social decision based on behavior. The left amygdale was activated during MrFa condition only, probably reflecting the behavioral significance of the face in the marital selection.

## O2P-G13 Rearing in enriched environment calms down male rat copulatory activities in open field

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In the present study, we examined effects of rearing in enriched environment on sexual behavior in male rats. After weaning at day 25, 7 male Wistar rats were housed together in a large cage equipped with several objects (reaning in enriched environment, EER). Additional 7 males were housed 2 or 3 in a conventional cage without particular objects (conventional rearing, CR). At the age of 8 weeks, all males were tested open field activity. EER males showed lower locomotor activity than CR males. Following the open field test, sexual behavior was evaluated. CR males showed hyperactivity in sexual behavior, while EER males showed paced sexual behavior, significantly increased number of intromissions and longer ejaculation latency. We suggest that EER males have comparable sexual motivation; rather CR males are emotionally affected by such queer place, resulting in enhanced sexual activity.

## O2P-G14 Altered Drosophila aggression, locomotion, and neuromuscular excitability by isolated rearing.

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Social isolation is known to trigger a variety of behavioral and physiological changes in many animals, but the effects of isolation have not been well documented in Drosophila. Here we report that isolated rearing can modify the behavior of both adults and larvae in Drosophila. Wandering 3rd instar larvae isolated since early 1st instar displayed a high frequency of turning but rarely a long stretch of straight movement. Adult female flies isolated after eclosion showed higher frequency of aggressive behaviors than flies reared in a group. We also found that isolated rearing markedly altered activity-dependent facilitation of synaptic transmission. Upon repetitive stimulation, neuromuscular junction of group-reared larvae generally displayed a gradual synaptic facilitation, whereas isolation-reared larvae showed a sudden jump of a far greater amount of transmitter release correlated with supernumerary firing of motor axons. Further mutational analysis in Drosophila may unravel the molecular mechanisms of behavioral and physiological changes induced by social isolation.

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## O2P-G15 Mice lacking motopsin/PRSS12 gene showed abnormal social behavior.

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Mental retardation is a frequent disorder affecting 1-3 % of the population. Recently it has been reported that the deficit of a serine protease, motopsin/prss12, causes non-syndromic mental retardation. Functional deficit of adaptive behaviors including daily-living skills and social skills is characteristic symptom of this disease, in addition to low intelligent quotient (IQ). To understand function of motopsin on social behavior, motopsin knockout mouse was behaviorally investigated. Motopsin mutant mouse prolonged social interaction in three kinds of tests, such as social behavior test, social recognition test, and resident-intruder test. There was no difference between the mutant and wild type mice in food localization test (olfactory test) or exploration test. Our results suggest that motopsin modulates social behavior.

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## O2P-G16 Dysfunction of dynamical modulation of decision making process depending on social context in autism spectrum disorder

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To pursuit dysfunctional mechanism of autism, we conducted a behavioral experiment by computer game, and compared autistic subjects (AS) with normal subjects (NS). Subjects played a competitive game with a computer opponent that always made its decision randomly. There were two experimental conditions. One was that subjects believed their opponent as human. Another was that subjects believed their opponent as computer. Two conditions were switched and iterated in the experiment. In the result, NS could consistently modulate their decision making process depending on the social context, their opponent is human or not. On the contrary, AS could not keep this modulation at the end of experiment. This result shows that AS have some difficulty in dynamical modulation of decision making process depending on social context.



## O2P-H01 Pituitary adenylate cyclase-activating polypeptide is associated with schizophrenia

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Pituitary adenylate cyclase-activating polypeptide (PACAP) is a neuropeptide with neurotransmission modulating activity. Here, we provide evidence that genetic variants of the genes encoding PACAP and its receptor, PAC1, are associated with schizophrenia. This allele of the PACAP gene, which is overrepresented in schizophrenia patients, was associated with reduced hippocampal volume and poorer memory performance, neurobiological traits related to risk for schizophrenia. Abnormal behaviors in PACAP knockout mice, including elevated locomotor activity and deficits in prepulse inhibition of the startle response, were reversed by treatment with an atypical antipsychotic, risperidone. These convergent data suggest that alterations in PACAP signaling might contribute to the pathogenesis of schizophrenia.

## O2P-H02 IL-1 receptor antagonist (IL-1Ra) KO mice show anxiety-like behavior

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It is well known that the expression of IL-1 mRNA is induced by stress. Lines of evidence show that IL-1 administration influences the release of monoamines and these effects are attenuated by the pre-administration of IL-1Ra. These results strongly suggest that IL-1 directly influences monoamine contents in the brain and trigger the mental disease, such as anxiety and depression. It is unclear, however, the precise roles of IL-1 in the development of psychological diseases. In this study, we used the IL-1Ra KO mice and examined various behavioral tests. Because of the absence of IL-1Ra, physiological levels of IL-1 in the brain may activate the receptor excessively resulting in the similar situation as stressed animals. Very interestingly, at 20 weeks, anxiety-like behavior was appeared and was ameliorated by the administration of Diazepam. Further mechanisms of the induction pathways are now fully investigating.

Grant-in-Aid for JSPS Fellows

## O2P-H03 Identification and functional characterization of TRAP1 as a candidate gene for major depression

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Major depression is a multifactorial disease that is susceptible to both environmental and genetic factors. We demonstrate an association between SNPs on tumor necrosis factor receptor-associated protein 1 (TRAP1) gene and major depression. To elucidate the molecular function of TRAP1, we analyzed the effect of TRAP1 knockdown *in vitro*.

We report that TRAP1 alters the strength of cell adhesion via an alteration of N-cadherin and catenin expression, raising the possibility that TRAP1 may play a role in mood regulation via a modulation of synaptic contact.

## O2P-H04 Chronic effects of methamphetamine, MDMA and 5-MeO-DiPT on raphe serotonergic neurons in rat organotypic mesencephalic slice culture

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Recent evidence suggests the involvement of raphe serotonergic neurons in drug dependence and addiction to the psychostimulant, methamphetamine (METH),and recreational drugs such as 3,4-methylenedioxymethamphetamine (MDMA) or 5-MeO-DiPT. In this study, we examined the chronic effects of METH, MDMA and 5-MeO-DiPT on serotonin release and serotonergic neurotoxicity in rat organotypic mesencephalic slice cultures containing the raphe nuclei. Acute treatment with METH and MDMA dose-dependently increased serotonin release, while 5-MeO-DiPT did not. Sustained treatment with METH or MDMA for 6 days followed by their challenge (24 hr later) significantly augmented their serotonin-releasing effects. Sustained treatments with 5-MeO-DiPT decreased tissue contents of serotonin and [$^3$H]citalopram binding, while METH and MDMA had little effect. These results suggest that chronic treatment with METH or MDMA induces serotonergic sensitization, while 5-MeO-DiPT produces serotonergic neurotoxicity without serotonin release.

## O2P-H05 Regulation of DISC1 through phosphorylation by ERK

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Disrupted-In-Schizophrenia 1 (DISC1) is a candidate gene for susceptibility to schizophrenia. Previously, we reported that Kinesin-1, a motor protein of anterograde axonal transport, as a novel DISC1-interacting protein. Kinesin-1 interacts with the NUDEL/LIS1/14-3-3epsilon complex or Grb2 through DISC1. The knockdown of DISC1 inhibits the accumulation of the NUDEL complex and Grb2 at the axons and axon elongation. DISC1 appears to regulate the localization of the NUDEL complex and Grb2 into the axons as a cargo receptor for axon elongation. However, it remains unknown how the functions of DISC1 are regulated. Here, we found that the phosphorylation of DISC1 was increased by treatment with a phosphatase inhibitor, Calyculin A, and this phosphorylation was canceled by treatment with an ERK inhibitor. DISC1 was directly phosphorylated by ERK in a cell-free system. The physiological meanings of DISC1 phosphorylation by ERK will be discussed.

※KAKENHI (18700363) (17024024) (15GS0319)

## O2P-H06 Neurogenesis correlates sensorimotor gating via activation of hippocampal Akt

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We have shown previously that irradiation in rat yields schizophrenic features comprising: 1) a decrease in Akt protein levels in hippocampus, 2) impaired prepulse inhibition of acoustic startle reflex, and 3) reduced neural stem cell proliferation in hippocampus. These results have suggested that neurogenesis abnormality may relate to hippocampal Akt down-regulation and sensorimotor gating dysfunction in rat. Here we show that intraventricular infusion of cytosine arabinoside (Ara-C) also yields the same features in rat. More, we find that hippocampal Akt transgene repairs impairment of prepulse inhibition in these rats by using adenovirus vector. These trials reveal that neurogenesis correlates sensorimotor gating via activation of hippocampal Akt, and which may be useful to establish the Akt transgene therapy for schizophrenia.

## O2P-H07 Identification of *YWHAE*, a gene encoding 14-3-3ε, as a novel susceptibility gene for schizophrenia

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Disrupted-In-Schizophrenia 1 (DISC1) is one of the candidate genes for the susceptibility to schizophrenia. We investigated the genetic association between DISC1 interacting molecules and schizophrenia in a Japanese population, and identified a promoter SNP for *YWHAE*, a gene encoding 14-3-3ε. The minor allele frequencies of this SNP were higher in controls. mRNA transcription and protein expression of 14-3-3ε were increased in the lymphocytes of healthy control subjects harboring heterozygous and homozygous minor allele. *Ywhae*$^{+/-}$ mice showed cognitive dysfunctions that are observed in schizophrenia patients. These results suggest that *YWHAE* is a novel susceptibility gene to schizophrenia that works protectively.

※KAKENHI15GS0319, KAKENHI17024024, 21st Century Centre of Excellence Program

## O2P-H08 Expression and genomic organization of methamphetamine-induced transcript *mrt3* and *as-mrt3* in the rat central nervous system

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Recently accumulated evidence has indicated that the behavioral sensitization induced by psychostimulants in human and rodents is associated with the gene expression that is involved in the functional and dysfunctional change of the neural plasticity. We have identified developmentally regulated methamphetamine responsive transcript *mrt3* from the rat cerebral cortex. Characterization of the genomic structure and gene expression suggested that *mrt3* and *as-mrt3*, which was located on the opposite strand of *mrt3*, were both expressed predominantly in the cerebral cortex in the rat. Cellular localization of *as-mrt3* product further suggested a possible role in the nuclear function including gene regulation and protein degradation in the brain. The expression of *mrt3* and *as-mrt3* in the cerebral cortex might be involved in the pathophysiology of initiation of behavioral sensitization and schizophrenic symptoms.

※KAKENHI18591274

## O2P-H09 Chronic administration of nicotine retards the development of morphine dependency and tolerance in mice

Abbas Haghparast, Nima Naderi, Abbas Khani, Amir Alizadeh, Fereshteh Motamedi

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Investigating the interaction between nicotinic and opioid receptors is of great interest to both basic mechanistic and clinical views. 80 male mice were rendered dependent on morphine. We used repeated injection of nicotine (0.001,0.01,0.1,1,2 mg/kg;ip) 15 min prior to each morphine application along with control groups. Tail Flick latency pre- and post-morphine injection and number of jumping after injection of naloxone (5 mg/kg;ip), 2 h after morphine application in the 4th day, were used as indexes for development of tolerance and dependency, respectively. Results showed that nicotine doses at 2,1 and 0.1 mg/kg significantly reversed the development of tolerance. The most effective dose of nicotine that significantly decreased the number of jumping is 0.1 mg/kg. So, our study indicates that repeated injection of nicotine reverses the development of morphine tolerance and dependency. Dose-response curve of nicotine vs. decrease in development of morphine dependency interestingly does not follow a unidirectional manner.

## O2P-H10 Nicotine and morphine interactions; new protocol for morphine dependency in mice

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Clinical use of morphine in pain management is controversial. Both nicotine and morphine are widely abused. We investigated the influence of nicotine on the development of morphine tolerance and dependence. Along with concurrent nicotine administration, 160 male mice were rendered dependent on morphine according both the method previously described by Marshall and Smith-Grahame (M groups) and 50% doses of morphine in the method (M50 groups). Tail flick latency before and after morphine injection and withdrawal Jumping test were used for the assessment of tolerance and dependency, respectively. We found: first, 50% doses of morphine in Marshall method is sufficient to induce dependency but not tolerance in mice; second, nicotine attenuates the development of tolerance in dose-dependent manner in M groups and significantly decreases whithdrawal jumping in both M and M50 groups in a biphasic profile (V-shape) manner.

## O2P-H11 A role of dorsolateral prefrontal cortex in cue-induced craving

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Concomitant activation of ACC and DLPFC has been linked to cue-reactivity, but their differences are not known. We studied the role of DLPFC using rTMS that reduces excitability of the targeted cortex. Ten smokers received four fMRI scans on different days, during which they viewed smoking-cue video. Each of four fMRI was scanned under different conditions of expectancy (non-expectant vs. expectant) and rTMS (true vs. sham) performed immediately before scan. Repeated TMS was targeted previously identified left DLPFC. In the true TMS condition, we found lowered self-reporting cue-induced craving and lack of cue-induced BOLD increase in the ACC and a correlation between craving and BOLD signal in the left DLPFC, both observed during the expectant and the sham rTMS conditions. The left DLPFC became less responsive to input from the contralateral DLPFC and medial temporal regions, but not to ACC. These findings suggest that DLPFC associates perceived drug availability with craving in a positive feed-forward manner.

## O2P-H12 CD38 can influence oxytocin release by regulating intracellular calcium concentration.

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CD38 has been identified as a transmembrane protein that catalyzes the formation of cyclic ADP-ribose and nicotinic acid adenine dinucleotide phosphate (NAADP) by ADP-ribosyl cyclase from NAD+ and NAD phosphate. cADPR and NAADP mobilize Ca2+ from ryanodine-sensitive intracellular Ca2+stores. However, the role of CD38-dependent cADPR or NAADP signaling pathway of Ca2+ regulation on posterior pituitary hormone release has not yet been described. Here we report that oxytocin is released by high potassium depolarization stimulation from the soma and presynaptic terminals of oxytocinergic neurons. Meanwhile, ryanodine and bromo-cyclic ADP ribose can decrease the amount of the oxytocin release due to depolarization from the posterior lobe of pituitary. Correspondingly, the calcium increase produced by high potassium depolarization can be decreased by ryanodine and bromo-cyclic ADP ribose too. Therefore we concluded that CD38-cADPR signaling pathway play a role in oxytocin release.

※Grant-in-Aid for COE Research



## O2P-H13 Presence of gabazine-resistant $GABA_A$ receptor currents and their modulation by neurosteroids in rat GnRH (LHRH) neurons

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GnRH neurons constitute the final common pathway in the neuroendocrine control of reproduction. In the regulation of GnRH neurons, GABA is likely to play a major role by acting extrasynaptic $GABA_A$ receptors ($GABA_A$Rs). To test this possibility, we recorded $GABA_A$R currents by means of perforated patch clamp technique in overnight-cultured GnRH neurons prepared from GnRH-EGFP transgenic rats. $GABA_A$R antagonist gabazine was used to distinguish extrasynaptic $GABA_A$R currents from synaptic ones. Low concentration of gabazine (500 nM) inhibited the $GABA_A$R currents by 40%, suggesting that GnRH neurons express substantial amount of extrasynaptic $GABA_A$Rs. Furthermore the $GABA_A$R currents were augmented by neurosteroids allopregnanolone and THDOC. The results suggest that extrasynaptic $GABA_A$Rs are expressed in GnRH neurons and neurosteroids modulate them, thereby regulating their cell excitability.

※KAKENHI16590180,18390070,18590226

## O2P-H14 Role of glucocorticoid receptor in vision and behavior

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The glucocorticoid receptor (GR) is a transcription factor involved in a broad range of biological processes, including stress and energy homeostasis. In spite of its well described functions, what is not clear is the behavioral consequence of the genetic ablation of its transcriptional activity. Here, we took advantage of a novel GR mutant with disrupted GR transcriptional activity. We found that the visual sensitivity, as measured by their optokinetic response (OKR), was unaltered under conditions of continuous light, but was severely perturbed by an exposure to darkness. Electrophysiological recordings localized the visual defect to the retina, with impaired cone photoreceptor response and bipolar cell response. In addition to the defects in visual adaptation, the mutant adult fish showed elevated freezing behavior when transferred to a confined space, presumably due to a increased stress response. Our results indicate that GR transactivation regulates adaptation to light changes in the retina and also a stress-induced behavioral response in adult zebrafish.

## O2P-H15 A physiological role for endogenous nociceptin in the regulation of food intake and weight gain

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Prior study shows that administration of the novel opioid peptide, nociceptin, affects pain, addiction, anxiety and feeding responses. Here, we studied nociceptin receptor knockout (NOP KO) mice to understand the physiological role of nociceptin in the control of feeding. We found that despite a slightly higher preference for high fat diet when presented under choice conditions when food-deprived, NOP KO mice consumed less of a high fat diet when presented ad libitum, yet tended towards higher body weights. In contrast, NOP KO mice consumed normal amounts of regular and high-sucrose diets presented ad libitum. We also found that NOP KO mice recovered their body weight very poorly following extended periods of food deprivation, though showed a normal anorectic response to peripheral leptin infusion. These data suggest that endogenous nociceptin plays a specific physiological role in controlling the metabolic and/or behavioral responses to food.

KAKENHI

## O2P-J01 Epigenetic regulation of critical period plasticity in visual cortex

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Neuronal circuits are shaped by experience during intervals of early postnatal brain development called critical periods (CP). We hypothesized that formation of CP windows reflects a change in epigenetic state using histone acetylation and DNA methylation. Histone H3 acetylation (AcH3) was observed in the pre-CP of developing visual cortex (VC), diminished by the peak of the CP and into adulthood. DNA demethylation (deMe) then peaked during the CP. We assayed two mouse models of delayed CP onset: dark-rearing from birth to adulthood (DR P56) and GAD 65 deletion (GAD65 KO). AcH3 and deMe were observed in both models. We tested our findings by causing histone hyperacetylation using valproic acid (VPA) in adult mice experiencing monocular deprivation (MD). Functional analysis revealed that VPA-injected adult mice had shifted their OD after MD. These results suggest a novel treatment for amylobia in adulthood.

※RIKEN BSI & Grant-In-Aid for Scientific Research (MEXT)

## O2P-J02 Effect of potassium on the transcription of GSK3 $\alpha$ and GSK3 $\beta$

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GSK3 $\alpha$ and GSK3 $\beta$ are known as key enzymes involved in Alzheimer's disease. Recently, it has been reported that the transcription of GSK3 $\beta$ was promoted with the APP fragment. However, the mechanism of GSK3 $\alpha$ and GSK3 $\beta$ gene transcription in physiological condition has been not clear. Then we analyzed the promoter activity of GSK3 $\alpha$ and GSK3 $\beta$ gene with the luciferase-reporter gene assay. The constructs containing the firefly luciferase gene directed by the promoter of GSK3 $\alpha$ and GSK3 $\beta$ were introduced stably into neuronal cells (SH-SY5Y) and non-neuronal cells (HEK293). The cells were treated with potassium chloride that induces depolarization. In SH-SY5Y cells, the promoter activities of GSK3 $\alpha$ and GSK3 $\beta$ were significantly changed comparing with cells without treatment. However, no change was observed in HEK293 cells. This suggests the neuron-specific factors involved in the transcription of GSK3 $\alpha$ and GSK3 $\beta$ genes. We discuss the molecular mechanism of GSK3 $\alpha$ and GSK3 $\beta$ gene expression.

## O2P-J03 Molecular characterization of MKL1/2, coactivators for SRF in morphology and transcription of neurons

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MKL(megakaryoblastic leukemia)1/2, which are able to bind to G-actin, function as SRF (serum response factor) co-activators play important roles in differentiating muscle cells. However, little is known about the roles of MKL1/2 in neuronal cells. Here we have demonstrated the expression and characterization of MKL1/2 in rodent brains. MKL1/2 are expressed in forebrain neurons and up-regulated in developing mouse brain. Overexpression of MKL1/2 in cortical neurons resulted in an increase of SRF transcriptional responses although the subcellular localization is mainly cytoplasmic. Furthermore, RNAi experiments revealed that endogenous MKL1/2 contributes to neuronal SRF-mediated transcription and MKL1 plays a crucial role in maintenance or formation of dendritic morphology. These findings suggest the linkage of change in neuronal cell shape to SRF-mediated gene expression.

※KAKENHI(17790056), Takeda Science Foundation, Narishige Neuroscience Research Foundation, Uehara Memorial Foundation

## O2P-J04 Imprinting of the cerebral cytochrome P450s and developmental neurotoxicity of lindane

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The present study attempted to understand developmental neurotoxicity of the pesticide by investigating alterations in the expression of cerebral cytochrome P450s (CYPs), known to be associated with several of the physiological functions of the brain, and behavioral effects in the offspring exposed in utero to low doses of lindane, an organochlorine insecticide. Prenatal exposure to low doses (1/1400th or 1/700th or 1/350th of LD50 of lindane) to pregnant dams from gestation day 5 to 21 (GD5-21) produced dose-dependent increase in the mRNA expression of cerebral and hepatic CYP1A1, 1A2, 2B1, 2B2 and 2E1 isoenzymes in the offspring during postnatal development. Similar increase was observed in the protein expression of these CYPs and the activity of the marker enzymes of these CYP isoforms. The persistence in the increase in expression of these CYPs upto adulthood in brain and liver of the exposed offspring suggested the potential of the pesticide to imprint the expression of CYPs in brain and liver of the offspring.

## O2P-J05 Spatiotemporal coding of gustatory information in hindbrain

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The peripheral taste nerves code taste qualities based on spatiotemporal activity patterns across the nerves. The gustatory information transmitted from the peripheral nerves is processed in hindbrain based on the neuron types which have multiple sensitivity to taste qualities. In order to study how the gustatory neuron types formed in the hindbrain reflect the temporal patterns of peripheral nerve activities, we propose a neural network model of the hindbrain. The short-term synaptic plasticity in the model can extract separately the spatiotemporal features of phasic and tonic responses evoked by the peripheral neurons. The firing pattern elicited by the phasic responses contributes to fast perception of taste qualities, whereas that elicited by the tonic response is useful for further analysis of taste quality and intensity.

## O2P-J06 G protein-coupled novel temperature sensing mechanism

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Animal receives environmental stimuli such as light, smell, and temperature by using sensory neurons. While light and smell are received by G protein-coupled receptors, temperature is received by TRP channel. Here we show novel temperature sensing mechanism through G protein-coupled signaling in *C. elegans*. The loss-of-function mutation in inhibitor of G protein, RGS, encoded by *eat-16* gene led to abnormal thermotaxis, which was caused by malfunction of AWC sensory neuron. Thermotactic defect in *eat-16* mutant was suppressed by the mutations in G protein(ODR-3)-coupled signaling in AWC. Calcium imaging revealed that AWC neuron in wild-type was activated by temperature changes. By contrast, AWC activity was significantly decreased in the G protein ODR-3 mutant. These results suggest that G protein-coupled signaling is required for thermosensation. We also found that TRP channel OSM-9 localizing at AWC cell body is involved in continuity of neural response.

※KAKENHI 18770005

## O2P-J07 An LCR *H* for the mouse odorant receptor (OR) genes acts in *cis*, and not in *trans*

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In the mouse, over 1000 OR genes are clustered at 44 different loci among most of the chromosomes. We have previously reported a locus control region (LCR) as a 2kb homology (*H*) sequence conserved between the mouse and human, that regulates the *MOR28* cluster. Now, we narrowed down the essential sequences in the *H* to a 124bp region. Mutation analyses of the 124bp region revealed 2 homeodomain sequences that are essential for the *H* activity. Recently, Axel's group reported that the *H* may act not only in *cis* but also in *trans*. A *trans*-acting LCR may help ensure the 1 neuron-1 OR rule if we assume that the *H* is the only one LCR in the OR gene family. However, our knockout experiment demonstrated that deletion of the *H* abolished the expression of 3 proximal zone 4 genes in the *MOR28* cluster, indicating the presence of another LCR for the downstream zone 1 genes. No other OR gene clusters, including the other allele of the *MOR28* cluster, were affected by the *H*-knockout. Based on these results, we propose that the OR genes are regulated by multiple LCRs that act in *cis*, and not in *trans*.

※KAKENHI(17570172)

## O2P-J08 A new candidate pheromone receptor contributing to the male courtship behavior of Drosophila

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Courtship behavior of Drosophila heavily depends on chemoreception. A gustatory receptor (Gr) gene, Gr32a, is a candidate for pheromone receptor. We report here that male courtship behavior is impaired when the Gr32a-expressing neurons are prevented from functioning. To block synaptic transmission in Gr32a-expressing neurons, the tetanus toxin (TNT) was expressed under the control of the Gr32a promoter (Gr32a>TNT) The depression of Gr32a-expressing neuron activities had no significant effects on the basic courtship activity. However, it leads to a specific defect in the wing extension/vibration step during the courtship. The control male flies normally extend one of their wings while another wing is rigidly placed in its closed position. On the contrary, the Gr32a>TNT flies often extend both wings simultaneously. The present result suggests that male flies determine which of the wings to extend or to close based on the lateralized pheromonal input from Gr32a-expressing neurons.

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## O2P-J09 Decoding an innate aversive quality from an odor map in the mouse olfactory bulb

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The binding signals of odorants received in the olfactory epithelium (OE) are converted to a topographic map of activated glomeruli in the OB. In order to study how the odor map is decoded, we generated mutant mice in which OSNs in a specific area of the OE were depleted by targeted expression of the diphtheria toxin gene. In the zone 1 (dorso-medial zone)-depleted animal, the antero-dorsal domain in the OB was devoid of axon termini and without glomerular structures. The zone 1-depleted mouse displayed no aversive responses to aversive compounds, although it was capable of detecting and discriminating them. In contrast, the class II -depleted mouse, in which the glomeruli were formed only in the most antero-dorsal part of zone1, demonstrated aversive behavior. These results indicate that zone 1 glomeruli in the OB have a separate role in decoding the odor map, and that there may be genetically-programmed, hard wired neural circuits for innately aversive odorants.



## O2P-J10 Behavioral state-dependent change of granule-to-mitral inhibition in the rat olfactory bulb

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Behavioral states regulate information processing modes at local neuronal circuits, which are reflected by the change in the local EEG pattern. Here we asked whether dendrodendritic synaptic interactions in the olfactory bulb depend on the brain states. In the brain of urethane-anesthetized rats that showed periodic alternation of neocortical EEG pattern, granule-to-mitral dendrodendritic synaptic inhibition was much larger during slow-wave state than during fast-wave state. The state-dependent shift in the dendrodendritic inhibition was observed also in freely behaving rats, being greatly enhanced during slow-wave sleep state compared to awake states. These results provide the evidence for the behavioral state-dependent modulation of the dendrodendritic synaptic interaction and suggest that olfactory bulb networks can engage in different information processing modes via the states of the dendrodendritic inhibition.

※KAKENHI(18100004, 17023012), JSPS Research Fellowships

## O2P-J11 A neural network model of the olfactory system of mice:Simulated attention behavior taking the odorant map into account

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An odor is composed of a combination of 400 thousands kinds of odorant molecules. However, it was found that the mice focus on a few odorant molecules to identify the odors, reducing the dimension of information. This behavior is called attention. Our research is aimed at elucidating the algorithm of attention and applying it to the odorant processing apparatus in the future. In this presentation, we propose a novel artificial neural network model based on the biological structure of the olfactory system. The experimental data of odorant map showing the relationship between the odorant molecules and the firing patterns of neurons are also taken into the account. The simulation results showed that the inhibition, which occurred on a part of the odorant map, could cause the attention.

※The 21st century COE program of JSPS on the Hyper Human Technology toward the 21st Century Industrial Revolution

## O2P-J12 The re-presentation of conditioned stimulus after acquisition of conditioned taste aversion increase ventral pallidum GABA release in rats.

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Previous our studies provided the evidence that $GABA_A$ receptors in the ventral pallidum (VP) are involved in palatability shift from ingestive to aversive in conditioned taste aversion (CTA). Present studies explored the possibility that the re-presentation of CS after acquisition of CTA might affect the GABA releasein the VP. Initially, rats received a pairing of 5 mM saccharin (CS) and an i.p. injection of 0.15M LiCl (conditioned group) or saline (unconditioned group). After this conditioning, microdialysis was carried out in the rats re-presented with CS via an intra-oral cannula. GABA was measured by HPLC with OPA-sulfite precolumn derivatization. The CS re-presentation rapidly induced aversive behaviors in conditioned group, but not in unconditioned group. The conditioned group showed significant increase in the GABA release (by 135%), the unconditioned group did not. These data suggest that palatability shift as a function of CTA increases the VP GABA release.

※KAKENHI(17730431)

## O2P-J13 Adaptor protein-dependent trafficking of high-affinity choline transporter

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High-affinity choline transporter (CHT) is efficiently transported to cholinergic axon terminals. We have previously shown that most of the CHT is associated with synaptic vesicles rather than synaptic plasma membrane. Here, we analyzed intracellular distribution of CHT in adaptor protein-3 (AP-3) deficient mouse (mocha mouse). In the mouse, endosomal granular structures were intensely labeled with CHT antibody, indicating possible deficit in CHT trafficking. Western blot analysis revealed that CHT on synaptic vesicle in mocha mice was decreased by 70% compared with wild-type mice. However no significant difference in synaptosomal choline uptake activity was detected, implicating the existence of large pool for CHT. Futermore most of CHT is found associated with synaptic-like micro-vesicles (SLMV) in CHT-overexpressed PC12 cells. The amounts of CHT detected on SLMV were greatly reduced by treating the cell with agents which halt AP-3, demonstrating AP-3 involvement in CHT trafficking.

## O2P-J14 Signal transport from synapse to the nucleus

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Long-lasting forms of synaptic plasticity requires gene expression. Recent studies showed that various kinds of transcription factors are located in synaptic sites of axons and/or dendrites, including CREB and NFκB, which are involved in synaptic plasticity. This indicates that signals should be transported from the synapse to the nucleus.Previously we reported that importin $\alpha$ is translocated from dendrites to the nucleus by stimulating cultured hippocampal neurons with KCl. In the present study, we focused on classical active nuclear import pathway, regulated by importins that recognize nuclear localization signals (NLS). We found that mutations in NLS of CREB and NFκB caused changes in the subcellular localization; these molecules widely distributed on neurites of cultured hippocampal neurons in addition to soma. We also investigated effects of importin on the subcellular localization of CERB and NFκB by using RNA interference.

## O2P-J15 Coupling profile of the metabotropic glutamate receptor1 is regulated by the C-terminal tail

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The metabotropic glutamate receptor 1 (mGluR1) is known to couple to different types of G proteins, such as Gq, Gs and Gi/o. We visualized the multi-signals via mGluR1 by simultaneous monitoring of [cAMP]$_i$ and [$Ca^{2+}$]$_i$ with a combination of a FRET-based senor and indo-1, respectively. We observed that in CHO cells mGluR1 $\alpha$ activated Gq and Gs pathways, while in HEK cells Gq and Gi/o pathways. In contrast, a short form splice variant, mGluR1 $\beta$, did not clearly activate Gs in CHO cells and Gi/o in HEK cells, although it activated Gq fully in both cells. The elimination of a positive charge cluster, RRKK located on the proximal C-tail, from mGluR1 $\beta$ restored the Gs and Gi/o couplings. These results suggested that the positive charge cluster suppresses the coupling of the short form mGluR1 (mGluR1 $\beta$) with Gs and Gi/o. Furthermore, as the positive charge cluster is also included in mGluR1 $\alpha$, we speculated that the region is masked by the long C-terminal tail in mGluR1 $\alpha$.

## O2P-J16 Phosphorylation of $GABA_B$ receptor R2 subunit by 5′AMP-activated protein kinase through opening NMDA receptor channel.

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Functional $GABA_B$ receptors are heterodimers formed $GABA_BR1$ and $GABA_BR2$. We have studied 5′-AMP activated protein kinase (AMPK) association to $GABA_B$ receptors intracellular domains. Although it is classically believed that AMPK is activated by intracellular 5′-AMP accumulated via ATP consumption, it has been appeared that calcium dependent pathway activates AMPK directly. In this study, we investigated whether $GABA_BR2$ phosphorylation by AMPK is regulated by intracellular calcium levels via opening of NMDA receptors in cultured cortical neurons. Exposure to NMDA for 15 min led to AMPK phsophorylation at T172, with concomitantly facilitating phosphorylation at S783 on $GABA_BR2$ subunit (p783). The facilitation of p783 was blocked by AP5 and MK-801. These data suggests that a fascination of excitatory transmission and/or an increase of intracellular calcium level may modulate $GABA_B$ receptor function through the activation of AMPK.

※The Nakajima Foundation

## O2P-K01 Efficient transduction of oligodendrocytes with AAV8 vectors

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Among various new adeno-associated virus (AAV) serotypes, serotype 8 vectors (AAV8) are of particular interest because they can transduce not only neurons but also glial cells in the murine brain. We evaluated the transduction characteristics of GFP-expressing AAV8 vectors in primary hippocampal cultures and in the adult rat brain. In mixed neuronal/glial cell cultures, prepared from the hippocampi of E17 Wistar Crj rats, 39% of Olig2-immunoreactive cells were positive for GFP. In Wistar rats, many GFP-positive oligodendrocytes were observed in the brain after injection of AAV8 vectors with the myelin basic protein promoter. AAV8 vectors transduce oligodendrocytes efficiently and thus are useful for researching dysmylinating or demylinating diseases of the brain.

※KAKENHI (18050401)

## O2P-K02 Lentiviral-vector-mediated gene rescue -Functional recovery by region-specific expression of CD38 gene-

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Most genes are expressed in variety of tissues, and therefore even if a simple knock-out of a gene leads to some abnormal phenotype, it is difficult to determine the region responsible for the abnormal phenotype. Here we show that lentiviral-vector-mediated re-expression of the human CD38 gene specifically into the hypothalamus rescued the plasma and central oxytocin concentration. Consistently, defects of social memory were restored in mice treated with lentiviral vectors expressing the human CD38, but not a mutant CD38 that contains one-third of the control enzyme activity. Thus, lentiviral-vector-mediated rescue of a defective gene at a specific site in a knock-out mouse is a powerful means to dissect the roles of a gene of unknown function.

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## O2P-K03 Development of dendritic membrane-targeting signals using lentiviral vectors

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We previously reported that lentivirus with synapsin I promoter expresses green fluorescent protein (GFP) specifically in neurons. The expression was, however, so weak that neuronal processes were not well visualized. In the present study, we tried to develop dendritic membrane-targeted GFP to label the infected neurons in a Golgi stain-like manner. Fatty acylation sites were first tested for membrane-targeting of GFP. GFP with Fyn N-terminal myristoylation/palmitoylation site (myrGFP) was localized to the dendritic membrane more efficiently than GFP with GAP43 N-terminal palmitoylation site. However, myrGFP was distributed at not only dendritic, but also axonal membranes. Thus, we next examined six kinds of putative dendrite-targeting signals. Of these signals, C-terminal of low density lipoprotein receptor ($LDLR_{CT}$) was the most efficient in targeting myrGFP to the dendritic membrane. Furthermore, we generated transgenic mice expressing myrGFP-$LDLR_{CT}$ and investigated the effect of the dendritic membrane-targeting signals.

## O2P-K04 Labeling history of neuronal activity and functional asymmetry of fasciculus retroflexus

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Experiences are encoded in neuronal circuits as their neuronal activity; however, to elucidate structure and function of the responsible circuits has been difficult. Immediate early genes (IEGs) promoters are useful for converting neuronal activity into gene expression. To label experiences, we designed genetic tracers with a fluorescent protein on cellular membrane (e.g. GAP43-Venus) under the control of the IEGs promoters (e.g. *zif268* promoter) and generated their transgenic mice. Because the GAP43-Venus has long life-time, it is accumulated on neurons activated; thus, the gene constructs operate to sum the neuronal activity and to label the experiences. The labeled sites and their fluorescent intensity represent quality and quantity of the experiences, respectively. Here we report that fasciculus retroflexus sinistra was transiently labeled asymmetrically on postnatal day 13 afterward in the transgenic mice, which indicates validity of the strategy and functional asymmetry of CNS.

※KAKENHI 13210021

## O2P-K05 Isolation of Song nuclei specific genes by using cDNA microarray in Bengalese finch

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Songbirds, like humans, are unusual animals in that they exhibit vocal learning and song syntax complexity. We studied the molecular mechanisms of song syntax complexity in the Bengalese finch using gene expression profiles with cDNA microarray. We constructed a Bengalese finch cDNA microarray containing 3,840 independent known genes. We tried to isolate the song-nuclei-specific genes using the cDNA microarray, as the song nuclei are neural substrate for birdsong. The HVC is an especially important nucleus in vocal learning and singing behavior. Therefore, we focused on isolating HVC-specific genes. The HVC and caudal nidopallium (cNidopallium: neighboring areas of the HVC) in adult male Bengalese finches were microdissected and the gene expression patterns were compared using two-color competitive microarray hybridization. We identified 34 genes highly expressed in the HVC and some genes were confirmed by in situ hybridization. Consequently, we found 15 genes that were expressed strongly in the HVC compared to the cNidopallium, and most of these genes were also expressed in other song nuclei.



## O2P-K06 Temporal relations of auditory selectivity among song-related thalamo-cortical brain loop nuclei in Bengalese finches

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There are a set of brain nuclei in songbirds that are related with song learning and production. Among these, HVC and mMAN represents a part of the thalamo-cortical loop connection, and output of HVC is fed to the input of mMAN via thalamus, output of mMAN then becomes input to HVC. Here we report first electrophysiological study of the auditory response of mMAN neurons and correlations with the activities of HVC neurons in anesthetized Bengalese finch males. The auditory responses of mMAN neurons showed strong BOS (bird's own song) selectivity that rivals to HVC. Event correlation data from simultaneous recordings of mMAN and HVC neurons showed that some activities of mMAN neurons preceded the firing of HVC neurons. These findings are in parallel with anatomical connection between the two nuclei and suggest that mMAN neurons could modulate some activities of HVC.

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## O2P-K07 Spatial temporal credit assignment neural network model

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In [1] I presented a model where replayed recurrent activity in an association layer generated at goals provided a novel way to form "context" cells representing a value function on experienced paths within the given action selection policy. Indeed replay of experienced place cells in CA3 has been observed by Foster and Wilson. The model is based on the loop projections from the hippocampus and cortex (pFC) to the striatum with feedback from SNr/VTA and uses a novel threshold LTP/LTD model of the D1/D2 dopamine receptors, compatible with Goto and Grace results of a balance of tonic and phasic dopamine at the hippocampal and pFC accumbens synapses. Here I show the modifyable action selection system[2] taught by the context cell learns the correct policy and makes a transition from exploratory to goal directed behaviour[3] in both egocentric and allocentric plus maze tasks with multiple variable reward magnitude goals. The observed response of task and expert neurons of the dorsal and ventral striatum, and some behavioural characteristics, are reproduced.[1] A.Ponzi, IEICE NC Technical Report. 103;163:19-24 (July 2006). [2] A.Ponzi, IJCNN 2007. [3] A.Ponzi, CNS 2007 abstract.

## O2P-K08 Can theta burst transcranial magnetic stimulation affect cortico-muscular synchronization in humans?

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Recently it has shown that a novel paradigm of repetitive transcranial magnetic stimulation (rTMS), "theta-burst stimulation" (TBS) can induce long-lasting suppression/facilitation of the neuronal excitability in humans, even with the stimulation duration shorter than those of conventional rTMS protocols. We examined effects of TBS on the functional coupling between left motor cortex and right first dorsal interosseous (FDI) muscle by means of the coherence between electroencephalography (EEG) and electromyography (EMG). Before and after the delivery of TBS over either primary motor area (PMA) or sensory motor area (SMA), EMG from FDI muscle and EEG from twenty scalp sites were recorded while the subject performed a weak isometric hand contraction. EEG-EMG coherence localizing for the C3 scalp site and at beta band (13-30Hz) significantly suppressed 30-60 minutes after the TBS on PMA, but not that on SMA, and recovered to the original level in 90-120 minutes.

## O2P-K09 Dynamic online target control of SSVEP-based brain-computer interface with multiple commands

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Brain computer interfaces (BCI) present a novel challenge to neuroscientists with their strict requirements for high reliability, real-time analysis and quantitative classification of multiple brain activity patterns. A BCI paradigm which is able to satisfy most of these requirements is the Steady-State Visual Evoked Potential (SSVEP) approach in which multiple flickering patterns evoke synchronized steady-state brain activity. In this study, we propose a multi-stage procedure for real time BCI with an implementation for up to 8 commands. Our EEG-based BCI system enables a user to navigate a small car on a screen in real time and to execute additional actions. This approach offers several novel points, such as integrated moving patterns for selective attention and minimal eye movement, as well as an online blind-source separation (BSS) unit for artifact rejection, improved feature selection and a fuzzy classifier. The modular and adaptive structure of the BCI platform allows an extension to an even higher number of commands, as well as to other BCI paradigms.

## O2P-K10 Single-trial based neural prediction of immediate and delayed go/no-go decisions.

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To develop an algorithm of neural mind reading, we simultaneously recorded from a small group (2 to 5) of single neurons in the superior colliculus (SC) while the monkey performed immediate and delayed go/no-go tasks, which required the monkey to either make a saccade toward a peripheral stimulus (go) or maintain fixation (no-go) depending upon stimulus color. We performed a regression analysis on the single-trial activity in order to generate a virtual decision function (VDF) which was designed to reflect the progress of decision-making. Post hoc analyses by VDF predicted the monkey's choice in approximately 90% of immediate and 85% of delayed trials. These results suggest that monitoring of SC activity has sufficient capacity to predict go/no-go decisions on a trial-by-trial basis, which is an ideal candidate to serve for a cognitive brain-machine interface (BMI).

Kakenhi (18019048, RPH), the Sumitomo Foundation (RPH) and the NIH Grant (R01-EY-08212, MAS).

## O2P-K11 Virtual force/tactile sensos for dynamic and cooperative interaction with a robot using user's biological signals

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This article proposes a new approach to virtual realization of force/tactile senses in machines possessing no such sensors. The key of our approach is that a robot exploits the user's biological signals, and therefore this approach is not dependent on the controlled object and is expected to be widely applied for a variety of machines including robots.This article describes an example robotic system comprised of an industrial robot manipulator, a motion capture system, and a surface EMG measurement apparatus.By making use of the user's surface EMG and postural information in real-time, we show that the robot that has no force/tactile sensors can behave similarly to the one possessing senses over its body.The advantage of our approach is highlighted by a task in which the robot and a user cooperatively hold and move a heavy load.

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## O2P-K12 A neuro-robotics study in the real world: Formation of the cognitive map by using theta phase coding

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The rodent hippocampus is known to represent the cognitive map. Theoretical models have been proposed for explaining how it is formed and the model by using theta phase coding in particular enables to form it only few minutes, as real-time computation. However, it is still unclear whether the computation is available for real-time cognitive map formation interacting with the real environment. We hypothesized that theta phase coding works well even in the real world and investigate it by using a mobile robot, Khepera, with colored floors. In the experiment, the one-time running behavior is successfully encoded into asymmetric connection in the hippocampal network, as the sequence of visual scenes. After a brief exploration, the associative connections are formed to represent the spatial relationship of places obtained from egocentric views. We report that theta phase coding is available for real-time cognitive map formation in the real environment with simple visual cues.

## O2P-K13 4-D imaging of secretory granule movements in chromaffin cells

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Our previous studies on the effects of mycalolide B (MB) or TPA in chromaffin cells implicated actin-myosin interaction and PKC in the regulation of vesicle recruitment for exocytosis. In this study, we characterized the vesicular dynamics in 4 dimensions (4-D) using a confocal microscope (LSM-5Live). We examined the effects of MB and TPA on the track motions of LysoTrackerGreen DND26-labeled secretory granules during each 2min of resting (Rst), depolarizing stimulation (Dep), and post-stimulation (P-Dep).

In Rst in TPA-treated cells, the vesicles moved within the larger space without changes in the velocity. The spatial movement of vesicles was increased in P-Dep as we reported previously, and the activation of the movement in P-Dep was decreased by MB.

These results support our previous findings that actin-myosin interaction and PKC are involved in the spatio-temporal regulation of the vesicle recruitment. Therefore, the spatial movement of the vesicles seems to be functionally linked to such a process for exocytosis.

## O2P-K14 Simulation study: Changes in the neural network of *C. elegans* induced by food-NaCl associative learning

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Typically, *C. elegans* moves toward NaCl (a soluble attractant). However, after exposure to NaCl in the absence of food, this nematode avoids the chemical (food-NaCl associative learning). To understand the changes induced by associative learning at the neural network level, we modeled a chemotactic network based on the actual neural connections and simulated the responses prior to and after food-NaCl associative learning on a computer. We estimated the changes that occurred in the nervous system by comparing the neural network connection weights prior to and after associative learning. The results revealed that signal transduction in AIY interneurons differed prior to and after food-NaCl associative learning. This corresponds to the experimental findings of previous studies suggesting that AIY neurons are important for food-NaCl associative learning.

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## O2P-K16 A Bayesian analysis of the time dependent irregularity of neuronal firing

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We have recently proposed a coefficient of local variation, termed Lv, for measuring the irregularity of neuronal firing. Cortical neurons have been classified into several firing types according to the values of Lv. This could be performed by confirming that the temporal variation of Lv for each neuron is small compared to the variation among neurons (Shinomoto, Shima and Tanji, Neural Computation, 15 (2003) 2823-2842). On the other hand, it was reported that the firing irregularity significantly changes in time during the behavioral task . In this contribution, we examine the apparent contradiction of those controversial issues by means of a Bayesian analysis: In our new framework, the rate and irregularity of the spikes are assumed to be slowly changing in time, and are determined under the Bayes method, given the spike data. We are going to apply this Bayes method to experimentally recorded spike trains by focusing on the degree of irregularity fluctuation.

KAKENHI(16300068 and 18020015)

# 抄録 Abstracts

## ポスター発表
## Poster Session Abstracts

## P1-a01 Effects univibrissae rearing on response properties of barrel cortical neurons in C-Fiber-depleted rats

s. Azizollahi[1], A. Shamsizade[1,2], V. Shey Bani[1], S. Arabzadeh[1], M. Afarinesh[1]

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C-fiber depletion increases low threshold somatosensory mechanoreceptive fields and decreases lateral inhibition in barrel cortex. In this study, we investigated the role of intact C-fibers in response properties of spared barrel neurons (layer IV) following experience dependent plasticity in barrel cortex. C-fiber depletion was induced by neonatal treatment with capsaicin (50 mg/kg I.P). Plucking of all whiskers but D2 started at first post partum day. Neuronal ON and OFF responses were computed following principal whisker (PW) and adjacent whisker (AW) deflections. In both c-fiber depleted and vehicle treated rats, whisker plucking increased ON responses to spared PW deflection. Neuronal ON responses to deflection of deprived AW were decreased in vehicle treated rats but in C-fiber depleted rats they did not decrease. Neuronal OFF responses showed similar result to ON responses. These results suggest that neonatal C-fiber depletion can modulate response properties of spared barrel neurons.

## P1-a02 Low concentration of nicotine triggers a novel regulatory pathway in neuromuscular synapses

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Nicotine in $10^{-8}$ M concentration was found to significantly reduce amplitude and quantal content of evoked end-plate potentials generated in short trains at stimulation frequencies of 4, 7 and 50 Hz in cut neuro-muscular mouse diaphragm preparation. Nicotine-induced inhibition was prevented by $\alpha$-cobratoxin, an antagonist of $\alpha$7-type neuronal nicotinic acetylcholine receptor (nAChR), and by apamin, small conductance calcium-activated potassium channel (SK-channel) antagonist, as well as found to be $Ca^{2+}$-dependent. This suggests a presence of a yet undescribed pool of presynaptic $\alpha$7 nACHRs, that can participate in a regulatory negative feedback pathway: activation of $\alpha$7 nAChRs by acetylcholine or other agonists, such as nicotine, results in a local $Ca^{2+}$ influx, coupled with activation of SK-channels. Resulting $K^+$ outflow hyperpolarizes presynaptic nerve terminal, reducing acetylcholine secretion in synaptic active zones. The suggested mechanism could be aimed at lowering mediator loss during prolonged high-frequency synaptic activity.

RFBR 02-04-48126

## P1-a03 Single action potential-evoked $Ca^{2+}$ transients at the calyx of Held presynaptic terminal

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$Ca^{2+}$ transient at presynaptic nerve terminals triggers transmitter release. Amplitude, kinetics and distribution of $Ca^{2+}$ transient regulate release modality, but the property of $Ca^{2+}$ transient within the nerve terminal remains elusive. To investigate this issue, we tried imaging for a single action potential-evoked $Ca^{2+}$ transient at a giant presynaptic terminal, the calyx of Held, using Oregon Green BAPTA 5N. Confocal spot measurement enabled detection of $Ca^{2+}$ gradients within the terminal. $Ca^{2+}$ transients were mainly observed at the synaptic site of the terminal, but the spatial distribution was sparser in older animals (P13-14 vs. P7-P8). Amplitude of the transient was smaller in older animals. The rise time of transients was equivalent to action potential duration, whereas decay time was regulated by $Ca^{2+}$ buffer concentration at the both age groups. These differences may underlie the developmental changes in synaptic efficacy at this synapse.

## P1-a04 Regulation of the differentiation of excitatory synapses by the SALM family of cell adhesion-like molecules

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Synaptic cell adhesion molecules (CAMs) are known to play key roles in various aspects of synaptic structures and functions. We herein report the identification of a family of cell adhesion-like molecules termed SALM that interacts with the abundant postsynaptic density (PSD) protein PSD-95. SALM2, a SALM isoform, distributes to excitatory, but not inhibitory, synaptic sites. Overexpression of SALM2 increases the number of excitatory synapses and dendritic spines. Bead-induced direct aggregation of SALM2 results in coclustering of PSD-95 and other postsynaptic proteins, including GKAP and AMPA receptors. Knockdown of SALM2 by RNA interference reduces the number of excitatory synapses and dendritic spines and the frequency, but not amplitude, of miniature excitatory postsynaptic currents. These results suggest that SALM2 is an important regulator of the differentiation of excitatory synapses.

## P1-a05 A 120 kDa protein localized in the synaptic membrane is cleaved by plasticity-related protease neuropsin

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Neuropsin (NP) is an extracellular serine protease known to participate in synaptic plasticity. Extracellular proteolysis of NP plays an important role in the modification of synaptic transmission and synaptic structure, but little is known about the molecular mechanism. Here, we report that NP specifically cleaves the 120 kDa protein, which was abundantly localized to the Triton X-100-insoluble fraction of the hippocampus, amygdala and olfactory bulb in the adult mice. When the activated recombinant NP added to crude synaptic membrane fraction, the 120 kDa protein was immediately cleaved and 37 kDa fragment was released in a dose- and time-dependent manner. The cleavage was blocked by serine protease inhibitor, leupeptin and PMSF, and NP STOP, but not other protease inhibitors, implying this step is NP specific proteolysis. These results indicate that 120 kDa protein could be an important factor of NP-dependent extracellular remodeling of synapse.

21st century COE research

## P1-a06 Impairment of afferent synaptic inputs for mechanical and cold sensation to substantia gelatinosa neurons of the rat spinal dorsal horn through cadherin-8 expressing synaptic glomeruli

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In vivo and slice patch-clamp recordings were made from spinal substantia gelatinosa neurons in wild and cad8-/- mice to compare the modality-dependent synaptic inputs with or without cad8. SG neurons with cad8 received synaptic inputs from menthol-sensitive afferents and exhibited a barrage of EPSCs in response to cutaneous pinch stimuli. In cad8-/- mice, the amplitude of C fiber-evoked EPSCs and the frequency of miniature EPSCs from menthol-sensitive (TRPM8 expressing) afferents were decreased. Behavioral analysis showed reduced sensitivities for mechanical and cold noxious stimuli in cad8-/- mice. The present results suggest that cad8 has a crucial role for forming functional synaptic connections between SG neurons and primary afferent fibers for mechanical and cold noxious sensation.

KAKENHI

## P1-a07 Optical imaging of glutamate release at individual hippocampal synapses

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At individual glutamatergic synapses, the amount of released glutamate per action potential determines the synaptic strength. However, the direct measurement of synaptically released glutamate has not been done. To measure glutamate release at single synapses, we developed a novel fluorescent glutamate probe called EOS (Glutamate Optical Sensor). Electrically evoked glutamate release was successfully detected at synapses in cultured hippocampal neurons. We found that released glutamate varied in amount among synapses. Also, changes in release probability by manipulating extracellular calcium concentration were associated with changes in the amount of glutamate released at individual synapses. Futhermore, phorbol ester increased the amount of glutamate released at individual synapses. These findings suggest that single hippocampal synapses contain several release sites. In summary, we evaluated the functional release properties of each individual synapses by using EOS.

## P1-a08 Characterization of a transneuronal cytokine family Cbln

Takatoshi Iijima[1], Eriko Miura[2], Keiko Matsuda[1], Yuichi Kamekawa[1], Masahiko Watanabe[2], Michisuke Yuzaki[1]

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Cbln1, a member of the C1q and tumor necrosis factor superfamily, plays crucial roles as a cerebellar granule cell-derived transneuronal regulator for synapse integrity and plasticity in Purkinje cells. Although other Cbln family members Cbln2-Cbln4 have distinct spatial and temporal patterns of expression throughout the central nervous system, their biochemical and biological properties have remained largely uncharacterized. Here, we demonstrated that Cbln2 and Cbln4 were secreted glycoproteins like Cbln1. In contrast, Cbln3 was not secreted when expressed alone, and retained in the endoplasmic reticulum or *cis*-Golgi. In wild-type granule cells expressing Cbln1 and Cbln3, Cbln3 proteins were secreted and reached postsynaptic sites on Purkinje cell dendrites, while Cbln3 was dramatically degraded in *cbln1*-null granule cells. These results indicate that Cbln2-Cbln4 may also serve as a trans-neuronal regulator of synaptic functions in various brain regions.

KAKENHI (18700324)

## P1-a09 Change in superfusion rate alters excitatory synaptic transmission in rat hippocampal slice

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In CA1 pyramidal neurons of hippocampal slice, increase in rate of superfusion of the medium (fast superfusion) markedly augments the amplitude of EPSC evoked by stimulation of Schaffer collateral. However, the fast superfusion did not affect both AMPA-induced current response and IPSC evoked by stimulation of striatum oriens in the same cell. Paired pulse ratio of the EPSC decreased during the fast superfusion and a rightward shift was observed in the peak of the amplitude distribution histogram of the evoked-EPSCs in low [Ca2+]o. Moreover, application of either A1 receptor antagonist DPCPX or ecto-ATPase blocker ARL67156 augmented the EPSC amplitude, and subsequent fast superfusion did not cause additional augmentation of the EPSC amplitude in the presence of DPCPX or ARL67156. These results suggest that major cause for the augmenting effect may be increase in glutamate release from the presynaptic terminal due to decrease in concentration of adenosine normaly existing nearby the synapse and inhibiting the transmitter release.

## P1-a10 Enhancement by phospholipase $A_2$ activation of glycinergic and GABAergic inhibitory transmission in rat substantia gelatinosa neurons

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Phospholipase $A_2$ ($PLA_2$) activation enhances glutamatergic excitatory transmission in substantia gelatinosa (SG) neurons. The present study examined a detail of the effect of melittin on inhibitory transmission in SG neurons in adult rat spinal cord slices by using the whole-cell patch-clamp technique. Melittin enhanced the amplitude and frequency of GABAergic and glycinergic spontaneous IPSCs (sIPSCs). The GABAergic but not glycinergic transmission enhancement was suppressed by tetrodotoxin or glutamate-receptor antagonists. These actions were reduced in extent by $PLA_2$ inhibitors. These results indicate that $PLA_2$ activation enhances glycinergic transmission in SG neurons and that GABAergic transmission is enhanced by the facilitation of excitatory transmission by $PLA_2$ activation and subsequent increases in glutamate-receptor activities resulting in the excitation of SG neurons. It remains to be examined what kinds of neurotransmitters are involved in the GABAergic transmission enhancement.

## P1-a11 Cbln1 directly induces excitatory presynaptic assembly in cerebellar Purkinje cells

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Cbln1 is a hexameric glycoprotein secreted from cerebellar granule cells. Although Cbln1-/- mice show impaired synapse formation and plasticity at parallel fiber-Purkinje cell synapses, its direct function has not been assessed. Here, we examined the effect of exogenous Cbln1 on Purkinje cell synapses. We found that Cbln1 in the medium increased synaptophysin-positive puncta that colocalized with dendrites of cultured Purkinje cells. Whole-cell patch-clamp recordings revealed that incubation of cerebellar slices with Cbln1 for as short as 8 hours could restore the reduced frequency, but not the amplitude, of miniature excitatory postsynaptic currents in Cbln1-/- Purkinje cells. Interestingly, mutant Cbln1, in which two cysteine residues were replaced with alanine to prevent hexamerization, could not rescue the structural and functional properties of synapses in Cbln1-/- Purkinje cells. These results indicate that Cbln1 forms a hexamer and directly induces excitatory presynaptic assembly.

## P1-a12 $Ca^{2+}$ clearance mechanisms in presynaptic terminals : $Ca^{2+}$ uptake to mitochondria started when $[Ca^{2+}]_i$ increased to a critical level

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To investigate the $Ca^{2+}$ clearance mechanisms in presynaptic terminals, we measured $Ca^{2+}$ dynamics during nerve stimulation in frog neuromuscular junctions. During 100Hz tetanus for 4 sec in a normal Ringer's solution (1.8mM $Ca^{2+}$, 0mM $Mg^{2+}$), $[Ca^{2+}]_i$ increased in two phases, steep rising for about 0.2 sec then slow increasing. When mitochondria were inhibited by rotenone and oligomycin, slow phase disappeared and $[Ca^{2+}]_i$ increased monotony with rapid rate. Membrane voltage imaging of mitochondria with TMRE suggest that $Ca^{2+}$ uptake to mitochondria accompanied by depolarization of membrane potential. When tetanus frequency was reduced to 20Hz, $[Ca^{2+}]_i$ dynamics did not affected by rotenone and oligomycin, however, CCCP increased $[Ca^{2+}]_i$ during tetanus. The effect of rotenone and oligomycin appeared when $[Ca^{2+}]_i$ increased more than 0.6 μM during 40Hz tetanus. Inhibition of $Ca^{2+}$ uptake to ER did not change $[Ca^{2+}]_i$ during tetanus at 40Hz. Each inhibition of plasma membrane $Ca^{2+}$ pump and $Na^+/Ca^{2+}$ exchanger increased $[Ca^{2+}]_i$ during tetanus at 20Hz.

## P1-a13 Differential distribution of parvalbumin positive boutons for two populations of pyramidal cells

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In rat perirhinal cortex, parvalbumin (PV+) boutons form a honeycomb at the border of layers (L) 1 and 2, with PV+ walls. By double labeling for PV and MAP2 (for dendrites), we found that apical dendrites of L2 pyramidal cells occurred mainly in the PV+ walls. As distal dendrites of L5 pyramidal cells occupy PV-sparse hollows, the dendrites of L2 cells are predicted to have more PV+ contacts than distal dendrites of L5 cells. Here, we used confocal analysis to quantify PV+ boutons (double labeled for VGAT) in contact with EGFP-expressing pyramidal cells in a 60 μm-wide zone of L2. L2 dendrites (n=11) had an average of 19.5 contacts (range=7-38), but L5 dendrites (n=11) had an average of only 5.0 (range=0-10). This confirms a differential density of PV+ boutons for L2 vs. L5 cells at the level of the L2 honeycomb. Boutons were sometimes clustered, but typically single, with 7-30 μm gaps. A next question concerns the relative perisomatic density of PV+ boutons for these two populations. We expect a population-specific proximal-distal ratio of PV+ boutons, significant for modes of synaptic integration.

## P1-a14 Enhancement by PAR-1 activation of glutamatergic excitatory transmission in adult rat substantia gelatinosa neurons

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We have previously reported that proteinase-activated receptor-1 (PAR-1) agonist peptide SFLLRN reversibly increases the frequency of spontaneous EPSC (sEPSC) without a change in the amplitude in substantia gelatinosa (SG) neurons. In the present study, we further examined the PAR-1 agonist mediated enhancement of excitatory transmission by applying the whole-cell patch-clamp technique to SG neurons in adult rat spinal cord slices. The presynaptic effect of SFLLRN was resistant to tetrodotoxin, and was not seen in the presence of PAR-1 antagonist peptide YFLLRNP. Peptide TFLLR, which is more selective to PAR-1 than SFLLRN, also increased sEPSC frequency without a change in its amplitude; this action was dose-dependent. Similar facilitatory effect was seen by endogenous PAR-1 agonist proteinase thrombin. These results indicate that PAR-1 activation in nerve terminals enhances the spontaneous release of L-glutamate onto SG neurons. It remains to be examined how PAR-1 activation results in enhancing L-glutamate release.

## P1-a15 Dendritic dimensions of cortical nonpyramidal cells

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Cortical nonpyramidal cells (NP) are composed of several dendritic types different in branching patterns. In addition, local morphologies are also important for the input interaction and transmittance to somata. We measured dendritic perimeter, cross sectional area and synapse density along dendrites of each NP subtype: a FS basket cell, double bouquet cell, Martinotti cell, and large basket cell. Many portions of the dendrites from each NP subtype were reconstructed three-dimensionally from serial ultra-thin sections. The synaptic densities on their dendrites were almost uniform from proximal to distal dendrites, but different between subtypes. At branching, a parent cross sectional area was almost equal to the sum of those of two daughter branches. Input conductance estimation using the circumference and cross sectional areas suggested impedance matching between parent and daughter branches at the node according to Rall's model. Importance of the impedance matching is studied by theoretical analysis.

KAKENHI

## P1-a16 Differential involvement of $GABA_A$ IPSPs in inter-columnar communication in the barrel and insular cortices

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In comparison with the barrel cortex, the primary gustatory area in the insular cortex is thought to have much less distinct functional columns. We examined the differences in the spatiotemporal pattern of excitation spread between the barrel and insular cortices, using voltage-sensitive dye imaging. The excitation evoked by stimulation of layer IV of the barrel cortex spread upwardly into layer II/III, where the excitation spread horizontally into neighboring columns partially, leaving the layer V/VI unexcited. By contrast, the excitation in layer IV of the insular cortex spread bi-directionally into layers II/III and V/VI, while the horizontal spread in layer II/III was much less prominent. Bicuculline application markedly enlarged the extent of horizontal spread in layer II/III of the barrel cortex, but not the case for the insular cortex. These results suggest that the involvement of $GABA_A$ receptors in the lateral inhibition in layers II/III is much more prominent in the barrel cortex than in the insular cortex.

## P1-b01 Firing pattern dependent specificity of cortical feed-forward subnetworks

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Previously, we reported that L5 pyramidal cells could be divided into three types by their firing patterns and were correlated with their subcortical target. Here, we examined whether these types of cells form specific functional subnetworks. Whole cell recordings were obtained from rat frontal cortical slices. First, we characterized physiological properties of L2/3 pyramidal cells. Like those of L5 cells, firing patterns of L2/3 cells were divided into three types showing no spike frequency adaptation or no frequency adaptation with burst at the initial phase or strong frequency adaptation during current injections. Dual recordings of L5 and 2/3 cells revealed all types of L5 cells received inputs from L2/3 cells fired with no spike adaptation. However, L2/3 cells fired with strong spike adaptation or burst made synapses with similar types of L5 cells, respectively. We also examined if these types of L5 cells share common inputs from L2/3 cells using dual recordings and single cell stimulation. Our results suggest networks from L2/3 to 5 have specificity depending on firing patterns.

KAKENHI(18700328)

## P1-b02 Evidence of nigral inhibitory inputs to GABAergic neurons in mouse superior colliculus

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The existence of direct inhibitory inputs from the substantia nigra pars reticulata (SNr), was examined in GABAergic neurons in the intermediate gray layer (SGI) of the superior colliculus (SC). For this purpose, we performed tract-tracing studies and whole-cell patch-clamp recordings using GAD67-GFP knock-in mice. Confocal fluorescence microscopic analysis revealed that after BDA injection into the SNr anterogradely labeled fibers from the SNr made synaptic contacts on GFP-positive SGI neurons. Voltage-clamp recordings showed that electrical stimulation of areas just ventral to the SC, where fibers from the SNr are located, evoked IPSCs in GFP-positive neurons. The IPSCs were monosynaptic and sensitive to a $GABA_A$ receptor antagonist. These results indicated that GABAergic SGI neurons receive nigral inibitory inputs and suggest that the SNr may not merely modulate the balance of excitation and inhibition in the SC local circuit.

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## P1-b03 $\alpha_2$-Adrenoceptor activation reduces GABAergic synaptic transmission in the developing mouse cerebellar cortex

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Our previous study has demonstrated that a selective $\alpha_2$-adrenoceptor agonist clonidine has a partial inhibitory effect on spontaneous IPSCs (sIPSCs) in mouse cerebellar Purkinje cells (PCs). Here, we found that the inhibitory effect of clonidine on sIPSCs was developmentally enhanced. Clonidine greatly reduced the frequency of spontaneous action potential (AP) in the molecular layer interneurons (MLIs), while the intensive attenuation was not age-dependently. To address the discrepancy between these inhibitory effects, we examined sIPSCs in PCs and AP in MLIs developmentally. During early postnatal days (<P11), only sIPSC was detectable and clonidine did not influence sIPSCs. Thereafter, AP appeared, and both the sIPSC frequency and the firing rate considerably increased. Together, the developmental increase in the clonidine-mediated inhibition of sIPSCs is attributed to an age-dependent increment of AP-derived sIPSCs and selective inhibition of MLIs' AP. Therefore, $\alpha_2$-adrenoceptor controls MLI activity and thus modulates PC activity.

KAKENHI (16700344)

## P1-b04 Analysis of innervation-dependent gene expression during synaptogenesis

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Reciprocal induction between pre- and postsynaptic cells is believed to be important for the differentiation of the synaptic structures. The induction process likely involves regulation of gene transcription, yet the identity and function of genes that are induced during this process remain largely unknown. Here, we aim to investigate the mechanisms of how innervation-dependent gene expression in the postsynaptic cell supports synaptic differentiation, using the neuromuscular system in Drosophila. We performed microarray analysis of individually isolated muscle cells to detect changes of gene expression in the postsynaptic cells. By comparing expression profiles of muscles before and after innervation and muscles in mutants that lack innervation, we identified 82 candidate genes that are potentially regulated by nerve innervation. These included extracellular matrix components, transcription factors and various enzymes. We are now performing genetic analyses of these genes to elucidate their roles in synaptic differentiation.

## P1-b05 SAD kinase directly phosphorylates the active zone proteins Bassoon and Piccolo.

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The presynaptic active zone (AZ) is the principal site where neurotransmitter release occurs in a $Ca^{2+}$-dependent manner. Several components of the AZ have been identified such as CAST, ELKS, Bassoon, Piccolo, RIM1 and Munc13-1. SAD is a serine/threonine kinase specifically expressed in the brain where it is tightly associated with synaptic vesicles and the AZ cytomatrix. Recently, we have reported that SAD kinase phosphorylates RIM1, but not Munc13-1, implicated in neurotransmitter release. In order to elucidate phosphorylation mechanisms at the AZ, we have attempted to search for other potential substrates by SAD. With a database search we found two consensus motifs possibly recognized by SAD, and then noticed the presence of these motifs in the AZ proteins Bassoon and Piccolo. In vitro phosphorylation assay indeed showed that SAD directly phosphorylates some regions of Bassoon and Piccolo. Our data suggest that a phosphorylation network may occur at the AZ, which might contribute to the assembly, maintenance, and function of the AZ.

## P1-b06 Interaction of complexin II with parkin

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ComplexinII (CPLX2) is soluble protein localized in presynapses, and regulates neurotransmitter release, but the mechanisms in detail have been obscure. To make clear the role of CPLX2 in presynapses, we screened the proteins interact with CPLX2 through the essential region for the preferential localization of CPLX2.
Pull down of solubilized rat brain P2 fraction with GST-CPLX2 fusion protein followed by proteome analysis by MALDI TOF MS revealed that several proteins bind to CPLX2. One of the candidates that were thought to function in presynapses, is Parkin which is a gene product of PARK2. The in vitro interaction assay by using Parkin and CPLX2 expressed in E. coli showed direct interaction of CPLX2 with Parkin. In addition, we obtained Ube1c and UBC7 as other candidates in proteome analysis and Yeast Two Hybrid analysis, respectively. These result show the possibility that the proteasome pathway with Parkin is involved in presynaptic function through the direct binding with CPLX2.

## P1-b07 An excitatory and inhibitory synapse ratio on cortical nonpyramidal cell subtypes of the rat

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Cortical GABAergic nonpyramidal cells regulate the activities of pyramidal cells. The nonpyramidal cells are also controlled by combined excitatory and inhibitory afferents. We investigated the ratio and density of excitatory and inhibitory synapses on nonpyramidal cell subtypes, such as parvalbumin (PV), calretinin (CR) and substance P receptor (SPR)-positive cell. These three subtypes were stained by preembedding immunohistochemistry. Successive ultrathin sections were also stained by postembedding GABA immunohistochemistry and reconstructed three-dimensionally. Individual nonpyramidal cells received both GABA-negative asymmetrical and GABA-positive symmetrical synapses, but their ratios varied between the subtypes. GABAergic terminals occupied ~3% among total dendritic synapses and ~25% among total somatic synapses in PV neurons, 35% and 74% in CR neurons, 17% and 16% in SPR neurons, respectively. These results indicate that the excitatory and inhibitory input balance in nonpyramidal cells is differentiated between the somata and dendrites, but also between the cell subtypes.

## P1-b08 Presynaptic release probability and GluR2 abundance at individual synapses in cultured hippocampal neurons

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Strength of an excitatory synapse, or synaptic weight, is dependent on both the presynaptic release probability (Pr) and the surface expression level of postsynaptic AMPA receptors. But it has not been clear how these factors are related or balanced at individual synapses. To address this question, we take advantage of live imaging in cultured hippocampal neurons. We estimate Pr by labeling presynaptic vesicles with a styryl dye, FM1-43, while we visualize postsynaptic surface AMPA receptors with anti-GluR2 antibody against the extracellular domain. We find no apparent correlation between Pr and GluR2 level. Such lack of correlation suggests that the balance of Pr and GluR2 abundance at individual synapses is neither fixed nor compensated in cultured hippocampal neurons.

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## P1-b09 Age-related changes in ionized calcium-binding adapter molecule 1 in the gerbil hippocampal CA1 region

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Microglia respond rapidly to a variety of alterations in the microenvironment of the brain and act as sensors for pathological events in the brain. we investigated the age-dependent changes in the levels of ionized calcium-binding adapter molecule 1(Iba-1), a microglial marker, in CA1 region of the gerbil hippocampus. Iba-1 immunoreactive microglia were detected in the hippocampal CA1 region of the postnatal month 1(PM 1) group. Iba-1 positive microglia were morphologically inactive between the PM 1 and PM 12 stages. Some were present in active form in the PM 18 and PM 24 groups. The Iba-1 protein levels in hippocampal CA1 homogenates were decreased in the PM 1 through PM 6 groups and increased in an age-dependent manner thereafter. This result may be associated with an age-dependent susceptibility to neurodegenerative diseases associated with the hippocampus.

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## P1-b10 Choline acetyltransferase of a peripheral type (pChAT) synthesizes acetylcholine in adult rat dorsal root ganglion

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Choline acetyltransferase of a peripheral type (pChAT) is an alternative splice variant of the well-studied ChAT protein (cChAT). Immunochemistry using a pChAT antiserum visualized known peripheral cholinergic cells that have been often difficult to detect with available cChAT antibodies. Moreover, the pChAT antiserum revealed neuronal structures that are thought to be non-cholinergic, as those in the dorsal root ganglion.

Here, we have examined possible acetylcholine production by pChAT in the DRG. Our analysis using Western blot and RT-PCR methods indicated that the ganglion neurons possessed pChAT, but never cChAT, mRNA and protein. Further neurochemical analysis showed that, the level of acetylcholine in the ganglion was as high as that in various brain regions known to receive cholinergic input. By using an immunoprecipitation method combined with laser captured microdissection, evidence was obtained that pChAT is an active enzyme capable of synthesizing acetylcholine in a subpopulation of primary sensory neurons.

## P1-b11 Nanoparticle induced neurotoxicity in the mice olfactory bulb: analysis of neurotransmitter and proinflammatory cytokine mRNA levels

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To investigate the neurotoxic effect of nanoparticles on neurotransmitters and proinflammatory cytokines in a mouse olfactory bulb, we performed in vivo microdialysis and real-time PCR methods. Eight-week-old male BALB/c mice were implanted with guide cannula in olfactory bulb and one week later, instilled carbon black (CB) intranasally. Six hours after the nanoparticle instillation, we injected lipoteichoic acid intraperitoneally. We found significantly increased extracellular glutamate, glycine levels and IL-1 $\beta$, TNF-$\alpha$ mRNA expressions in olfactory bulb of CB instilled mice. These findings indicate that nanoparticle CB may trigger neurotoxicity by modulating neurotransmitters and proinflammatory cytokines synergistically with bacterial cell wall component LTA in the mice olfactory bulb.

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## P1-b12 Effect of retinoic acid on neurosteroidogenesis in cultured rat hippocampus

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Hippocampus synthesizes steroid hormones (neurosteroids). Neurosteroids may modulate memory and some other functions. The regulation mechanism of neurosteroidogenesis, however, has not been clarified. Here we analyzed effect of retinoic acid on rat hippocampal sex steroid synthesis and mRNA contents of steroidogenic enzymes.

The hippocampal slices from 10 days old rat were incubated with 0.01 to 10μM of 9-cis-retinoic acid. Testosterone content in the slices was increased by the 48 hours treatment with 0.1μM retinoic acid. Among the steroidogenic enzymes, mRNA of P450 17$\alpha$ was increased by the treatment. P450 17$\alpha$ might be a regulatory step for testosterone biosynthesis. mRNA of P450arom was increased by the incubation with 1μM retinoic acid. Estradiol content was, however, not affected by the treatment. Retinoic acid can be a physiological regulator for testosterone synthesis in the rat hippocampus.

## P1-b13 Aromatic derivatives of DOPS could induce DOPAergic responses

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We hypothesize 3,4-dihydroxyphenylalanine (DOPA) as a neurotransmitter. DOPA microinjected into the nucleus tractus solitarii (NTS) elicited hypotension and bradycardia, although L-*threo*-3,4-dihydroxyphenylserine (DOPS), an analog of DOPA, did not. Non-enzymatic derivatives of DOPA and DOPS also caused these DOPAergic responses. By an ODS column, we obtained active fractions, A11 and S12 from non-enzymatic derivatives of DOPA and DOPS, respectively. Phosphate was contaminated in A11, whereas DOPS was in S12. By a size-exclusion column, we obtained active fractions, A113 and S122 from A11 and S12, respectively. The measurement of NMR and UV absorption suggested that S122 contained a benzene ring. Glycine and 3, 4-dihydroxybenzaldehyde (DHBALD) were non-enzymatically generated from DOPS. However, these derivatives did not cause DOPAergic responses. Thus, active derivatives of DOPS were predicted to be aromatic compounds different form DHBALD.

KAKENHI(16390068)

## P1-b14 Spatiotemporal observation of glutamate and GABA release from developing cerebellar cortex

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Glutamate and GABA are known to play important roles as modulators in the survival and development of cerebellar neurons. We recently reported the spatial nature of these releases from developing cerebellar slices with an enzyme-linked assay system. Whereas this system was useful, it was not adapted for temporal observation because the transmitter images were given by subtraction of intrinsic fluorescent images. The intrinsic fluorescence might change anytime. In the presence, we developed a new system to detect glutamate and GABA release without subtraction. In the new system, only fluorescent molecules resulting from released transmitters would be excited by weak evanescent light. Using this system, the rapid increase of glutamate release with AMPA was appeared after P6. On the other, the effect of bafilomycin, a VGAT inhibitor on GABA release, was appeared slowly but successively. We suggest this system would become a good tool to transmitter detection.

Toyoaki Scholarship Foundation

## P1-b15 Subcellular arrangement of molecules for 2-arachidonoyl-glycerol-mediated retrograde signaling in the striatum

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Endocannabinoids mediate retrograde signals for suppression of transmitter release at synapses of medium spiny (MS) neurons. Diacylglycerol lipase-$\alpha$ (DAGL $\alpha$), an enzyme for synthesis of an endocannabinoid, 2-arachidonoyl-glycerol (2-AG), metabotropic glutamate receptor 5 (mGluR5), and muscarinic acetylcholine receptor 1 (M1) were distributed on the somatodendritic surface of MS neurons. DAGL $\alpha$ and mGluR5 levels were highest in spines, while M1 level was lowest in spines. Depolarization-induced suppression of inhibition was enhanced by mGluR5 or M1 activation, but that of excitation was enhanced only by mGluR5 activation. These suppressions were abolished by the DAG lipase inhibitor tetrahydrolipstatin. CB1 receptor, a target of 2-AG, was expressed at specific synapses of MS neurons. Thus, 2-AG-mediated retrograde signaling modulates MS neuron's excitability depending on cortical activity and cholinergic tone.

## P1-b16 New system for released glutamate detection using the covalent immobilization of glutamate dehydrogenase and evanescent light

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Glutamate plays important roles for cerebellar differentiations and functions. Recently we reported the spatial nature of glutamate releases from acute cerebellar slices with specific enzyme-linked assay systems. Whereas our system was useful for detection of glutamate release, the measurement procedure was complex as making a human error. In addition, irradiated ultraviolet light was so toxic that the fluorescence decreased during serial detection. Now we have developed new system for released glutamate detection. Reacting enzyme, glutamate dehydrogenase was immobilized on glass surface with covalent bond, and excitation ultraviolet light was provided from the glass surface as weak evanescent light. Using this new system, glutamate release could be detected easily and successively without damages.

Research for Promoting Technological Seed

## P1-b17 Role of magnesium in neural process of rat cultured neurons

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Magnesium ion ($Mg^{2+}$) has been long identified as an essential ion as regards cell and body functions. Its deficiency indicated significant effects on various biological functions. Recent studies indicate that $Mg^{2+}$ plays an important role in brain disease and injury. However, the mechanism of $Mg^{2+}$ effects on neural process is not well understood, unlike those of $Ca^{2+}$. Here we investigated the effect of $Mg^{2+}$ on activities of rat cultured cortical/hippocampal neurons by using polymer electrode array and optical measurements, such as multi-photon microscope. Flow-cytometric analysis was also carried. Neural activity and the intracellular $Ca^{2+}$ concentration are largely affected by $Mg^{2+}$ removal. Intracellular magnesium concentration was investigated by $Mg^{2+}$ indicator dye, KMG-20-AM. Results are indicated different effects of the $Mg^{2+}$ removal on cortical and hippocampal neurons reflecting the difference in their magnesium requirement. Further experiments will be required.

## P1-b18 Non-neuronal GABA release in external granular layer and transient VGAT distribution

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GABA is not only an important neurotransmitter but also one of the trophic factors for developing brains. In cerebellum, immature granule cells in EGL are excited by GABA, whereas a small number of GABergic neurons are distributed there. We have reported GABA release from developing cerebellar cortex and observed GABA was released in both EGL and the layer under Purkinje layer of newborn cerebellum. GABA release in EGL was disappeared with development, and affected with glutamate treatment. Another GABA release increased with migration of GABergic neurons, and was not affected with glutamate. Using VGAT-Venus transgenic rat, we observed transient distribution of VGAT in EGL of newborn cerebellum. We speculated GABA would be transported to EGL by some transporters like VGAT.

Toyoaki Scholarship Foundation

## P1-b19 Steroid synthesis and synaptocrinology in the hippocampal synapses

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The endogenous synthesis of androgens and estrogens is elucidated for the hippocampus of adult male rats. By means of HPLC, we observed that testosterone (T) was converted into dihydrotestosterone (DHT) and 17 $\beta$-estradiol (E2). Hippocampal E2 concentration was much higher than that in plasma, which was enough to change the long-term depression and density of dendritic spines in CA1 and CA3 region. Immunohistochemistry and immunogold electron microscopy revealed that the steroidogenic enzymes were localized in the principal neurons (pyramidal cells and granulle cells) of hippocampus. Especially, P45017 $\alpha$ and P450arom were localized in pre- and post synaptic compartments in the principal neurons. These results suggest that steroids synthesized in the hippocampal synapse change the synaptic transmission and the state of synapse rapidly.

## P1-b20 Characterization of glutamatergic neurons in the rat midbrain raphe nuclei

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We previously reported that almost all vesicular glutamate transporter 3 (VGLUT3)-positive cells showed immunoreactivity for GABA in the neocortex and choline acetyltransferase (ChAT) in the neostriatum. In the dorsal and median raphe nuclei, about 80% of VGLUT3-positive cells also displayed immunoreactivity for 5-hydroxytryptamine (5HT), but the others were negative for 5HT. To characterize these VGLUT3-positive/5HT-negative cells, we performed double fluorescence staining using several markers. Firstly, all VGLUT3-expressing cells were positive for neuronal nuclear antigen (NeuN) but not for glial fibrillary acidic protein (GFAP), indicating that VGLUT3 was expressed only in neuronal cells. Secondly, all VGLUT3-expressing cells were negative for both glutamic acid decarboxylase (GAD67) and tyrosine hydroxylase (TH). These results suggest that VGLUT3-positive/5HT-negative neurons in the raphe nuclei utilize mainly glutamate as a neurotransmitter.

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## P1-b21 Development of a potent and selective GPR7 agonist: a systematic structure-activity study of neuropeptide B

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Neuropeptide B(NPB) has been recently identified as an endogenous ligand for the orphan G protein coupled receptors GPR7 and GPR8. Previous studies imply that the neuropeptide-receptor pathway comprising NPB and GPR7 may be involved in feeding, weight regulation and pain response through either direct or indirect actions in the central nervous system. In order to identify useful experimental tools to address physiological roles of NPB as neurotransmitter via GPR7, we prepared a series of Br(-)NPB-23-NH2 analogs and carried out the systematic structure-activity studied of NPB. Interestingly, several NPB analogs in which the consecutive amino acids from Pro4 to Val13 were replaced with several units of 5-aminovaleric acid(Ava) linkers retained their potent binding and functional activities for GPR7. Among the Ava-substituted NPB-analogs, we identified several synthetic NPB analogs with improved potency and selectivity for GPR7. These NPB analogs may be useful pharmacological tools to elucidate the physiological roles of GPR7.

## P1-b22 Regulatory mechanism of relaxin3 gene expression

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Relaxin3 is a novel member of relaxin, which is expressed in the brain and testis. In the brain, the distribution of relaxin3 was confined to the gray matter of midline dorsal pons, called the nucleus incerutus. These relaxin3 neurons were responsive to the corticotropin-releasing factor stimuli, and the expression of relaxin3 was increased by the restraint stress. Then, we investigated the regulatory mechanism and signal transduction pathway of the relaxin3 expression. We found the expression of endogenous relaxin3 in neuroblastoma cell line (Neuro2a), and used this cell line for the reporter assay. We constructed the fluorescence reporter vector combining the 5'-flanking region of relaxin3 gene with the upstream of the EGFP coding sequence. We observed that the expression of reporter gene was enhanced by the treatment with dbcAMP and forskolin in Neuro2a cells. Furthermore, we performed the promoter analysis of relaxin3 gene and identified its regulatory region. In this meeting, we discuss the transcriptional regulatory mechanism of relaxin3 gene.

## P1-b23 Study of D-serine function in vivo by establishing and analysis of serine racemase knockout mouse

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As an endogenous ligand of the *N*-methyl-D-aspartate (NMDA)-type glutamate receptors (GluRs), D-Ser has been thought to be involved in synaptic plasticity and associated with pathophysiology of schizophrenia. D-Ser is sythesized by serine racemase (SR) from L-Ser. To investigate the function of D-Ser in vivo, we have recently established SR knockout (KO) mouse with pure C57BL/6 genetic background. The concentration of D-Ser in the cerebral cortex and hippocampus of SR KO mouse decreased to about 10% of normal level. Our results indicate that SR is the main enzyme to produce D-Ser in the brain. In the adult wild-type mouse, SR was mainly detected in neurons, but not in astrocytes which have been considered to secrete D-serine. Using our SR KO mouse, we are investigating the effect of reduced D-serine on function of NMDA-type GluRs and pathophysiology of schizophrenia.

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## P1-b24 The role of $GABA_A$ receptor-mediated actions of taurine in developing cerebral cortex

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It is known that $GABA_A$ receptor (R)-mediate actions are known to modulate radial migration of cortical plate (CP) cells. Paradoxically, however, GAD65, 67 double knock-out mice showed normal cortical architecture. This implies compensatory mechanism either in receptors or agonists. We used GAD67-GFP knock-in mice to investigate the effects of ambient GABA. It is known that taurine concentration peaks in embryonic days. We thus investigate the influence of the ambient taurine to CP cells by inhibiting taurine metabolism using D-cysteine sulfinate (D-CSA). The CP cells which exist in ventricular zone at E14 were labeled by in utero electroporation. The distribution of labeled cells was analyzed at E17. The radial migration was facilitated in taurine decreased homozygous GAD67-GFP knock-in mice. This result indicates taurine may have supplemental effect for GABA, that could play critical protective role in pathological condition of GABAergic system.

## P1-b25 Differential expression of SNAP-25 family proteins in mouse brain

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SNAP-25 plays essential role in neurotransmitter release by exocytosis as a t-SNARE protein. SNAP-25 is also identified as a major protein in fast axonal transport. Two splicing isoforms, SNAP-25a and SNAP-25b, are expressed in neurons. In non-neuronal cells as well as in neurons, SNAP-23 is expressed as a t-SNARE for exocytosis. To know the functional roles of these SNAP-25 family proteins, specific antibodies were generated. Quantitative analysis revealed that the amount of SNAP-25b (10.8 pmol/mg) was much larger than those of SNAP-25a (0.9 pmol/mg) and SNAP-23 (1.1 pmol/mg) in adult mouse hippocampus. SNAP-25 was distributed in synapse-enriched regions and the expression was continuously increased during early postnatal period. On the other hand, SNAP-25a was enriched in nerve tracts and the expression was increased transiently during one to two week after birth. The expression of SNAP-23 was relatively constant in during postnatal period. These results suggested each SNAP-25 family proteins have specific roles in brain function.

## P1-b26 Expression of an acyl-CoA synthetase, lipidosin, in astrocytes and its up-regulation during experimental remyelination.

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Lipidosin is a long-chain acyl-CoA synthetase expressed in the organs that are selectively damaged in X-linked ALD. Immunohistochemistry showed that lipidosin immunoreactivity was colocalized with that of GFAP, but not with PGP 9.5, and lipidosin-producing cells detected by an antisense probe specific for lipidosin mRNA were also GFAP-immunopositive. These data indicate that lipidosin is expressed in astrocytes, but not in neurons. Immunoelectron microscopic analysis revealed that lipidosin immunoreactivity was widely distributed from astrocytic perivascular endfeet to perisynaptic processes. Lipidosin immunoreactivity was much increased during the recovery phase from experimental demyelination. These data suggest that lipidosin may be involved in lipid metabolism mediated by astocytes during the process of remyelination.

## P1-b27 Aberration of glutamatergic neurons in X-ray irradiated mouse embryonic cortex

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Although prenatal X-ray irradiation induces severe defects of neuronal migration and layering in the embryonic cerebral cortex, no study has focused on aberration of glutamatergic neurons by the irradiation. In ICR mouse embryos irradiated with X-ray (1.5 Gy) on embryonic day (E) 9 or E13, cortical glutamatergic neurons were examined on E11, E13, E15 and E17 by immunostaining for vesicular glutamate transporters (VGLUT1 and VGLUT2), glutamatergic neuron markers. Little aberration was seen in the cortex of E9-irradiated embryos except the delayed cortical plate (CP) formation at any age examined. In the E13-irradiated embryos, cortical afferent glutamatergic axons obliquely traversed CP and ran up to the superficial layer. Fiber bundles in both cortical afferent and efferent trajectories expanded in the intermediate zone. These results show that the susceptible period to the X-ray irradiation of developing mouse glutamatergic neurons is between E9 and E13, with different impacts depending on the time of irradiation.

## P1-b28 Sigma-1 receptor expression in rat brain during postnatal development

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σ-1 receptors (Sig1R) appear to play a significant role in a variety of physiological processes, including neuronal development. In this study, we investigated the developmental changes of Sig1R expression in rat frontal cortex, striatum, hippocampus and cerebellum from postnatal day 1 to 12 months by using immunoblot analysis. In all regions, the levels of Sig1R expression were highest at postnatal day 1, and were lowered during development. In striatum, Sig1R levels were decreased rapidly after birth, meanwhile decreased from 3-weeks and 1-week postnatal in hippocampus and cerebellum, respectively. The levels of Sig1R expression were lowest at adult rat (older than 8-weeks) in all brain regions. In addition, after subchronic treatment of Sig1R agonist, fluvoxamine in adult rat (8-weeks), the level of Sig1R expression significantly increased in striatum but not in cerebellum. These results indicate that the Sig1R expressions were differentially regulated in brain regions and developmental stages.

## P1-b29 Cajal-Retzius cells express vesicular glutamate transporter 2 (VGLUT2) during the mouse corticogenesis

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Despite glutamate plays a crucial role in neuronal growth and migration, little is known about when and where glutamate acts in the fetal brain. Therefore, we studied ontogeny of VGLUT1 and VGLUT2, the best markers of glutamatergic neurons, in the mouse fetal cerebral cortex. In situ hybridization revealed that VGLUT1 mRNA (*VGLUT1*) expression occurs in preplate (PP) cells at E10 but not in marginal zone (MZ) cells from E15 onward, whereas *VGLUT2* expression persists in the PP/MZ cells from E10 to E17. In accordance with this result, fluorescence in situ hybridization combined with immunofluorescence staining showed that *VGLUT1* exists in microtubule-associating protein 2-positive/reelin-negative pioneer neurons, while *VGLUT2* exists in reelin-positive Cajal-Retzius cells in the PP/MZ. These results show that Cajal-Retzius cells and pioneer neurons are glutamatergic, suggesting that VGLUT1 and VGLUT2 differentially play distinct roles in glutamate-mediated events mediated by these earliest-born cells before the initiation of synaptogenesis.

## P1-b30 Localization and characterization of an orphan G protein-coupled receptor expressed in the nervous system

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G protein-coupled receptors (GPCRs) comprise large and diverse gene families and are potential targets for pharmacological therapeutics. The superfamily of mammalian GPCRs has been divided into three families (( family A, rhodopsin receptor-like; family B secretin receptor-like; family C, metabotropic glutamate receptor (mGluR)-like)) based on thier homology. In the nervous system, amino acids including dipeptides function as neurotransmitters and modulators through the action of GPCRs. The mGluRs, calcium sensing receptor (CaR) and $GABA_BRs$ have all been shown to be involved in important central and peripheral physiological functions. In addition, several lines of pharmacological evidence indicate that additional mGluR, CaR and $GABA_BR$ subtypes might exist. GPRC5B has been identified as the candidate, but the function is still unknown. In the present study, we examined the gene expression in the nervous system in detail, and tried to identify the activators by expression assays using calcium imaging and Xenopus oocyte electrophysiology.

## P1-b31 $M_1$ subtype of muscarinic acetylcholine receptor is preferentially expressed in the principal neurons of the mouse telencephalon

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The $M_1$ subtype of muscarinic acetylcholine receptor ($M_1$ mAChR) is a $G_{q/11}$-protein coupled receptor, and its activation is known to exert various effects on neural functions. In the present study, we examined cellular and subcellular localization of $M_1$ mAChR in the mouse telencephalon. Immunofluorescence with specific antibody revealed that $M_1$ mAChR was abundantly expressed in the cerebral cortex, the hippocampus, and the striatum. In the cerebral cortex and the hippocampus, intense immunoreactivities for $M_1$ mAChR were observed in the perikarya and dendrites of pyramidal neurons, but not in GAD67/65-positive interneurons. In the striatum, intense signals were observed in the perikarya and dendrites of medium spiny neurons, but not in parvalubmin-, nNOS-, or CHT-positive interneurons. These results suggest that $M_1$ mAChR is preferentially expressed in principal neurons, but not in interneurons, in the telencephalon.

## P1-b32 IP3 receptor binding protein, IRBIT, specifically binds to and activates sodium bicarbonate cotransporter 1

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We previously identified an IP3R binding protein, termed IRBIT. Since IRBIT is released from IP3R, we hypothesized that IRBIT is a signaling molecule and regulates downstream target molecules in response to the production of IP3. In this study, we succeeded in identifying Na+/HCO3- cotransporter, NBC1, as an IRBIT binding protein. Of the two major splicing variants of NBC1, pNBC1 and kNBC1, IRBIT was specifically bind to pNBC1 and not to bind to kNBC1 at all. Furthermore, an electrophysiological analysis in Xenopus oocytes revealed that pNBC1 requires co-expression of IRBIT to manifest substantial activity comparable to that of kNBC1, which displays substantial activity independently of IRBIT. These results strongly suggest that pNBC1 is the target molecule of IRBIT and that IRBIT plays an important role in pH regulation through pNBC1.

## P1-b33 The critical role of transmembrane prolines in melanin-concentrating hormone receptor

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The melanin-concentrating hormone receptor 1 (MCH1R), a G protein-coupled receptor (GPCR), plays important roles in energy balance and stress-induced responses. GPCR transmembrane (TM) proline (Pro) has been implicated as molecular hinges for proper binding and activation. By single-substitution mutations of these Pro to Ala, the ability to form key structural and functional configurations in MCH1R was modified. Western blotting analysis showed that the extent of glycosylation was dramatically reduced in all of mutations. Mutation of Pro within TM 2, 4, 5, and 6 led to a complete loss of induction for calcium mobilization. On the other hands, Pro mutation in highly conserved motif (NPxxY) in TM7 still activates signaling. We further identify other amino acid residues that severely impaired receptor activity. Our result will provide useful information to aid understanding of the dynamic mechanisms of MCH1R activity.

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## P1-b34 Postnatal development of the KCC2-localization and GABA network in the mouse somatosensory cortex

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In the CNS, GABA actions developmenally shift from depolarization to hyperpolarization as a result of decreasing the intracellular chloride ion concentration by the expression of KCC2. In the present study, we focused on the mouse somatosensory cortex. First, to clarify the time-course of changes in the GABA actions during cortical development, we investigated the developmental localization of the KCC2 *in the mouse* somatosensory cortex using specific antibodies against KCC2. Second, to reveal the relationship between the changes in GABA actions and development of GABA network, we examined the developmental localization of GABA and VGAT, which relates to the vesicular release of GABA in the immature and mature synapses, in the same area. These The KCC2 was expressed in the sequential order of the differentiation of neuron when GABA synapses were formed, indicating the onset of GABAergic synaptic inhibition. Furthermore, as demonstrated in the cerebellum, GABA might be released by exocitosis before the synapse formation, the onset of KCC2 expression might be concomitant to the GABAergic synapses.

## P1-b35 Involvement of $P2X_7$ receptor in the regulation of glutamate transporters in cultured spinal microglia

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Spinal microglia expressing certain types of glutamate transporters function in the modulation of neuropathogenesis. In this study, we examined the effect of ATP, which is a potent modulator in the neuron-glia interaction, on the regulation of glutamate transporters in cultured spinal microglia. We detected the expression of both GLAST and GLT-1 in those cells. Preincubation with ATP or BzATP significantly inhibited the uptake of glutamate. These actions were blocked by pretreatment with a selective $P2X_7$ antagonist, a MEK-ERK inhibitor, or an antioxidant. The extracellular $Ca^{2+}$ or $Na^+$ did not have any roles in the effect of BzATP. Incubation with BzATP led to a marked decrease in the Vmax, but not the Km, of glutamate transport. These results firstly indicated that $P2X_7$ receptor in spinal microglia has an important role in the regulation of glutamate transport via activation of the ERK cascade and production of oxidants.

KAKENHI(17791322)

## P1-b36 Inhibition of selective serotonin reuptake inhibitors on vesicular monoamine transporter 2

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Effects of widely used antidepressants, selective serotonin reuptake inhibitors (SSRIs), on neuronal vesicular monoamine transporter (VMAT2) were largely unknown. In the present study, we investigated the effects of SSRIs on VMAT2 activities by measuring ATP-dependent [$^3$H]dopamine (DA) uptake into synaptic vesicles purified from rat striatum. SSRIs used in this study (fluoxetine, paroxetine, fluvoxamine) showed potent inhibition of vesicular [$^3$H]DA uptake *in vitro*. To investigate how these SSRIs work, we performed kinetic analysis. Although reserpine, a typical VMAT2 inhibitor, inhibited VMAT2 in an uncompetitive manner, fluoxetine inhibited VMAT2 in a noncompetitive manner. These results suggest that there is a novel mechanism underlying antidepressive or side effects of SSRIs.

## P1-b37 PKA may regulate the binding of GluRδ2 to its scaffolding protein, Delphilin

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GluRδ2 is selectively expressed in cerebellar Purkinje cells and plays an important role in motor learning, motor coordination and long-term depression. Delphilin is selectively expressed in Purkinje cell-parallel fiber synapses and specifically interacts with GluRδ2 C-terminus via its PDZ domain. SPR analyses showed that Delphilin PDZ bound to GluRδ2 C-terminal peptide (DPDRGTSI), but not to its phosphopeptides (DPDRGpTSI and DPDRGTpSI). We showed the incorporation of phosphate into Thr at -2 and Ser at -1 of GluRδ2 C-terminus by PKA in vitro. In the heterologous expression experiments, Delphilin coimmunoprecipitated with GluRδ2 was dramatically decreased under the PKA activated condition. Thus, phosphorylation of -2T and/or -1S of GluRδ2 C-terminus by PKA may regulate the binding of GluRδ2 to its scaffolding protein, Delphilin.

## P1-b38 Molecular basis of the autonomic regulation of mammalian pinealocytes with focus on nicotinic receptors

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Nicotinic responses of the principal cells (ddy mice and Wistar rats) in the pineal gland (pinealocytes) of mammals were studied using a whole-cell patch-electrode. When external saline containing 10 μM nicotine was applied from a "Y tube" to pinealocytes, definite inward currents were induced (holding potential, -50 mV). Other nicotinic agonists, including ACh (10 μM), choline (1 mM) and cytisine (10 μM), also induced inward currents. The *I-V relationship* of the induced currents showed marked inward rectification with reversal potential of about -5 mV. Interestingly, the magnitude of the response of pinealocytes to nicotinic agonists was considerably lower during the period of development after birth. To further study the possibility that nicotinic synaptic transmission in pinealocytes may occur in vivo, external saline containing 20 mM KCl was applied from a "Y tube" to pinealocytes, but no typical postsynaptic currents were detected from the present preparation.

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## P1-b39 Asymmetric localization of Notch2 on the microvillous surface in choroid plexus epithelial cells

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Notch family molecules are transmembrane receptors that play various roles in contact-dependent cell-cell interactions in a wide range of organs. In the brain, Notch2, but not the other members of Notch, is expressed in the choroid plexus (CP) at an exceptionally high level. We immunohistochemically examined the cellular and subcellular localization of Notch2 protein in the CP using confocal and electron microscopy. Unexpectedly, Notch2 was asymmetrically localized on the apical and microvillous surface of epithelial cells in the CP of both postnatal and adult rats. This localization pattern of Notch2 suggests its novel and unknown role independent of contact with adjacent cells in the CP. In organotypic cultures of the CP, the addition of anti-Notch2 antibody resulted in deformation of microvilli in epithelial cells, which suggests a role of Notch2 in the maintenance of the microvillous structure in CP epithelial cells.

## P1-b40 Delphilin, the PDZ domain-containing protein, interacts with the monocarboxylate transporter 2

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Delphilin, which interacts with a glutamate receptor (GluR) δ2 subunit, is a postsynaptic density scaffolding protein at the cerebellar parallel fiber-Purkinje cell synapses. Delphilin specifically interacts with the GluRδ2 C-terminus via its PDZ domain. As a number of PDZ-containing scaffolding proteins bind to several membrane proteins, we expected that Delphilin may also have other binding partners besides GluRδ2. To search for the link between Delphilin and other binding proteins, we carried out screening among candidate membrane proteins localized in Purkinje cells by surface plasmon resonance analyses. As a result, we found that the C-terminus of the monocarboxylate transporter (MCT) 2 binds specifically and significantly with Delphilin PDZ and there is a probable existence of GluRδ2-Delphilin-MCT2 complex in synaptic membranes. Now, we are further analyzing the interaction.

## P1-b41 Regulatory mechanism for P2X7 receptor activation by arachidonic acid and/or it's metabolites in mouse cultured astrocytes under resting conditions

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We recently reported that in mouse astrocytes under resting conditions, P2X7 receptor (P2X7R), of which pore formation is involved in neurotransmitter release and cell death, is activated partially by ATP released by themselves, suggesting that P2X7R plays important roles to maintain brain homeostasis under not only pathophysiological but also physiological conditions. Because in the preliminary study, several NSAIDs could increase YO-PRO-1 uptake, a P2X7R-forming pore marker, we examined regulatory mechanism for P2X7R activation focusing on arachidonic acid and its metabolites. In addition to NSAIDs, YO-PRO-1 uptake by astrocytes was increased by inhibitors for lipoxygenase and phospholipase $A_2$, and these increased uptakes were inhibited by oxATP, a selective P2X7R antagonist, while the uptake was decreased by antagonists for PPARγ. Therefore, it was suggested that PPARγ was, at least in part, involved in regulation of P2X7R activity in mouse astrocytes under resting conditions.

## P1-b42 A cell surface ligand of astrocytes promotes excitatory synapse-formation through PKC-activation in neurons

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We previously demonstrated that astrocytes affect excitatory synapse-formation by the contact with neurons. Local contact with astrocytes through neuronal integrin elicited protein kinase C (PKC)-activation in neurons.The resulting propagation of PKC signaling in a neuron is a significant basis to explain the underlying mechanism for global maturation of neurons following local adhesion of astrocytes. Despite these findings, the ligand molecule(s) on astrocytes for the integrin reception has been remained unknown. Recently, we have identified one candidate of such molecules on astrocytes.The molecule which abundantly exists on the cell surface of astrocytes. Interestingly, similarly to astrocytes, HeLa cells overexpressing the ligand molecule on the cell surface could phosphorylate MARCKS, a PKC substrate, meaning PKC-activation in a neuron.Also, excitatory synapse-formation was promoted in neurons by contact with the HeLa cells.Our study demonstrates active involvement of astrocytes in neuronal maturation through the ligand molecule.

## P1-b43 Functional characterization of choline transport in rat astrocyte

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Choline is an organic cation that plays an essential role in plasma membrane formation and cell growth. In brain, choline also contributes to neurotransmission as a precursor of acetylcholine (ACh). Glia cells are reported to possess choline transport system, little is known about its function. In this study, we examined the molecular and functional characterization of choline transport system in rat astrocyte CTX TNA2 cell line. Choline uptake into CTX TNA2 cells was strongly enhanced in the absence of $Na^+$. Choline uptake was significantly decreased by alkalinization of the intracellular condition with ammonium chloride. Collapsing intracellular $H^+$ condition by protonophores, CCCP and FCCP, inhibited choline uptake. RT-PCR demonstrated that CTX TNA cells expressed mRNA for $Na^+$-independent transporter, choline transporter like protein-1 (CTL-1), mainly CTL-1a subtype. These results suggest that choline transport system functionate in astrocyte, coupling with certain hydrogen exchange system.

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## P1-b44 Glial $Na_x$ channels control lactate signaling to neurons for brain [$Na^+$] sensing

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Sodium (Na) homeostasis is crucial for life and Na-levels in body fluids are constantly monitored in the brain. The subfornical organ (SFO) is the center of the sensing responsible for the control of salt-intake behavior, where $Na_x$ channels are expressed in specific glial cells as the Na-level sensor. Here, we show direct interaction between $Na_x$ channels and $\alpha$ subunits of $Na^+/K^+$-ATPase, which brings about Na-dependent activation of the metabolic state of the glial cells. The metabolic enhancement leading to extensive lactate production was observed in the SFO of wild-type mice, but not of the $Na_x$-knockout mice. Furthermore, lactate, as well as Na, stimulated the activity of GABAergic neurons in the SFO. These results suggest that the information on a physiological increase of the Na level in body fluids sensed by $Na_x$ in glial cells is transmitted to neurons by lactate as a mediator to regulate neural activities of the SFO.

## P1-b45 Effect of bisphenol A on the protein level of glutamine synthetase in primary-cultured astroglial cells

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Rat astroglial cells were grown during the last 12 hours of culture in either calf serum-free or calf serum-containing medium. After the addition of bisphenol A (BPA; 4.5 μM), an estrogenic disrupting chemical, to each medium, the cells were incubated for additional 12 hours. The protein fractions obtained from the cultured cells were employed for the Western blot analysis of glial fibrillary acidic protein (GFAP) and glutamine synthetase (GS). The protein level of GS increased in the serum-containing medium as compared to that in the serum-free medium. By the addition of BPA to the serum-free medium, the protein levels of GS increased. However, by the addition of BPA to the serum-containing medium, the protein level of GS decreased. Both in serum-free and -containing medium, the addition of BPA had no effect on the protein level of GFAP. These results suggested that BPA disturbed the calf serum mediated increasing effect on the protein level of GS.

## P1-b46 Role of Annexin2 in lysolecithin-induced demyelination of the peripheral nervous system

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Annexin2 (AX2) is a Ca2+ binding multifunctional protein, which exerts potent anti-inflammatory effects through inhibition of phospholipase A2. Previously, we have reported the characteristic localizations of AX2 in paranodal regions and Schmidt-Lanterman incisures in murine sciatic nerves. However, a role of this protein in the peripheral nervous system (PNS) in normal and pathological conditions is still uncertain.To elucidate the role of AX2 in demyelination and remyelination of the PNS, the expression patterns of AX2 in the lysolecithin-treated mouse sciatic nerves were examined by immunohistological and western blot analyses. Histological observation revealed swelling of myelin membranes in lysolecithin-treated nerves. In these nerves, AX2 was markedly accumulated in the paranodal regions. The changes of AX2 suggest that AX2 may have a role for demyelination and remyelination.

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## P1-b47 Distribution of paranodal proteins in myelinated nerves from Shambling mice with a *Caspr1* mutation

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Caspr1 is a transmembrane protein that locates at paranodes of myelinated nerves and makes a complex with contactin and neurofascin (NF155) forming paranodal junctions. Recently we found that mutated Caspr1 proteins, lacking transmembrane and intracellular domains, were expressed in spontaneous mutant mice, Shambling (*shm*). Our EM study revealed that paranodal junctions were disrupted in *shm* myelinated nerves. We here studied whether the *shm* mutation affects expression and distribution of paranodal proteins. Caspr1 staining was found at the paranodal region from *shm* sciatic nerves. The staining pattern, however, varies among paranodes, showing that three-fourth paranodes lacks the Caspr1. Contactin and NF155 was distributed similar to Caspr1 at the paranodal region. In contrast, protein 4.1B that binds with intracellular domain of Caspr1 was located independently from Caspr1 at the paranode. These findings suggest that altered distribution of paranodal proteins causes to disruption of paranodal junctions in *shm* mice.

## P1-b48 Characterization of neurons in the mouse subfornical organ by retrograde labeling

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The subfornical organ (SFO) is the center of sensing sodium (Na) levels in body fluids, where $Na_x$ channels function as the Na-level sensor for the control of salt-intake behavior. Here, we characterized neuronal types in the SFO by immunohistochemistry and retrograde labeling from target regions. When cholera toxin B subunit was injected into the paraventricular nucleus and supraoptic nucleus, the main loci of production of an antidiuretic hormone, arginine-vasopressin, the neurons labeled retrogradely in the SFO showed overlapping with hyperactivated (Fos-immunopositive) neurons under dehydrated conditions. The colocalization frequency did not differ between wild-type and $Na_x$-knockout mice. Furthermore, the Fos-positive neurons in the SFO were not GABAergic neurons, which are Na-dependently stimulated by $Na_x$-positive glial cells. These results suggest that the SFO neurons projecting to the neuroendocrine systems are independent of the $Na_x$ signaling.

## P1-c01 Effect of Amphotericin B on cellular functions of cultured microglia

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Amphotericin B (AmB), a polyene antibiotic, is reported to have therapeutic effects on prion diseases, in which microglia have been suggested to play important roles by proliferating and producing various factors. We examined the effect of AmB on mRNA expression levels of several factors in rat primary microglia using the reverse transcription-polymerase chain reaction. AmB could activate the cell, similarly as lipopolysaccharide (LPS), to induce nitric oxide synthase and pro-inflammatory cytokines, tumor necrosis factor-$\alpha$, interleukin (IL)-1$\beta$, and IL-6. AmB and LPS down-regulated the level of ciliary neurotrophic factor (CNTF). However, AmB, but not LPS, up-regulated the levels of glial cell-line derived neurotrophic factor (GDNF) and brain-derived neurotrophic factor (BDNF). These results suggest possible AmB effects on the pathological mechanism of prion diseases by controlling microglial cellular functions, such as neurotrophic factors production.

## P1-c02 Phorbol 12-myristate 13-acetate suppresses $Ca^{2+}$-dependent peptide hormone release from clonal rat glioma C6 cells

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Glial cells release various bioactive substances such as growth factors and neurotransmitters by exocytosis, and these functions are quite important for maintaining and modulating brain functions. To know the regulatory mechanisms in glial cells, we transiently transfected human growth hormone (hGH) in cultured rat glioma C6 cells. Just like in PC12 cells, the enhanced green fluorescent protein (EGFP)-tagged hGH expressed in C6, was distributed in granular structure in cytoplasm. Ionomycin induced a profound release of hGH in a $Ca^{2+}$-dependent manner. Phorbol 12-myristate 13-acetate (PMA), an activator of protein kinase C (PKC), inhibited the release of hGH from C6 cells, and this suppression was reversed by a PKC inhibitor, bisindolylmaleimide I (BIS). PMA caused a mobility shift of SNAP-23 in SDS-PAGE and this change was suppressed by BIS. These results suggested that the release of peptide from glial cells was negatively regulated by a PKC-dependent mechanism through the phosphorylation of SNAP-23.

## P1-c03 Intracellular labeling of single cortical astrocytes in vivo

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As recent experimental evidence suggests glial may participate in neural information processing, there is a need to monitor the physiology of glial cells in vivo in the matured brain. Concurrently, identification of the recorded glial cells has become important as glial cells are heterogeneous. Morphological recovery and identification of the in vivo recorded glial cells has been difficult due to the leak of the tracer through gap junctions. Here we demonstrate that use of large fragment biotinylated dextran amine (BDA) is an effective way to label a single glial cell recorded with a sharp microelectrode in vivo. Furthermore, the tracer signal amplification was achieved by a combination of avidin biotinylated horseradish peroxidase macromolecular complex (ABC) and tyramide based methods, making multiple immunohistochemistry feasible. Post-hoc immunohistochemistry show that most (>70%) of the blindly recorded cortical non-spiking cells were astrocytes, but other glial cells such as oligo-dendrocytes and microglia like cells can also be recorded.

KAKENHI (18053026)

## P1-c04 Role of Iba1 in cytokine production in macrophages and microglia

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Ionized calcium binding adaptor molecule 1 (Iba1) is an EF hand calcium binding protein expressed selectively in microglia/macrophages (MΦ). Iba1 is implicated in the mobility and phagocytosis of these cells, but its role in cytokine production is not clear. In this study, by using Iba1 knock out (KO) mice, we examined whether Iba1 is involved in the release of cytokines (IL-12 p40, IL-10) as well as nitric oxide (NO) in peritoneal MΦ and primary cultured microglia. In LPS-stimulated Iba1 KO MΦ, the release of IL-12 p40 was lower, whereas IL-10 release was higher than in wild-type. LPS-induced NO release and iNOS expression were also less in Iba1 KO MΦ. In microglia, Iba1 deficiency caused the similar decrease in IL-12 p40 release and iNOS expression. These results indicate that Iba1 may play an important role in inflammatory response by enhancing pro-inflammatory IL-12/NO release and by suppressing anti-inflammatory IL-10 release.

KAKENHI(18053017)

## P1-c05 Astroglial cell assemblies

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Over the past years, many studies have focused only on neurons, and astrocytes are considered to be non-excitable passive cells. Recent studies have revealed that they are active participants that may exert significant control over a huge CNS network. However, many studies have focused on properties of individual astrocytes, and little is known about their collective behavior. What are their properties as communication elements in a neuro-glial network? We therefore performed immunohistochemistry and revealed that their physical locations are arranged at almost regular intervals of 20-30 μm. Furthermore, we observed spontaneous activities of hippocampal astrocytes for as long as 1hr using $Ca^{2+}$imaging techniques. The cross-correlogram between all pairs of approximately 200 astrocytes showed that astrocytes in closer vicinity tended to exhibit more correlated activities. These properties are clearly different from neurons, and our findings will lead to better understanding of neuro-glial network function.

## P1-c06 ZNRF1 regulates glutamine synthetase protein expression in Schwann cells via a ubiquitin-proteasome pathway

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Ubiquitin-proteasome system has been implicated in a variety of neuronal functions. We have previously identified a novel E3 ubiquitin ligase, zinc finger/Ring finger 1 (ZNRF1), which is up-regulated in Schwann cells of degenerating peripheral nerves. Here we have identified glutamine synthetase (GS) as a protein targeted by the ZNRF1-dependent proteasomal degradation mechanism. In neuro2a cells, forced expression of ZNRF1 reduced the GS protein level in a dose-dependent manner, and this effect was blocked by a proteasome inhibitor. In a mouse sciatic nerve transection model, injury induced ZNRF1 expression in a segment distal to the injury site resulted in the decreased expression of GS protein, while its mRNA remains the same. These findings suggest that, unlike most other proteins characterizing Schwann cell phenotypes, GS expression is regulated post-translationally by ZNRF1 E3 ligase-dependent ubiquitin-proteasome degradation mechanism.

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## P1-c07 Forskolin up-regulates expression of mRNA for a glutamine transporter in cultured rat neocortical astrocytes

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The view that the glutamine (Gln) transporter (GlnT) is a member of the system A transporter superfamily with ability to fuel the glutamate/Gln cycle at glutamatergic nerve terminals is prevailing. In the present study, we have attempted to demonstrate the possible modulation of GlnT gene expression in response to cAMP in cultured neocortical astrocytes. An RT-PCR analysis revealed that sustained exposure to the adenylyl cyclase activator forskolin for 24 h led to a significant increase in GlnT mRNA expression in astrocytes. Forskolin drastically stimulated the promoter activity of GlnT in a manner sensitive to a protein kinase A inhibitor in the C6 glioma cells, moreover, while deletion mutation analysis revealed that the stimulation of GlnT promoter activity was mediated by CRE/AP-1 like site on the GlnT gene promoter. These results suggest that cAMP would up-regulate mRNA expression of GlnT through the facilitation of its promoter activity at CRE/AP-1 like site in cultured rat neocortical astrocytes.

## P1-c08 Promoted function by cAMP signals of a glutamine transporter expressed in cultured rat neocortical astrocytes

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The neurotransmitter glutamate is supposed to be recycled through the glutamine (Gln)/glutamate cycle that involves Gln transporter (GlnT) belonging to the system A transporter family. In line with our previous findings, in this study, we have attempted to demonstrate the possible functional modulation of GlnT in response to cAMP response element binding protein (CREB) in cultured rat neocortical astrocytes. The exposure to the adenylyl cyclase activator forskolin induced phosphorylation of CREB with a concomitant increase in [$^3$H]Gln accumulation sensitive to a selective system A transport inhibitor in a concentration-dependent manner. Moreover, ChIP assay revealed that exposure to forskolin led to the upregulation of GlnT mRNA when determined on experiments using forward and backward primers targeted to the GlnT promoter region. These results suggest that phosphorylation of CREB would lead to promoted function of GlnT through upregulation after the interaction with the promoter in cultured astrocytes.

## P1-c09 Extracellular S100B increases the amplitude of kainate-induced gamma oscillation *in vivo*

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S100B is predominantly synthesized by astrocytes and secreted to the extracellular space. The secreted S100B exerts various effects on neurons, astrocytes and microglia depending on the concentration. We have shown that the amplitude of kainate-induced gamma oscillation in CA1 str. radiatum is significantly smaller in S100B knockout mice (Sakatani et al., 2007). To assess the contribution of extracellular S100B to the gamma oscillation, we combined local field potential recording and local infusion of dimeric S100B at CA1 str. radiatum in S100B KO mice. Local infusion of low concentration (100 nM) S100B did not have a significant effect on kainite-induced gamma oscillation. At a high concentration (10 μM) of S100B, local infusion of S100B resulted in an increase of the gamma oscillation. In a separate set of experiments, anti-S100B antibody was locally applied in wild-type mice, resulting in a decrease of the gamma oscillation amplitude. Our results suggest that extracellular S100B increases the amplitude of kainate-induced gamma oscillation.

KAKENHI(18053026)

## P1-c10 Activity-dependent glial coverage in the rat nucleus of tractus solitarius during postnatal development

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Glial cells contribute to maturation of synapses in developing nervous system. We have previously reported that, in the rat caudal nucleus of tractus solitaries (cNTS) during postnatal development, the decrease of axosomatic synapse number in small cell soma appears to depend on increase of glial coverage by astrocytic processes. However, the factors influencing the increase of glial coverage in cNTS remain unknown. In the present study, to elucidate whether the neural activity facilitate glial coverage of small cell soma in cNTS, the effects of activity blockade in vivo were examined. Deprivation of activity with intraperitoneal injection of MK801, NMDA receptor antagonist, at P5-P8 caused less matured glial coverage in small cell somata at P10 and P21. These results suggest that glial differentiation around soma requires neural activities. Activity-dependent pathway involving astrocytes might play a role in decrease of axosomatic synapse number in small cell during developing cNTS.

## P1-c11 The sensitivity of differentiated astrocytes to hydrogen sulfide is diminished in the reactive astrocytes

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Hydrogen sulfide ($H_2S$), which enhances the induction of hippocampal long-term potentiation and induces calcium waves in astrocytes, has been proposed to be a synaptic modulator in the brain. Here we show that differentiated astrocytes acquire sensitivity to $H_2S$ that is diminished by their transformation into reactive astrocytes. Although NaHS, a donor of $H_2S$, did not increase the intracellular concentration of $Ca^{2+}$ in progenitors, exposure to leukemia inhibitory factor, which induces differentiation into glial fibrillary acidic protein-positive astrocytes, increased the sensitivity to NaHS. In contrast, epidermal growth factor (EGF), transforming growth factor $\alpha$, dibutyryl cyclic AMP and interleukin-1 $\beta$ induced the conversion to reactive astrocytes with diminished sensitivity to NaHS. This effect of EGF was inhibited by cycloheximide, indicating that de novo protein synthesis was required for the suppression of NaHS sensitivity.

## P1-c12 Identification of apoE as a ligand for LDL receptor-related protein 4 (LRP4)

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We examined whether apoE is a ligand for LRP4 in the rat brain. The distribution pattern of apoE was similar to that of LRP4, since apoE was distributed in a punctate manner on the cell surface throughout neurons including in somas and dendrites. Most of apoE and LRP4 were co-localized in cortical neurons in culture and apoE and LRP4 were co-immunoprecipitated. Thus, apoE was demonstrated to be a ligand for LRP4. Next, we examined the effect of antibody against the ligand-binding domain of LRP4 (anti-LB). Anti-LB applied to neuronal cells in culture down-regulated MAP2-immunoreactive neurons, reduced the viability of neurons and impaired synaptic integrity. This effect was considered to be due to blockade of binding to LRP4 protein of extraneuronal ligands, such as apoE/cholesterol, since anti-LB suppressed binding of apoE to the LRP4 heterologously expressed in Cos7 cells. These results suggest a possible role of LRP4 working as a receptor from extracellular signals in the maintenance of the viability of neurons and synapse integrity.

KAKENHI(16047211, 17500252)

## P1-c13 mRNA exparession profile of claudin subtypes in mouse blood-brain barrier

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Tight junctions (TJs) are an important component of the blood-brain barrier (BBB). To understand the contribution of each claudin subtype to TJ formation, we have measured the mRNA expression levels of claudin-1 to -23 in highly purified mouse brain capillary endothelial cells (BCECs). Mouse BCECs were isolated with anti-PECAM-1 antibody by magnetic cell sorting. Expression of Tie-2, Mdr1a and GLUT1 mRNAs was concentrated in the isolated fraction. Among claudin subtypes, claudin-5 was most highly expressed, at a level which was at least 593-fold greater than that of claudin-1, -3 or -12. Expression of mRNAs of claudin-8, -10, -15, -17, -19, -20, -22 or -23 was also concentrated in the isolated fraction, suggesting these subtypes are expressed in mouse BCECs. The levels of claudin-10 and -22 mRNAs were comparable with that of occludin mRNA. These results indicate that claudin-5 is the most abundant claudin subtype in mouse BCECs, and are consistent with the idea that claudin-10 and -22 are involved in TJ formation at the BBB in cooperation with claudin-5.

## P1-c14 BBB kit (Blood-Brain Barrier in vitro Reconstruction Model)

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The present study was designed to establish the in vitro BBB model, by co-culturing with BBB cells, rat brain microvascular endothelial cells (RBEC), rat pericytes and rat astrocytes. BBB cells were cultured on Transwell™. Transendothelial electrical resistance (TEER) and permeability of sodium fluorescein (Na-F, 376Da) were measured to estimate barrier and tight junctions (TJs) functions. TJs-related proteins were analyzed with immunohistochemical and Western-blotting techniques. A significant increase in TEER was observed in the model of EPA which was a BBB kit composed of RBEC and pericytes co-cultured in-contact and astrocytes out-of-contact, with a reduction of Na-F permeability. Claudin-5, a TJs protein was richly expressed in EPA-type BBB kit. Thus, our BBB kit can be used for basic research, as well as for drug transport assays.

## P1-c15 Contribution of increase in brain serotonin (5-HT) upon increase in blood 5-HT induced by 5-HTP administration in rat

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Our previous studies showed that augmented brain 5-HT can cross the blood-brain barrier (BBB) through 5-HT transporter from the brain to the peripheral blood in rats with gastrointestinal resection to elevate brain 5-HT alone. We found that SSRI pretreatment abolished the increase in blood 5-HT level, although there was a clear augmentation of brain 5-HT level in those rats. In the present study, we examined changes in blood 5-HT level when brain 5-HT level was elevated by intravenous administration of 5-hydroxytryptophan in intact rat. A microdialysis method and HPLC system were used to determine the brain 5-HT levels in samples collected from the frontal cortex. Blood 5-HT levels were determined from whole blood, collected from the central vein. The increase in blood 5-HT level with SSRI pretreatment was lower than that without SSRI pretreatment, suggesting that brain 5-HT would contribute in part to the increase in total blood 5-HT level, via 5-HT transporter in BBB.

## P1-c16 Identification of novel UCH-L1-potentiating compounds by *in silico* drug screening

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Ubiquitin C-terminal hydrolase-L1 (UCH-L1) is a de-ubiquitinating enzyme and is a component of the ubiquitin-proteasome system. It was demonstrated that enhancement of UCH-L1 activity rescues $\beta$-amyloid-induced decreases of synaptic function and contextual memory (Gong, B., et. al, *Cell*, 2006, 126, p.775). In this study, we attempted to identify UCH-L1-binding compounds. We performed *in silico* drug screening using human UCH-L1 crystal structure data and a virtual compound library including 26,891 compounds (ChemBridge Corp.). The *in silico* drug screening was implemented by DOCK software (first screening) and GOLD software (second screening). We identified two new compounds which potentiate UCH-L1 activity by 1.3-fold. The novel UCH-L1-potentiating compounds may be useful for the drug development of Alzheimer's disease.

## P1-c17 A Japanese herbal medicine Ninjinyoueito ingredients, Aurantii nobilis pericarpium, rescue from aging-induced demyelination

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Our previous findings showed that FCRγ/Fyn-MBP cascade is a potential therapeutic target for remyelination by Ninjinyoueito (NYT).NYT is constituent of a mixture of 12 raw medicinal plants.To elucidate which compound is more potential medicinal plants for the recovery form demyelination, 4 groups of mixture (Mix A, B, C, D)consists of 17 compounds isolated from NYT have been tested.The levels of phosphorylated 21.5 kDa MBP decreased markedly during demyelination induced by aging. The decrease of 21.5kDa P-MBP level in aged mouse brain was recovered after treatment with Mix D (hesperidin and narirutin, major constituents of Aurantiinobilis pericarpium) for two months. These results suggest that Mix D rescue from aging-induces demyelination.

## P1-c18 Calcium Channel Blocker Peptides Isolated from Tarantula Venom : identification, characterization and utilization

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Some of the neurological and cardiac diseases, such as epilepsy, arrhythmia and hypertension, are caused by dysfunction of ion channels. Toxins that recognize ion channel subtypes are versatile tools for channel studies, and thus contribute to drug discovery. In this study, we screened tarantula venom to identify calcium channel blocker peptides that specifically recognize a variety of the channel subfamilies.Three peptides were identified that inhibit different types of calcium channel. One of the peptides (35 amino acids) suppressed the $Ca_v2.1$ (P/Q-type) current. The sequence is identical with that of ω-GsTx SIA. The second peptide (known as GsAFII) with 30 amino acid residues blocked L-type $Ca^{2+}$ channel ($Ca_v1.2$). We also isolated a novel peptide, which specifically targets the T-type $Ca^{2+}$ channel ($Ca_v3.1$). The peptide has sequence homology with sodium channel blocker peptides. These spider venom peptides so far identified have different structural features with different cysteine motifs, ICK.

NEDO

## P1-c19 Effects of N-(2-propylpentanoyl) urea on impairment of learning and memory induced by common carotid artery occlusion in mice

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Effects of a new valproate analog, N-(2-propylpentanoyl) urea (VPU), on impairment of learning and memory induced by 2 vessel occlusion (2VO) were investigated. Intraperitoneal administration of VPU at the dose of 50 mg/kg significantly improved a deficit of performance in MWM test in 2VO mice on day 5. Accordingly, administration of VPU either 50 or 100 mg/kg could significantly ameliorate learning and memory deficit in step-down test as well. Number of survival neurons in CA1 of hippocampus in VPU-treated group seemed to be higher than those observed in 2VO group. Treatment by VPU significantly abolished an increase in MDA induced by 2VO. Based on the results obtained, it is suggestive that VPU could exert neuroprotective effect against impairment of learning and memory induced by 2VO resulting in an improvement in MWM and step-down tests observed. Decrease of oxidative stress, seen as a reduction of MDA, could probably underlie protection of neuronal cell death in hippocampus as well as an improvement in behavioral tests.

## P1-c20 Minocycline attenuates neuronal cell death and improves cognitive impairment in Alzheimer's disease models

minji Kim, Yoori Choi, Hye-Sun Kim, Ki Young Shin, Eun-Mee Kim, Yoo-Hun Suh

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Minocycline is a semi-synthetic tetracycline antibiotic that has been reported to have significant neuroprotective effects. In this study, we demonstrate that minocycline has neuroprotective effects in in vitro and in vivo Alzheimer's disease models. Minocycline was found to attenuate the increases in the phosphorylation of eIF2$\alpha$ induced by A$\beta$ peptide1-42 treatment in NGF-differentiated PC 12 cells. In addition, increases in the phosphorylation of eIF2$\alpha$ were attenuated by administration of minocycline in Tg2576 mice which harbor mutated human APP695 gene including the Swedish double mutation and A$\beta$ peptide1-42-infused rats. We found that minocycline administration attenuated deficits in learning and memory in A$\beta$ peptide1-42-infused rats. Increased phosphorylated state of eIF2$\alpha$ is observed in Alzheimer's disease patients' brains and may result in impairment of cognitive functions. Based on these results, minocycline may prove to be a good candidate as an effective therapeutic agent for Alzheimer's disease.

## P1-c21 Critical role of SDF-1/CXCR4 in the development of boundary cap cells and CNS/PNS segregation

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The central and peripheral nervous system (CNS and PNS) are segregated compartments, connected only by motor and sensory axons via specialized interfaces, namely the motor exit point (MEP) and dorsal root entry zones (DREZ), respectively. The CNS/PNS interfaces allow the passage of axons but prevent cell mixing. How these interfaces are formed and function in CNS/PNS segregation remain largely unknown. A subset of neural crest cells is strategically positioned at DREZ/MEP forming the so-called boundary cap (BC). Here we show that DREZ BC cells express CXCR4, a receptor of the chemokine SDF-1. Analysis of SDF-1 knockout mice embryos by immunohistochemistry showed that the BCs in SDF-1$^{-/-}$ were malformed as evidenced by their irregular shape, excessive cell numbers and loose association with the dorsal roots. Strikingly, many BC cells "emigrate" into the neural tube and express PNS lineage markers within the CNS. Our results reveal a critical role of SDF-1/CXCR4 in the making of an integral CNS/PNS interface.

KAKENHI

## P1-c22 Analysis of beta2 Chimaerin in rostrocaudal polarity formation of the optic tectum

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We have been interested in rostrocaudal polarity formation of the chick optic tectum. It accepted that En2 plays a pivotal role in rostrocaudal polarity formation. We have isolated beta2 Chimaerin during screening of the molecules that are repressed by En2 at the mesencephalon. beta2 Chimaerin functions as a Rac-GAP that is activated by PKC-diacylglycerol/phorbolestel signal. *beta2 Chimaein* is expressed in the mesencephalon with rostrocaudal polarity and in the diencephalon. Misexpression of siRNA of *beta2 Chimaein* upregulated *EphrinA2* expression, suggesting involvement of beta2 Chimaerin in rostrocaudal polarity formation of the tectum.

Grant-in-Aid for JSPS Fellows

## P1-c23 Developmental processes in the ferret LGN independent of input from retina and visual cortex

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Visual mammals such as monkeys and ferrets have highly developed lateral geniculate nucleus (LGN), which contains a variety of relay neurons and layered structure. The LGN receives retinal ganglion cell (RGC) axons, which are segregated into layers during development. Although mechanisms of RGC axon segregation in the LGN have been well examined, those underlying development of LGN neurons are largely unknown. Previously we identified molecules expressed in the ferret LGN, which can be used as developmental timing markers including vGluT2 and PCP4. We found that development of LGN neurons proceeds even without retinal activities. We successfully established slice culture using the ferret LGN, in which *in vivo* developmental processes of the LGN were recapitulated at least in part. In contrast to RGC segregation in the LGN, our results suggest that developmental processes of LGN neurons do not require input from retina and visual cortex. We will discuss "LGN-autonomous" and "LGN-non-autonomous" developmental processes in the LGN.

KAKENHI, 21COE

## P1-c24 Functional analysis of the *Sufatase FP3* gene during zebrafish embryonic development

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Heparan sulfate proteoglycans (HSPGs) play a crucial role in mediating important signaling by Wnt, FGF, and other heparin-binding factors. Interaction of HSPG with these signaling molecules depends on specific sulfation patterns in heparan sulfate chains. Novel sulfatases, SulfFP1 and SulfFP2, have heparan sulfate 6-*O*-endosulfatase activity, suggesting that they regulate cell surface signaling by altering specific sulfation patterns. Recently we isolated a third homologue, *SulfFP3*, from zebrafish. SulfFP3 has about 56% and 71% overall amino acid homology with SulfFP1 and SulfFP2, respectively. At 24 hr postfertilization, *SulfFP3* is expressed in the ventral region of the spinal cord and brain. In order to examine the function of *SulfFP3*, we knocked down *SulfFP3* by microinjecting antisense morpholino oligonucleotides into embryos. Preliminary results suggest that blocking of the SulfFP3 function results in morphological defects during embryonic development.

KAKENHI (18060005)

## P1-c25 The role of Strawberry notch family genes in the central nervous system development of zebrafish

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We studied *Strawberry notch* (*Sno*) homologues of zebrafish to understand functions of this gene family in the vertebrate brain development. Zebrafish has three *sno* family genes, *sno1*,*sno2a* and *sno2b*. Whole mount *in situ* hybridization showed that *sno1* and *sno2a* exhibited very similar expression pattern during early embryonic stages. Embryos injected with morpholino antisense oligonucleotide (MO) against *sno1* or *sno2a* showed morphological abnormality in the brain. Because *Sno* is involved in regulation of gene expression in *Drosophila*, we analyzed effects of *sno* MO on expression of neural markers. The expression of *delta*genes was unaffected in the morphants at the 1-somaite stage, while it was significantly reduced at the late tailbud stage. These morphants exhibited highly reduced expression of *rora2* and *er81*, and delayed expression of *islet* . These data suggested that *strawberry notch* genes may play important roles in neuronal differentiation and maturation in zebrafish.

## P1-c26 The cloning of zebrafish purpurin gene and its expression in early developing retina

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Purpurin a retina specific 22kDa protein, was originally discovered in developing chicken retina as a cell adhesion molecule. We recently cloned goldfish purpurin cDNA as a trigger molecule for optic nerve regeneration. However the expression and function of purpurin have not been studied in developing fish retina. Here we report the identification and expression analyses of the zebrafish purpurin during retinal development. We identified an 854 bp complete zebrafish purpurin cDNA. The levels of purpurin mRNA increased in the ONL at 3-7 dpf. Purpurin protein was diffusely located in all retinal layers. Furthermore we cloned a 4.2 kbp genomic DNA contained a 1.9 kbp 5′flanking upstream region for zebrafish purpurin. A 1.9kbp promoter fragment was cloned upstream of the GFP cDNA and injected into 1 cell stage of zebrafish embryos. GFP expression was detected in the ONL at 3 dpf. The molecular mechanism of purpurin transcription is in now progress.

## P1-c27 The role of sonic hedgehog signaling in the developing cerebral cortex

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The secreted protein Sonic hedgehog (SHH) plays important roles in morphogenesis of various embryonic tissues. It is widely known that *Shh* from the notochord and floor plate ventralizes the developing neural tube. It has also been reported that *Shh* is expressed in the dorsal telencephalon after midgestational stages. The data from in vitro experiments suggested that Shh maintains neural stem cell characters and promotes proliferation. However, the localization of *Shh* in the developing dorsal telencephalon has not been clarified. In this study, we found that SHH is expressed in the dorsal telencephalon of mouse embryo. To explore role of Shh signaling, we conditionally knocked out *Shh* gene in a dorsal telencephalon-specific manner. The mutant embryos showed the hypotrophic telencephalon. There was a reduction in the population of proliferative cells and differentiated neurons at early neurogenesis stages. Our results suggest that Shh signaling in the dorsal telencephalon during neurogenesis maintains cortical neural precursor identity and stem cell niche.

## P1-c28 Length of the frontal lobe from the anterior pole of the frontal lobe to the central sulcus in human brain

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In according to the cerebral hemisphere moving more caudally in the developmental process, the central sulcus which indicates the anterior-posterior length of the frontal lobe moves caudally at the same time. In our previous study, the distances from the anterior pole of the frontal lobe to the posterior pole of the occipital lobe were longer on the left side (85%) than on the right side. In the present study, the distance from the anterior pole of the frontal lobe to the central sulcus was measured from the anterior pole of the frontal lobe to the central sulcus on the 13 human brains which medical students used in the neuroanatomical exercise. The standard point of the central sulcus was situated on the highest sulcus of the marginal zone of the hemisphere. The distances in the 3 brains were same on both side, but these in the other brains were longer on the left side than on the right side. The results show that the frontal lobe extends more caudally on the left side than on the right side.

## P1-c29 Expression pattern of Tsukushi during mouse development

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We previouly identified a novel secreted factor, Tsukushi (TSK), which functions as a BMP inhibitor during chick gastrulation (Ohta et al., 2004; Ohta et al., 2006). Recently we found that TSK plays a role in balancing the retinal stem cell proliferation and differentiation in the ciliary marginal zone. In order to better understand TSK roles during vertebrate development, we generated the knockout mice by inserting a lacZ gene into exon 1 of the TSK gene. We have performed lacZ staining to investigate its expression patterns and found that the lacZ expression is observed in various regions including the eye and the brain during embryonic and postnatal development. Based on these results, we will analyze immunohistochemically the knockout mice. We would like to discuss possible roles of TSK during mouse development.

## P1-c30 Role of Wnt signaling in the mushroom body development

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The Drosophila mushroom body (MB) is a paired neuropile structure required for olfactory memory and composed of three distinct types of neurons; γ, $\alpha'/\beta'$, $\alpha/\beta$ Kenyon cells (KCs). Kenyon cells have their cell bodies in the dorsoposterior part of the brain and project their axons anteriorly forming a massive fiber tract. At the frontal margin of the brain, KC axons bifurcate to form discrete dorsal and medial lobes. In this study, we demonstrate that Wnt signaling is required for the proper formation of the MB structure. Major components of Wnt signaling are Wnt, Frizzled (Fz) receptor, cytoplasmic protein Dishevelled (Dsh). $\alpha/\beta$ lobes were lost or reduced in *fz* or *dsh* hypomorphic mutation. Moreover, *fz* mutation in *dsh* hypomorphic background drastically enhanced the phenotype; $\alpha/\beta$ KC axons failed to project. In addition, mutation of *wntless*, which is required for Wnt secretion, showed phenotypes similar to *fz* and/or *dsh* mutation. We are currently studying autonomy of the Wnt signaling in the MB development.

KAKENHI (18022010)

## P1-c31 Involvement of DMAP1 in the neural migration and differentiation in zebrafish

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The *freeze frame* (*fzf*) mutation ectopically induces the Isl1-GFP-positive cells in the discrete region of CNS such as anterior margin of the optic tectum and dorsal part of the spinal cord. Furthermore, in the rhombomere 3, subset of the posterior trigeminal motor neurons showed defects in differentiation and subsequent cell migration to make the motor nucleus in the white matter. We found that the locus of *fzf* encodes the zebrafish orthologue of *dmap1* (DNMT1 associated protein1), and *fzf* caused the missense mutation (W151R) in *dmap1*. Previous reports demonstrated that DMAP1 interacts with several molecules such as DNMT1 (DNA methyltransferase1), RGS6 (Regulators of G protein Signaling 6). We are examinning the relationship of these molecules with DMAP1 in the developing CNS of zebrafish embryos.

## P1-c32 The chemokine SDF-1 α regulates axonal elongation and pathfinding from entorhinal cortex to dentate gyrus in hippocampus

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SDF-1 α (stromal cell-derived factor 1 α) plays critical roles in cell migration and axonal pathfinding. In this study, we observed that SDF-1 α induced neurite elongation in cultured entorhinal neurons. Fibers of entorhinal cortex(EC) project to hippocampal dentate gyrus(DG) in vivo. Therefore, we suspect that SDF-1 α attracts fibers from EC to DG. To check this possibility, we used co-cultures of slices from EC of GFP transgenic rats and hippocampus of wild rats. In this slice culture system, we are able to detect the projection of GFP-labeled axons from EC to DG. In this system, we observed that blocking of SDF-1 α caused reduction of projecting fibers from EC to DG. These results suggest that SDF-1 α plays critical roles in the axonal pathfinding step from EC to DG.

## P1-c33 Neurotrophin-3 influences cortical laminar formation during development of mouse cerebral cortex

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Laminar formation of cerebral cortex requires precisely regulated generation and migration of the cortical progenitor cells. We have revealed that brain-derived neurotrophic factor (BDNF), a member of neurotrophin family, altered the phenotypes of newly-generated cortical neurons with localization and gene-expression profile. In this study, we examined the effects of neurotrophin-3 (NT-3), another member of neurotrophins, on the cortical progenitor cells labeled with 5-bromodeoxy uridine (BrdU) after injection into the ventricular space of 13.5-day-old embryonic mice. The total number of BrdU+ cells in the whole cortex was increased. The BrdU+ cells were slightly decreased in layer IV, and increased in deeper layers at postnatal 6 days at low dosages of NT-3 (0.3-1.0 mg). However, at high dosage (3 mg) the BrdU+ cells were increased between layer II and V, predominant in layer IV. BDNF could not mimic these effects. These observations suggested that NT-3 would be involved in the decision processes of neuronal laminar fate in different ways from BDNF.

## P1-c34 Analysis of the downstream effectors for a transcriptional repressor RP58 in the embryonic cerebral cortex

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RP58 is a transcriptional repressor containing zinc finger and POZ domain. The mice deficient for RP58 gene show severe abnormalities in the cerebral cortical layer formation, suggesting its crucial role in cerebral development. The RP58 gene was highly expressed throughout the developing cortical plate, subventricular zone, hippocampus and amygdala. To identify RP58 effectors in the cortex, we performed micro-array analysis using RNA isolated from WT and RP58(-/-) cortex of E16. Among the genes that were up-regulated in mutant, we searched putative RP58 binding sites in the promoter sequences and identified several genes including Calretinin, Neurogenin 2, LTBP 2 and Tac 1 as candidates for downstream effectors of RP58. To ask whether these genes are practically regulated by RP58 in vivo, we performed Q-PCR and reporter assay using RP58 expression vector and promoter-luciferase constructs in cultured cells.

## P1-c35 RP58 may be involved in regionalization of neocortex and hippocampus

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To understand the function of a transcriptional repressor protein RP58, we generated mice lacking the RP58 gene. We found that expression of calretinin (CR) is enhanced in the mutant cortical region. The CR-positive cells are NeuN positive and Tbr1 negative, and correspond to late-born cortical neurons by BrdU labeling experiment, suggesting that the mutant late-born neurons migrate to cortical region and show CR immunoreactivity. Expression of $\alpha$-crystalline, a marker of hippocampal plate, is detected in the mutant medio-dorsal cortical region, suggesting that the boundary between neocortex and hippocampus shifts laterally in the mutant cerebral cortex. Therefore, the mutant increased CR-positive cells in the cortex may correspond to CR-positive cells observed in the wild-type hippocampal region.

## P1-c36 Microarray analysis of neurospheres treated with mood stabilizers.

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Neural stem cells (NSCs) are present in the subventricular zone in the adult brain and provide neurons in the olfactory bulb through the rostral migratory stream. We have shown that mood stabilizing drugs enhance the self-renewal of NSCs and expand the size of NSC pool in the adult brain. In this study, we performed microarray analysis using neurospheres which were cultured in the presence of mood stabilizers. Several genes were isolated, which were upregulated or downregulated by the drug treatment and we focused a gene that is downregulated more than two fold by both lithium and valproic acid treatment. The gene contains a WW domain and seems to act as a transcriptional coactivator, but its function in the nervous system is not known. By in situ hybridization analysis, the expression of the gene is detected in the tissue surrounding the lateral ventricle of the embryonic and adult brain, where NSCs exist. We are investigating the function of the gene in the NSCs.

## P1-c37 Profiling of gene expression required for mouse/*Drosophila* neurogenesis, which are identified by *Drosophila* RNAi Screening

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We have been performing genome-wide RNAi screening of *Drosophila* genes by injecting dsRNA into embryos (Andrej I. V., Alessandra C. R., Paola P., Siuk Y. et al., PNAS 2004/ Koizumi K., Higashida H., Yoo S. et al., PNAS, 2007). 65 genes including 34 novel genes required for embryonic neurogenesis were screened among 7312 so far (around 50% of total *Drosophila* genes). Semi-quantitative RT-PCR study of these genes during embryogenesis is now undergoing to figure out genes required stage specifically. To search genes involved in human developmental disorders, Mammalian counterparts of these *Drosophila* genes are also studied. We found some of human homologues cause human developmental disorders by their mutation and another homologues exist in putative autistic locus inferred from linkage analysis of autism patients. RT-PCR analysis of these genes in mouse embryonic brain will be also shown.

Grant-in-Aid for COE Research

## P1-c38 Decreased expression of Olig2 in response to $\beta$-amyloid accelerates the death of neural stem/progenitor cells.

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Despite increased neurogenic differentiation markers in hippocampal CA1 in AD, neurons are not replaced in CA1 and neocortex in the disease. A$\beta$ might contribute to deteriorate brain microenvironment for neurogenesis and survival of immature neurons. We examined whether A$\beta$ alters the expression of cell fate determinants in cerebral cortical cultures and in an AD mouse model (PrP-APPSW). The up-regulation of Mash1 and down-regulation of Olig2 were found in cerebral cortical cultures treated with A$\beta$1-42. A$\beta$ increased in the proportion of Olig2-negative/ mash1-expressing cells, which are not labeled with cell-specific markers. The decrease in Olig2+ cells was also observed in cerebral cortex of adult PrP-APPSW mice. However, Mash1+ cells were not detected in either wild type or PrP-APPSW mice. Cotransfection experiments with Mash1 cDNA and Olig2 siRNA revealed that OLig2 may decide the fate of Mash1-overexpressing cells to differentiation or death. We conclude that down-regulation of Olig2 by A$\beta$ in Mash1-overexpressing cells switch the cell fate to death.

## P1-c39 Expression pattern of Olig2 in the rat olfactory bulb following olfactory epithelium lesion

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Our previous study showed that nestin positive reactive astrocytes appeared in glomeruli of the olfactory bulb at 3~7 days after the olfactory epithelium lesion. To clarify the detail of astroglial response in the olfactory bulb, immunohistochemistry for the transcription factor olig2 was carried out. At 3~7 days after the lesion, glial progenitor cells with nuclear Olig2 expression significantly increased in the glomerular layer compared to those of the control animals that were sparsely distributed. Nestin positive reactive astrocytes showed the translocation of Olig2 expression into the cytoplasm. Thus the reactive astrocytes are thought to be derived from glial progenitor cells. Swift conversion from progenitors to reactive astrocytes following the lesion may play a crucial role on reinnervation in the olfactory bulb.

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## P1-c40 Analysis of *Drosophila* C2HC-type Zn finger factor dNZF in neuronal differentiation

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Neural zinc finger factor (NZF)/myelin transcription factor (MyT) gene family is constituted of transcription factors with DNA-binding domains of a C2HC-type (Cys-$X_4$-Cys-$X_{12}$-His-$X_5$-Cys) Zn finger conserved from *C. elegans* to human. The functions of the NZF/MyT gene family remain elusive, although *Xenopus* MyT1 was shown to play a critical role in neuronal differentiation. To elucidate NZF/MyT functions, we previously identified a *Drosophila* homolog dNZF and generated *dnzf* mutants which almost die at the pharate adult stage. Immunohistochemical analysis revealed that dNZF is expressed in a subset of neurons. To identify differentially expressed transcripts between wild-type and dnzf mutant flies, we performed differential display analysis using Genefishing kit. Finally, we identified 7 genes of which differential expression between them was confirmed by real-time quantitative RT-PCR. Moreover, we have made a *dnzf*-GAL4 line in which GAL4 was inserted into near the transcriptional start site of the *dnzf* gene using targeted transposition.

## P1-c41 BDNF supports the survival of neuronal progenitors differentiated from neural stem cells in vitro

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Generation of differentiated neurons and glial cells from neural stem cells (NSCs) or neural progenitor cells (NPCs) is a complex process involving interplay between intrinsic cellular programs and extrinsic cues such as growth factors. Brain-derived neurotrophic factor (BDNF) is the most plausible candidate to regulate cortical progenitor cells, because of its expression in the NSCs/NPCs during cortical neurogenesis. Although BDNF increases number of neurons generated from NSCs/NPCs after withdrawal of FGF-2, in vitro, the mechanism underlying this effect is largely unknown. NSCs/NPCs derived from the mouse cerebral cortex of 14-day-old embryos were cultured in the defined medium supplemented with FGF-2 (10 ng/ml) and/or BDNF (100 ng/ml) with a passage of every 3-4day, and the cells were characterized by proliferation rate, cell type and survival rate. Results demonstrated that BDNF facilitates survival of NPCs differentiated from NSCs, judged from longer doubling time, limited proliferation activity and preference to neuronal differentiation.

## P1-c42 Intrathecal infusion of growth factors stimulates NG2-expressing progenitors in mutant *SOD1*-transgenic rats

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NG2-expressing ($NG2^+$) cells, previously known as oligodendrocyte progenitor cells, constitute the largest proliferating cell population in the adult central nervous system. Recent reports have shown that the $NG2^+$ cells are intrinsically multipotent and have the ability to generate neurons. In the present study, we examined the $NG2^+$ cells in the spinal cord of mutant *SOD1*-transgenic (Tg) rats characterized by adult-onset selective motoneuron degeneration. In addition, we studied whether exogenous growth factors could promote the $NG2^+$ cell proliferation in the degenerating spinal cord. In parallel with the motoneuron loss, the $NG2^+$ cells proliferated significantly as compared with wild-type rats. However, the number of newly-generated mature oligodendrocytes was not significantly different between Tg and wild-type rats. Moreover, intrathecal infusion of EGF and FGF-2 in Tg rats significantly promoted the proliferation of $NG2^+$ cells. Therefore, the $NG2^+$ cells may be a potential target to develop a restoration therapy for neurodegenerative diseases.

KAKENHI (18790586)

## P1-c43 Enhanced expression of TGF $\beta$ 1 in cell cycle blocker-induced neuronal differentiation of neurosphere

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We have shown that pre-treatment with deferoxamine (DFO: cell cycle blocker in G1/S phase) increase the number of neurons from neural stem/progenitor cells (NPCs) with relation to prolonged enhancement of $p27^{kip1}$ mRNA expression. To investigate the molecular mechanism of neuronal differentiation by cell cycle blocker in detail, we focused on trasnforming growth factor beta (TGF $\beta$). As TGF $\beta$ 1 increases transcription of CKIs such as $p21^{cip1}$ and $p27^{kip1}$ and inhibits cell proliferation, we investigated the expression of TGF $\beta$ mRNAs at the end point of DFO treatment and at 24 hours after differentiation when $p27^{kip1}$ mRNA elevation has started. Expression of TGF $\beta$ 1 mRNA was significantly elevated after the differentiation, although it was not altered during DFO treatment. On the other hand, different TGF $\beta$ isoforms expression did not change. Data suggest that enhancement of TGF $\beta$ 1 expression along with $p27^{kip1}$ elevation would be included in the mechanism of neuronal differentiation followed by cell cycle arrest.

## P1-c44 Astrocyte-specific labeling with a recombinant lentiviral vector carrying DsRed protein driven by a human glial fibrillary acidic protein promoter

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We have constructed a recombinant lentiviral vector carrying DsRed protein driven by a hGFAP promoter. We checked expression patterns of cell type-specific markers in DsRed-positive cells 1 day after infection of primary cultured cortical cells with this lentiviral vector. In DsRed-positive cells, the expression pattern was as follows: 100 ± 0.32% for GFAP (+), 98.1 ± 1.15% for S100 $\beta$ (+), 100 ± 2.61% for GLAST (+), 81.6 % ± 3.31% for Nestin (+), 0.08 ± 0.43% for Tuj1 (+), and 0.00 ± 0.00% for O4 (+), indicating that this lentiviral vector achieved astrocyte-specific gene targeting. Currently we are investigating the in vivo expression pattern of these markers in DsRed-positive cells after direct injection of this lentiviral vector into the subventricular zone and white matter. This technique will be a strong tool for long term monitoring of morphology and differentiation of astrocytes and/or radial glia.

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## P1-c45 Characterization of the reeler Cerebral Cortex by expression of molecular markers

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The cerebral cortex is the six-layered structure. Previous birth-date labeling experiments suggested that the lamination pattern of the cerebral cortex of reeler, the Reelin deficient mutant mouse, is reversed. Recent studies identified genes expressed in the cerebral cortex in a layer specific manner. In this study, we carried out in situ hybridization using such marker genes to characterize the malformation of the reeler cortex. We found that distribution of cells expressing these markers did not exhibit reversed pattern of lamination. Double staining of the normal brain by detection of BrdU (birthdate labeling) and the layer markers showed relationships between marker expression and birthdate of the cortical neurons. However, such coupling of birthdate and specific gene expression was disrupted in reeler cortex. These observations suggest involvement of Reelin signal in regulation of birthdate of the cortical neurons.

## P1-c46 Notch signaling inhibits glial differentiation of the EGF-responsive cells in developing mouse cerebellum

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Notch signaling plays key roles in cell fate determination.We examined the effect of Notch signaling on differentiation of the EGF-responsive proliferating cells in early postnatal mouse cerebellum. The EGF-responsive cells expressed GFAP weakly and nestin strongly. These cells stopped dividing and differentiated into GFAP-strongly-positive and nestin-negative cells in response to CNTF and BMP2. Over-expression of Notch intracellular domain (NICD) inhibited stronger expression of GFAP induced by CNTF and BMP2 and maintained nestin expression even in the presence of these factors. Most of NICD-over-expressing cells were labeled with BrdU even in the presence of CNTF or BMP2. These findings suggest that persistent activation of Notch signaling in EGF-responsive cells inhibits glial differentiation of these cells induced by CNTF and BMP2 and maintain these cells in the proliferative state.

## P1-c47 Massive apoptosis during neurogenesis induced by XRCC4 inhibition

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Neural cell fate is determined by expression of proteins specific to neurons or glia cells. Transcription factor cascades and chromatin remodeling induce expression of these proteins. In addition, it has been hypothesized that genomic DNA rearrangement is essential for neurogenesis. In this study, Wstern blot analysis revealed that nestin, a marker of neural progenitor cells, appeared after increase of both histone H2AX phosphorylation, a marker of DSBs, and Xrcc4 involved in NHEJ. Inhibition of Xrcc4 protein by siRNA resulted in massive apoptosis of induced neural cells. Apoptosis was only found when the P19 cells with siRNA were treated to differentiate into neural cells. In conclusion, our data suggests that DSBs are induced before cell fate determination of neural progenitor cells and NHEJ plays a critical role in the process of neural cell fate determination.

## P1-c48 Analysis of BRINP-interacting proteins

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Once differentiated from stem cells, neurons no longer proliferate, but extend nerve fibers to form neural circuitry. The mechanism suppressing neuronal cell-division is not known. We recently identified a novel neuron-specific protein family, BRINP (BRINP1, 2, 3) that possesses no similarity with other proteins but suppresses cell-cycle progression and causes cell death in proliferating cells. In this study, we searched for proteins that interact with BRINPs and analyzed their characteristics. By utilizing the two-hybrid system, we identified several BRINP1-interacting proteins including Cyclin-dependent kinase 5 (CDK5) from a mouse brain cDNA library. IP-Western blotting experiment on CDK5/BRINP-expressed 3T3 cells confirmed the binding of CDK5 to all three BRINPs. However, strength of the binding varied with different BRINPs, i.e. BRINP3 > BRINP2 >> BRINP1. Further characterization of functional domains of BRINPs responsible for the CDK5-binding and cell-cycle suppression will be discussed.

## P1-d01 glial cells missing (gcm) genes in the induction of neural stem cells

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Neural stem cells (NSCs) first appear in E8.5 mouse embryos, but not in earlier embryos although neural tissue exists at E7.5. We propose that primitive NSCs exist in the E5.5-7.5 epiblast, which later differentiate to definitive NSCs. Here, we investigated roles of gcm-1/2 genes in the induction of NSCs. Because gcm-1/2 double mutant embryos developed apparently normal until E7.5 but died around E8.5, we cultured cells from E7.5 epiblast and let primitive NSCs differentiate to definitive NSCs in vitro. The efficiency to obtain neurospheres (derived from definitive NSCs) was much lower in gcm-1/2 double mutants than in littermate controls. Unexpectedly, when neurospheres were cultured under the differentiation condition, gcm-1/2$^{-/-}$ neurospheres as well as control neurospheres differentiated into neurons, astrocytes, and oligodendrocytes. These results suggest that gcm genes play an important role in the development of mammalian nervous system but that those genes are not essential for the generation of glial cells.

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## P1-d02 Activation of endogenous neural stem cells in intracerebral hemorrhage model rat

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We have developed internal capsule (IC) hemorrhage model rat which has a small hematoma but relatively severe motor dysfunction. We investigated the activation of endogenous neural stem cells (NSCs) in this model. To evaluate proliferation of NSCs, BrdU was administered for 14 days after the lesion. In the subventricular zone, BrdU-labeled cells increased in lesion group (147.0 ± 25.5, n=3, $p<0.05$) compared to control (37.7 ± 12.7, n=3). To evaluate migration of NSCs, BrdU was administered from 2 days to 2hours before the lesion. In the dorsal striatum adjacent to corpus callosum (dSTR), BrdU-labeled cells increased in lesion group (43.0 ± 3.6, n=4, $p<0.01$) compared to control (15.0 ± 1.0, n=4). Furthermore, DCX-positive cells in the dSTR and in the peri-hemorrhagic area also increased in lesion group (10.1 ± 1.4, n=8, $p<0.01$, 6.5 ± 1.0, n=7, $p<0.05$) compared to control (6.5 ± 1.0, n=8, 0.0 ± 0.0, n=6). These data suggested that proliferation and migration of endogenous NSCs are activated after intracerebral hemorrhage.

## P1-d03 *Mbh1* is involved in cerebellar granule cell differentiation downstream of a proneural gene, *Math1*

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Cerebellar granule cells (CGCs) are one of the major neuronal populations in the cerebellum. CGC differentiation is initiated by expression of a proneural gene, *Math1*, genetic ablation of which results in the failure of the external granule layer (EGL) formation. Its downstream targets required for proper CGC differentiation remain unidentified, however. Using transgenic mice and chromatin immunoprecipitation assay, we have previously shown that a homeobox gene, *Mbh1*, is a potential downstream target of Math1 in differentiating CGCs. Here we further indicate a role of *Mbh1* in the early differentiation of CGCs. Delivery of *Math1*-specific siRNAs by *in vivo* electroporation knocks down Math1 protein levels in CGC precursors, which causes downregulation of *Mbh1* as well as aberrant EGL formation. In addition, transfection of a dominant negative form of *Mbh1* abrogates EGL formation and downregulates NeuroD and TAG-1, markers for differentiating CGCs. These data suggest that *Mbh1* function is essential for proper CGC differentiation.

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## P1-d04 Changes in expression of genes and proteins during neural differentiation from embryonic stem cells by Neural Stem Sphere method

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Mouse embryonic stem (ES) cells unidirectionally differentiate into neurons via neural stem cells by Neural Stem Sphere (NSS) method. Here, we analyzed changes in expression of genes and proteins during the differentiation. We identified vimentin, creatine kinase, galectin and some proteins, which were up-regulated in neural stem cells and down-regulated in ES cells and neurons by proteomic analysis. We also measured expression level of the corresponding gene by real-time RT-PCR and compared the levels with those of the proteins.

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## P1-d05 Super selective neuronal differentiation and application of cynomolgus embryonic stem cells

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Primate ES cells have rather different character from rodent ones, but it is inevitable to elucidate mechanism for stable culture, purification and induction into object-oriented differentiation, because human ES cells might show wide similarity to cynomolgus ones. We improved the way of large scale culture maintaining totipotency without contacting feeder cells indispensable for primate ES cells. Our refibed super selective induction method for dopaminergic neurons enabled us to transplant induced neuronal precursor cells in vivo as xenograft which survive without forming tumor such as teratoma for long period with more accuracy and reliability. Their immunohistological, eletrophysiological and ethological evaluation suggests high stability, activity, ability to make neural network system and potentiality to improve clinical symptom of Parkinsonism. Fully functional neural network with other types of induced neurons is under development.

## P1-d06 Isolation of neural stem cells from damaged rat cerebral cortex after traumatic brain injury

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In this study, we isolated and cultured nestin-positive cells from tissue around cerebral cortical damaged area after traumatic brain injury (TBI) in the rat, and examined the possibility of differentiation into neurons or glia.Nestin-positive cells were seen around the damaged area at 1 , 3 and 7 days after rat TBI. Tissue isolated from around the damaged area at 3 days after injury and spheres was cultured with bFGF and EGF. These spheres could not be isolated at 1 and 7 days after injury. These isolated spheres consisted of nestin positive neural stem cells. Neurospheres differentiated into Tuj1-, GFAP- and O4-positive cells after 4 days in culture without bFGF and EGF. These results indicate that isolated and cultured neurospheres can differentiate into neurons and glia. An increase in nestin-positive cells around a cerebral cortical damaged area might contribute to neurogenesis and neuroplasticity.

## P1-d07 Rat and human glioma cells express NG2 proteoglycan and immune cell markers

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Many gliomas respond to platelet-derived growth factor. Involvement of NG2 chondroitin sulfate proteoglycan (NG2) has been reported in platelet-derived growth factor (PDGF)-mediated signaling by sequestrating PDGF. In this study, RT-PCR, immunoblotting and immunocytochemistry revealed that rat C6 glioma and several human glioma cell lines expressed NG2 as well as PDGFalpha receptor. Furthermore, C6 glioma and some human glioma cells expressed macrophage-markers such as CD68 and Iba1. The glioma cells possessed activities of esterase and phagocytosis to some extent. C6 glioma also expressed mRNAs encoding lymphocyte markers, CD4, CD8 and CD200. Such expression patterns of the glioma cells are distinct from those of astrocytes and microglia. Similar Iba1+/NG2+ cells, which may be blood-borne, are found in ischemic core of rat brain after middle cerebral artery occlusion. These observations may shed light to study on the lineage and nature of glioma.

## P1-d08 New method for investigating guidance effects of chondroitin sulfates *in vitro*

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Chondroitin sulfates (CSs), carbohydrate side chains of chondroitin sulfate proteoglycans, are involved in formation of retinotectal pathway in chick embryos, delimiting a border of the pathway and chronological sorting of the axons in the optic tract. CSs exhibit structural diversity with various disaccharide units combined; however, it has not been shown whether the CSs affect the growth cones directly or not. In addition, there have been few studies about the structural diversity and functional specificity. A method called the spot assay, examining growth of the axons around the spot border, was not suitable for the study about CSs. Here we established a method to elucidate effects of the CSs and their structural diversity and functional specificity. We investigate behaviors of the growth cones in contact with the polystyrene beads coated with chondroitin sulfate phosphatidylethanolamine.

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## P1-d09 The origin and development of the precerebellar neurons in the spinal trigeminal nucleus

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Precerebellar neurons, generated in the lower rhombic lip (LRL), form several brainstem nuclei, including the pontine gray, reticulotegmental, external cuneate, lateral reticular, and inferior olivary nuclei. In addition to these major nuclei, several others, including the spinal trigeminal nucleus, also project to the cerebellum, but their origin remains unknown. Here we show that a subpopulation of the interpolar subnucleus of the spinal trigeminal nucleus (Sp5I), which project to the cerebellum, is derived from the LRL. By introducing an *EGFP* gene into the LRL by *in utero* electroporation, we labeled precerebellar neurons *in vivo*. Some of the EGFP-labeled neurons migrated on the surface of the medulla and formed nuclei in the contralateral Sp5I region. These neurons expressed a precerebellar neuronal marker gene, and sent their axons to the ipsilateral cerebellum. Furthermore, these neurons were localized in Sp5I in the adult brain. These data strongly suggest that precerebellar neurons in Sp5I are generated in the LRL.

## P1-d10 Real-time imaging of precerebellar neurons during migration and early steps of nucleogenesis

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By observing a series of fixed preparations, we have previously documented the migratory behavior of precerebellar (PC) neurons, which change migratory direction from tangential to radial at defined positions to form nuclei. Here, we performed real-time imaging of PC neuron subsets, pontine grey nucleus/ reticulotegmental nucleus (PGN/RTN) neurons, in mice embryos to gain insight into the mechanisms regulating their migration and nucleogenesis. PGN/RTN neurons were labeled by in vivo electroporation of EGFP plasmid into the lower rhombic lip, and live imaging was performed on coronal slices. We found that 1) the rate of radially migrating cells was slower than that of tangentially migrating ones; 2) some PGN/RTN neurons radially migrated past the presumptive nuclear region and then reversed back towards the region. All migratory behaviors observed in live preparations were consistent with morphologies of PGN/RTN neurons in fixed tissues. We speculate that an attractive activity from their final destinations may be involved in PGN/RTN nucleogenesis.

## P1-d11 GABA and glutamate regulate the differentiation of newborn hippocampal granule cells

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Newborn granule cells (GCs) migrate from the tertiary dentate matrix (tdm) of dentate hilus to the granule cell layer in the early postnatal hippocampus. In this study, we investigated whether GABA and glutamate regulate the differentiation of newborn GCs in neonates because GABAergic neurons are abundant in tdm and GCs receive excitatory inputs from the entorhinal cortex. Hippocampal slices were obtained from postnatal six-day-old rats. The hilar region of slice including tdm was dissociated to prepare the primary culture of immature GCs. At DiV4, cultured GCs were classified into three developmental stages from I to III. Chronic application of 1 mM GABA significantly increased the number of stage III cells. Although 50 μM glutamate did not affect the developmental stage of GCs, it increased dendritic branches of stage III cells. These results indicate that both GABA and glutamate affect the differentiation of immature GCs, but have different roles.

## P1-d12 Termination of lesion-induced barrel plasticity is independent of oligodendrocyte differentiation in the mouse somatosensory cortex

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A critical period (CP) for lesion-induced barrel plasticity ends at a specific time point during development. To elucidate mechanisms responsible for termination of the CP for barrel plasticity, we investigated the role of oligodendrocytes (OLs) in the mouse barrel cortex (S1). We found that OL differentiation proceeds markedly at the end of the CP. Interestingly, although OL differentiation was blocked in Olig1-null mice, the CP normally terminated at P5 even in the absence of cytoarchitectonic changes in layer 4 of S1. Thus, our results indicate that OL differentiation occurs at the end of the CP, but is not intrinsically necessary for termination of barrel plasticity. A recent report showed that genetic deletion of Nogo, which is expressed in OLs, extends the CP for ocular dominance in the visual cortex. Thus, different cortical areas may utilize distinct mechanisms for termination of CPs.

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## P1-d13 Slingshot regulates primary dendrite formation in rat hippocampal neurons

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Neurons extend axons and highly branched dendrites during development. It remains to be known how the number and shape of dendritic arbors are determined in the developing nervous system. We examined the role of cofilin on the number and morphology of dendrites in cultured rat hippocampal neurons. Cofilin is a key regulator of actin dynamics by stimulating actin filament depolymerizaton and severing. Cofilin is inactivated by LIM-kinase (LIMK) and activated by Slingshot (SSH) phosphatases. In the earlier developmental stage (before 7 DIV), overexpression of SSH1 induced the abnormal accumulation of actin filaments in the tip of neurites. In the later developmental stage (after 14 DIV), knockdown of SSH1 increased the number of primary dendrites, in contrast to control cells which did not sprout the new dendrites. These results suggest that SSH1 negatively regulates the de novo formation of primary dendrites in the later developmental stage. We also examined the roles of cofilin, LIMK and SSH in neurite extension in PC12 cells and chick DRG neurons.

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## P1-d14 Layer-specific gene expression in developing rat retrosplenial cortex

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The granular retrosplenial cortex (GRS) in rats has a highly modular organization in layer 1 (L), where dendritic bundles of L2 neurons are detected from postnatal day 5 (P5), by GluR2/3 and of L5 neurons from P3, by OCAM. To investigate the molecular mechanisms of this temporal and spatial specificity, we carried out gene expression profiling where L2 and 5 were excised in GRS and, for comparison, in barrel cortex: at P3 (before formation of L2 dendritic bundles), at P12 (after their formation), and at P28 (adult condition). One group of genes was highly expressed both in L2 and 5 at P3. The expression in L5 remained, but that in L2 was transitory. Another group of genes was highly and specifically expressed in L2, but not in L5 of GRS at P3. Of these, the most highly expressed (>64-fold) is *NT-3*, (known to regulate dendritic growth in developing cerebral cortex), as well as some genes in the MAPK cascade. Thus, we propose that NT-3 and its downstream MAPK cascade are involved in dendritic bundle formation.

## P1-d15 Cytoplasmic domain of Protocadhrin-α is essential for establishment of normal serotonergic projection

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*Protocadherin-α (Pcdha)* is a subfamily of cadherin superfamily and consists of multiple isoforms genereated from a unique gene cluster. *In situ* hybridization study revealed that the strong signals were detected in several brain regions including the cerebral cortex, hippocampus, Purkinje cells of the cerebellum, and serotonergic (5-HT) neurons of the raphe nuclei. In this study, we focused on the functional roles of *Pcdha* family in the development of 5-HT neurons. We used *Pcdha* mutant mice in which the cytoplasmic domain of Pcdha protein is partially truncated, and examined the morphology of 5-HT neurons and fibers in the mutant mice. 5-HT fibers in some regions such as the hippocampus prominently increased in the mutant mice, but in the other regions such as the dorsal striatum apparently decreased. These results demonstrate that cytoplasmic domain of Pcdha is essential for the normal development of 5-HT system in the mouse brain.

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## P1-d16 UNC-51 regulates axonal and dendritic patterning in the Drosophila brain

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The conserved serine/threonine kinase UNC-51 has been implicated in axonal development in *C. elegans* and mouse. We have previously shown that UNC-51 regulates kinesin-dependent synaptic vesicle transport in segmental nerves interacting with UNC-76, a kinesin-1 adaptor. Here, we investigate developmental functions of UNC-51 in wiring complex brain networks focusing on the mushroom body (MB) and the antennal lobe (AL). We show that UNC-51 mutations result in defective axonal development, in which MB lobe and core structures are markedly disrupted. In addition, a diverse array of neuronal molecules such as Fas II, Syt and NC82 is mislocalized, which is reminiscent of the previous observation in segmental nerves. Moreover, loss of *unc-51* leads to significant impairment of dendritic patterning in AL with irregular glomerular arborization and occasional overshooting. These data suggest that, apart from axonal transport, UNC-51 may regulate diverse neuronal processes such as axonal guidance, fasciculation and dendritic patterning.

## P1-d17 Leukocyte cell-derived chemotaxin 2 (LECT2) regulates neuritic extension and branching

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We have previously demonstrated that LECT2 was remarkably localized in the soma and neurites in cultured neurons until 3 days in culture. To clarify the role of LECT2 in neurons, we compared the outgrowth of axons and dendrites in cultured neurons from LECT2-KO mice (KO) with those from wild-type mice (WT). After 4 days in culture, the length of the axons and dendrites of KO were significantly shorter than those of WT. On the other hand, after 14 days in culture, the axonal branching of KO was inferior to that of WT; although there were no significant differences in the length of axons and dendrites between KO and WT. Furthermore, immunostaining for LECT2 demonstrated that LECT2 was expressed in the soma and neurites after 4 days in culture, and only in the soma after 14 days in culture. These results suggest that LECT2 regulates the neuritic extension and participates in the regulation of axonal branching.

## P1-d18 Enhancement of axon and dendrite outgrowth by drebrin expression at early developmental stages in hippocampal neurons

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We have previously shown that drebrin E is primarily localized in the actin arc of growth cones at early developmental stages. In this study, we examined the effect of overexpression of drebrin in neurite outgrowth. We prepared the primary cultures of rat hippocampus using Banker Methods. At 0 day in vitro, the cells were transfected with GFP-tagged drebrin E or A cDNA. After 48 hours transfection, the cells were fixed and analyzed their morphology. Both of GFP-drebrin E and A accumulated in the tips of neurites. Overexpression of drebrin diminished the size of growth cones, and increased the axon length and the number of dendrite. These data suggest that the drebrin E plays a role in the outgrowth of axons and dendrites at early developmental stages.

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## P1-d19 Receptor-type protein tyrosine phosphataseζ(PTPζ) associates with DNER and regulates its subcellular localization

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PTPζ uses a heparin-binding growth factor pleiotrophin as a ligand and play important roles in the morphogenesis of cerebellar Purkinje cells. We previously showed that PTPζ interacted with Delta/Notch-like EGF-related receptor, DNER. Several tyrosine residues in the cytoplasmic region are phosphorylated, and act as putative substrate of PTPζ. In the Neuro-2A cells transfectants expressing DNER, this protein was actively internalized depending on the tyrosin-based and dileucine-type sorting motifs. Pleiotrophin suppressed the internalization and led to the accumulation of DNER on the plasma membrane. Though DNER expression suppressed retinoic acid-induced neurite extension of Neuro-2A cells, pleiotrophin stimulation reversed this effect. These observations suggest that pleiotrophin-PTPζ signaling regulates morphogenesis of neurons by modifying the intracellular transport of DNER.

## P1-d20 Expression of chondroitin sulfate sulfotransferases in the developing mouse cerebellum

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Chondroitin sulfate (CS) chains are modified by various sulfotransferases generating diverse sulfation patterns, which are assumed to be involved in the selective binding to various proteins such as growth factors. Our previous study suggested that CS-D type structure generated by UST is important for the development of Purkinje cells. In this study, we analyzed the expression patterns of several CS sulfotransferases (UST, GalNAc4-6ST, D4ST) in the postnatal mouse cerebellum. While the expression of UST gene increased from P1 to P14, that of GalNAc4S-6ST and D4ST genes decreased during this period. Using in situ hybridization, it was revealed that these genes were expressed by Purkinje cells, Bergmann glia, and granular cells. These results suggest that the structural variation of CS chains may regulate Purkinje cell-Bergmann glia interaction and granule cell migration.

## P1-d21 The role of sonic hedgehog in dendritic spine formation

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During development of vertebrate central nervous system (CNS), Sonic hedgehog plays essential roles in various processes including dorsoventral patterning of the neural tube, proliferation of neural progenitors and guidance of growing axons. Here, we found that Sonic hedgehog induced dendritic spine formation in primary cultured neurons. Sonic hedgehog and its receptors Patched1 and Smoothened were expressed in developing cerebellar Purkinje neurons and hippocampal pyramidal neurons at early postnatal stages. Sonic hedgehog treatment significantly enhanced spine maturation in hippocampal neurons. Conversely, inhibition of Sonic hedgehog pathway by a specific inhibitor cyclopamine retarded spine maturation. GST-pull down assays revealed that smoothened bound to molecules localized at post-synaptic density via the cytoplasmic tail, suggesting its localization in the post-synaptic membrane. These data suggest that Sonic hedgehog might be involved in spine formation in CNS dendrites.

## P1-d22 Essential role of Rac1 in development of cerebral cortex and hippocampus

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Neuronal development requires highly precise regulation of actin-based structures to achieve coordinated migration and precise wiring of neurons. Rac1, a member of the Rho family of small GTPases, is assumed to regulate neuronal cell migration and neurite outgrowth through actin cytoskeletal reorganization. However, it is poorly understood how Rac1 contributes to these cellular functions because of the embryonic lethality of Rac1-deficient mice. In this study, we show that conditional deletion of Rac1 in the dorsal telencephalon, including cerebral cortex, hippocampus and olfactory bulb, results in abnormal cortical and hippocampal morphogenesis. The mutant mice showed partially abnormal layering of cerebral cortex and striking reduction of commissural axons, such as the corpus callosum and the anterior commissure. These results suggest that Rac1 requires neuronal cell migration and proper axonal targeting in cerebral cortex.

## P1-d23 Role of reactive oxygen species on cell migration in neural progenitor cells

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In the development of the cerebral cortex, neural progenitor cells (NPC) are known to migrate from the ventricular zone to the cortical plate. However, the molecules regulating this migration have not well elucidated. Recently, it has become clear that reactive oxygen species (ROS) work as secondary messengers in various cells. In the present study, we examined whether ROS is involved in migration of NPC *in vitro*. Phenylephrine, a selective $\alpha_1$-adrenoceptor agonist, induced migration of NPC derived from mouse brain at embryonic day 14. N-acetyl-L-cycteine, a ROS scavenger, inhibited the phenylephrine-induced migration. DCF assay revealed that stimulation with phenylephrine induced ROS production. We next examined the source of ROS. Apocynin, an NAD(P)H oxidase inhibitor, attenuated the phenylephrine-induced migration. Furthermore, we studied the effect of an inhibitor of Rac, which is required for NAD(P)H oxidase activation. NSC23766 inhibited the migration in a dose-dependent manner. These results suggest that ROS produced by NAD(P)H oxidase regulates $\alpha_1$-adrenoceptor-mediated migration in NPC.

## P1-d24 Roles of pre- and post-synaptic neuron activity in interhemispheric connection development in the mouse visual cortex

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Targeted neuronal silencing using ion channels becomes a useful technique to elucidate the mechanism for activity-dependent neuronal wiring. By using a potassium channel Kir2.1, we investigated the role of neuronal activity in the formation of interhemispheric (callosal) axon projections. The region- and layer-specific projection pattern of callosal axons from one cortical hemisphere to the other was visualized by unilateral in vivo GFP electroporation. To explore the role of neuronal activity in callosal axon development, Kir2.1 was expressed in either callosal projection neurons or their target postsynaptic neurons. Exogenous Kir2.1 expression in cortical neurons reduced their firing rate. Kir2.1 expression in projection neurons disturbed their axon arbor growth, while that in postsynaptic neurons had limited effect on the pattern of presynaptic axon projections. These results suggest that targeted Kir2.1 expression is a useful method to reveal distinct effects of pre- and post-synaptic activity-blockade on cortical wiring.

## P1-d25 ARD1-NAT1 complex regulates neuronal dendritic arborization through $\alpha$-tubulin acetylation

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A regulation of microtubule (MT) organization is critical for the dendritic development. Expression of ARD1 and NAT1, an N-acetyltransferase complex, increased during dendritic development, and both proteins were co-localized with MT in dendrites. We investigated whether the complex functions on dendritic development through $\alpha$-tubulin acetylation. Alpha-tubulin acetylation at Lys40 was critical to the dendritic extension. ARD1-NAT1 complex enhanced a transfer of acetyl group to purified MT fraction. ARD1 is a catalytic subunit of the N-acetyltransferase, and knockdown of the ARD1 reduced the acetylation level of $\alpha$-tubulin in HEK293T and dendritic extension in cultured neurons. Overexpression of dominant-negative form of ARD1 also limited the dendritic arborization. We propose that the regulation of $\alpha$-tubulin acetylation by ARD1-NAT1 pathway is involved in the dendritic development.

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## P1-d26 Involvement of BIG-2 in convergence of olfactory axons to target glomeruli

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Olfactory sensory neurons (OSNs) expressing a given odorant receptor (OR) converge their axons onto a few topographically fixed glomeruli, resulting in establishment of the odor map on the olfactory bulb (OB). Here we report that BIG-2 (contactin 4), a GPI-anchored axonal glycoprotein belonging to the immunoglobulin superfamily, is expressed by a subpopulation of OSNs. Immunohistochemical staining of mouse OB sections revealed a mosaic pattern of glomerular arrangement with strongly BIG-2-positive, weakly positive, and negative axon terminals, which is overlapping but different from mosaic expression patterns of Kirrel2 and ephrin-A5. There is a close correlation between the expression level of BIG-2 protein and the choice of OR in individual OSNs. In BIG-2-deficient mice, OSNs expressing a given OR frequently innervate multiple glomeruli at ectopic locations. These results suggest that BIG-2 is one of the axon guidance molecules playing crucial roles in the formation and maintenance of functional odor map in the OB.

KAKENHI 16300105

## P1-d27 Molecular mechanism that regulates medially oriented axonal growth of upper layer neurons in cortical development

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During development layer 2/3 cells extend axons medially toward the contralateral cortex. Here, we studied the mechanism for axon outgrowth of layer 2/3 cells in cortical slice culture. To label these axons, a yellow fluorescent protein-encoding plasmid was introduced into the ventricular zone cells by *in utero* electroporation when layer 2/3 cells are born. Within a day after transfection, a cortical strip containing the labeled region was dissected and cultured. After 3 days *in vitro*, most labeled cells were found to extend axons medially. This medially oriented growth was disrupted when the lateral portion of the cortical slice was removed or when the slice was cut shorter along the mediolateral axis. These results indicate that medially oriented growth of layer 2/3 cell axons was controlled by a repulsive molecule, which is localized laterally or distributed in a lateral high- medial low gradient fashion in the cortex.

KAKENHI(18021021)

## P1-d28 A possible involvement of Netrin-1 as a guidance cue for hippocampal mossy fibers

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Hippocampal mossy fibers (MFs), axons of dentate granule cells (GCs), run through the stratum lucidum and make synaptic contacts with CA3 pyramidal cells. We previously proposed the existence of chemoattractants in CA3 but the molecules have not been revealed. In this study, we utilized an explant coculture system and examined whether Netrin-1, a guidance molecule, guides MFs to CA3. CA3 mini-slices and dissociated GCs were prepared from postnatal six-day-old rat hippocampus and cocultured in collagen gels. After three days in vitro, almost all MF axons (89.58 ± 5.72%) projected toward CA3 slices in control cultures, whereas about half of them (41.53 ± 13.84 %) did not show any direction preference in the presence of function-blocking anti-Netrin-1 antibody. Theses results suggest that Netrin-1 serves as a chemoattractant for MFs.

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## P1-d29 Possible role of histone deacetylase 6 in brain

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HDAC6, a microtubule-associated deacetylase, has been thought to regulate microtubule-stability by reversible acetylation. As HDAC6 is abundant in brain tissue, HDAC6 might contribute to acquire proper function and maintenance in brain. To elucidate this, we examined its expression profile in whole brain. In human brain, HDAC6 is expressed by most of neurons. However, HDAC6 is abundant in neurons composing cerebellar nuclei, locus ceruleus, and substantia nigra. HDAC6 is also abundant glial cells and neuronal fibers. Intrestingly, strong expression of HDAC6 was observed in reactive-astrocytes and granule cells moving in the cerebellar molecular layer, suggesting that HDAC6 is required for cytoskeletal rearrangement. We also examined the subcellular localization of HDAC6 in rat cultured neuron. HDAC6 localizes to not only perinuclear region, but also neuritis and spines. Those evidences suggest that HDAC6 may play an important role in the construction of cytoskeleton in neurons, which in turn, contribute to the formation of synapse and neuronal network.

## P1-d30 The role of neural activities of thalamic and cortical neurons in thalamocortical axon branching

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Thalamocortical (TC) axons form elaborate branches in the developing cortex. Our recent study using cocultures of the thalamus and cortex has shown that neural activity plays a role in TC axon branching. Here we assessed the relative importance of neural activities of the thalamus and cortex for TC axon branching by overexpressing inwardly rectifying $K^+$ channel (Kir) 2.1, in either thalamic or cortical cells in the coculture preparations. Electrophysiological recordings revealed that spontaneous firing, which appears during culturing, was substantially decreased in Kir2.1-transfected neurons. When thalamic neurons were transfected with Kir2.1, the TC axons formed little branching in the cocultured cortex. TC axons invading the cortical region with many Kir2.1-expressing neurons also formed less branching. These results suggest that neural activities of both thalamic and cortical neurons are required for TC axon branching.

KAKENHI(18300105)

## P1-d31 Nerve injury induces the expression of syndecan-1 heparan sulfate proteoglycan

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Heparan sulfate proteoglycans, which bear long chains of heparan sulfate glycosaminoglycan, play significant roles during embryogenesis, including the formation of th CNS. However, their involvement in nerve regeneration has not yet been clarified. Previously, we reported that nerve injury induces the expression of EXT2, a glycosyltransferase required for heparan sulfate synthsis, heparan sulfate glycosaminoglycan and syndecan-1 heparan sulfate proteoglycan. In this study, we examined the expression of syndecan-1 after hypoglossal nerve injury. The upregulation of syndecan-1 mRNA and protein were detected using in situ hybridization and immunohistochemistry in injured hypoglossal motoneurons, and strong signals were also detected in the proximal stump of injured nerve in immunohistochemistry. These result indicate that syndecan-1 is upregulated in injured motoneuron, transported to nerve lesion and might be involved in nerve regeneration.

## P1-d32 Regeneration of nigrostiatal dopaminergic axons by transplantation of olfactory ensheathing cells is accompanied by elimination of the fibrotic scar formation in the lesion site

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Transplantation of olfactory ensheathing cells (OECs) into the lesion site of the central nervous system is known to enhance regeneration of transected axons and functional recovery. To examine the mechanism underlying the axonal regeneration by OEC transplantation, the nigrostriatal dopaminergic axons were unilaterally transected in rats and cultured OECs mixed with fibroblasts were transplanted into the lesion site. In mice transected and transplanted with OECs and fibroblasts, numerous dopaminergic axons were regenerated through the transplanted tissue which was filled with p75-immunoreactive OECs and fibronectin-immunoreactive fibroblasts. In the lesion site of transplanted animals, deposition of type IV collagen and the formation of a fibrotic scar were completely prevented. From these results we propose that axonal regeneration which occurs after OEC transplantation is mediated by the elimination of the fibrotic scar.

KAKENHI18500271

## P1-d33 Regeneration of optic nerve axons in zebrafish

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The CNS of teleost fish is capable of spontaneous axon regeneration after nerve fiber injury. The mechanisms in neurons that allow successful axonal regeneration are not fully understood. Both extrinsic factors from the neuronal environment and intrinsic neuronal factors seem to play a role in successful nerve regeneration. Zebrafish retina is a useful model for the study of axonal promoting factors. In this study, we examined effects of conditioned media from neurons or cells on axonal regeneration from cultured zebrafish retinal explant. Neurite outgrowth from the explant retina was observed in the medium 10% FBS but not in the medium containing 1% FBS. However, conditioned medium from rat macrophages promoted axonal regeneration in 1% FBS up to the same level as in 10% FBS.

## P1-d34 Upregulation of galectins in retina and its possible role in fish optic nerve regeneration

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Unlike mammals, fish optic nerve can regenerate its axons after lesion. We have investigated these regeneration mechanisms using zebrafish and goldfish visual system. We observed that two types of galectin, galectin-1 and galectin-3, were upregulated in both retinas after optic nerve lesion. These proteins are known to have the ability to promote cell-cell interactions. By in situ hybridization and immunohistochemical study, galectin-1 expression was initially increased in all retinal nuclear layers, and then concentrated in the ganglion cell layer during optic nerve regeneration. Galectin-3 showed a similar expression pattern in the retina during nerve regeneration. The time of increased levels of galectins corresponded with the axonal elongation stage after lesion. Furthermore, addition of recombinant galectin protein induced neurite outgrowth in culture. These data indicate that upregulation of galectins is an impotant event for regrowing optic nerve axons.

## P1-d35 Cell survival and axonal regeneration of injured retinal ganglion cells by purpurin

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The retinal ganglion cells (RGCs) in mammals cannot regrow their axons and become apoptotic after nerve injury. However, goldfish RGCs can survive and regenerate optic nerve after injury. We attempt to block the cell death and accelerate axon outgrowth of rat RGCs by useful molecules derived from goldfish after injury. We cloned purpurin from axotomized retina 5 days previously, a secretory retinol binding protein as the early stage molecule of regeneration. The purpurin mRNA was dramatically increased in the photoreceptor cells by 2 days after injury. Then it secreted toward to RGCs revealing by immunohistochemitry. In the goldfish retinal explant culture, purpurin induced axon elongation through retinoic acid signaling pathway. Furthermore, purpurin showed a promoting activity of cell adhesion and survival to adult rat RGCs. We are now in progress to investigate axonal elongation of rat RGCs by purpurin.

## P1-d36 New canie spinal cord injury model free from laminectory

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Rehabilitation is essential for functional recovery after a spinal cord injury. It requires long period and effort. To establish efficient programs for rehabilitation, we explored a new canine spinal cord injury model free from laminectomy using a balloon. After surgical operation, these dogs moved using with forelimbs alone, and showed spastic tonus in their hindlimbs. Pathological examination of the damaged spinal cord showed the formation of cavities surrounded by scar tissue containing high level of collagen. One and a half years after operation, the electromyograms of forelimb and hindlimb were measured. Forelimb integrated electromyogram(IEMG) was from 0.0106 to 0.0387, whereas, hindlimb IEMG was from 0.0036 to 0.0280. Recovery from paralysis was not observed clearly because it was the case which didn't undergo systematic rehabilitation.

## P1-d37 Neuroprotective effects of transplanted olfactory ensheathing cells on axotimized retinal ganglion cells in adult rats

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Although olfactory ensheathing cells (OECs) have been successfully used to enhance axon regeneration within the injured central nervous system, no evidence could demonstrate any in vivo protective effect of such macroglial cells on injured central neurons. In the present study, we investigated the effect of OECs on neuronal survival of axotomized retinal ganglion cells (RGCs) in adult rats. OECs suspension or the same volume of DMEM/F12 medium was injected into the ocular stump of the orbitally transected ON in the experimental or control group. All animals were allowed to survive for 2, 7 or 14 days respectively following operation and the numbers of FluoroGold-labeled RGCs were counted. A significantly higher number ($P<0.01$) was detected at 7 day in the experimental group than in the control group. Thus, transplanted OECs are capable of protecting injured RGCs at a certain period after ON transection.

## P1-d38 Difference in regeneration and survival between OFF-center ganglion cells and ON-center cells in cat retina

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By detecting dendritic ramification in the IPL we examined whether fewer OFF-retinal ganglion cells (RGCs) survived axotomy and regenerated axons than ON-RGCs, and whether axonal regeneration of OFF-RGCs was promoted when number of regenerating RGCs (r-RGCs) was increased with drugs. Ratio of ON-RGCs versus OFF-RGCs was 1.26 (ON 56%/OFF 44%) in the normal retinae. In RGCs surviving axotomy, ratio of ON-cells versus OFF-cells increased to 3.1, 75%/24%, indicating ON cell dominance. In surviving RGCs of the retinae injected with nipradilol (NO donor, beta blocker) the ratio was improved to 1.2, 55%/45%. In r-RGCs of saline injected retinae (control), the ratio increased to 2.61, 72%/28%. An intravitreal injection of combination of BDNF+CNTF+forskolin increased only the number of OFF-r-RGCs. Nipradilol injection increased both ON- and OFF-r-RGCs. Immunohistochemistry showed denser trkB in the inner IPL of intact retina. Thus lower surviving ability of OFF-RGCs may be attributed to lower density of trkB. Smaller ability to survive axotomy of OFF RGCs may result in smaller regeneration ability

## P1-d39 Neurons and astrocytes exhibit lower activities of global genome nucleotide excision repair than do fibroblasts

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Nucleotide excision repair (NER) is a DNA repair pathway which eliminates various types of helix-distorting DNA damage including some forms of oxidative damage and UV-induced photoproducts. To understand why patients with NER-defective disorders develop progressive neurological abnormalities, we investigated NER capabilities in neural cells. Primary cultured neurons and astrocytes derived from rat embryonic brains were prepared in mixed-cell cultures, and fibroblasts from the same embryos were cultured for comparison. Determination of damage in situ using antibodies was used to measure NER capabilities in neural cells, which were identified by staining with specific cell markers. The results demonstrate that neural cells have significantly lower NER capabilities than fibroblasts. That result was consistent with the finding that levels of an NER-related protein (PCNA) recruited at the UV-damage sites were lower in neural cells than in fibroblasts.

## P1-d40 Primary sensorimotor activation during affected hand grasping after hemiparetic stroke: A cross-sectional NIRS study

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Motor recovery after stroke may be attributable to cerebral reorganization. We investigated the laterality of primary sensorimotor cortex (SM1) activation using NIRS between patients with good (n=3) and poor (n=5) recovery at chronic stage after stroke. NIRS probes were placed on the scalp overlying both sensorimotor cortices, and the cerebral Hb changes were recorded during 5 cycles of 15 s unilateral hand grasping and 30 s resting periods. We defined a contralaterality index as CI=(Nc-Ni)/(Nc+Ni), where Nc and Ni denote the numbers of channels that show significant changes in Δ[Oxy-Hb] of contra- and ipsi-lateral hemispheres, respectively. The good recovery patients showed a contralateral predominant pattern (CI=0.48±0.13), while CI of poor recovery patients was -0.33±0.20 during affected hand grasping. This result suggests the relationship between SM1 laterality and the degrees of recovery after stroke.

JST/CREST

## P1-d41 Responses of oligodendrocyte progenitors in the spinal cord following dorsal rhizotomy in the adult rat

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In the adult spinal cord, oligodendrocyte progenitors (OPs) are distributed throughout both the white and gray matter, but little is known concerning the potential role of OPCs. We examined the response of OPs to the degeneration of the central processes of primary sensory neurons induced by dorsal root transection. OPs were identified by staining with antibodies against NG2 and Olig2. Axonal degeneration resulted in a prompt response of OPs in the dorsal funiculus and dorsal horn, consisting of morphological transformation, cellular proliferation, and upregulation of NG2 expression days 2-4 after surgery. Olig2 staining was exclusively nuclear until 3 weeks after surgery, and thereafter a clear cytoplasmic pattern of Olig2 staining was found in numerous cells in the dorsal funiculus. The majority of cells with nuclear Olig2 co-expressed GFAP. These results suggest that axonal degeneration induces a unique sequence of changes of OPs and that a subpopulation of the newly generated OPs differentiate into astrocytes.

## P1-d42 Large-scale change in the corticofugal projections in rats with neonatal hemi-decortication

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To study the mechanism of functional recovery of forelimb movements in rats that received unilateral decortication at PND5, descending projections from sensorimotor cortex (SMC) of the remaining hemisphere was studied neuroanatomically and electrophysiologically at their adult stage. After injection of biotinylated dextran amine, massive aberrant terminations were observed in the contralateral red nucleus and pontine nucleus, and ipsilateral dorsal column nucleus and gray matter at the cervical segments. Electrical stimulation of the remaining medullary pyramid evoked clear aberrant negative field potentials in dorsal horn of ipsilateral cervical segments and polysynaptic EPSPs in ipsilateral forelimb motoneurons. These aberrant corticofugal projections may underlie the functional restoration of forelimb movements in these animals.

JST, CREST, Kawaguchi

## P1-d43 Exercise has a neuroprotective effect in parkinsonian rat model

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We examined the effects of compulsive exercise in rat model of parkinsonian disease. After 6-hydroxydopamine injection into the striatum, the rats were forced to run on the treadmill for 30 min each day. The exercise group displayed a significant decrease in amphetamine-induced rotational behavior and a significant preservation of tyrosine hydroxylase (TH)-positive fibers in the striatum and TH-positive neurons in the substantia nigra pars compacta (SNc) compared with non-exercised one. The great number of the Bromodeoxyuridine-positive cells and Doublecortin-positive cells were found in the subventricular zone in the exercise group. Some of them were migrated toward the lesioned striatum and differentiated into neurons. The mechanism is considered that neurotrophic factors, especially GDNF or neurotrophins, increase throughout the striatum or SNc by exercise. These results suggest that exercise in the parkinsonian disease may have a neuroprotective effect or enhance the neurogenesis.

## P1-d44 Developmental changes in mouse striatal neurons differ to those seen in rat

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Medium spiny neurons (MSN) of the mammalian striatum are thought to integrate and filter cortical input, and hence play a major role in behavior. Whilst much basal ganglia research has been performed in rats thus far, the advent of genetic techniques favors the mouse model. In order to determine the developmental changes occurring in mouse MSNs, we performed whole-cell recordings and labeled individual neurons in mouse brain slices at different ages. Our analyses thus far indicate significant differences in morphometric and physiological parameters between age groups. Furthermore, developmental trends in our results differ with those reported in rats. In particular, we report the presence of an inward rectifying potassium current ($K_{ir}$) in all MSNs of mice as young as postnatal day 10. $K_{ir}$ is an important determinant of membrane potential states, and hence vital to the role of MSNs in filtering cortical input. Our results suggest that systematic characterization of mouse striatal neurons may be an important step in basal ganglia research.

University of Sydney Research and Development Grant

## P1-d45 Sensory basis of motor program selection in the cricket behavior

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Crickets react differently to an air puff stimulus, that is, running and/or jumping on the ground, swimming when on a water surface and flying when suspended in the air. Leg ablated crickets occasionally react to an air puff by flying even when they are placed on a water surface. Sensory inputs from legs are thus important for the behavioral selection. Some small hair sensilla on the legs were equipped with 2-5 sensory neurons. All such hair sensilla had a tiny pore at the tip. These findings suggest that they are chemosensitive hairs. Electrophysiological investigations proved that some of the sensilla were accompanied with water receptor cells. Therefore, such sensilla are thought to play an important role for the behavioral selection between swimming and flying. On the other hand, crickets placed on a high viscosity methylcellulose solution sometimes show walking. Therefore, the resistance force from substrate to the legs was thought to be important for the initiation of walking.

## P1-d46 Neurons expressing GAD 67 and glycine transporter 2 mRNA in and around the trigeminal motor nucleus of the rat

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The trigeminal motor nucleus (Vm) and its surrounding neuropil contain commissural GABAergic and glycinergic neurons that send axons to contralateral Vm. In the present study we used digoxigenin labeled probes to detect GAD 67 and glycine transporter 2 (GLYT 2) mRNA by *in situ* hybridization. As a result, expressions of GAD 67 and GLYT 2 mRNAs were observed in and around Vm. Some neurons expressed GAD 67 mRNA or GLYT 2 mRNA, respectively, and some neurons expressed both mRNAs. Previous studies have shown that GAD 67 and GLYT 2 are often co-localized in trigeminal premotoneurons. Our results also indicate that some neurons in and around Vm coexpress GAD 67 and GLYT 2 mRNA.

## P1-d47 Development of a quantitative evaluation system for motor function using wrist movement

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We developed a new system for quantitative evaluation of motor function and propose a method that characterizes abnormal movements in various nervous diseases. As an experimental task, we asked subjects to perform various wrist movements such as step-tracking wrist movements and visually guided tracking movements. The participants included patients with spinocerebellar degeneration or Parkinson's disease as well as normal controls. We recorded two-degree-of-freedom wrist joint movement and activities of four wrist prime movers (ECR, ECU, FCU, FCR) simultaneously. To analyze relationship between muscle activities and movement dynamics, we estimated dynamics of the wrist joint from the four muscle activities using a dynamic equation. Our results demonstrated that there is enough information in the activities of the four muscles to explain the location, speed and acceleration of the wrist joint. Thus, it is possible with our system to characterize abnormal movements in various neurological disorders quantitatively.

KAKENHI(18700202)

## P1-d48 The striatofugal system from patch and matrix compartments in rat neostriatum

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The striatum can be divided into two compartments, patch and matrix, which are known to have distinct chemical compositions and connections. Here, neurons located in either patch or matrix were infected with recombinant sindbis virus , and their labeled axons were entirely reconstructed with a camera lucida. Some reconstructed axons originating from matrix arborized into the three main striatal targets, while some reconstructed axons originating from patch terminated at only external segment of globus pallidus. These data are inconsist with the previous concept of a dual striatofugal system: direct/indirect and patch/matrix, and call for a reevaluation of the organization striatofugal projection system.

## P1-e01 Functional connection from Mauthner cell to homologous reticulospinal neurons in the goldfish hindbrain

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Teleost hindbrain contains segmentally arranged reticulospinal neurons(RSNs). Paired large RSNs in the fourth segment(r4), Mauthner(M) cells, are known to initiate escape behavior. Activation of the M-cell produces only the initial phase of escape, suggesting involvement of other RSNs in the control of the subsequent body movement. Here, we investigated the physiological connection between M-cell and the RSNs in r5(MiD2cm, MiD2i) by paired recordings in adult goldfish. The intraaxonal activation of M-cell produced inhibitory postsynaptic potentials in the ipsilateral RSNs with onset latency of 1.1msec. This inhibition is strong enough to stop bursting of the postsynaptic RSNs. By contrast, the activation of M-cell elicited long-lasting depolarization in these neurons on the contralateral side. No apparent response was induced in the M-cell by firing of the RSNs in r5. The connection pattern from M-cell to the RSNs in r5 was repeated in r6, suggesting duplication of the wiring pattern in the two neighboring segments.

KAKENHI (17023029) (18300134)

## P1-e02 Time-domain coding of somatosensory reflex gain during arm movements

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Somatosensory reflex gain during movement is known to be modulated according to given tasks and environments anticipatorily, before movement initiation. It is unclear, however, whether the reflex gain modulation is completed before movement start or the gain is set toward its target (i.e., dynamical environment) in movement. Here we investigated temporal feature of anticipatory reflex gain modulation when subjects performed planar reaching movement with different force fields in the later part of movement. A small perturbation to induce arm reflex response was applied at several timings before the arm encountered the force field. We found clearer reflex gain modulation according to the force field directions as the perturbation was applied closer the force fields. An additional experiment further showed that the reflex modulation was developed in relation to the temporal distance to the force fields rather than the spatial distance. These findings suggest that somatosensory reflex gain is coded in time domain for upcoming environments, independently of movement execution.

## P1-e03 The relationship between the sense of self-agency and visuo-motor incongruency

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When one acts physically or mentally, one always feels that I act myself, which is the sense of self-agency. A sense of self-agency is the feeling of causing our own actions. Franck et al. (2001) reported that when there is a discrepancy in visuomotor perception, we might not feel a sense of self-agency. Participants moved a mouse device and viewed resultant feedback, which was biased either temporally or spatially. They then judged whether there was a bias or whether they felt they had moved the cursor on their own. We found that the sense of self-agency is felt even when a perceptible visuomotor temporal bias occurs, but that it is not felt when a perceptible angular bias is presented. The sense of self-agency may be brought about by the detection of a contingent relationship between several modalities (Van den Bos & Jeannerod, 2002). The amount of contingency in visuomotor information in the temporally delayed condition may be sufficient to enable one to feel a sense of self-agency. We would not identify as ourselves things that move simultaneously.

## P1-e04 Influence of finger somatosensory information on the coordination of bimanual finger movements

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Human frequently perform movements using right and left fingers. In this study, we investigated the coordinated control mechanisms in finger movements. Subjects performed in-phase and anti-phase cyclic movements by forefingers. During these movements, to explore the cross talk information between inter-finger control systems, we changed the motion visual feedback gain or viscosity force field gain of the left finger tip. The external load change in left finger resulted in a less effect on the right finger movement, but the amplitude change in the left finger had much effect on the right finger movement. Next, to explore the effect of arm configuration, subjects were instructed to performed in-phase movements tasks under different distance between both hands. The effects became stronger as the distance between both fingers was closer. These results suggest coordinated system is much influenced by the somatosensory feedback form forefingers and depended on arm configuration.

## P1-e05 Information analysis of Monkey AIP neurons during hand manipulation task

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We analyzed spike activities of the monkey anterior intraparietal area (AIP) to reveal how the brain represents information related to hand manipulation. During the hand manipulation task, AIP neurons show selectivity to the 3D shape, orientation and size of the objects to be grasped and/or the hand configuration to grasp them. Here, we calculated mutual information (MI) between spike frequency of single neurons and various clusters of 6 objects, and examined how MI changed according to different task periods (i.e., fixation, reaching and grasping) for each type of clustering. MI took peak values in different periods dependent on clustering types: For some neurons, MI for rough clustering had the peaks in the fixation period before reaching, while MI for fine clustering had the peaks in the reaching or grasping period. This suggests that AIP neurons may represent information relevant to the hand manipulation task in a hierarchical manner, that is, different grains of information in different periods.

## P1-e06 Hindlimb locomotor pattern evoked by the midbrain stimulation in decerebrate, paralyzed rabbits

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In decerebrate rabbits, stimulation to the midbrain cuneiform nucleus (CNF) evokes hopping movements of the hindlimbs. The movements are characterized by nonalternation of bilateral hindlimbs and co-contraction of extensors and flexors at the late support phase. This study aimed to know functional involvements of sensory feedbacks in generating such hopping pattern. After decerebration, the rabbits were paralyzed with pancuronium bromide and artificially ventilated. To evoke locomotion, 50-Hz stimuli (30-110μA, 0.2ms duration, 10-15sec) were applied to the CNF. Electroneurograms (ENG) were recorded from extensor and flexor nerves of the hindlimbs. During stimulation, extensor and flexor ENGs showed rhythmic activities. But nonalternation of bilateral limbs and co-activation of extensors and flexors were not always observed. This suggests that both spinal locomotor pattern generators and sensory feedback signals from active body parts are necessary for generating the full hopping pattern.

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## P1-e07 Quantitative analysis of inputs to projection neurons from the neocortex or the thalamus in the rat neostriatum

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Projection neurons in the neostriatum are composed of at least two groups: striatopallidal neuron group and striatonigral neuron group. These projection neurons resceive excitatory inputs from the neocortex and the thalamus. In this study, we quantitatively analysed the relation between the two groups of the projection neurons and two kinds of inputs in the neostriatum. Dendrites of projection neurons in the neostriatum were visualized by a recombinant virus vector which labeled the infected neurons in a Golgi stain-like fashion. Axon terminals input to projection neurons in the neostriatum from the neocortex or the thalamus were stained by the immunofluorescence method. The appositions of the dendrites to the inputs were observed three-dimensionally with a confocal laser-scanning microscope. Some appositions were examined electron-microscopically and calculated the densities of inputs.

KAKENHI(17791305)(18019017)

## P1-e08 Effects of task requirements on the brain activity in the coincident timing task

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Subjects intercepted a moving visual target on the screen at the designated point using a cursor manipulated by handgrip force. Timing- and position (=force) errors were converted to the score. Two conditions of low- and high task requirement were prepared in each block of trials to manipulate psychological pressure. In the high-requirement condition (condition P), if the subject could not obtain a preset criterion score, the total score of that block was nullified, while if he could, obtained score was doubled. In the low-requirement condition (condition C), obtained score was kept intact. Anxiety level was higher in condition P and in failed block. Timing error decreased and force error increased in condition P. An fMRI imaging revealed enhanced activity of amygdala and SMA, and depressed activity of prefrontal cortex in condition P, suggesting high psychological pressure and anxiety modifies the working memory and motor planning.

KAKENHI(16300206)

## P1-e09 Balance control of the virtual inverted pendulum: effects of experimentally introduced time delays

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To understand how humans use open- and closed-loop control in visually guided movement, we constructed a virtual inverted pendulum (VIP); its dynamics was simulated within a computer (its temporal characteristics could be changed), and subjects could stand it in a display by moving a mouse according to visual feedback. The present study examined effects of time delay (0~400 ms) additionally introduced between subject's action and movement of the VIP bottom on balance control of the VIP (that was equivalent to a 1 m-rod in the gravity field of 0.2 G (G=9.8m/s$^2$)). As results, (1) in control (time delay, 0 ms) subjects intermittently moved the VIP. Each action showed bell-shaped velocity profile with duration of ca. 150 ms ("go" phase). Between subsequent "go" phases a "stop" phase was intercalated, in which the VIP bottom remained still. (2) Introduction extra time delays decreased occurrence of "stop" phase. Appearance of continuous "go" without intercalated "stop" could be related to the open-loop control based on the prediction of on-going planned action.

## P1-e10 Cerebellar complex spikes encode movement in intrinsic coordinate frames

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What triggers complex spikes (CS) of cerebellar Purkinje cells (PC) is a crucial issue for understanding the cerebellum. There is a well-known spiky increase of the CS discharge around onsets of visually guided forelimb movements. We examined whether the increased discharge is triggered by the visual stimulus or any other intrinsic signals, such as motor commands. We used a wrist movement task that introduced a delay between the presentation of a target and the go signal for movement initiation. The CS discharge was tightly linked to the movement onset rather than to the go signal. Furthermore, directional selectivity of the CS discharge covaried for a change in posture, indicating encoding in intrinsic coordinate frames. These results suggested that the discharge of the CS is triggered by an internal signal, such as efference copies, rather than a visual stimulus associated with the go signal.

PRESTO/JST, MESSC (SK).

## P1-e11 Rhythm learning in the monkey

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Rhythm is a fundamental aspect of our motor behaviors. It is important not only in music, but also in any skilled behaviors. However, the neural basis of rhythm is largely to be studied. To study the neural mechanisms of rhythmic motor control, we have trained two monkeys on a rhythmic button press task. In this task, the monkeys were required to press an LED button when it flashed with or without a tone. The button flashed six times (beats) with an isochronous rhythm (inter-beat interval: 750, 1000, or 1500 ms), a complex rhythm (combination of two intervals), or random intervals. The monkeys did learn the rhythms as indicated by the shortening of the reaction time. The monkeys' performance was compared to that of human subjects. Both monkeys and humans learned faster rhythms better. For the complex rhythms, both species performed better with the 750/1500 ms intervals (1:2 rhythm) than with the 750/1000 ms intervals (1:1.33 rhythm). These results suggest that the monkeys and humans share the basic mechanisms of rhythmic motor control.

## P1-e12 Continuous information flow decreases the activity of continuous information flow decreases the activity of supplementary motor area (SMA) in coincident timing task

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The environment surrounding us is abundant. Therefore, different types of information can specify the same event. For example, a certain time in the future can be specified by two consecutive onsets of stimuli as well as by continuous time-to-arrival information. We investigated human brain activity in interval and interceptive timing tasks, in which discrete and continuous information specifying the same timing was provided, respectively. Brain activity was recorded by 1.5 T fMRI when participants performed the tasks. Results showed that the SMA was activated more strongly in the interval timing task than in the interceptive timing task, suggesting that different neural networks are activated for timing control depending on different information provided to specify the same timing.

KAKENHI(17500416)

## P1-e13 Activity of pursuit neurons in the caudal part of the frontal eye fields (FEF) during static roll-tilt

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To examine whether coordinate frames representing FEF pursuit signals are orbital or earth-vertical, we compared preferred directions of FEF pursuit neurons during upright and static, whole-body roll-tilt in head- and trunk-restrained monkeys. Preferred directions (re monkeys' head/trunk axis) of virtually all FEF pursuit neurons tested (n=21) were similar during upright and static whole-body roll-tilt. The slight shift of preferred directions of the majority of neurons could be accounted for by ocular counter-rolling. Mean (±SD, n=29) discharge rates during upright and right ear down and left ear down conditions were 14(±7), 15(±7), and 15(±8) spikes/s. Visual motion preferred directions were also similar between upright and 40° right ear down. These results indicate that pursuit neurons in the caudal FEF do not signal static roll-tilt and that they code pursuit signals in head/trunk-centered coordinates.

KAKENHI (18300130)

## P1-e14 Membrane properties of prepositus inhibitory neurons in VGAT-Venus transgenic rats

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The prepositus hypoglossi nucleus (PHN) is an important center for controlling horizontal eye movements. In this study, to clarify electrophysiological properties of inhibitory PHN neurons, we investigated membrane properties of PHN neurons in vesicular GABA transporter (VGAT)-Venus transgenic rat in which inhibitory neurons expressed a fluorescent molecule, Venus, using whole-cell patch clamp technique. Comparison of membrane properties between Venus-positive and -negative PHN neurons revealed that a single, fast afterhyperpolarization, firing patterns with a delay in the first spike generation, and those with the longer first interspike interval than the second one were preferential properties in Venus-positive neurons. These membrane properties will be useful for identifying inhibitory PHN neurons.

KAKENHI (18021005, 18019007)

## P1-e15 Cerebellar plasticity suggested by the facilitation and transfer of saccadic adaptation

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Feed-forward associative learning (FFAL) theory proposed by the author (2005) connects cerebellar cell activities with motor behaviors under the cerebellar adaptive control. There have been at least two challenges to the theory from experimental results of saccadic adaptation. One is the adaptive facilitation (Kojima et al., 2004). Faster rate of gain decrease suddenly reduced to a normal rate during a second set of gain decrease trials. Not LTP but de-LTD of synapses from parallel fibers to Purkinje cells is required for this rate-inflection to take place from the viewpoint of the theory. Another one is the transfer of saccadic adaptation (Kaku et al., 2006). The vectorial transfer component of saccades in different directions is almost equivalent to a vectorial adaptive component of the adapted saccades in accordance with what the FFAL theory tells. However, precise examination of the data revealed slight difference in these vectorial components. Under the assumptions that the LTD will become greater at the synapses with more powerful connectivity that has been induced by memory transfer, the FFAL theory can also explain the difference.

## P1-e16 A model of small involunatay eye movements to reproduce squre-wave jerks

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Square-wave jerks (SWJ) are pairs of involuntary microsaccades that take the eye off from the fixation target and regress to it after 100-200 ms (Leigh & Zee, 1983). In this study, we proposed a mathematical model of small involuntary eye movements which can reproduce SWJs based on a saccadic neural system model (Broomhead et al, 2000). Because SWJs occur in the darkened room (Shaffer et al, 2003), we supposed that second microsaccades which correct the eye back to the fixation target arise without any relation to the retinal information. If saccades are not planned, the fixation position is corrected based on the saccadic distance information stored in the resettable neural integrator (RNI) immediately after a involuntary microsaccade has been generated. RNI automatically discharges the integrated signals and sends back to the superior colliculus. We implemented this algorithm into a saccadic neural system model. As a result, our model can reproduce SWJs suggesting that RNI may play a role in accurate gazing.

## P1-e17 An active role of MST in maintaining visual stability during saccades in the natural environment

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In order to investigate the neural substrate maintaining visual stability during saccades in the natural environment, we measured human brain activity during visual search and that of visually guided saccades that reproduce eye movement pattern of precedent search. We found dissociable activation patterns within hMT+. In area MST, activations during visual search were significantly lower than those during visually guided saccades, both with and without background pattern. In contrast, MT showed no different activations between visual search and visually guided saccades. The lower MST activation during visual search suggests that MST does not passively code retinal slip accompanying saccades. Rather MST is most likely involved in the neural system responsible for saccadic suppression, and MST activity may depend on whether retinal slip is caused by internally driven saccades such as visual search or externally driven (visually guided) saccades.

## P1-e18 Responsiveness of primate cerebellar hemispheric lobule VII to saccade and smooth pursuit eye movements

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Our previous studies revealed that distinct areas in cerebellar hemisphere lobule 7 (H7) are involved in saccade and smooth pursuit eye movement control. Here, we analyzed simple spike activities of H7 Purkinje cell (P-cell) during visual guided saccades and pursuits in two monkeys. Two-thirds of recorded H-7 P-cells increased discharges transiently before the onset of saccades, without any direction or amplitude preference. They also showed phasic or tonic increase of discharges before the onsets of pursuits, with a slight preference in the downward direction. No particular eye velocity sensitivities were recognized in most of them. In 20% of them, micro-stimulations of recorded sites induced either slow or quick eye movements with long latencies. These results suggest that H-7 plays a role in the control of saccades and smooth pursuits, involving their initiation timing.

## P1-e19 Visual responses of monkey cerebellar hemispheric lobule VII during saccade eye movement preparation

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The cerebellum plays an important role in movement control. Our previous study reveals that the cerebellar hemispheric lobule-VII (H7) is involved in saccades and smooth pursuit eye movements. In order to examine the possible role of H7 in the preparation of saccades, we analyzed activities of simple spikes from H7 Purkinje cells while the monkey performed visual (VGS) and memory (MGS) guided saccades. We compared saccade cue- and movement-related activities between VGS and MGS. In VGS, most of H7 neurons showed increase of discharges at saccade go signals and subsequent saccades. In MGS, they increased discharges to the brief presentations of saccade target cue while fixating on the central point, and at saccade go signals and actual saccades. These neurons also showed transient changes of activities to the flashing of central fixation point even in the absence of saccades. These results suggest that H7 receives not only eye movement-related signals but also saccade-related visual signals.

SORT, JST,

## P1-e20 Properties of the anticipatory convergence eye movement

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The convergence eye movement is a disjunctive eye movement to an approaching visual target. Also convergence eye movements preceding visual motion are found in human and cat. We trained cats (n=7) to start convergence by an alarm signal (buzzer sound or combination of buzz and blinking of LED), preceding target movement by 4s. After training, ocular convergence was elicited by the alarm signal before target movement in 60% of trials. In three cats, we used training with obliquely approaching target. After training, asymmetrical anticipatory eye movements were observed. The lateral suprasylvian area (LS) is one of the extrastriate cortices of cats which receives visual information from V1 and microstimulation in the LS evoked convergence eye movements. Localised lesion in the LS decreased the amplitude of visually-evoked convergence eye movements and occurrence of anticipatory convergence eye movement. Based on these findings and related LS neuronal activities, we will summarise the properties of anticipatory convergence eye movements in both species and the role of the extrastriate visual system in cat.

## P1-e21 Involvement of superior colliculus in visual-oculomotor processing after unilateral V1 lesion

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Superior colliculus (SC) is supposed to be a critical region for visuo-motor transformation related to saccade execution and many SC neurons exhibit visual and/or saccade-related activities. Monkeys with damage to the primary visual cortex (V1) can make saccades to visual stimuli in their affected visual field. To examine the neuronal basis for this residual ability, we recorded neuronal activities in SC of the monkey with unilateral V1 lesion during visually guided saccades. In the ipsilesional SC we found visual responses to the target in their response field, while their peak firing rate decreased and their onset and peak latencies were delayed, compared with those of contralesional SC. Our results show that ipsilesional SC can be driven by visual stimuli and would be involved in visual-oculomotor processing, but somewhat in a different manner from the intact hemifield.

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## P1-e22 Neck proprioceptive inputs to pursuit neurons in the caudal frontal eye fields (FEF)

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The smooth-pursuit system moves the eyes accurately to track slowly moving objects. By applying passive rotation of the trunk against the stationary head, we examined responses of 150 FEF pursuit neurons to passive trunk rotation when the monkeys fixated a stationary spot in space. Compared to the high percentage of horizontal pursuit neurons responding to horizontal trunk rotation (84/108=78%), only minority of pursuit neurons with vertical or oblique preferred directions were activated by horizontal trunk rotation (9/42=21%), indicating that neck proprioceptive responses are direction specific. The average sensitivity of horizontal pursuit neurons was 0.56 sp /s/deg/s. During ramp trunk rotation, neck proprioceptive responses exhibited velocity and position components with the modal latency of 75 ms. Modulation during smooth-pursuit and neck proprioceptive responses added linearly, suggesting that neck inputs contribute to discharge modulation of FEF pursuit neurons during smooth-pursuit if the trunk was rotated.

KAKENHI(18300130)

## P1-e23 Latency of vestibular responses of pursuit neurons in the caudal frontal eye fields (FEF) to whole body rotation

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We examined the latency and time course of FEF pursuit neuron discharge to whole body step-rotation in head-fixed monkeys. Pursuit neurons were classified either as gaze-velocity or eye/head-velocity neurons based on the previous criteria. Responses to whole body rotation were tested during cancellation of the vestibulo-ocular reflex (VORc), VORx1 and chair steps in complete darkness (VORd). About 70% of pursuit neurons responded during VORd with latencies < 80 ms. The initial responses were similar in the 3 VOR tasks and were also similar between the 2 groups of neurons. During VORc and x1, discharge of the 2 groups diverged at 90ms following chair rotation onset, consistent with pursuit latencies. The time course of discharge rate difference of the 2 groups between VORc and x1 was predicted by the modulation associated with pursuit. These results suggest involvement of the FEF in integration of vestibular inputs and pursuit signals.

KAKENHI(18300130), KAKENHI(18700377)

## P1-e24 The effect of a symbolic cue on human smooth pursuit initiation

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We studied effects of a symbolic cue informing the future pursuit target on smooth pursuit initiation in 4 human subjects. Each trial started when a central fixation spot was presented on a CRT monitor. After the subject fixated the spot, two small random-dot patches (centered 3 deg left and right of the center) and a central cue stimulus were presented for 100-1000 ms (cue-duration). Then, the cue and one of the patches disappeared and the remaining patch, which the subject was instructed to pursue, moved toward the center. Experiments were executed in two separate blocks. In one, an arrow-cue indicating the future pursuit target was presented, and a meaningless diamond-cue in the other. We compared the initial pursuit responses between two different cues. When the cue-duration was 300 ms or longer, the pursuit responses obtained with the arrow-cue were significantly larger than those obtained with the diamond-cue, suggesting that it is the time spent to choose the target based on the symbolic cue.

KAKENHI (17700309)

## P1-e25 Amphiphysin Ir participates in receptor-mediated endocytosis in ARPE-19

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Amphiphysin Ir (amph Ir) is a retina-specific splice variant of amphiphysin I with a putative role in synaptic vesicle endocytosis. In human retina, amph Ir is present in the ribbon synapses and the pigment epithelium (RPE), suggesting roles of amph Ir not only in the visual transduction but also in maintaining the health of neural retina. Using RNAi and pulse- chase assays with endocytic markers, we addressed the possible involvement of amph Ir in endocytosis in a cell line ARPE-19, which is commonly used as a model of human RPE. Amph Ir-specific siRNA clearly interfered with the endocytic pathway of transferrin (Tsfn) . In many cells with reduced amph Ir expression, Tsfn uptake still occurred after 20 min of chase, but instead of accumulating in perinuclear regions, it accumulated in small punctate structures dispersed in the cytoplasm, with some enrichment at the cell periphery. Immunoreactivities to clathrin heavy chain or dynamin overlapped to some extent with the Tsfn-containing puncta. These results suggest that amph Ir is involved in the clathrin-dependent, receptor-mediated endocytosis in human RPE.

## P1-e26 Selective responses of neurons in the intraparietal sulcus of the monkey during a visual grouping task guided by attention

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To study neural mechanisms of visual grouping of discrete objects, we designed a grouping detection task and recorded neuron activities from the lateral bank of the intraparietal sulcus (IPS) and area V4. Visual stimulus composed of 5 square black or white dots placed on gray background to form a cross. Monkeys had to release the lever when three dots with the same contrast were aligned either horizontally or vertically (target). To biase grouping in one orientation, we controlled attention either by using a visual cue or by biasing orientation within trial blocks. In both IPS and V4, many neurons showed selective responses to the target but the selectivity was quite different between two areas. In IPS, most neurons (71 of 104) showed greater selectivity to target orientation that was a key feature to the task rather than the contrast. In contrast, most V4 neurons (26 of 34) showed greater selectivity to contrast. These results suggest that IPS neurons play an important role in visual grouping guided by attention.

## P1-e27 Discrete color-related subregions in the monkey inferior temporal cortex

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Single neuron recordings have shown that there are many color-sensitive neurons in the inferior temporal (IT) cortex, but it is still unclear how they distribute in this area. We examined color-related responses in broad brain area, including the IT cortex, by using fMRI in the alert macaque monkey. We measured the BOLD responses to chromatic stimuli (colored gratings and Mondrian-like patterns) and achromatic stimluli (same patterns without color) while the monkey was performing a fixation task. Color-biased responses were observed in LGN, V1, V2, V3 and V4. In addition, some color-biased patches were distributed in the IT cortex; around the posterior middle temporal sulcus, in the posterior bank of the superior temporal sulcus and in the anterior middle temporal sulcus. Their positions were partly overlapped with shape-biased patches detected in a separate experiment. Our results support the hypothesis that color-sensitive neurons are concentrated in multiple sub-regions in the IT cortex.

KAKENHI(17022040)

## P1-e28 Influence of visual saliency in monkey visual cortex

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Recent studies suggest that a detection of a visually salient object in a cluttered scene involves processing in multiple areas in occipital, parietal and frontal cortices. To clarify the whole picture of this network, we measured the influence of the saliency on the activities in the monkey brain by using fMRI. We presented an array stimulus containing 8 disks during a fixation task; in some arrays one of the disks was unique in color (singleton), in others all were identical. We found that the presence of the singleton in the array enhanced the responses in V2/V3/V4, LIP and FEF. The enhancement was stronger in the contralateral hemisphere in V2/V3/V4 and part of LIP, but not in the other areas, suggesting these areas have different functions. Interestingly, we also found a strong response enhancement with the contralateral bias on the posterior bank of the superior temporal sulcus, which was anterior to the motion-sensitive region identified by a separate experiment. This sheds new light on the neural system involved in the detection of the salient object.

## P1-e29 Functional effects of 5-HT1B and 5-HT2A receptors in macaque V1

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We recently reported that mRNAs of serotonin receptor subtypes, 5-HT1B and 5-HT2A, are specifically expressed in the primary visual cortex (V1) of macaque monkey. To elucidate functional roles of these receptors in vivo, we performed single-unit recordings of visual responses from 101 V1 neurons with microiontophoretic administration of specific drugs for each receptor subtypes in anesthetized and paralyzed monkeys. The effects of activating these receptors seemed to be bi-directional depending on the activity levels of each neuron. The effect of agonist for 5-HT1B receptor, CP93129, was more facilitatory when the firing rate was high, but more suppressive when the firing rate was low. The effect of DOI, agonist for 5-HT2A receptor, was opposite in direction. We also found that activation of these receptors affects the relative timing of firings to visual stimulation. We concluded that these receptor subtypes control the gain and the timing of visual responses complementarily.

## P1-e30 Signal propagation properties in the visual cortex evoked by flush photolysis of a caged glutamate

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Spatio-temporal properties of signal propagation in the visual cortical circuit have not fully understood. Previously, we studied the signal propagation pattern induced by externally applied electrical stimulation to the visual cortex. In this study, we used a UV laser photolysis of the caged glutamate for stimulation, since it mimics the excitation induced by the synaptic transmission. The induced exciation was measured by $Ca^{2+}$ imaging system.

When the photostimulation was applied to layer 4, which receives inputs from LGN, the transient intracellular $Ca^{2+}$ ($[Ca^{2+}]_i$) elevation was observed. The high $[Ca^{2+}]_i$ region spread horizontally from the stimulus site in layer 4. The high $[Ca^{2+}]_i$ region also spread vertically to layer 2/3 and layer 5 and then spread horizontally in these layers. The horizontal spread of high $[Ca^{2+}]_i$ region in layer 2/3 was more restricted than those in layer 4 or 5, and sometimes a hotspot was observed in layer 2/3. When the photostimulation was applied to layer 2/3 or layer 5, the transient $[Ca^{2+}]_i$ elevation was observed only within the same layer of the stimulus site.

## P1-e31 Characteristics of neuronal responses in cortical areas MT/MST of monkey during pursuit eye movements

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To study neuronal mechanisms of our ability to perceive visual motion in space independent of pursuit eye movements, we studied responses of neurons in the cortical areas MT/MST of monkeys to textured images, either random-dot pattern or check pattern, which were presented briefly and moved at various speeds during smooth pursuit and stationary fixation. During pursuit, most MST neurons were more sensitive to the actual motion of the textured image than to the motion of its retinal image, whereas most MT neurons showed the reverse tendency.

When a check pattern was used and its temporal frequency exceeded 20Hz on the retina, neuronal responses in both areas decreased irrespective of fixation or pursuit eye movement. The decreased neuronal responses at higher temporal frequencies on the retina support the idea that the MST neurons code visual motion in space while compensating for the eye movements by utilizing the retina-based information obtained from the area MT and the information encoding eye movements.

KAKENHI17022019

## P1-e32 Match to heading tasks influence MST neuronal responses to optic flow

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When moving through our environment we monitor our heading to match our established path. We studied the influence of a match to heading task on optic flow responses in MST neurons. Two monkeys distinguished between sustained or reversed headings. We first presented large-field optic flow simulating one of eight headings. After a blank-screen delay, a second optic flow stimulus presented either the matching or the opposite heading. Two-thirds of the neurons (65%, 67/103) showed significant heading selectivity; half of which (31%, 32/103) also showed stimulus condition effects. These were equally divided between neurons with decreased responses amplitudes to either the match or the opposite test stimulus. An additional 7% (7/103) of the neurons showed significant heading by condition interaction effects, suggesting a change in heading selectivity, typically as a loss of selectivity in response to either the match or opposite stimuli. These findings suggest top-down directional influences on optic flow responses in MST that might contribute to recognizing heading deviations.

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## P1-e33 Express visual search

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The neural mechanisms underlying oddball visual search have been studied in the frontal and parietal cortices and in the superior colliculus. The initial visual responses to the target and the distractor were indiscriminable in most neurons in these areas but then responses became distinguishable about 120 ms after the stimulus. However, it was also reported that V1 cells distinguished oddball stimulus from homogeneous background stimuli around the peak of initial visual responses (about 60 ms after stimulus onset; Knierim and VanEssen,1992). We expected that monkeys might be able to use such initial discrimination signal under appropriate conditions. We introduced a temporal gap between the fixation spot offset and the stimulus onset. Then, in the color and shape visual search with a gap of 200 ms, the monkey made express saccades (reaction time between 65 and 90 ms) to the target, respectively, in 50.2% and 47.2% of the trials. These results suggest that the animal can utilize the difference in the initial visual response for visual search under the gap condition.

CREST,JST

## P1-e34 A Physiological role of gap junctions in firing properties of retinal AII amacrine cells

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Retinal AII amacrine cells (AII cells) generate TTX-sensitive repetitive spikes that are dependent on glutamatergic synaptic input and are reciprocally connected via gap junctions. To examine physiological roles of the gap junctions, we performed whole-cell patch clamping of AII cells in the mouse retinal slice. In current-clamp mode, we observed large fluctuations of membrane potential in AII cells. Adding gap junction blockers to the external solution diminished the voltage fluctuations, suggesting that the fluctuations are originated from the activities of neighboring AII cells. In addition, under these conditions, AII cells fired spikes at the highest frequency even when a small amount of current injection (about 10 pA) was applied at a membrane potential of about -65 mV. This is possibly because the gap junction blockers made the input resistance higher by closing the shunt via gap junctions. Our data suggested that gap junctional connections of AII cells might have a crucial role to respond wide range of input, probably by controlling shunt current.

## P1-e35 Developmental change of activity-dependent regulation of mGluR1 $\alpha$ in the lateral geniculate nucleus

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The neural connections from the lateral geniculate nucleus (LGN) to the primary visual cortex (V1) shows significant plasticity responding to visual experience in early life. To determine whether similar plasticity operates in other connections in the visual system, we explored the plasticity of feedback projection from V1 to the LGN in rat. We examined effects of cortical inactivation on the density of type 1 metabotropic glutamate receptor $\alpha$ (mGluR1 $\alpha$) and vesicular glutamate transporter 1 (vGluT1) in the LGN which locate postsynaptically and presynaptically at synapses of the feedback projection, respectively. The immunohistochemical signal for mGluR1 $\alpha$, but not vGluT1 in the LGN significantly decreased following pharmacological inactivation of V1. This effect was observed in both young and adult rats. These results suggest that mGluR1 $\alpha$ in the LGN is maintained by cortical activity at any age.

KAKENHI(17023033)

## P1-e36 Neural mechanism of biological motion processing: An MEG study

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Many neuroimaging studies have identified the brain region that was activated by biological motion (BM) perception, however, its neural dynamics still remains unclear. Unlike previous studies, we adopted "double stimulus presentation" method to extract magnetoencephalographic (MEG) response relating to processing of BM perception. We presented scrambled point-light walker (PLW) as a first stimulus (S1) and then presented upright PLW or inverted PLW as a second stimulus (S2) sequentially. The MEG response was observed for S1 at around 280 msec, and the peak latency of S2 was at around 380 msec from each stimulus onset. Additionally, in S2, the peak amplitude of PLW stimulus was significantly larger than that of scrambled PLW and the dipole was estimated more anterior to that in S1. These findings suggest that MEG response to human figure and action was enhanced in occipitotemporal region and PLW stimulus would be processed after general motion processing.

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## P1-e37 BDNF protein level is increased by inhibition of PKA, but not ERK, CaMK, PI3K, p38MAPK or Cdk5 in developing visual cortex

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BDNF, PKA and ERK are necessary for ocular dominance plasticity in the visual cortex. Although PKA and ERK might regulate the plasticity by modulating BDNF protein level, little is known about regulation of BDNF protein level in developing visual cortex. Therefore we measured BDNF levels in the visual cortex of young rats treated with various kinase inhibitors. Unexpectedly, PKA inhibitor increased both BDNF protein and its mRNA levels, although PKA is elucidated to upregulate BDNF expression through CREB and ERK phosphorylation. Inhibitors of ERK, CaMKs, p38MAPK, PI3K or Cdk5 had no effect. PKA inhibitor decreased phosphorylation of ERK but not CREB. Therefore PKA might negatively regulates BDNF expression without affecting CREB in vivo.

KAKENHI(15700303, 17023033)

## P1-e38 Laminar specificity of callosal connections in the rat striate cortex

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In lateral part of rat striate cortex, which represents central visual space, reciprocal callosal linkages are organized in a topographic manner. Callosal projecting cells of this region distribute in the supra- and infragranular layers. Although previous investigations revealed overall distribution of callosal cells and their axon terminations, laminar specificity of the callosal projections has not been characterized. Therefore, we made small injections of antero- or retrograde tracers into the supra- or infragranular layers in the lateral striate cortex of one hemisphere. In supragranular injections, retrogradely labeled cells distributed mainly in layer II/III and IV of contralateral striate cortex. Antrogradely labeled axons densely branched in layer II/III. In contrast, in infragranular injections, labeled cells distributed mainly in layer IV, V and VI. Afferent axons terminated mainly in the infragranular layers. These results suggest the laminar specific segregation of callosal connectivity in the lateral striate cortex.

## P1-e39 Inconsistency and uncertainty in the locations of human visual areas in Talairach space

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By combining brain imaging and information theory, we measured the amount of alignment inconsistency for each visual area in Talairach space and the amount of uncertainty about which area resides there. The inconsistency and uncertainty were quantified as the occurrence probabilities of 13 visual areas and the entropy of the probabilities, respectively. The overall average probability and entropy were found to be ~0.2 and ~1.0 bits, respectively, indicating substantial inconsistency and uncertainty. There was some inhomogeneity between areas. The probability and entropy maps generated here can be readily beneficial to any application in which it is desirable to predict the areas that are most probably present at an anatomical point and to know the uncertainty associated with this prediction.

the 21st Century COE Program (D-2 to Kyoto University) , MEXT, Japan

## P1-e40 On the direct activation of retinal ganglion cells by extracellular electrical stimulation - computational and experimental analyses -

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Recently, clinical trials of the retinal prostheses have succeeded in restoring partial visual perception in the blind patients suffering from degenerative retinal diseases. In those trials, retinal ganglion cells are directly/indirectly activated by extracellular electrical stimulation delivered from the micro-electrodes ocularly implanted. In the present study, we elucidated effects of the direct current stimulation on the spike firing of retinal ganglion cells, in a biophysical neuron model and dissociated neurons in vitro. The computer simulation suggested that not only the charge (charge density) but also the waveform of current stimuli are important factors for effectively eliciting the spike firing. The results of computer simulation were supported experimentally by the perforated-patch whole-cell recording from the isolated rat retinal ganglion cells. Furthermore, effects of the spatial location of stimulation electrode on the spike firing were examined.

KAKENHI17700398

## P1-e41 Chronic recording of neural activity of LGN in behaving rat.

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The lateral geniculate nucleus (LGN) of the thalamus conveys visual information to the primary visual cortex. Because activity of LGN neurons is elicited by retinal inputs, amount of LGN activity are expected to represent strength of visual inputs. Also, visual deprivation is assumed to decrease the LGN activity and leads to experience-driven cortical plasticity in young animals. However, it is not clear whether neural activity of LGN faithfully represents the input strength in awake condition. In order to test this hypothesis in behaving animals, we implanted a wire electrode to the LGN of rat chronically and recorded neural activity in behaving condition. Strobe flash stimuli effectively elicited visual responses of LGN neurons. Unexpectedly, however, we found no quantitative difference in mean firing rate of ongoing activity between the light and dark environment. In addition, abrupt increase of activity of short duration was accompanied by exploratory whisking of rats. These results suggest that neural activity of LGN does not simply represent the quantity of visual inputs in behaving rats.

KAKENHI(17023033)

## P1-e42 Effect of noise on pattern discrimination

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It has been shown that noise can improve detection of sensory stimuli in some non-linear systems including humans. In the previous study we showed that visual contrast detection sensitivity can be improved by adding a certain amount of random visual noise. Here we investigated whether visual pattern discrimination can also be improved by adding such visual noise to simple patterns of light. Discrimination of vertical and horizontal lines of light was measured by a two-alternative forced-choice (2AFC) procedure in six participants. Correct response rate was 58.3% without overlapping noise and increased to 86.7% with noise intensity at -10dB and 0 dB. Combined with our previous data, these findings strongly suggest that noise can improve pattern discrimination in human visual perception.

## P1-e43 Spontaneous membrane voltage changes in retinal neurons dissociated by a new method

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Recent studies showed that the vibro-dissociation method, which utilizes mechanical vibration without enzyme, is useful to isolate neurons from various CNS regions, yet never to vertebrate retinas. In this study, we have developed a method for dissociating mammalian retinal neurons by purely mechanical means, combining tissue incubation in low-$Ca^{2+}$ solution and the vibro-dissociation technique. Based on phase-contrast microscopy, rat retinal neurons dissociated by this method were in morphologically good condition, and could be identified as ganglion cells. In perforated-patch whole-cell recordings, the isolated cells showed spikes in response to depolarizing current injections, and showed the activation of voltage-gated membrane conductances in response to depolarization steps. Furthermore, under current-clamp, the ganglion cells showed spontaneous IPSP-like voltage fluctuation, suggesting a possibility that we can analyze not only the nonlinear membrane properties of single neuron, but also properties of signal transmission in single synaptic boutons.

KAKENHI17700398

## P1-e44 Expression pattern of voltage-gated sodium channel subtypes in the rat retina

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Retinal ganglion cells (RGCs) and subsets of retinal amacrine cells can generate TTX-sensitive action potentials evoked by light stimulus. In RGCs, various types of voltage-gated sodium channel a subunits ($Na_v$-$\alpha$) mRNAs ($Na_v1.1$, $Na_v1.2$, $Na_v1.3$, and $Na_v1.6$) are expressed (1). However, subtypes of $Na_v$-$\alpha$ expressed in the amacrine cells have not been identified. To examine the specific $Na_v$-$\alpha$ subtypes expressed in the amacrine cells, we applied *in situ* hybridization (ISH) on the rat retina with the RNA probes that recognize TTX-sensitive $Na_v$-$\alpha$ isoforms. RGCs expressed $Na_v1.1$, $Na_v1.2$, $Na_v1.3$, and $Na_v1.6$ as reported by Fjell et al. (1). However, $Na_v1.1$ was expressed not only in RGCs, but also in the inner nuclear layer (INL). $Na_v1.1$-positive cells in INL were located on the INL/IPL (inner plexiform layer) border. AII amacrine cells are located close to INL/IPL border, and these cells can be labeled with antibodies to parvalbumin. We applied double staining using ISH technique and immunohistochemistry, and revealed that most of AII amacrine cells expressed $Na_v1.1$.

(1) Fjell et al., Mol Brain Res 50:197-204, 1997

KAKENHI15500289

## P1-e45 Dynamic role of inhibitory circuits in ocular dominance plasticity: A network model of the developing visual cortex

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Y.Y-S. & T.K.H. have shown that the experience-dependent plasticity in V1 following monocular deprivation dynamically modulates inhibitory inputs that was probably mediated by inversely altering thalamocortical input onto both pyramidal (PY) and inter (IN) neurons. Here, we tested this hypothesis by constructing a computational model of the local visual cortical circuit. We examined the range of synaptic conductances at the afferent synapses from contra- and ipsi- lateral eyes that allows our model to replicate the experimentally observed response. We found that the model can replicate the observed spike bias values only if the synapses onto the IN are plastic. Interestingly, they are first biased toward input from the deprived eye, and then biased toward input from the non-deprived eye.T.K.H & T.F thank financial support from the MEXT.

17022036, 17021047

## P1-e46 Modifications in perineuronal nets of rat vestibular nuclear neurons during postnatal development and in injury

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Perineuronal net (PN), the extracellular matrix around neurons, has been shown to be involved in neural plasticity. To determine if PN in the vestibular nuclei (VN) plays any role on plastic change of central vestibular network, we studied PN in two states of functional modifications in the vestibular system which are postnatal development of VN and compensation of VN neuronal activity after unilateral labyrinthectomy (UL). Staining with lectin WFA was used to identify PN around VN neurons. In postnatal VN, fuzzy PN was detectable by P3. They became consolidated from P9 onwards. The number of VN neurons with PN also increased with age, reaching adult level by P21. With UL, the PN on the lesioned side of adult rats became fuzzy at 3-5 days post-lesion but resumed normal by 11-14 days after UL. PN on the labyrinth-intact side, however, showed the compact network. Our findings suggest that structural changes of PN around VN neurons contribute to plasticity of central vestibular system.

## P1-e47 Responsiveness of thalamic neurons to rotational and translational motions in the horizontal plane

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To identify the thalamic region(s) that relays vestibular signals towards the cerebral cortex, immunostaining was performed on rats that were subjected to rotational or translational stimulation along the horizontal plane. Neuronal activation was defined by Fos expression. Both canal- and otolith-related neurons were found in the subparafascicular nucleus(SPF). Fos-immunoreactive(ir) neurons activated by horizontal canal or otolithic inputs were intermingled throughout the SPF, with a higher number in its middle portion. Stationary and labyrinthectomized controls showed only sporadically scattered Fos-ir neurons in the SPF. Given that vestibular inputs from both ears converge at the vestibular nucleus, experiments were designed to determine whether the vestibular thalamus on one side receive input from both inner ears. In rats with unilateral labyrinthectomy for 2 weeks, symmetric Fos pattern was found on both sides of the SPF after stimulations. Our findings suggest that horizontal head movement signals arising from one ear are processed in both sides of the SPF.

## P1-e48 Developmental changes in excitatory and inhibitory transmission at central vestibular synapses of rats

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Signal transmission in the adult vestibular nucleus (VN) is regulated by excitatory and inhibitory synapses. Little is known about synaptic activities in developing VN. Patch clamp recordings in VN neurons of postnatal rats revealed that both the number and amplitude of GABAergic miniature IPSC increased with age while the decay time decreased. Corresponding parameters of glutamatergic miniature EPSC (mEPSC) did not change with age. These mEPSCs were equally contributed by AMPA and NMDA components before P9 but were dominated by AMPA component at P21. For evoked EPSC (eEPSC), we observed a developmental decrease in NMDA component and a reciprocal increase in the quantal contribution of AMPA component. The reduction in NMDA-only synapses coincided with the increase in AMPA/NMDA ratio. We further found that NMDA receptor-mediated eEPSC was contributed predominantly by NR2B subunit in neonates but by NR2A subunit after P7. Our findings document changes in synaptic events during the maturation of VN neurons. [Supported by HKRGC]

## P1-f01 Effect of implicit response in Auditory MEG measurement

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We presented a continuous base tone (CBT is A4=440[Hz] pure tone) to subjects during 1[s] long for each experimental session. It is well known that unimodal responses exist in the MEG waves at only the beginning after the onset of CBT, even if the CBT exists. Despite of the existence of CBT, MEG responses look to have been gone to the background noise after the onset response. Although such silent activity period might be showing perceptual acclimatization, corresponding representation or activity should exist in the brain as long as the existence of introspective perception. In order to investigate the feature of such silent activity period, we added an extra tone (ET) of another frequency to CBT, 500[ms] after the CBT presentation. In the present study, the cosine component of fixed A1 dipole for ET was decreasing and the latency was increasing, as the frequency approaches to CBTs. It has been suggested that some kind of implicit activity exists in the silent activity period in MEG responses.

## P1-f02 Neural substrates involved in auditory feedback and feed-forward controls of pitch modulation

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Subjects (n=17, right handed) were required to vocalize /a/ for 5 s in an fMRI experiment. In transformed auditory feedback condition (TAF), the feedback voice pitch was shifted and the subjects were required to hold the pitch of the feedback voice constant by changing the original pitch. In the pitch modulation condition under masking condition (Mm), they were required to modulate their pitch indicated by visual cues without vocal feedback. In non-TAF condition (with vocal feedback) and vocalization condition under masking (M, without vocal feedback), they merely vocalized /a/ continuously. The comparison between the TAF and the non-TAF conditions revealed significant activations in the superior temporal gyrus, the insula and the inferior pariental lobule, while that between the Mm and M conditions revealed a significant activation in the basal ganglia. Feedback and feed-forward control of pitch modulation are thus suggested to be supported by different neural networks.

JST to S. Koyama

## P1-f03 Neural Responses to Gap Embedded in Continuous Sound: Simultaneous Recordings of MEG and EEG.

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Gaps in continuous sounds play important roles for speech perception: perception of voiceless consonants, (/k/,/p/,/t/) and, segments between spoken sentences. Our previous ERPs studies showed that ERPs for onsets of gaps differ for different gap durations. In order to investigate neural sources for the gap responses, we measured EEG (Cz, T5, T6, nose tip reference) and MEG(a 306-channel whole head MEG system, Elekta Neuromag Oy, Finland) simultaneously using gap stimuli (16, 64, 256 ms) embedded in a continuous tone from eight healthy adults. As results, both of ERPs and MEG showed deflection peaking 100 ms after the gap onsets (16, 64, 256ms) and the gap end (256 ms only) (N1 and N1m component). N1m showed a clear right hemisphere dominance. A positive peak followed the N1 in the EEG responses while no corresponding peak was observed in MEG responses. EEG and MEG are thus shown to reflect different aspects of neural activity associated with gap perception.

JST (Brain Science and Education)

## P1-f04 Neural mechanism of corticofugal modulation of frequency processing in bat auditory system

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Most species of bats making echolocation use Doppler-shifted frequency of ultrasonic echo pulse to measure the velocity of target. To perform the fine-frequency analysis, the feedback signals from cortex to subcortical and peripheral areas are needed. The feedback signals are known to modulate the tuning property of subcortical neurons. However, the neural mechanism for modulation of the tuning property is poorly understood. To address this issue, we present a neural model for detecting Doppler-shifted frequency of echo sound reflecting from a target. We show here that the direction of best frequency shifts of subcortical neurons is determined by a single mechanism consisting of two components, or facilitation and inhibition. We also propose a functional role of the best frequency shift in extracting the information of target velocity under background signal reflecting from trees.

## P1-f05 Effect of a change in amplitude at sound onset on activities of auditory cortical neurons in the Mongolian gerbils

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The onset of sound is important for auditory nerve responses. However, in most auditory experiments rising parts of sound stimuli have not been paid attention to. In this study we focused on neural responses to constant frequency (CF) sounds in AC(Auditory Cortex) of Mongolian gerbils (Meriones unguiculatus) when the rising speed at the onset was varied. Because of the logarithmic factor of auditory sensation, we used the stimuli that had several constant logarithmic rising speeds (100 dB/s to 3200 dB/s) and several peak sound pressure levels. The frequencies of sounds corresponding to the responses were different by the rising speed of sound stimuli. In 100 dB/s rising speed, more than 6000 Hz Stimuli hadn't generated auditory responses, but in 3200 dB/s rising speed, had generated. And the latency was also different by the frequencies of sound stimuli at slow rising speed. These results cannot assert that rising speed decide the generations of the auditory responses, though suggest that the threshold have not been controlled by sound pressure level or the frequency.

## P1-f06 Neural responses of inferior colliculus to echolocation sounds in Japanese house bat (*Pipistrellus abramus*).

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In our laboratory, we found compensation for intensity of the echo by recording the returning echoes from flying bats (Pipistrellus abramus) with telemetry microphones. This result suggested the existence of neural mechanisms that proved the echo-intensity compensation. A number of neurophysiological studies for intensities of pulse and echo were conducted, however, the echo intensity compensation had hardly investigated. In this study, we recorded the single unit responses of inferior colliculus (IC) in the awake bats to returning echoes presented under free field stimulation, and investigated the relationship between the results and echo-intensity compensation behavior.

## P1-f07 *Arc* mRNA expression pattern in female zebra finch auditory pathway

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Male song discrimination mechanism in the female brain is an open question in the songbird study. To answer this question, we utilized the catFISH (cellular compartment analysis of temporal activity by fluorescent *in situ* hybridization) for *Arc* mRNA. *Arc* is one of effector immediate early genes, and expression of *Arc* is synaptic-activity dependent. *Arc* mRNA is not translated at the cell bodies but transferred to the peripheral of the neurons and translated locally. Because this process is time-dependent, two populations of neurons that are activated at different timings can be discriminated with *Arc* mRNA expression by FISH. Two conspecific songs were applied sequentially with 50 min interval to the female zebra finch. Then, the expression of *Arc* was investigated. We found that caudal medial mesopallium and hippocampal formation are the candidates for song discrimination regions. In these regions, some neurons activated by two songs are overlapped. These findings provide the neural coding strategy of auditory pathway for song discrimination.

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## P1-f08 P2X1 in the inferior colliculus

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P2X receptors are ionotropic receptors activated by extracellular ATP. P2X receptors in the dorsal root ganglion are thought to be related to the nociception. P2X receptors are also reported to be distributed throughout the central nervous system. We explored P2X1 expression in the inferior colliculus (IC) which is known as brainstem auditory center by immunohistochemistry. P2X1 immunoreactive cells were found in the central and the external nucleus of the IC as large or medial sized multipolar cells. They seemed to disappear in the late developmental period: The IC of 8 weeks old rats had many positive cells, while the IC of 12 weeks old rats had no positive cells. We also found sparse but distinct P2X1 immunoreactive fibers in the central nucleus of the IC. They seemed to appear with late maturation between 8 week and 12 week. Double immunofluorecence study with tracer technique showed that these fibers were corticocollicular. These findings suggest that ATP from glial cells in the IC modulates auditory processing.

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## P1-f09 Effects of musical experience on the automatic auditory feature processing

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Music consists of different features of sound information. Although they are believed to be processed differently, its detailed brain mechanisms and effects of learning are still unclear. We studied the effects of musical training on preattentive discriminating processes of the sound features using auditory mismatch fields (MMFs), which are evoked by deviant sounds embedded in a repetitve standard sounds. During four different oddball tasks to detect the frequency, rhythm, chord and timbre information processing, MMFs were measured in eight musicians and nine nonmusicians using magnetoencephalography. Irrespective of the musical experience, MMF waveforms were different among the sound features. However, musicians generally showed the left hemispheric dominance, whereas nonmusicians showed the right dominance, especially in frequency and chord. These results suggest that the auditory feature processing is modulated by musical experience.

## P1-f10 A rat model for investigating speech sound recognition in humans: discrimination learning of synthetic speech sounds dependent on the presence of the auditory cortex

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The first and second formants of vowels are essential for vowel recognition. We have reported that discrimination learning between vowel-like sounds with multiple formants requires the presence of the auditory association cortex in rats. In the present study, we investigated discrimination learning of voiced stop consonants /b, d, g/, which are characterized by transition of multiple formants. Water-deprived rats were trained to discriminate between two synthesized stop consonants. Licking a spout during presentation of a consonant was rewarded with water while the other was not. Bilateral lesions of the auditory cortex impaired discrimination learning between stop consonants with multiple formant transitions, while discrimination between single formant transitions was not affected. These results suggest that discrimination learning between synthetic speech sounds in rats may serve as an animal model for investigating speech sound recognition in humans.

## P1-f11 Functional connections between the core and belt fields of the guinea pig auditory cortex observed by optical imaging

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Functional connections between the core and belt fields of the guinea pig auditory cortex were investigated using voltage-sensitive-dye optical imaging and local cortical inhibition and activation by muscimol and bicuculline (GABAa agonist and antagonist, respectively). Inhibition of AI in the core fields by muscimol resulted in a decrease of responses to pure tones in the ventroanterior (VA), ventromedial (VM), ventroposterior (VP) and posterior (P) belt fields. Inhibition of the dorsocaudal field (DC) in the core fields resulted in a decrease of the response in VM, P and VP. Activation of AI by bicuculline induced an increase of the response in DC, VA and VM and a decrease of the response in P and VP. Activation of DC induced an increase of the response in VM, VP and P but no change in the response in AI and VA. These results suggest that AI and DC respectively send differential excitatory and inhibitory outputs to each belt fields of the auditory cortex.

## P1-f12 Ongoing and spontaneous cortical representations of auditory attributes

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The ongoing and spontaneous activities have been considered to be essential and important in understanding the brain function. These activities have been investigated in the visual and somatosensory cortices and shown to have strong spatial coherence which accounts for the cortical attributes. In this study, we examined whether the ongoing and spontaneous activities in the auditory cortex represents the auditory attributes by using voltage sensitive dye imaging. We applied the independent component analysis to remove physiological noises from observed data, the scaled signal-plus-noise model to extract the ongoing activity from the neural activity, and the coherence map to reveal the coherence in the cortical activity. Here we report that the ongoing and spontaneous activities in guinea pig primary auditory cortex represent the auditory attribute corresponding to the tonotopy.

## P1-f13 A study of the influences on tastes by oral noxious stimuli

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Capsaicin, a pungent substance in chili peppers, activates transient receptor potential (TRP) V1, and menthol that evokes cool sensation, an ingredient of mint, activates TRPM8. On the other hand, allyl isothiocyanate and cinnamaldehyde, ingredients of mustard and cinnamon, respectively, activate TRPA1. These TRPs are recognized as the channels that receive harmful stimuli. While we daily eat these chemical substances, the sensations evoked by these substances are not tastes, but noxious stimuli. It remains unknown how these substances influence tastes. In this study, we investigated the influences on taste by oral noxious stimuli. Capsaicin, menthol, allyl isothiocyanate, and cinnamaldehyde were chosen as the representatives of noxios substances, and the influences of these substances on taste sensations were electrophysiologically studied. Additionally, we examined the distributions of TRPs in the gustatory system using *in situ* hybridization and immunohistochemistry. In this conference, we show the results and discuss the influences on taste by oral noxious stimuli.

KAKENHI (18689041)

## P1-f14 Amphibian V1R gene family is expressed in the *Xenopus* main olfactory system

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Over 180 members of the V1R pheromone receptor genes are expressed specifically in the rodent vomeronasal organ (VNO). In contrast, only two V1R genes were identified in fishes that lack VNO. The VNO was appeared in amphibians phylogenetically. In order to determine at what point during evolution the V1R genes function in the VNO, we analyzed the *Xenopus* 21 V1R genes, and found that the expression was not detected in the VNO, but detected in the main olfactory epithelium (MOE), suggesting that V1R switched its main expression site from MOE to VNO after the divergence between amphibians and terrestrial vertebrates. In addition, we found that the V1R-expressing cells coexpressed Gi2 in the MOE. The V1R-Gi2 signal transduction pathway, which seems to be important in rodent VNO, is likely to exist in amphibian MOE. Our results suggest that amphibians represent an intermediate stage between fish and mammals during the evolution of V1R-mediated pheromone sensing.

Basic Research Program

## P1-f15 Vomeronasal axons play a key role in the glomerular formation of the accessory olfactory bulb.

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Rodent vomeronasal receptor neurons (VRN) project to multiple glomeruli in the accessory olfactory bulb (AOB). We examined how a zinc-finger gene Fez-like (Fezl), BDNF, and Neuropilin-2 (Npn-2) gene control the projection and segregation of VRNs in the AOB of mice. In Fezl-deficient mice, axonal projections of VRN to the AOB were completely lost and no glomerular arbors of the Mitral/ tufted cell (MT cell) were formed. Npn-2 affected in targeting of VRN axons to the AOB and branching of MT cell glomerular tufts. BDNF deficient mice had little changes on VRN projection, nor glomerular formation. These suggest that VRN projections have strong influences on the glomerular formation in the AOB.

## P1-f16 Transgenic mice expressing tamoxifen-inducible Cre recombinase in mitral/tufted cells of the olfactory bulb

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To understand how mitral/tufted (M/T) cells in the olfactory bulb receive and process peripheral olfactory information, the genetic visualization and manipulation of M/T cells at a single-cell level should be essential. Here, we generated mice harboring tamoxifen-inducible Cre recombinase-modified estrogen receptor fusion transgene under the control of the M/T cell-specific protocadherin 21 (pcdh21) gene promoter, designated pcdh21-CreER. By crossing pcdh21-CreER mice to several lines of Cre reporter mice, administration of tamoxifen was shown to induce reporter gene expression in dose-dependent manner, while the basal frequency of recombination varied among reporter lines. Recombination was restricted to a small subset of M/T cells under administration of low dose of tamoxifen, enabling us to visualize the Golgi stain-like morphology of single M/T cells. These results show that pcdh21-CreER mice can be used to the analysis of detailed morphology and synaptic connections of M/T cells, and genetic manipulation of a small subset of M/T cells in a predominantly wild-type background.

## P1-f17 Close co-ordination of binasal inputs in the rat anterior olfactory nucleus

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Individual sensory systems including olfactory system have two sensory organs to monitor environmental cues. What is the functional significance of having two, right and left, olfactory epithelia?

Anterior olfactory nucleus (AON) of the olfactory cortex has commissural fiber connection with contra-lateral AON, suggesting the interaction between right and left AON. Here, we recorded extracellular single-unit activity of AON neurons and examined the responses to the odor stimulation of ipsi- and contra-lateral olfactory epithelium.

We found that most neurons in the AON received converging excitatory inputs from both olfactory epithelia. In addition, individual AON neurons showed a close co-ordination of odorant selectivity between ipsi- and contra-lateral nasal inputs. Furthermore, when ipsi-lateral nostril was obstructed, the response to the contra-lateral odor stimulation increased a few minutes after the obstruction, indicating that unilateral nasal obstruction induces a rapid switch over of nasal inputs to the AON.

## P1-f18 Olfactory detection threshold of ordinary volatile chemicals in mice

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We aimed to establish an animal model of olfactory hypersensitivity to volatile organic compounds (VOC) and to apply the model for pathophysiological understanding of VOC-related human disorders such as sick house syndrome or multiple chemical sensitivities. The first step of our attempts was to develop a system for evaluating olfactory detection threshold of VOC gas in mice. We attached odor-presenting hardware composed of an odor-sniffing glass port, solenoid valves, diaphragm pumps and polyethylene gas bags to a standard operant chamber, and we developed software for controlling the equipments. Male mice (C3H/He) were trained for the task of pressing the lever (right or left one) corresponding to sniffed gas (VOC-mixed air or clean air), and then they performed multiple operant learning tasks to discriminate between toluene gas with various concentrations and clean air. This study is ongoing, and we found that the detection threshold of toluene gas in mice is under 5 ppb, which is at least a hundred times smaller than that in humans reported previously.

## P1-f19 Effects of centrifugal inputs on distribution of c-Fos immunoreactive neurons in the olfactory bulb

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To examine a possible role of centrifugal inputs in the olfactory bulb activity, distribution of bulbar neurons expressing an immediate early gene product, c-Fos, was immunohistochemically studied in adult rats in which lesion had been made in the unilateral olfactory peduncle including the anterior limb of the anterior commissure and/or the anterior olfactory nucleus. Neurons in the juxtaglomerular region, the superficial external plexiform layer, the mitral cell layer and the granule cell layer (GCL) showed varying degrees of immunoreactivity in animals sacrificed in the ambient air. In the GCL, the major target region of centrifugal fibers, diffuse bands of immunoreactive granule cells and patches of nonreactive granule cells that were interposed in between were noted. Preliminary experiments revealed that lesion of the olfactory peduncle made distribution of immunoreactive granule cells more uniform, suggesting that centrifugal inputs confine the distribution of activated granule cells.

## P1-f20 Activity-dependent expression of semapholin 7A in the mouse olfactory bulb

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In the mouse, olfactory sensory neurons (OSNs) expressing the same odorant receptor (OR) converge their axons to a specific set of glomeruli in the olfactory bulb. Within glomeruli, OSN axons synapse onto dendrites of projection neurons, mitral/tufted (M/T) cells. However, it is largely unknown how the neuronal identities of M/T cells are established. It was recently shown that a set of adhesive/repulsive molecules, whose expression levels are regulated by ORs and neuronal activity, define the neuronal identity code and contribute to the axon sorting of OSNs. We found that semapholin 7A (sema7A) is expressed in an activity-dependent manner not only in OSNs but also in M/T cells. Since it has been shown that Sema7A is involved in the extension of the lateral olfactory tract, we postulate that a similar mechanism to that for the axon sorting of OSNs may contribute to the neuronal connection of M/T cells in an activity-dependent manner.

## P1-f21 The largest subtype of murine odorant receptors responsive to a pair of enantiomers is more sensitive to a more potent partner of the enantiomers in human perception

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In the previous study, we classified ca. 2,740 murine olfactory receptor neurons (mORNs) into >41 subtypes in combination of the best odorants and the 2nd best odorants among a pair of carvone enantiomers and 23 cognate or non-cognate odorants. The result showed that receptor codes were characteristic of carvone enantiomers especially near the threshold concentration, where the largest subtype of mORNs responsive to carvone(s) was more sensitive to R(-)-carvone that is more potent than S(+)-carvone in human perception. Here, we examined relative sensitivities of mORNs for another pair of enantiomers showing greater human threshold difference. The result showed more remarkable superiority of the mORNs sensitive to the human-sensitive enantiomer in population. These results support our hypothesis of the receptor-sensitivity-dependent odor information coding that can explain odor constancy through a range of stimulus intensity.

## P1-f22 Possible roles of *sema6A* in the mitral cell layer formation

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The transgenic strategy is a powerful approach to uncover the molecular mechanisms of axon guidance in the mammalian olfactory system. In the previous study, using the H region, an enhancer of the olfactory receptor (OR) gene, we devised a mosaic analysis system that induces the transgenic expression of a particular gene of interest in a subset of OSNs. During a course of our experiments, we obtained a transgenic mutant mouse that demonstrated defects in the axonal projection of OSNs and mitral cell layer formation. This mutant appears to be generated by an accidental insertion of the transgene into a chromosomal locus coding for an axon guidance molecule. Closing analysis reveled that *sema6A* gene for a transmenbrane semaphorin protein, located near the transgene integration site. In situ hybridization analysis revealed that Sema6A was specifically upregulated in the olfactory epithelium of the mutant mouse. These results indicate that *sema6A* plays important roles in mitral cell layer formation and axonal projection of OSNs.

## P1-f23 Combinatorial code of cell- recognition molecules for the axonal segregation of olfactory sensory neurons.

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In the mouse olfactory system, each olfactory sensory neuron(OSN) expresses only one functional odorant receptor(OR) gene out of ~1000 members. OSNs expressing the same OR gene converge their axons on a specific pair of gromeruli in the olfactory bulb, although they are dispersed in the olfactory epithelium. In the previous study, we identified several pairs of cell-recognition molecules, e.g., Kirrel2/Kirrrel3 and EphA5/ephrin-A5, whose transcription levels are complementary to each other and regulated by OR-dependent neuronal activity. These molecules appear to be involved in segregating axons by their adhesive/repulsive interactions. We further searched for additional cell-recognition molecules whose expression levels are uniquely correlated with the expressed OR species.

## P1-f24 Decreased interneurons in the *Pax6* mutant mouse olfactory bulb

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Pax6 is a transcriptional factor that plays essential roles in brain development. Previously, we reported decreased number of interneurons in the limbic system of *Pax6* heterozygous mutant mice (*Sey/+*, Haba et al., Neurosci. Res. Suppl. 2006). In the present study, we further investigated defects of GABAergic interneurons in the *Sey/+* olfactory bulb. Immunohistochemical studies revealed that the number of parvalbumin (PV)- and somatostatin (SM)-positive GABAergic interneurons were remarkably decreased in the external plexiform layer of the *Sey/+* olfactory bulb compared to those in the wild type. Pax6 expression was detected in all PV- and SM-positive interneurons, suggesting that loss of these types of neurons are caused by cell-autonomous defects in the *Sey/+* interneurons. Furthermore, BrdU pulse-chaise analysis revealed that PV- and SM-positive interneurons were only generated during the embryonic period. These results suggest that Pax6 is essential for generation and/or maintenance of specific subtypes of GABAegic interneurons in the developing olfactory bulb.

CREST, JST

## P1-f25 Major urinary proteins increase the number of nestin-positive cells in mouse vomeronasal cell cultures

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The vomeronasal organ (VNO) retains a stem cell population, which continually generates new neurons throughout life. Stowers et al. reported that the vomeronasal neuron (VSN) in the TRPC2$^{-/-}$ mouse not only fails to signal, but also fails to regenerate VSNs. These findings suggest that active signaling plays an important role in the regeneration of VNO stem cells. To address this issue, we examine the effect of natural stimuli on cell proliferation and/or survival using the mouse VNO cell culture, which was prepared from newborn mice. Major urinary proteins (MUP) including their bound-ligands were purified from urine of female Balb/c mice as natural stimulants, which are sufficiently polymorphic proteins to code for individuality. Immunocytochemical staining showed that when MUPs plus their ligands were added to the mouse VNO cell culture and maintained for 2 weeks, the number of nestin-positive cells significantly increased. This supports our hypothesis that MUPs enhance the regeneration of VSNs.

## P1-f26 Oxytocin facilitates the induction of long-term potentiation in the accessory olfactory bulb

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We have shown that the mitral to granule cell synapse in the accessory olfactory bulb (AOB) is critical site for olfactory learning in mice, in which a female forms a memory to the pheromonal signal of the male that mates with her. The formation of this memory depends on mating-induced release of noradrenaline (NA) in the AOB. In support of this, we have also shown in slice preparations that NA gates long-term potentiation (LTP) at the mitral to granule cell synapse. In addition to NA, the neuropeptide oxytocin (OT) has been implicated in a number of social behaviours, including maternal care, affiliation, and social attachment. Recently, mapping OT receptor gene expression using an OT receptor-lacZ reporter mouse revealed the expression of OT receptors in the AOB. This raises the possibility that OT is involved in the olfactory learning in mice. To test this possibility, we examined whether or not OT modulates LTP at the mitral to granule cell synapse in slice preparations. The results show that OT facilitates the induction of LTP at the mitral to granule cell synapse in the AOB.

## P1-f27 The reception of ESP peptides in rodent vomeronasal neurons

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We recently identified a sex-specific peptide, named ESP1 (exocrine gland-secreting peptide 1), that was secreted in tears of male mice. The ESP1 is transferred to the female vomeronasal organ (VNO) wherein it elicits an electrical response. ESP1 also induces c-Fos expression in a subset of vomeronasal sensory neurons (VSNs) expressing a receptor, called V2Rp5. The ESP1 gene is a member of a multigene family that consists of 38 genes. Fifteen out of 38 ESPs were expressed in exocrine glands and had an activity in inducing electrical responses in VSNs. Moreover, 10 orthologous ESP genes were found in rat genome, one of which was expressed only in a female exocrine gland. These results indicate that ESP peptides convey various pheromonal information including sex and species via the VNO in rodents. Elucidation of functions of ESP peptides and their receptive mechanisms in the vomeronasal system will provide a molecular basis for pheromonal communication in rodents.

PROBRAIN

## P1-f28 Olfactory computation using spatiotemporal pattern of network activity for odor representation

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In olfactory system, it is important to clarify how dynamic responses of network solve the computational problems. We propose a computational model in which the concentration-invariant odor information is represented by the spatiotemporal activity of network. As a model for the primal center, we designed a network consisting of projection neurons (PNs) and interneurons, in that a spatiotemporal order of the PN activities depends on an order of input magnitude for each PN. The network activity can represent the invariant odor information because in the biological network the order of the magnitude of odor input for each PN would be kept regardless of odor concentration. The represented odor information is easily evaluated and stored by another network model for the higher olfactory center. In our model, transforming the input spatial pattern into the invariant spatiotemporal order is essential for representing the odor information and output patterns of the proposed networks are consistent with those of the biological ones.

KAKENHI17075003

## P1-f29 Histamine modulates olfactory response in the olfactory epithelium

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Parosmia, a smelling disorder, is a symptom of allergic rhinitis. In allergic patients, histamine and leukotriene are inflammatory mediators. Here, we hypothesized that histamine modify olfactory information. First, we confirmed the expression of histamine receptor in olfactory epithelium using immunohistochemical technique. $H_1$ receptor was mainly expressed in dendrite and soma of olfactory receptor cells (ORCs). On the other hand, weak signal of $H_2$ receptor was observed at dendrite of ORCs. Next, we measured the histamine response in isolated ORCs using a calcium imaging technique. ORCs were isolated enzymatically and were loaded with fura-2. A bath application of 100 μM histamine increased the intracellular calcium concentration in 30% of ORCs. Olfaction is determined by the combination of ORC responses. Therefore, we suggest that histamine-induced changes of the combination of ORC responses might be responsible for parosmia in patients with allergic rhinitis.

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## P1-f30 Plasticity for functional restoration of forelimb movements following neonatal unilateral decortication.

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It is generally believed that juvenile brain is resistant to injury. As an experimental model to study the plasticity of the juvenile brain, we studied the functional compensation of motor functions in rats that received unilateral decortication at neonatal stage. Five Wistar rats received unilateral decortication on the 5th day after birth. Behavioral observation was started using "reach and grip task" in 10th to 14th week after birth. The affected forelimb showed successful movements, although the ability was poorer than the intact forelimb. After 6 weeks of training, the sensorimotor cortex of the remaining hemisphere was lesioned. Then, the performance of both forelimb movements was severely impaired. These results suggested that after neonatal unilateral decortication, the forelimb movements on the affected side were restored by the remaining hemisphere.

supported by CREST, JST, JAPAN

## P1-f31 Role of visual information on prism adaptation of throwing movements: The effect of visual information of the thrown ball

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Humans can adapt to a visually-distorted environment through making dozens of movement in the distorted environment (i.e., prism adaptation). The authors ran a behavioral experiment on the prism adaptation of a throwing task, asking what portion of the visual information is essential for the adaptation of visuomotor transformation. Specifically, we removed some part of visual information of the thrown ball by an electronic shutter controlled synchronized with the ball movement, and examined the effect of the timing of removal on the magnitude of prism adaptation. Significant aftereffect was observed when no visual information was provided after the ball hit the target only if some part of the ball trajectory was exposed to the subjects. This suggests the possibilities that our brain utilizes the difference between the predicted and actual trajectories of the flying ball, or the difference between the target and the impact position estimated from the flying trajectory, for acquiring the new visuomotor relationship.

## P1-f32 Expert pianists exploit an efficient agonist-antagonist muscular synergy when striking the keys on the piano.

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The upper limb's agonist-antagonist muscular synergy for the production of joint torques in striking the keys on the piano was investigated in 7 pianists and 7 novice players. Kinematic and EMG recordings were made while these subjects performed striking the key by lifting the arm at some height. To initiate the downswing, flexion muscular torque at the elbow that had counteracted with gravity was decreased. Associated with this, activity of the elbow extensors (agonist) occurred much earlier for the novices than the experts. Instead, the experts decreased the postural activity of elbow flexors (antagonist) before anti-gravity torque was decreased. These data indicated that the novices produced joint torque to extend the elbow joint by actively contracting its agonists, whereas the experts made it by releasing postural contraction of the antagonists to take advantage of gravitational force. These differences were noted at all levels of sound loudness. The findings suggest that there is a muscular synergy to strike the key with less muscular effort only for the experts.

## P1-f33 Remodeling of sensory nerve projection onto Mauthner cell during development in zebrafish larva

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The Mauthner(M-) cells, paired large reticulospinal neurons in teleost hindbrain, are known to trigger fast escape from sudden noxious stimuli.In adult goldfish and zebrafish, sound stimulus effectively elicits escape.
However, early larva can escape from tactile stimulus, but not from sound stimulus.In the present study, we examined the innervations of auditory(VIII) and trigeminal(V) nerves onto M-cell with in vivo whole cell recording from zebrafish larva.
After 5 days post fertilization(dpf), sound or electrical stimulation of VIII nerve elicited fast inward currents in the M-cell mediated by electrical and chemical synapses.Electrical stimulation of V nerve induced only small currents with longer latency.In contrast, at 2dpf, head skin shock evoked postsynaptic inward currents whereas sound didn't.Therefore, M-cell receives stronger inputs from V nerve at 2dpf and from VIII nerve after 5dpf.
Thus, it is suggested that effective sensory input to excite M-cells switches from tactile to auditory input during development so that fish can perform fast escape from distant predators and noxious stimuli.

KAKENHI(17023029,18300134)

## P1-f34 Increase of intermuscular coherence independent of cortical activity during recovery from corticospinal tract lesion in monkeys

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For the purpose of clarifying the neural mechanism of functional recovery after spinal cord injury, we investigated intermusclar and cortico-muscular coupling between the LFP of the primary motor cottex (M1) and EMGs of various forelimb muscles during precision grip in monkeys with lesion of the corticospinal tract (CST). Before CST lesion, one monkey showed cortico-muscular coherence at 17 Hz with intrinsic hand muscles, but no intermusclar coherence was observed, while other 2nd monkey showed no cortico-muscular and intermuscular coherence. EMGs of antagonist muscle pair showed long term synchrony (first postoperarive week), then oscillation (<3rd week), then this oscillation became coupled at 30-40Hz (<1 month). Such intermuscular oscillatory coupling increased afterwards. However, cortico-muscular coupling disappeared after lesion; 30-40 Hz oscillatory activity was not found in M1. We suggest that the intermuscular coupling at 30-40 Hz that develops in parallel to functional recovery is always not mediated via M1 and direct corticomotoneuronal connection.

CREST, JST

## P1-f35 Gene expression of GAP-43 in the monkey motor cortex after lesion of the lateral corticospinal tract

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Precision grip recovers even after complete lesion of the lateral corticospinal tract (L-CST) in the monkey (Sasaki et al., 2004). To investigate molecular bases of the functional recovery, we focused on the gene expression of GAP-43, a marker for synaptic sprouting. While the hybridization signals for GAP-43 mRNA were weak in layers II and III of the motor cortex (M1) in the normal monkeys, moderate to intense signals were frequently observed throughout layers II to VI in the monkey that showed recovery after the L-CST lesion. The enhanced expression was observed not only in M1 but also in the premotor and supplementary motor area. These results suggest that GAP-43-mediated plastic change occurs in various motor related areas during recovery after L-CST lesion.

CREST of JST

## P1-f36 Quantitative analysis of corticospinal projection from digit area of motor cortex of rhesus monkey

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Intraspinal axonal trajectories of corticospinal tract (CST) neurons from digit area in the primary motor cortex were quantitatively analyzed. In two adult rhesus monkeys, biotinylated dextran amine was injected into digit area. Among the stained CST axons, those in the lateral funiculus contralateral to injection was 93±6 % and in the ipsilateral side was 4.2±4.0 % in the C2-Th2 segments. Two point eight ±2.6 % of axons passed in the ipsilateral ventral funiculus. The axons on the contralateral side mainly projected to the lamina VII of Rexed in all the segments between the C1 to Th2, and additionally to lamina X in the cervical enlargement (CE; C6-Th1). On the ipsilateral side, axons mainly projected to lamina VII at the C1-C4 and to lamina VIII at CE, respectively. CST axons crossing the midline were increased in CE. These results indicated the differential projection pattern of CST axons between midcervical segments and CE.

## P1-f37 Willed visuomotor imagery alleviates deafferentation pain: Possible implications of mirror visual feedback treatment

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Following lesions in somatosensory pathways, deafferentation pain often occurs. Patients report that the pain is of a complex nature. Anecdotal evidence has suggested that mirror visual feedback (MVF) can alleviate the pain, so we examined the possibility of using MVF to treat deafferentation pain and categorized pain descriptors into exteroception associated pain and proprioception associated pain. After MVF, the patients · use of proprioceptive pain descriptors decreased significantly. The pain amelioration and the decrease of proprioceptive pain descriptors were closely linked to the emergence of willed visuomotor imagery of the affected limb, suggesting that MVF seems to compensate for somatosensory insufficiency of the deafferentated limb. With better understanding of the nature of the pain, treatment strategies could incorporate methods that produce proper visuomotor imagery.

KAKENHI (18591702)

## P1-f38 Gene expression profile of inflammatory molecules in the nucleus tractus solitarii of the spontaneously hypertensive rat (SHR)

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Recently, we identified that junctional adhesion molecule-1 (JAM-1) was over expressed in endothelial cells in the nucleus of the solitary tract (NTS) of SHR compared to normotensive Wistar-Kyoto rat. In the present study, we have assessed the level of gene expression of other major adhesion molecules and cytokines in the NTS of SHR relative to WKY by performing real-time RT-PCR. Gene expression of inter-leukin-6 (IL-6) was significantly lower in the NTS of SHR compared to the WKY (SHR: 0.41±0.12, n=5; WKY: 1.03±0.12, n=5, $p<0.01$). In contrast, expression of adhesion molecules including: intercellular adhesion molecule-1, vascular cell adhesion molecule-1 and junctional adhesion molecule-3 were not different. Our data suggest that the NTS of the SHR exhibits a specific inflammatory state and the high expression level of JAM-1 mRNA and lowered IL-6 mRNA in the NTS of the SHR is a unique characteristic.

British Heart Foundation

## P1-f39 Influence of emotion regulation on autonomic activity during anticipatory anxiety

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Awareness of the internal state of one's body state relates to intensity of negative emotions. However, a technique includes direct, intuitive, experiential awareness of experience in the moment is known to treat negative emotions. In this study, we examined the effects of emotion regulation for aversive stimulus and its anticipation. Heart rate and skin conductance responses were used to measure autonomic activity. We hypothesized that those who learned adaptive emotion regulation technique can reduce anticipatory anxiety and better recovery after aversive stimulus.

## P1-f40 Neuronal expression of c-Fos protein in the brain after intraperitoneal injection of apelin-12 in Wistar rats

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The present study was aimed to survey central neurons that may be activated after intraperitoneal injection of apelin-12 (AP12, one of apelin peptide homologues), by examining the distribution of neurons expressing c-Fos. AP12 has been known to induce gastric acid secretion as well as other physiological functions such as regulation of ciculation and body fluid. In the test experiment, AP12 (800 μg/kg) was intraperitoneally injected in Wistar rats. One and a half hours after the injection, rats were perfused with saline and fixed. The brain was removed and sectioned (40 μm). Every fourth section was processed for detection of c-Fos by using ABC methods. Intraperitoneally injected AP12 induced neural expression of c-Fos in the paraventricular hypothalamic nucleus and other several nuclei in the brain. The present results suggest that AP12 stimulates central neurons that may play a role in the maintenance of body fluid homeostasis as well as other physiological functions.

## P1-f42 Involvement of GABA receptor mechanisms in the control of noradrenaline release in the median preoptic nucleus in response to volume expansion in rats

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In urethane-anesthetized rats. volume expansion (VE) caused by intravenous infusion of 4% Ficoll (1% body wt) significantly reduced the NA release in the median preoptic nucleus (MnPO) area that accompanied an elevation in mean arterial pressure. The VE-induced reduction in the NA release was significantly attenuated by perfusion with either bicuculline (10 μM), a $GABA_A$ receptor antagonist, or phaclofen (10 μM), a $GABA_B$ receptor antagonist, through a microdialysis probe. The amount of the antagonist effects was much greater in the bicuculline-treated group than in the phaclofen-treated group. These results show the contribution of the GABAergic system to the regulation of NA release in the MnPO area in response to VE.

## P1-f43 Capsiate topically activates peripheral sympathetic nervous system

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Capsiate (CST), a much less pungent capsaicin analog, was identified in fruits from CH-19 sweet, a cultivar of chili pepper. CST was found to be a TRPV1 channel receptor agonist and to induce enhancement of energy expenditure (EEE) as capsaicin (CAP). As CST is easily hydrolyzed and never detected in peripheral circulation after oral administration, the observed EEE by CST could solely be attributed to the neural reflexes originated in gastrointestinal TRPV1 stimulation. We studied contribution of either neural or endocrine output (epinephrine (Epi) release from adrenal gland) to EEE. Epi levels in rats increased 15 min after CAP administration but not by CST. CST, however, increased MHPG, an NE metabolite, suggesting that EEE by CST is induced by sympathetic nervous output but not by endocrine one, which is different from EEE by CAP where endocrine output contributes. CST ingestion increased NE turnover rate to 2-fold in brown adipose tissue while no effects on those in heart and pancreas, indicating that the projection loci of CST-originated reflexes are not ubiquitous but topical.

## P1-f44 Capsiate enhances sympathetic nerve activity of brown adipose tissue

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Capsiate (CST) is a capsaicin (CAP)-like ingredient of CH-19 sweet, a non-pungent cultivar of chili pepper. CST was found to activate TRPV1 receptor and enhance oxygen consumption. Unlike CAP, orally administrated CST is not absorbed due to its vulnerability to hydrolysis. In this study, to investigate the involvement of sympathetic nerve activity (SNA) for a rise of oxygen consumption, we directly monitored efferent SNA projecting to the brown adipose tissue (BAT) in anesthetized rats. Intra-gastric CST administration enhanced BAT SNA gradually and continually for more than 3 hrs in a dose dependent manner, while CAP at the maximum dose never induced an equivalent magnitude of the response. This increment of SNA both by CST and CAP was abolished by blocking sympathetic ganglionic transmission. The difference in the pattern of SNA response between CST and CAP may at least partly be due to an inability of CST to induce epinephrine secretion from the adrenal medulla.

## P1-f45 Chelonian spinal nerve-sympathetic ganglial complex: A synapsin I and synaptic vesicle protein immunohistochemical study.

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A tyrosine hydroxylase-immunoreactive (TH-ir) nerve cell mass was found in the dorsal nerve belonging to the spinal nerve ganglion of the red-eared slider, *Trachemys scripta elegans*. These TH-ir nerve cells were multipolar neurons and did not contain any structures that could be chromaffin granules. We speculated that the TH-ir nerve cell mass was composed of sympathetic postganglionic neurons. We immunohistochemically investigated the synapses localization in the spinal nerve ganglia using antibodies against the Synapsin I and the Synaptic vesicle protein 2. Synapse-immunopositive structures were distributed in the TH-ir nerve mass but not in the spinal nerve ganglion proper. These structures were interpreted as synapses between preganglionic fibers and postganglionic neurons. On this basis, we suggested that the TH-ir nerve cell are not adrenergic sensory ganglion neurons but are sympathetic postganglionic neurons.

## P1-f46 Respiratory neurons in the caudal VRG of the monkey

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The basic breathing rhythm takes place in the medulla. However, little is known about the location and firing patterns of medullary respiratory neurons in the monkey. This study was carried out to investigate the respiratory neurons in the monkey brainstem caudal to the obex. Experiment was performed on 1 adult monkey, anesthetized with sodium pentobarbital. Phrenic nerve discharges did not show the post-inspiratory discharges. Twelve extracellular recordings were made. All neurons were expiratory (E) neurons (12/12). Eleven of 12 E neurons were augmenting type and 1 neuron was unclassified. Ten of 12 neurons extended their descending axons in S2 spinal level. Recording sites were found in the region of the nucleus retroambigualis. The evidence in the present study suggests that the function of E neurons might be involved in the important role of co-ordinating respiration and motor functions in the lumbar and the sacral spinal cord.

## P1-f47 Effects of local heating of body surface on physiological functions in Japanese women sensitive to the cold

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A steaming heat pack was applied to the abdomen or lower back of normal control subjects and women sensitive to the cold while physiological functions including EEGs, ECG, blood pressure (BP), and local skin and tympanic temperature as well as subjective feeling were monitored in a low temperature environment (20-21C°). The results indicated that local heating in the abdomen and back increased $\alpha$-wave power, surface temperature in the toe and subjective comfortable sensation, but did not change tympanic temperature. Analyses of heart rate variability indicated that sympathetic activity was higher while parasympathetic activity was lower in the normal control than women sensitive to the cold, suggesting abnormal autonomic responses to low temperature environment in women sensitive to the cold.

## P1-f48 Circadian profile of ventilation in histamine H1 receptors lacking mice

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We examined the circadian profile of ventilation and metabolism in mice, and the hypothesis that activation of the histamine H1 receptor is required for the ventilatory variation throughout a day. Ventilation, aerobic metabolism, and electroencephalogram were measured in unanesthetized state of H1 receptor-knockout (H1RKO) and wild-type mice. Both genotypes showed daily oscillations in minute ventilation (VE) and oxygen consumption (VO2), with greater values during the dark period than the light period. In the light period, H1RKO mice increased VE and CO2 excretion (VCO2), but did not change VO2 compared with wild-type mice, suggesting that genotype differences in VE are responsible for VCO2 differences. In the dark period, H1RKO mice did not change VE, but elevated VCO2 compared with WT mice, showing reduced ratio of VE to VCO2. Electroencephalogram results show a depressed arousal state in the dark period of H1RKO mice. We conclude that histamine modulates circadian profile of ventilation by affecting metabolism and arousal state via H1 receptors.

## P1-g01 Activation of COX isozymes in spinally projecting neurons in the PVN and LC by centrally administered CRF in rats

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Recently, we reported the involvement of brain cyclooxygenase (COX) in centrally administered corticotropin-releasing factor (CRF)-induced activation of the sympatho-adrenomedullary outflow in rats. In this study, we examined the expression of brain COX isozymes in the spinally projecting neurons using rats injected with monosynaptic retrograde tracer into the spinal intermediolateral cell column. Retrogradely labeled cells were detected in the paraventricular hypothalamic nucleus (PVN), locus coeruleus (LC), raphe nuclei and rostral ventrolateral medulla (RVLM). After central administration of CRF, co-expression of COX-1/c-Fos in the PVN-spinal neurons and of COX-2/c-Fos in the LC-spinal neurons were observed, while co-expression of COXs/c-Fos was not observed in the raphe- and RVLM-spinal neurons. These data suggest that COX-1 in the PVN and COX-2 in the LC play roles in the CRF-induced activation of the central sympatho-adrenomedullary outflow in rats.

## P1-g02 Neonatal cerebellum broadly displays respiratory activities

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We recorded respiratory activities in newborn rat cerebellum-pons-medulla (-spinal cord) block preparations using optical imaging with voltage-sensitive dye. Cycle triggering with C4 inspiratory activity revealed optical responses broadly in the cerebellum covering the lateral part of VII-VIII lobules, paraflocculus (PFL), lateral end of vermis and the inferior olive. Optical recording further revealed that pulmonary stretch evoked activities even more broadly in the cerebellum via a vagus nerve. We also showed with whole cell recording that Purkinje cells exhibit respiratory modulation of simple spike discharges. When DiI was injected into the inferior olive, it was anterogradely transported to the respiratory active parts of the cerebellum, whereas it retrogradely labeled neurons located ventrally to the ambiguus nucleus. Since conjunction of climbing fiber signals from the inferior olive and mossy fiber signals from vagus nerve is expected to induce long-term depression, the present results suggest that, during neonatal stages, a major portion of the cerebellum plays a role in adaptive control of respiration.

KAKENHI 18500318

## P1-g03 Identification of new antigenic candidates in autoimmune neuropathies by serological proteome analysis

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In order to identify new antigenic candidate proteins present in peripheral nervous tissue, we performed proteome mapping and immunoproteomics analysis using patients sera of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), Guillain-Barre syndrome (GBS), $\alpha$-myelin-associated glycoprotein neuropathy ($\alpha$-MAG), and leprosy patients. Over 50 of antigenic protein reacted on immunoblots membrane with CIDP, GBS and $\alpha$-MAG sera. More than 30 spots were successfully matched with stained gels. The majority of the peripheral nervous proteins which is immunoreactive with patients sera belong to the following categories; neural function, protein synthesis and processing, signal transduction, metabolism and transporters. Particularly prominent is the protein related redox reaction, such as GST-pi protein, MnSOD, peroxiredoxin-1 and thioredoxin peroxidase 2 isoform c, reacted on immunoblots membrane with GBS sera. In conclusion, serological proteome analysis may provide a useful tool for the identification of novel autoimmune antigens, whose relevance to pathogenically events in autoimmune neuropathies remains to be defined.

## P1-g04 Chronological changes of CD4$^+$ and CD8$^+$ T cell subsets in the periphery and CNS during the course of EAE

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To determine the contribution of CD8$^+$ T cells to the pathophysiology of experimental autoimmune encephalomyelitis (EAE), an animal model of multiple sclerosis, we examined CD4$^+$ and CD8$^+$ T cell responses in the spleen and CNS of EAE mice. The increase in splenic CD4$^+$ T cells was more pronounced than that of CD8$^+$ T cells. In contrast, the number of both CD4$^+$ and CD8$^+$ T cells in the CNS was associated with increasing disease severity, however, myelin oligodendrocyte glycoprotein (MOG)-specific cells were distributed in all cellular and anatomic compartments examined. PCR analysis suggested that both CD4+ and CD8+ T cells produced interferon-γ (IFN-γ) and tumor necrosis factor-α (TNF-α), while interleukin-17 (IL-17) was produced mainly by CD4+ T cells. Taking together, our data suggest that CD4$^+$ T cells play a critical role in EAE induction, whereas CD8$^+$ T cells may play an important role in EAE progression and maintenance.

## P1-g05 The effects of viscero-somatic interactions on thalamic mast cell recruitment in rat developing cystitis

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Mast cells (MCs) accessing brain parenchyma through the blood-brain barrier in healthy animals are limited to pre-cortical sensory relays, the olfactory bulb and thalamus. We have demonstrated that the unilateral repetitive stimulation of the abdominal skin generates controlateral thalamic asymmetry in the distribution of MCs in the rostralmost part of the midline thalamus, the paraventricular pars anterior and reuniens nuclei subregion of animals injected with cyclophosphamide, in strict relation with cystitis genesis. Data are probably related to abnormal visceral/somatic interactions. Thalamic MC asymmetry is restricted to the brain region associated with visceral/vagal inputs, via the nucleus of the solitary tract, and somatic inputs, via the medial contingent of the spinothalamic tract inputs, and takes its origin from abdominal skin where cystitis generates secondary abdominal skin hyperesthesia leading to referred pain in man. We suggest thalamic MCs may play a role in integrative and cognitive sensory processes, including some aspects of visceral pain.

## P1-g06 Alteration of systemic/brain tryptophan metabolism and modulation of anxiety-related behavior in tryptophan 2,3-dioxygenase (TDO)-deficient mice

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The tryptophan (Trp) and its metabolites such as serotonin (5-HT) play a critical role in physiological condition and psychiatric diseases. TDO and indolamine 2,3-dioxygenase (IDO) are the initial two rate-limiting enzymes for Trp metabolism. Despite the importance of this enzymatic step, the contribution of TDO and IDO in vivo is poorly understood. Here, we generated mice deficient for *tdo* (*Tdo*$^{-/-}$) and studied the physiological roles of TDO in vivo. *Tdo*$^{-/-}$ mice showed markedly increased plasma levels of Trp and 5-hydroxyindoleacetic acid (5-HIAA) compared with wild-type littermates. *Tdo*$^{-/-}$ mice also showed increased levels of brain Trp, 5-HT and 5-HIAA in the presence of IDO. In addition, behavioral analyses revealed an anxiolytic modulation in *Tdo*$^{-/-}$ mice. These findings demonstrated that TDO plays an essential role in regulating the levels of both systemic and brain Trp metabolites, and functions as a dominant regulator of the serotonergic pathway, and contributes to mood control.

## P1-g07 Healthy carriers of the 5HTTLPR s/s genotype are romantic?

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Serotonin transporter (5HTT) function has a role in the development of negative mood states. Healthy carriers of the short (s) allele of the promoter region of the 5HTT gene have shown stronger activations of the emotion-related brain regions, such as the amygdala, during viewing the aversive stimuli, compared to the carriers of the long (l) allele. The present study investigated whether the 5HTT gene-linked polymorphic region (5HTTLPR) also affected the sensitivity to positive stimuli or events. When the healthy carriers of the 5HTTLPR s/s genotype watched the film of the person whom they are in love with, several brain regions, such as the orbitofrontal cortex and hypothalamus, were activated strongly and they become positive, relative to the carriers of the l/s and l/l genotypes. Therefore, s/s carriers may also be more sensitive to love-related events and may be head over heels in love with the loved one.

## P1-g08 Two types of social buffering differentially attenuates conditioned fear responses in male rats

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In this study, we assessed the ability of two types of social buffering to attenuate the conditioned fear responses in Wistar male rats. After being fear conditioned to the auditory conditioned stimuli, the subject was housed for one day either solitary or with an unfamiliar associate (Pair-housing). On the next day, the subject underwent the fear expression test either solitary or with an unfamiliar associate (Pair-exposing). The Pair-housing suppressed the autonomic response, whereas the Pair-exposing diminished the freezing behavior. In addition, the Pair-housing and Pair-exposing additively attenuated the fear responses. These results suggest that the two types of social buffering differentially attenuated the conditioned fear responses depending on the timing of an associate's accompaniment. In addition, the Fos analyses suggest the role of the infralimbic region of the prefrontal cortex and basal amygdala in each social buffering.

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## P1-g09 A possible role for an adaptor protein, X11L/mint2, in risk assessment

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X11L/Mint2 is a brain-specific adaptor protein that interacts with many synaptic molecules. We previously demonstrated that a lack of X11L alters the metabolism of its binding protein, amyloid precursor protein, via sorting mechanisms. Thus, we hypothesized that X11L is involved in integrating neuronal information through regulating the sorting or anchoring of synaptic machineries. To test this hypothesis, we examined the phenotypes of X11L-deficient mice at molecular, cellular, and behavioral levels. The mutant mice were normal in conventional behavioral test batteries assessing emotional and cognitive abilities. The mutant mice had behavioral deficits, however, when the environmental conditions were changed in some behavioral tests, such as home cage feeding behavior, elevated plus maze, and marble burying tests. These behaviors reflect conflicts and environmental conditions might determine the conflict level. The data suggest that X11L has a role in neuronal mechanisms that are involved in risk assessment.

KAKENHI(18700403)

## P1-g10 Behavioral characteristics of ASK1 KO mice

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Apoptosis signal-regulating kinase 1(ASK1) is a stress response factor activated by various stresses. However, little is known about its role in brain functions. Immunohistochemical analysis of wild type mice (C57/bl 6J) revealed the presence of high density ASK1 protein in broad area of the brain. We applied line of behavioral tests to ASK1 KO mice (e.g. the tail suspension test, the elevated plus maze test). Compared to wild type, ASK1 KO mice showed significantly higher activities in the novel environment and daily movement remained high in the home cage. In addition, achievement of the rotarod test was also high in ASK1 KO mice. These results suggest that ASK1 may be involved in emotion-related motor functions.

## P1-g11 Mechanisms of increase in anxiety by overexpressing of CaMKII in forebrain

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Previous studies have suggested important roles of α Calcium/Calmodulin dependent protein kinase II ( α CaMKII) in emotional behaviors. To understand roles of α CaMKII in emotional behaviors, we have investigated transgenic mice overexpressing α CaMKII in forebrain and found that mutant mice showed increase in anxiety. Taken together with findings from CaMKII KO mice, we suggest positive correlation of CaMKII expression and anxiety-related behavior. To investigate mechanisms of increase in anxiety led by CaMKII overexpression, we next analyzed the expression profiles of mutant mice using micro array and found that mutant mice showed abnormal expression levels of genes (Osr2 and Calbindin) in dentate gyrus of hippocampus. We also found the correlation of onset between increase in anxiety and decrease in Calbindin expression in dentate gyrus of male mutant mice. To further understand the abnormalities of dentate gyrus function of mutant mice, we are now examining adult neurogenesis in dentate gyrus.

## P1-g12 Chemical property of the neural pathways from the central amygdaloid nucleus to the nucleus retroambiguus via the midbrain periaqueductal gray in the rat

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The midbrain periaqueductal gray (PAG) is involved in producing vocalization and lordosis, sending its projection fibers to the nucleus retroambiguus (NRA) that contains premotor neurons concerning vocalization and lordosis. Our previous study indicated that the axon terminals from the central amygdaloid nucleus (ACe) make synaptic contact with the PAG neurons projecting to the NRA. In the present study, we demonstrated that large numbers of NRA-projecting PAG neurons expressed vesicular glutamate transporter 2 mRNA but not glutamic acid decarboxylase 67 mRNA by using retrograde tract-tracing combined with *in situ* hybridization technique. Furthermore, it was revealed that most of the ACe terminals in the PAG exhibited GABA immunoreactivity by using a combination of anterograde tract-tracing and immunohistochemical techniques. The data suggest that the ACe exerts a GABAergic inhibitory action upon glutamatergic excitatory PAG neurons that project to the NRA.

## P1-g13 Organization of the regions surrounding the medial geniculate nucleus

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The posterior thalamic regions surrounding the medial geniculate nucleus have been implicated in various functions, such as classical fear conditioning, nociception, and sexual behavior. Despite growing interest, there is some controversy over the parcellation of these regions, which makes it difficult to examine the neuronal circuits involving them. In the present study, we examined these posterior thalamic regions of mice with the immunohistochemical and tract-tracing methods. The multiple fluorescent immunohistochemistry revealed their basic cyto- and chemoarchitecture. The injections of retrograde tracer Fluoro-Gold into several cortical and subcortical regions resulted in the labeling patterns characteristic of the individual injection sites. Especially, the injections into three amygdalar nuclei (lateral, central, medial) displayed prominently different patterns of labeling, in good correspondence with the chemoarchitecture. On the basis of these results we propose the novel parcellation of those regions.

## P1-g14 Brain activity for reward prediction on ambiguous perception

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It is unclear how our brain predicts reward and punishing outcome on perception of ambiguous stimuli in environment. To investigate relationship between perceptually ambiguous stimuli and reward processing on them, we examined reward-predicting brain activity on high and low coherency random dot motion stimuli by using fMRI. First, we trained subjects to establish a contingency between particular direction of motion stimuli and delivery of reward. Then, we presented high and low coherence motion stimuli, and asked subjects to judge direction of the motion. Juice or saliva was followed by stimulus direction associated with reward or neutral outcome, respectively. The data at the time of cue presentation showed that activation in the caudate was correlated with reward prediction based on stimulus direction that was influenced by coherence level, whereas reward-predicting activation based on subject's performance was observed in the putamen. The results suggested that stimulus-based and perception-based reward prediction could be dissociated in the basal ganglia.

## P1-g15 Analysis of local field potentials in the hippocampus of piglets during rest

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Under halothane anesthesia, a pair of tungsten electrodes was implanted into the temporal hippocampus of piglets and fixed on the surface of the skull with a transmitter using dental cement. After recovery from surgical procedures, we recorded local field potentials (LFPs) by radiotelemetry in unrestrained conditions. When the piglets were rest at daytime. The two types of theta, 1 (7-12 Hz) and 2 (4-6 Hz) were present in the LFPs. In addition, beta(12-30 Hz) or gamma(30-80 Hz) activity was often recorded in the hippocampus, which may represent attentive immobility in the piglets during lying down.

## P1-g16 The regional difference in temperature related sensations

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In the present study, we examined the regional difference in temperature-related sensations, which can be divided into "temperature sensation" and "thermal comfort/discomfort". Subjects sitting in a 32-33 °C (hot) or 21-22 °C (cold) environment were locally cooled and warmed with water perfused stimulators. The areas stimulated were the face, chest, abdomen and thigh. Temperature sensation and thermal comfort/discomfort of stimulated areas and those of whole body were reported by the subjects. While there is no significant regional difference in local temperature sensation, there are significant differences in local thermal comfort/discomfort among the four areas. The neuronal wiring in the central nervous system to produce thermal comfort/discomfort would differ among body regions.

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## P1-g17 Analyses of running wheel activity (RWA) in aromatase-knockout (ArKO) mice

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Estrogen (E)-deficient ArKO mice show disrupted reproductive behaviors, but their non-reproductive (NR) behaviors, such as activity, anxiety and learning, are not well defined. In the present study, as part of ongoing analyses of estrogenic regulation of NR behaviors, we examined RWA of ArKO mice. RWA of gonadally intact ArKO mice of both sexes was significantly lower than those of wild-type (WT) and heterozygous (HZ) mice. Chronic E treatment at a total dose of 1 or 5μg after gonadectomy increased RWA in ArKO males to the levels similar to those of WT and HZ mice. In females, E treatment failed to increased RWA of ArKO mice to the levels seen in WT and HZ mice especially at the dose of 1μg, which was generally more effective than 5μg. These results suggest that E facilitate RWA in both sexes, although the dose response to E may be different between sexes. Interestingly, within-day patterns of RWA of both sexes of ArKO mice was different from those of WT and HZ regardless of hormonal conditions, suggesting that they may be determined by an organizational effect of E.

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## P1-g18 Orexinergic neurons mediate excitatory signals to Purkinje cells in a descrete microzone of the cerebellum

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We previously reported in rabbits that electric stimulation of the classic defense areas in the hypothalamus and the mesencephalic periaqueductal gray (PAG) leads to excitation of Purkinje cells in a microzone of the cerebellar flocculus (folium-p). Because the excitation is blocked by orexin receptor-1 antagonists, we suggest that orexinergic neurons in the hypothalamus and their axons passing through PAG are its sources. In this study, to support this assertion, we further demonstrate that the excitation was virtually abolished when orexinergic neurons had been destroyed by intrahypothalamic injection of saporin conjugated with orexin B. The defense area stimulation also induces bicucullin-sensitive inhibition in folium-p Purkinje cells. Because this inhibition was less affected than the excitation by saporin, it may be mediated, at least in part, by non-orexinergic fibers that excite inhibitory interneurons in the cerebellum or release GABA from themselves.

## P1-g19 Role of estrogen receptor b (ERb) and sexual experience in female reproductive behavior behavior in mice

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Female mice and rats are highly receptive on the day of estrus but their receptivity greatly diminishes by the day after estrus. Previously, we found that unlike wild-type (WT), ERb deficient (bERKO) mice continued to show a high level of sexual receptivity even on the day after estrus. These findings led us to hypothesize that a lack of ERb might interfere with an inhibitory action of progesterone (P). In the present study, we tested this hypothesis in mice ovariectomized and treated (sc) with estradiol benzoate (EB) and P. In the test done at EB48h+P4-7h (Test-1), bERKO and WT mice showed similar levels of sexual receptivity, whereas on the next day (Test-2) only bERKO mice were receptive. Interestingly, these genotype differences were abolished by weekly repetition of the same procedure for a few weeks, i.e., with sexual experience, WT females also started to show high receptivity even in the Test-2. These results suggest that ERb may be responsible for fine-tuning of a timing of sexual receptivity during estrous cycles in sexually naïve mice.

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## P1-g20 Anxiolytic and antidepressant effects of estradiol in postpartum rats after weaning pup

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A decline in E2 at delivery and lactation is thought to be involved in anxiety, depression and postpartum psychosis. It, however, remains unclear which E2-receptor subtype mediates those psychological disorders, ER $\alpha$ or ER $\beta$. Our first study demonstrated that primiparous rats at 3wks-postpartum showed anxiogenic and depressive response compared to diestrus nulliparou females in elevated plus maze (EP), pre-pulse inhibition (PPI) and forced swimming (FS) tests. In the second experiment, to determine whether E2's anxiolytic and antidepressant actions in the 3wks-postpartum is mediated by ER $\beta$. we examined the effect of ER-subtype selective agonist on behavioral tests after injection (s.c.) with selective ER $\beta$ agonist, diaryl-propio-nitrille (DPN; 1.0 mg/kg), ER $\alpha$ selective agonist propyl-pyrazole-triol (PPT; 1.0 mg/kg), 17 $\beta$ - estradiol (0.25 mg/kg), or vehicle once daily for 4 days. As a result, we confirmed the DPN's anxiolytic and antidepressant actions in EP, PPI and FZ tests with 3-wks postpartum rats.

## P1-g21 Dopamine metabolism in the nucleus accumbens induced by injection of CART peptide into the ventral tegmental area in rtas

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Cocaine- and amphetamine-regulated transcript (CART) is a putative neurotransmitter associated with the rewarding and reinforcing properties of both natural and unnatural stimuli. The mesolimbic dopamine (DA) system is a critical pathway involved in mediating reward and reinforcement, and the importance of CART-containing neurons within the mesolimbic DA system has been suggested. In the present study, effects of CART (55-102) peptide injected into the ventral tegmental area (VTA) were investigated on DA metabolism in the nucleus accumbens schell (NAS). Bilateral injections of CART peptide into the VTA did not affect DA concentration, but increased the concentration of DOPAC and HVA, DA metabolites. Pretreatment of sulpiride, a DA $D_2$ receptor antagonist, abolished the increase of DA metabolism induced by CART peptide. The results suggest that CART peptide plays a role in the mesolimbic DA system mediating reward and reinforcement.

## P1-g22 A new test to measure the strength of visual exploration in common marmoset

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To assess the motivational strength of visual exploration in common marmoset, we developed a new test. The apparatus consisted of a testing box and stimulus chamber with a black wall between them. A small hole was made in the wall so that a stimulus was visible via the hole. We measured frequency and duration of visual exploration response (peeping at a stimulus through the hole) under six stimulus conditions (4 individual marmosets and 2 inanimate objects) and an empty condition. Both frequency and duration of response increased when another marmoset was in the stimulus chamber but not increased when an inanimate object was in the chamber in comparison with when the chamber was empty. Such higher rate of visual exploration response continued at least five consecutive experimental days. Furthermore, this apparatus was installed easily to a home cage. We propose this newly developed test to assess the motivational strength of visual exploration in common marmoset.

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## P1-g23 Theta EEG power enhancement for encoding novel scene at attentional blink period

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Theta activity is one of the prominent oscillatory pattern in rodents and humans. Recent studies reported the functional importance of the cortical network emergence with the theta oscillation during various cognitive tasks. To investigate the functional relevance of the theta oscillation in human for encoding the working memory, we performed EEG measurements by using a rapid serial visual presentation (RSVP) task. The RSVP task had two targets in its visual stimuli stream, and the subjects had to identify the both targets by judging whether or not the probe stimuli were presented in the stream. We used four digit numbers as first target, and novel natural scenes as second target. The modulation of theta EEG power was identified in association with the second target presentation by applying a subsequent memory analysis. The results showed that the frontal medial theta activity (centered on 3Hz) concerns with the encoding of the novel scenes at the situation without the stimulus-driven attention.

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## P1-g24 Enhancement of memory formation and LTP by up-regulation of CREB activity

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Previous genetic studies have shown that the loss of function of transcription factor CREB leads to an impairment of memory consolidation, indicating that CREB is an essential molecule for this process. To further understand roles of CREB in memory consolidation, we examined effects of up-regulation of CREB activity on memory consolidation. We have generated transgenic mice expressing dominant active CREB mutant (CREB Y134F; MCB, 20, 4320-, 2000) in forebrain under the control of $\alpha$ CaMKII promoter. These mutant mice showed significant increase in c-fos expression in hippocampus and amygdala. Behavioral analyses indicated that increase in CREB activity enhances consolidation of fear, spatial and recognition memories. Consistent with this, these mutant mice also showed the enhancement of long-term potentiation (LTP) in CA1 region of hippocampus. Taken together with previous studies that blocking CREB function disrupts memory consolidation, we concluded that CREB functions as a molecular switch to form memory.

## P1-g25 Comparison of hippocampal LTP induction and maintenance between monkeys and rodents

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Long-term potentiation (LTP) is a form of synaptic plasticity, which has been studied extensively in the rodent. However, information about LTP in the primate hippocampus is limited. We have recently been developed a technique to record evoked potentials in the monkey hippocampus. Using this model, we investigated the induction and maintenance of hippocampal LTP in monkeys and compared its properties with those of rats. Stimulation and recording electrodes were implanted in the perforant pathway and dentate gyrus, respectively. The effects of high frequency stimulation (HFS) on evoked potentials were tested in an awake condition. As results, HFS reliably induced LTP in the hippocampus of both species. LTP was maintained stably for more than 4 weeks in monkeys, whereas it declined to the basal level within a week in rats. This could underlie the difference of the course of memory consolidation between the 2 species.

## P1-g26 The Drosophila cell-adhesion molecule Klingon is required for long-term memory and is regulated by Notch signaling activity

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Although cell adhesion molecules have been implicated in long-lasting synaptic plasticity, their contribution to the long-lasting memory is not clear. In Drosophila, long-lasting memory can be dissected into a translation-dependent long-term memory (LTM) or a translation-independent anesthesia resistant memory (ARM). A behavioral screen for defects in long-lasting memory identified the ruslan (rus) mutant, which is disrupted in klingon (klg), a member of the Drosophila immunoglobulin superfamily of cell adhesion molecules. Strikingly, Klg is regulated by the Notch signaling pathway, and Notch induced enhancement of LTM is disrupted by the klg mutation. Klg is significantly localized at the juncture between neuropil and glia in the adult brain. Furthermore, LTM was restored in klg mutant when klg is expressed either in neuronal or glial cells, suggesting that Notch regulates Klg expression to establish neuron-glia communication required for LTM formation.

## P1-g27 Activity-dependent organization of associative neuronal dynamics of the hippocampal CA3 network in vitro

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To investigate contribution of the recurrent synapses on the associative memory function of the hippocampus, we recorded simultaneously multi-unit activities from two groups of CA3 neurons in a hippocampal slice by using two tetrodes with a separation of 0.4-mm. When we stimulated locally one of the groups of neurons by micro-iontophoresis of glutamate, putative mono- or polysynaptic activities were observed in the other group of neurons (associative response, AR). Attempting to enhance the AR, we stimulated simultaneously and repetitively both of groups by the micro-iontophoresis of glutamate (paired stimulation, PS). As a result, the number of AR-positive neuron was increased significantly. The PS facilitated switching from AR-negative to AR-positive neuron. These data suggest that the associative neuronal activities reorganize the neural network in the hippocampal CA3 region by modifying their recurrent synapses.

## P1-g28 The role of NO in the formation of long-term memory in cockroaches

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It was suggested that long-term memory formation requires activation of NO signaling pathway in crickets. However, the use of free-moving insects has some difficulties in studying the mechanisms by which NO pathway induces long-term memory formation. previously In *Periplaneta americana*, we reported that the responses of salivary neurons to an odor increased by repeated paring of odor with sucrose solution and that this effects were retained at least 1 day after conditioning. Here, we investigated the role of NO pathway for the long-term memory formation in cockroaches using pharmacological method. Conditioning effect 1 day after conditioning was impaired by injection of L-NAME, inhibitor of NO synthase. In contrast, injection of saline had no effect on retention 1 day after conditioning. Memory acquisition and retention 30 minute after conditioning was not affected by injection of L-NAME or saline. Our findings indicate that NO pathway is necessary for long-term memory formation in cockroaches.

## P1-g29 ERK is required for fear memory formation

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The Raf/MEK/ERK cascade plays a central role in various situation of signal transduction in eukaryotic cells. To clarify a role of ERK in memory formation, we generated transgenic mice expressing dominant negative MEK1 (dnMEK1) in forebrain inducibly with tetracycline-regulated gene expression system. In contextual fear conditioning test, the transgenic mice displayed a significant impairment in freezing response at 24 hours after shock. However, doxycycline-treated transgenic mice displayed no deficits in freezing response. Next, we reactivated dnMEK1 expression by depleting doxycycline after the mice had learned the task. After a 6-weeks period, dnMEK1 expression was restored and the freezing response of transgenic mice was markedly impaired. These results indicate that ERK cascade plays important roles in not only memory consolidation but also memory retrieval.

## P1-g30 Mechanisms of memory reconsolidation associated with memory enhancement in passive avoidance task

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Recent findings showed that memory retrieval induces destabilization of reactivated memory and that reactivated memory is re-stabilized through new gene expression-dependent reconsolidation. However, the significance of memory reconsolidation still remains unknown. To understand this, we have examined the effects of re-exposure on memory reconsolidation using light-dark passive avoidance task. This task is thought to allow us to discriminate between reconsolidation and extinction phases at the time point when mice enter dark box from light box. We found that re-exposure to light box enhanced passive avoidance memory and that protein synthesis inhibition or inhibition of CREB activity blocked both enhancement and reconsolidation of avoidance memory, suggesting that memory reconsolidation is associated with memory enhancement. More importantly, protein synthesis inhibition in hippocampus blocked only enhancement of avoidance memory, indicating the hippocampus is required for only enhancement but not reconsolidation.

## P1-g31 Roles of gene expression regulation in consolidation and reconsolidation of object recognition memory

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Memory consolidation is thought to require gene expression. Recent studies have shown that when conditioned fear memory is retrieved, fear memory becomes labile and requires protein synthesis-dependent reconsolidation to re-stabilize reactivated memory. We have investigated the roles of gene expression in consolidation and reconsolidation of fear memory and shown that transcription factor CREB is required for consolidation and reconsolidation of fear memory. In this study, to understand the impacts of gene expression regulation on object recognition memory, we examined a role of novel protein synthesis and CREB using pharmacology and mouse genetics. Here we showed that protein synthesis inhibition by anisomycin (ANI) disrupts consolidation and reconsolidation of object recognition memory. We found that transgenic mice expressing dominant active CREB mutant show the enhancement of consolidation of object recognition memory. These findings indicate crucial roles of new gene expression in consolidation and reconsolidation of object recognition memory.

## P1-g32 Optical imaging of dendritic propagating potential in heterosynaptic associative LTP of rat hippocampal CA1 pyramidal neurons

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In contrast to Hebb type learning rule, in spatiotemporal learning rule (STLR) proposed by Tsukada (1996), synaptic strength is modified by both spatial coincidence and the temporal summation of synaptic inputs without back propagating action potentials (BPAP). When a pair of strong and weak stimulation is applied simultaneously in hippocampal CA1, heterosynaptic associative LTP, one of the physiological evidence of STLR, is induced in both stimulation sites. Our previous study showed that heterosynaptic associative LTP could be induced under decreased-BPAP condition using low TTX application. However, the mechanism of LTP was unclear. In this study, we recorded dynamic responses in both strong and weak input site under low TTX condition by using voltage-sensitive dye. From this result, it is clear that EPSP produced by strong stimulation propagates along the dendrite and affects the synaptic strength of weak input site.

COE program of JSPS

## P1-g33 Transcription factor BMAL1 plays critical roles in memory formation

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bHLH-PAS transcription factor BMAL1 ubiquitously expresses in brain. BMAL1 functions by forming a heterodimer with either CLOCK or NPAS2, which has been known to play important roles in control of circadian rhythm and memory formation, respectively. To understand roles of BMAL1 in learning and memory formation, we generated conditional mutant mice that enable to induce the inhibition of BMAL1 function in forebrain. These mutant mice express a dominant negative mutant of BMAL1 (BMAL1 R91A) that forms a heterodimer with CLOCK but loses the binding activity with E-box (Hosoda et al, 2004), under the control of tetracycline-dependent promoter. Behavioral analyses showed that these mutant mice exhibit severe impairments of formation of contextual fear, social recognition and spatial memories but showed normal immediate memory of social recognition. In contrast, transgenic mice suppressed BMAL1 R91A expression by doxycycline revealed normal memory formation. These results indicate that BMAL1 plays critical roles in memory formation.

## P1-g34 Roles of retinoic acid receptors in learning and memory

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Retinoic acid regulates gene expression through their nuclear receptors (retinoic acid receptor; RARs and retinoid X receptor; RXRs) that express in a wide range of brain. Previous studies have provided evidence that retinoic acid deficient mice and RARs knock out mice show impairments of LTP and spatial memory. To further clarify the roles of RARs in learning and memory, we generated several types of conditional mutant mice that overexpress RAR $\alpha$ in a forebrain. Because biochemical analyses show that overexpression of RAR $\alpha$ led to increase in expression of target gene of RARs, retinoid signaling pathway is thought to be activated in forebrain of these mutant mice. Behavioral analyses revealed that RAR $\alpha$ overexpression mice showed enhancement of contextual fear and spatial memories. Taken together with previous findings, we suggest that RAR $\alpha$ plays crucial roles in learning and memory and functions as a positive regulator for learning and memory.

## P1-g35 Mechanism of reconsolidation and extinction of spatial memory

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Previous our studies using Morris Water maze revealed two distinct time-dependent phases following memory retrieval: reconsolidation and extinction phases (Suzuki et al., 2004). To understand regulation of memory stability following retrieval, we have investigated mechanisms of reconsolidation and extinction of spatial memory using Morris water maze. We first analyzed a role of new gene expression and N-methyl-D-aspartate receptor (NMDAR) function in hippocampus in reconsolidation of spatial memory and found that infusing protein synthesis inhibitor anisomycin or NMDAR antagonist D-AP5 into hippocampus immediately after probe trial disrupts reactivated spatial memory. These data suggest that retrieval of spatial memory triggers reconsolidation of spatial memory, and that this process requires protein synthesis and NMDAR function. We are now investigating molecular mechanism of destabilization of reactivated spatial memory in hippocampus and mechanisms of extinction of spatial memory.

## P1-g36 Mechanisms of reconsolidation and extinction of contextual fear memory

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Retrieval of conditioned fear memory by conditioned stimulus (CS) initiates two processes; reconsolidation or extinction. Protein synthesis inhibition following short re-exposure to CS disrupted fear memory but inhibition of protein synthesis following longer re-exposure blocked only extinction without affecting fear memory. To further understand the relationship between reconsolidation and extinction, we first examined roles of CREB using conditional mutant mice that enable to inhibit CREB activity and found that similarly with results using protein synthesis inhibitor, CREB is required for reconsolidation and extinction of contextual fear memory. We next investigated brain regions increasing phosphorylated CREB at S133 in reconsolidation and extinction phases. We observed increase in phospho-CREB in hippocampus and amygdala following short re-exposure to CS inducing reconsolidation, and amygdala and prefrontal cortex following longer re-exposure inducing extinction, respectively, indicating that duration of re-exposure determines brain regions activated for memory processing.

## P1-g37 Use of intron and exon probes to characterize the expression of immediate early genes

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Immediate early genes (IEGs) such as *c-fos* and *zif268* are frequently used as markers for active neurons. To improve time resolution in IEG expression analysis we performed *in situ* hybridization with probes against the intronic regions of IEGs, which specifically detect pre-mRNA, and compared the time course of pre-mRNA expression to mRNA expression detected with probes against the exon, which detects mature mRNAs. Our results revealed several differences in spatial and temporal expression patterns among IEGs, suggesting that every IEG has its own expression profile. Furthermore, taking advantage of the temporal difference between pre-mRNA and mRNA appearance we found that for some IEGs neurons expressing the same IEG changed depending on the stimuli applied. This method will provide us opportunities to compare activated neuronal populations in wider areas in the brain by choosing suitable IEGs for particular brain regions of interest.

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## P1-g38 Attractor variability of neuronal population activity in hippocampus and neocortex of rats during conditional association learning

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Some studies have suggested neuronal activities change through learning process. However, it is unclear whether previously acquired task-related neuronal activity is consistently preserved or not. For investigating this problem, neuronal activities of hippocampus and medial prefrontal cortex in rats were recorded by using multi-electrodes techniques during a conditional auditory-spatial discrimination task, which demands association learning between a tone and a position to be responded. Neuronal population activity was firstly recorded during asymptotic performance of this task and then changes of discharge rates and synchronization in the same neuronal population were examined through reversal learning and relearning processes. Relearning must require the same information processing as in the original learning whereas it has been interfered by reversal learning. Thus, we would discuss about flexibility of population coding in the hippocampal memory system.

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## P1-g39 Functional analysis of the Rho GTPase-activating protein RICS in the nervous system

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In neuronal systems, Rho GTPase-activating proteins (RhoGAPs) and their substrates, Rho GTPases, have been implicated in regulating multiple processes such as axonal growth and guidance, dendritic elaboration and formation of synapses. RICS is mainly expressed in the brain and functions as a RhoGAP protein for Cdc42 and Rac1 *in vitro*. To examine the biological function of RICS, we disrupted the RICS gene in mice. RICS$^{-/-}$ mice developed normally and were fertile. However, when cultured *in vitro*, Cdc42 activity in RICS$^{-/-}$ neurons was higher than that in wild-type neurons. Consistent with this finding, hippocampal neurons derived from RICS$^{-/-}$ mice bore longer neurites than those from wild-type mice. Furthermore, to investigate the behavioral significance of RICS$^{-/-}$ mice, we subjected these mice to a battery of behavioral tests. RICS$^{-/-}$ mice showed deficits in cued fear conditioning. These findings suggest that RICS plays an important role not only in neurite extension but also in the formation of fear memory.

## P1-g40 A cooperative dynamics of network of neural oscillators between the neuronal activity and the synatptic weight.

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In general, the activity of neurons highly depends on the structure of neural network, namely, the synaptic connections among the neurons. On the other hand, this network structure is also gradually changing, because the synaptic connection is modulated by its plasticity depending on the current neuronal activity. This cooperative dynamics between the neuronal activity and the synaptic connection is essential for realizing high adaptability in brain. However, it is almost unclear what type of functional network structure is organized through such a cooperative dynamics.Thus, we investigate this issue in the modeling study. Considering an oscillatory activity of neurons, we show that the system can be described by the both dynamics of the general phase oscillators and synaptic weights. This system exhibits various types of dynamical behaviors according to the rule of synaptic plasticity.

KAKENHI (18047014, 18019019, 18300079), CREST

## P1-g41 Involvement of the *Calcineurin B2* gene in *Drosophila* courtship memory

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Courtship conditioning is a classical conditioning paradigm in *Drosophila* that involves only physiological stimuli and is able to induce long-term memory (LTM) lasting several days. Since the activation of transcriptional factors or new protein synthesis is required for LTM-formation, it is possible that expression level of genes regulating LTM-formation increases after the conditioning. We have performed DNA microarray analyses to compare transcriptional changes induced by courtship conditioning in wild-type males, and identified a *Calcineurin B2* (*CanB2*) that encodes regulatory subunit of calcineurin. Calcineurin is a $Ca^{2+}$/calmodulin dependent serine/threonine protein phosphatase and affects various behavior or neuronal plasticity in animal. We measured LTM of *CanB2* homozygous mutants by courtship conditioning, and confirmed that the homozygous mutants show the defective LTM but the LTM in heterozygous mutants are normal. Taken together, our study suggests that the formation of long-term courtship memory depends on the expression level of *CanB2*.

## P1-g42 Singing-related activity in basal ganglia of juvenile songbird

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Although the striatal nucleus Area X is clearly required for normal song learning, its function in this process remains unclear. As a first step toward understanding the role of the basal ganglia in this easily quantified form of motor learning, we have begun to characterize the activity patterns of Area X neurons in juvenile songbirds during the sensorimotor learning period. We recorded single-unit activity from Area X of male juvenile zebra finches (from 50 to 70 days of age) when the birds produce immature and highly variable "plastic" songs. We found that the level of activity of Area X neurons was consistently increased during singing. This increase began before initiation of singing. These neurons did not respond to playback of the bird's own song, or to hearing the tutor sing in awake condition. Some neurons that were active during singing also showed occasional phasic increases of activity during sleep, possibly similar to previously described "replay" of song-generation activity in song system motor nuclei.

## P1-g43 Neuronal activity of the nucleus accumbens and the globus pallidus in a decision making task

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Prediction of forthcoming reward for candidates of actions is a critical step in decision making. In this study, we recorded neural activity from the nucleus accumbens (NA) and the globus pallidus (GP) of rats during a decision making task. In this task, a rat should select left or right nose-poking, which is followed by a food reward at variable probabilities. We developed a reinforcement learning model which predicts rat's next choice, and estimated the reward predicted for each action upon each trial for analysis of neuron firing. During 1sec before execution of action, 15% of NA neurons and 28% of VP neurons showed correlation with reward prediction. During the action, 67% of NA and 61% of VP neurons showed action selectivity. The proportions of neurons coding reward predictions and actions were not significantly different between NA and GP. This result suggests that the transformation from expectation of reward to action selection is achieved by dynamics of the cortico-basal ganglia loop including NA and GP, rather than in a particular region.

## P1-g44 Relationship between prefrontal and hippocampal neuronal spike activities in anesthetized rats

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The prefrontal cortex (PFC) and hippocampus (Hip) are important brain regions for learning and memory. The anatomical connection between both regions is well known. The aim of this study was to investigate possible functional connections between these regions based on the correlation of neuronal activities. A cross-correlation histogram was constructed based on each pair of PFC and Hip neuronal spike trains which were simultaneously recorded in urethane-anesthetized rats. The pattern of cross-correlogram changed depending on the location of the electrodes. The cross-correlogram corresponding to monosynaptic projection from Hip to PFC was also seen. These results suggest the neuronal connectivity is reflected in the cross-correlogram changes and could be analyzed based on spontaneous firing patterns of PFC and Hip. The method using the present study may be useful in investigating neuronal connectivity between the PFC and Hip for learning and memory in conscious animals.

## P1-g45 *Nitric oxide synthase* dsRNA interfere with long-term memory formation in an insect

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Cyclic AMP pathway plays an essential role in long-term memory (LTM) formation. In insects, nitric oxide (NO)/cyclic GMP (cGMP) pathway is also involved in LTM formation. We suggested NO/cGMP pathway is upstream of cAMP/PKA pathway in pharmacological study in crickets (Matsumoto et al. 2006). We attempted to use dsRNA to investigate signaling cascade underlying the formation of LTM, and show that *nitric oxide synthase (NOS)* dsRNA is effective to interfere with LTM formation. Nympal crickets were injected with dsRNA before conditioning. At 30min and 24h after conditioning, we performed preference test. We found that *NOS* dsRNA interfered with LTM formation, without affecting short-term memory retention.

## P1-g46 Relationship between hippocampal and prefrontal local field potential power changes related to neuronal connectivity in anesthetized rats

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To investigate whether neural connectivity between the hippocampus (Hip) and prefrontal cortex (PFC) is reflected in the relationship between power changes in these local field potentials (LFPs), we recorded Hip and PFC extracellular spike activities (spikes) and LFPs simultaneously in urethane-anesthetized rats. The cross-correlation (CC) histogram between the Hip and PFC spikes was analyzed for each pair of recording sites. Recorded LFPs were filtered with 8-16, 16-32, 32-64, 64-128, and 128-256 Hz band-pass filters, and then the CC between the Hip and PFC LFP power changes was analyzed in each frequency band. These analyses revealed that the features of the CC histogram for neuronal spikes were similar to those of the CC for the 32-64 or 64-128 Hz LFP power changes. These results suggest that the neuronal connectivity is also reflected in the feature of the CC for the gamma-band power changes between the Hip and PFC. Similar methods may be useful for learning and memory studies on LFPs.

## P1-g47 Olfactory learning deficits and suppression of age-related memory impairment by a mutation in *chico*,a *Drosophila* insulin receptor substrate

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Age-related memory impairment (AMI) is a phenotype of brain aging. The molecular mechanisms underlying AMI remain elusive. Given that regulation of lifespan by insulin/insulin-like growth factor signaling (IIS) and intrinsic linkage between AMI and organismal aging, one candidate signaling pathway underlying AMI is IIS. Here we show that *chico*, which mediates *Drosophila* IIS, plays an essential role in olfactory learning and AMI. We found that *chico* null mutants exhibit learning deficits after olfactory conditioning. In addition, *chico* null mutation suppresses AMI. In contrast to *chico*, increasing IIS activity in the mushroom bodies (MBs), structures important for olfactory memory and AMI, causes premature AMI. Inconsistent with previous study, however, *chico* mutation did not extend female lifespan in our w(CS) genetic background. These results separate the brain aging from the organismal aging and demonstrate a biological role of IIS in the MBs both for learning and for AMI.

## P1-g48 The relation of the spatial cognition deficit and the hippocampal heterotopic neurons in the prenatal MAM-treated rats

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Offspring resulting from prenatal exposure to Methylazoxymethanol (MAM), a DNA methylating agent, on the embryonic day 15 are characterized by malformations in hippocampus, loss of lamination in neocortex, and microcephaly. We had reported that MAM-treated rats showed impaired reference memory in Morris water maze during adult period. In this study, we estimated reference- and working-memory of MAM-treated rats during weaning period, using Morris water maze task (Stage1) and delayed-matching-to-place (DMTP) task (Stage2). We further examined whether heterotopic neurons in hippocampus and neocortex have normal neural network, by injection of FluoroGold (FG) into septum and cervical spinal cord. MAM-treated group showed significantly longer escape latency in Stage1 and lower correct response ratio in Stage2 than control. Also, FG-labeled neurons were observed in both heterotopic regions. These results suggest that spatial cognition deficit in MAM-treated rat could not result from functional decline of heterotopic neurons.

## P1-h01 Role of monkey prefrontal cortex in temporal processing

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To examine how the brain represents the length of the elapsed time, we devised a time-reproducing task. The task required a monkey to plan button press based on the estimates of the elapsed time. After the monkey became able to discriminate among several different time lengths (1 and 5 seconds for monkey M; 2, 4 and 7 seconds for monkey J), single neuron recording was performed in the prefrontal cortex, chiefly in area 9. We found that a group of cells displayed time length-specific activity. Further, we inactivated area 9 by injecting a small amount of muscimol (a GABA agonist). After the injections, the number of errors increased significantly during the performance of the task. These results indicate that area 9 plays a critical role in the temporal processing.

## P1-h02 Effects of eye contact on social attention and their neural basis investigated by fNIRS and EEGs

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We investigated effects of eye contact on social attention and their neural basis by functional near infrared spectroscopy (fNIRS) and EEGs. A facial stimulus with a gaze directing to the left or right side of a display was presented. The subjects were required to indicate the location of a target stimulus which appeared at the left or right side of the display after the facial stimulus. Response latency to detect the target was shorter when gaze direction and location of the target was congruent. Effect of the gaze direction was more prominent when a facial stimulus with eye contact was presented. Now we are analyzing fNIRS and EEG data during this task, and the results indicated some heterogeneity of the cortex in social attention.

CREST

## P1-h03 Behavioral paradigm for neurophysiological studies of metamemory in monkeys

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Recent behavioral studies have demonstrated that macaque monkeys possess the metamnemonic ability to monitor their own memory. In addition, recent neuropsychological as well as neuroimaging studies have revealed prefrontal contributions to metamemory. However, neural mechanisms underlying metamemory is not yet known. Therefore, we developed a behavioral paradigm that requires the ability of metamemory, using oculomotor delayed matching-to-sample tasks, in order to examine the neural mechanisms underlying metamemory in the prefrontal cortex. In this paradigm, monkeys are sometimes allowed to decide whether or not to take a test on their own, depending upon their own cognitive states, and are sometimes forced to take a test. Better performance on voluntarily taken than on forced tests indicates the metamnemonic ability in monkeys. We argue that the present paradigm can be an effective tool for examining neuronal substrates of metamemory through single-unit recordings in monkeys.

KAKENHI (17300103)

## P1-h04 Dopamine responses to complex reward-predicting stimuli

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Midbrain dopamine neurons (DA) are suggested to encode reward prediction error signals. Previous studies found that the DA discriminated reward-predicting stimuli from nonreward-predicting stimuli with 50-110 ms latency. However, it still remains unclear whether and how the DA respond to complex reward-predicting stimuli, which take time to discriminate. To examine this issue, we used random dot motion direction as reward-indicating stimulus feature, which is believed to require cortical processing, and recorded the DA activity of two Japanese monkeys when they performed random dot motion discrimination task with asymmetric reward schedule. We found that the asymmetric reward schedule modulated their task performance. Furthermore, the reward asymmetry didn't affect the early component of the DA responses, but did the late component. Thus, we concluded that the DA calculated prediction errors based on continuously incoming information available at the moment.

Human Frontier Science Program

## P1-h05 Neuronal coding of reinforcers during multi-step choice task in the striatum

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To study neuronal coding of reinforcers during multi-step choice task in the striatum, we trained two monkeys to choose 1 of 3 target buttons in a trial-and-error basis. Correct choices were followed by high-tone beep and reward water, while incorrect choices were followed by low-tone beep without reward. Correct choice rates at first (N1), second (N2), third (N3) choices were 33, 50, 85 %, respectively. Once correct button was hit, monkeys got one more reward by choosing the known button (R1, 95 %). We recorded 239 projection neurons and 149 cholinergic interneurons based on their discharge properties. Cholinergic interneurons (105) responded to the beeps at N1, N2 and N3 trials with similar magnitudes, but showed almost no responses at R1 trials. Projection neurons responded to the beeps (143) with varied magnitudes depending on the reward probability (26), or responded differentially between R1 trials and other trials (24). Our results revealed distinct participations of projection neurons and cholinergic neurons in the reinforcement processes of reward-based decision and action selection.

KAKENHI(17022032)

## P1-h06 Risk-sensitive choice in chicks

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Several different factors of food items have been shown to be responsible for foraging choice, i.e., amount, proximity (either temporal or spatial), and consumption cost. In weeks-old chicks of domestic chicken (Gallus gallus), these factors proved to be represented in distinct neuronal assemblies in the pallio-striatal network (arcopallium and nucleus accumbens), suggesting that they are neuro-ecologically separable. In natural situation, however, food items may vary also in their availability, so that the chicks face risks. In this study, we developed a novel task in which risk-sensitive choice is precisely measured. Subjects are confronted with a pair of feeders, one serving a consistent profitability of p pellets of millet and another serving either 10 or 0 pellets each at equal probability. Results of behavioral titration revealed a consistent equilibrium value of p = ca. 3 pellets (i.e., considerably lower than the expected value of 5 pellets), indicative of risk-aversion. The present results show that the chick serves an ideal model for studying the neural basis of risk-sensitivity.

Kakenhi (18019015)

## P1-h07 Neural correlates of antisaccades in the primate motor thalamus

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Antisaccade task has been used to investigate the neural mechanisms of volitional motor control. In this task, subjects are required to make a saccade to an unmarked location opposite to a flashed stimulus. Previous studies demonstrated that neurons in the supplementary eye field (SEF) and the basal ganglia showed greater activity before antisaccades than prosaccades, while neurons in the superior colliculus (SC) showed less activity for antisaccades than prosaccades. Considering these observations and recent imaging studies, we postulated that cortical-subcortical network through the motor thalamus is essential for both the preparation and execution of antisaccades. We so far recorded from 68 single neurons in the VA/VL thalamus when two monkeys performed the pro/anti saccade tasks. The activity of more than one third of individual neurons was statistically different between the tasks, and the population as a whole showed a significant preference for antisaccades. These results suggest that the basal ganglia-thalamo-cortical circuitry plays roles in the generation of antisaccades.

KAKENHI, PRESTO

## P1-h08 Influence of rhythm perception on finger tapping

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When listening to music, we often spontaneously synchronize our body movement to rhythm. Generally, rhythm is what has a temporal rule, and we could perceive rhythm from a sequence of equally-spaced tones such as metronome sounds. In the present study, we investigated the effect of tone sequences on a finger-tapping task in which subjects were required to synchronize their tapping to the sequences. The onset of tapping in response to equally-spaced tones was faster than the onset of a tone. Subjects seemed to perceive the rhythmic structure of the equally-spaced tones and estimated when they would hear the next tone. The subjects were then required to do the similar tapping task with a tone sequence in which the first tone of each triplet was louder than the others; we intended the subjects to perceive the rhythm of triple times. Tapping in response to the louder tones were stronger than those to the other tones. The results suggested that the perception of a rhythm pattern, which has regular level changes, influenced the way of finger tapping.

CREST,JST

## P1-h09 Selective impairment of reward-based adaptive choice of actions by intra-striatal injection of dopamine D1 receptor antagonist

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We showed that projection neurons in monkey striatum learn to encode action-specific reward values based on the reward prediction errors in several trials previously experienced (Samejima et al., 2005). To know the involvement of dopamine in reward-based adaptive choice of actions, we injected dopamine D1 (SCH23390, 10μg/μl, 1.6-3.2μl) or D2 receptor antagonist (Eticropride hydrochloride, 6μg/μl, 1.6-3.2μl) in the striatum of 2 monkeys performing a free-choice task between left- and right-handle turns for probabilistic reward. When the D1 antagonist was injected, monkeys had difficulties to choose higher-valued actions constantly by fluctuating between the two actions. In contrast, when D2 antagonist was injected, such effects were not observed. The effects of D1 antagonist were prominent when injected in the middle rostro-caudal level of the striatum. These results suggest that dopamine and D1 receptor activation in the striatum play indispensable roles in reward-based adaptive choice of actions.

KAKENHI(17022032, 1800383)

## P1-h10 Behavioral analysis of Japanese monkeys during a numerosity-based operation task

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Behavioral studies in primates have suggested the existence of two distinct elements in numerical system: approximate and precise numerical system. Although the approximate numerical system has been well studied in monkeys, it is still controversial how the precise numerical system is operated in the monkeys. To investigate this issue, we trained two monkeys to perform a numerosity-based operation task. We examined the effect of numerosity on the performance rate and the reaction time from the perspective of the object index theory, which was proposed to account for the precise number system. As a measure of numerosity, we calculated the total number of object index used in our task. We found that two distinct number systems exist in macaques with the critical object index number of 4.

## P1-h11 Pop-out of gaze

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When a gaze directed toward us exists in our visual field, our eyes are frequently and automatically captured by such a gaze. This implies that we analyze information embedded in gazes before our attention becomes fully engaged with it. Direction of gaze is an important, socially communicative signal between humans. Previous studies have shown that human visual processing is composed of an early, capacity-independent preattentive stage and a late, capacity-dependent attentive stage. We can know whether a visual feature mainly relies on preattentive or attentive processing by a visual search diagnostics in which subjects are instructed to seek out a discrepant stimulus from a matrix of visual stimuli. In this study, using visual search diagnostics, we report that a gaze directed toward some subjects could be *popped out* of a matrix of averted gazes, implying that, for some subjects, information about gaze direction is processed in the capacity-independent preattentive stage. This finding highlights the importance of the social meanings which are embedded in visual stimuli even for early-stage naturalistic visual processing.

KAKENHI (16500260; 18020012)

## P1-h12 Transition of goal representation of putative pyramidal neurons and interneurons in the primate prefrontal cortex

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To investigate functional difference between cell-types in the lateral prefrontal cortex(LPFC), we examined temporal pattern of activity and fluctuation in spike trains of putative pyramidal cells (PNs) and inhibitory interneurons (INs) ,while monkeys are engaged in a path-planning task. In this task, the animals were required to plan multi-step of movements to each final goals. During a delay period, we found that representation of final goals was gradually shifted to that of immediate goals or the 1st goal. We compared temporal pattern of activity of putative PNs and INs and found that PNs showed earlier transition of representation of goals than INs. Furthermore, temporal variance was significantly higher during the delay period than during the initial control period. These findings suggest that two cell-types in the LPFC show different contribution to the transition of goal representation.

## P1-h13 Cortical mechanism of positive self-concept : an fMRI study

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The aim of this study is to identify the cortical mechanism of self-concept. Twenty seven subjects were participated. Written informed consent was obtained from each subject. The adjective stimuli describing personal trait were shown and the participants were asked to judge whether the adjective describes him/herself. After the measurement,they were asked to evaluate the degree of its preferability of the adjective. Medial frontal gyrus was activated in conventional subtraction, self judgment VS positive/negative judgment. Significant correlation between cortical activation and self-evaluated positive self-concept was observed in light postcentral gyrus, and no significant correlation for negative self-concept.

## P1-h14 Self and other's body part representation in parietal visuo-tactile bimodal areas

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The ability to recognize other's action plays an important role in social life of primates. Recent cognitive neuroscience studies implied that the brain can use self body image as a framework for understanding others' action. Some human imaging studies suggested that self and other's action representation was represented in the posterior parietal cortex. However, physiologically it is still unclear how other's body parts are encoded in the parietal cortex. We reported that some visuo-tactile bimodal neurons in area VIP had receptive fields <RFs> on the monkey's face, showed visual response during the monkey's observation of the experimenter was being stimulated on corresponding face part. Here, we extended recording sites including area 7b to find representation of other's body parts by visuo-tactile bimodal neurons. The results showed some of 7b bimodal neurons had visual RFs on the experimenter's forearm or fingers tip like of VIP neurons. The location of RFs on both self and other's body strictly matched. We will discuss these findings and relate it to existing ideas about social cognition.

KAKENHI (18047026)

## P1-h15 Choosing a smaller number or a larger number in the number comparison task elicited different responses in the parietal region -A NIRSstudy-

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"Number comparison task" was often used for the study of neural mechanism of number processing. In this task, two numbers are displayed and usually, subjects have to select a larger one. Behavioral study showed the distance between 2 numbers was crucial for response time and fMRI study showed the involvement of the parietal cortex in this task. By using this task, we studied the effect of choosing a larger or a smaller number on the reaction time and neural activity change by NIRS. The results showed significantly longer reaction time and the bigger change of $HbO_2$ level in the parietal region when the difference between two numbers was small. The consistency of NIRS results with previous fMRI studies validated the use of NIRS. Our new finding is that when subjects had to select a smaller number, significantly longer reaction time and a bigger $HbO_2$ change in the parietal region were observed. This result indicates that human has tendency to choose a larger number and parietal cortex is involved in this process.

COE from MEXT Japan

## P1-h16 Neural activity in the human brain involved in strategic rule identification

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To investigate brain regions involved in identifying behavioral rules under uncertain situations, we measured brain activation by using functional magnetic resonance imaging, while participants performed a rule-based two-choice task. In this task, subjects were required to identify abstract behavioral rules by testing the validity of two potential rules. After the discovery of an appropriate rule, subjects selected one of two choice cards in conforming to that rule. We analyzed the brain activity focusing on the time period that corresponded to the identification of the behavioral rule based on the judgment of either positive or negative feedback signals to subjects' response. We found active foci involved in rule-identification in the posterior medial frontal cortex and the striatum.

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## P1-h17 Cue-period activity of monkey prefrontal neurons during a duration-discrimination task

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The prefrontal cortex (PFC) is one of areas involved in time perception; however, roles of this area remains unclear. We recorded single neuronal activity from the PFC of two monkeys while they performed a duration-discrimination task. In the task, two visual cues (squares in blue or red) in different durations were presented consecutively followed by delay periods and the subjects then chose one of the cues presented longer in duration. We focused analysis on the first cue (C1) activity, because the neuronal activity of the second cue period (C2) may carry temporal information of both the C1 and C2. We found that prefrontal neurons showed activity that was related to temporal processing. For instance, a group of neurons exhibited phasic responses after constant intervals from the C1 onset. Our results suggest that the PFC contributes to compare current C1 durations with template duration that is learned after repeated trials, due to sort the C1s into possibly long or short cues in the present task.

KAKENHI (18500314)

## P1-h18 Effect on neurocognition and mental fatigue after BCAA administrations

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Mental fatigue, a functional disturbance of the central nervous system, causes change in neurocognition. We observed a decreased performance in spatial learning and exercise in rats injected with L-tryptophan. Interestingly, rats injected with tryptophan, but subjected to musical stimuli increased the time to exhaustion, compared with a group subjected to white noise. The water maze performance test, after administration of muscimol, was effective in fatigued rats. In humans, to investigate the prevention of mental fatigue, sensation of fatigue was evaluated by scoring the fatigue-related items of the Profile of Mood States instrument. A mental fatigue task, used in Uchida-Kraepelin's arithmetic, a test of continuous addition for 30 min used in neuropharmacological studies. We observed a significant improvement in the group that received BCAA cf.placebo. Our findings demonstrate that mental fatigue can be diminished by inhibiting the system L-amino acid transporter for uptake of tryptophan. Also, it is tempting to speculate that spatial memory is enhanced through GABAergic visual function. This work was supported by Tezukayama Gakuen.

## P1-h19 Neural network model for visual reconstruction using the morphoelectrotonic transform theory

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In our cognition, mutual circulation with internal model on external world of our situation has been caused based on present input information. To clarify the integration of such information, neural network model with dynamical changed variously and flexibly becomes important.We construct the model of the visual reconstruction from input information. The relations between objects are simulated from the correlation of input pulse and associative memory. This associative memory is composed of the synchronous associative memory network, and extracts the relations between objects by the phase obtained from input pulse sequence.And, the relation and the bind between objects are characterized by the morphoelectrotonic transform (Zador, 1995). Binding of the object is reconstructed the relation of the associative memory, and the correlation of the object's modality is learned.In this simulation, the visual reconstruction of the modality between three objects, the difference of time of input object and the input order were able to be shown by the interval of the pulse sequences of the color.

## P1-h20 Neural representation of information measures in the monkey frontal cortex

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Humans behave so as to gain greater information. Information theory and experimental psychology propose different measures for information value: Shannon information, probability gain, and absolute impact. To examine neural representation of information value, we tested the three information measures by recording neural activity of monkeys. They were trained to perform tasks that provided them with different values of information about reward location. Results: The monkeys almost always chose the most informative alternative. During performing the tasks, the neural activities showed significant proportionality to Shannon information and probability gain. The proportionality was greater with the former than with the latter. These suggest that information value may be dominantly represented by Shannon information in the brain.

## P1-h21 Early hierarchical syntactic processing in Broca's area for merging words

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The current linguistic theory assumes a syntactic merging operation, which forms, when applied to an object (noun phrase, NP) and a transitive verb (V), a new higher-order hierarchical level (verb phrase, VP). However, it remains to be elucidated how such syntactic processing is related to the neural dynamics in a cortical region. Using MEG, we measured brain activity from 10 subjects during each of four tasks: syntax, semantics, evaluation, and memory tasks. Sentence stimuli consisted of NP and V (transitive or intransitive). We mapped the temporal activity using minimum norm estimates, which were corrected for multiple comparisons with permutation tests ($P < 0.05$). Under the transitive-verb condition, but not under the intransitive-verb condition, the activity was selectively enhanced in the syntax task as early as 130 ms after the verb onset, and it was localized in the left inferior frontal gyrus (area 45). These results suggest that Broca's area is engaged in the early preparatory processing of hierarchical syntactic structures.

CREST, JST

## P1-h22 Chronic treatment of methylphenidate injures behavioral/cognitive functions by degeneration of dopaminergic neuron in rats

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Methylphenidate (MP) is a commonly used drug to treat ADHD. In human and animal studies, MP exerts similar rewarding and discriminatory effects like as other stimulants including cocaine and methamphetamine. However little is known about the long-term effects of MP on the neural functions. We here assessed whether chronic MP treatments (1 or 3 mg/kg, 2 weeks, p.o.) affect on the vulnerability of dopamine (DA) neurons and behavioral/cognitive function in rats. Immunoblot analysis revealed that DAT significantly decreased in a dose-dependent manner in the mPFC. It was also verified that TH immunoreactive neurons decreased in the VTA following chronic MP treatment. Furthermore, we found that MP-treated rats deficit cognitive function measured by novel object recognition task. These results suggest that chronic MP treatment induces dysfunction of DA neurons, resulting in deficits of behavioral/cognitive brain functions.

Tohoku University 21st Century COE Program `CRESCENDO`

## P1-h23 Hemispheric differences in the activation patterns in the infant brain during auditory stream processing

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We used multi-channel NIRS to investigate how the infant brain processes auditory streams. We prepared three types of tone sequences as stimuli; tone1: random sequences, tone4: four successive tones construct a chromatic scale, and tone12: twelve successive tones construct a chromatic scale. In a single trial of each condition, 96 tones in a chromatic scale from C4 to C6 were presented (50 ms per tone). Participants were quietly sleeping 3- to 4-month-olds (n = 26). First, we found bilateral activation in temporal regions under all conditions. One-way ANOVAs among conditions revealed functional differentiation between the hemispheres. The right temporoparietal region showed greater activation if the pitch processing load increased (tone1 > tone4, tone12), whereas the left homologous region exhibited greater activation if the auditory segregation load increased (tone1, tone12 > tone4). Our findings suggested that distinct and cooperative processes in the left and right hemispheres of infants perform auditory stream segregation using pitch information.

CREST/JST

## P1-h24 Cerebral clearance of human amyloid-β peptide (1-40) across the blood-brain barrier is reduced by complex formation of activated α2-macroglobulin

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Low-density lipoprotein receptor-related protein-1 (LRP-1) ligands such as activated α2-macroglobulin (α2M*) have been reported to act as a carrier of the LRP-1 mediated Aβ clearance by in vitro studies. The present study investigated the in vivo effect of the α2M* complex on human Aβ(1-40) (hAβ(1-40)) elimination from the rat brain across the blood-brain barrier (BBB). Pre-incubation with α2M* reduced the elimination of [$^{125}$I]hAβ(1-40) compared with that of [$^{125}$I]hAβ(1-40) without pre-incubation, while pre-administration of α2M* in the rat brain did not significantly inhibit at the same concentration. The elimination rate of purified [$^{125}$I]hAβ(1-40)/α2M* complexes was reduced by 85.0% compared with [$^{125}$I]hAβ(1-40) monomers. [$^{125}$I]α2M* was not eliminated from the rat brain, suggesting that LRP-1 does not play a major role in the brain-to-blood efflux transport at the BBB. These findings indicate that α2M* complex formation reduces the brain-to-blood efflux clearance of hAβ(1-40) at the BBB.

## P1-h25 The roles of X11 and X11L in APP metabolism

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X11 (X11α) and X11L (X11β), neuron-specific adaptor protein, associate with the cytoplasmic domain of amyloid β-protein precursor (APP) and suppress APP metabolism. Altered metabolism of APP is strongly implicated in Alzheimer disease pathogenesis. Although we have produced X11L-deficient mice and reported the facilitation of β-secretase cleavage of APP in these mice (J. Biol. Chem. [2006] 281, 37853), the functions of X11 and X11L in APP metabolism are still unclear in detail. Present study mainly focused on three aspects to resolve these issues: 1) role of endogenous X11 in endogenous APP metabolism regulation; 2) functional divergence between X11 and X11L; and 3) effect of X11 and X11L-double deficiency on metabolism and subcellular localization of APP. To elucidate these issues, we produced X11/X11L- double deficient mice and analysed phenotypes in vivo and in primary cultured neurons of these mutant mice.

## P1-h26 Novel intracellular domain fragment of Alzheimer amyloid precursor protein (AICD) via γ-secretase γ-site cleavage

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Accumulation and deposition of fibrillar Aβ is considered the primary cause of Alzheimer's disease. Aβ is derived from Alzheimer amyloid precursor protein (APP) by sequential proteolytic cleavage involving β- and γ-secretase. Recently, γ-secretase was shown to cleave near the cytoplasmic membrane boundary of APP (termed ε-site cleavage), as well as in the middle of the membrane domain (termed γ-site cleavage). The relationship between γ-site and ε-site cleavage is still unclear. In this study, to elucidate the relationship between γ-site and ε-site cleavage, the generation of AICD in membrane fractions of cultured cells was analyzed. We found that a novel AICD was generated via direct γ-site cleavage and was rigidly associated with the membrane. These results indicate that γ-site and ε-site cleavage are regulated differently and that Aβ is directly generated via γ-secretase γ-site cleavage.

a grant for the Intractable Diesease Division, the Ministry of Health and Welfare in Japan

## P1-h27 Effects of chronic treatment with donepezil via α7-nicotinic receptor and phosphatidylinositol 3-kinase cascade

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Previously, we showed that chronic donepezil treatment (10 μM, 4 days) up-regulates nicotinic receptors (nAChRs), facilitates nicotine-induced $Ca^{2+}$ influx and sensitizes the neurons to the neuroprotective action of donepezil in primary culture of rat cortical neurons. Here we show that concomitant chronic treatment with donepezil and α7-nAChR antagonist or inhibitors of kinases of the phosphatidylinositol 3-kinase (PI3K) pathway inhibited the α7-nAChR up-regulation and sensitization to the neuroprotective effect of donepezil. Furthermore, concomitant treatment with donepezil and α7-nAChR antagonist suppressed the marked facilitation of the nicotine-induced $Ca^{2+}$ influx. Our results suggest that α7-nAChR and PI3K pathway play significant roles in mediating the effects of chronic donepezil treatment.

KAKENHI(18890220)

## P1-h28 Analysis of lipoprotein in cerebral spinal fluid (CSF) of Alzheimer's disease (AD) patients using HPLC

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We present lipoprotein analysis of CSF of patients with AD using HPLC. Samples were taken from in-patients with their written consent or that of their families. 74 samples were analyzed including 37 AD and other diseases (aged 79.0±10.0). CSF was obtained by lumbar puncture and was kept at 4 °C. A 200 µl aliquot of CSF was separated by gel filtration on two tandem connected TSKgel LipopropakXL columns, and proteins, cholesterol and phospholipids were monitored using on-line reaction HPLC system. LDL-C in CSF of the AD, FTD and cerebral hemorrhage (CH) groups was higher than that of control, although TC in CSF, HDL-C in CSF was not. The high LDL-C in CSF level in AD, FTD and CH samples suggested breakdown of the blood brain barrier and we will investigate the diagnostic implications.

## P1-h29 N-cadherin enhances extracellular secretion of A $\beta$

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We have previously reported that N-cadherin-mediated cell-cell contact promotes cell-surface expression of PS1/γ-secretase. In the present report, we evaluated whether N-cadherin-based contact modulates A $\beta$ production via affecting subcellular distribution of PS1/γ-secretase. Here, we demonstrate that the inhibition of N-cadherin-mediated cell-cell contact in primary neurons reduces extracellular release of A $\beta$. Conversely, stable expression of N-cadherin into Chinese Hamster Ovary cells, expressing Swedish mutant of human amyloid precursor protein led to enhanced A $\beta$ secretion. Moreover, expression of deletion mutant PS1, lacking interaction domain with N-cadherin, reduced the secretion of A $\beta$ species, compared to the expression of wtPS1. These results indicate that N-cadherin-mediated synaptic contact modulates subcellular distribution of PS1/γ-secretase, as well as A $\beta$ secretion via PS1/N-cadherin interaction.

## P1-h30 A screening for novel components of gamma-secretase complex using by in vivo crosslink method

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It is well known that Amyloid beta protein (A-beta) accumulates in Alzheimers Disease (AD) brain, and is involved in the pathogenesis of AD. Gamma-Secretase cleaves the Amyloid Precursor Protein (APP) in its transmembrane domain, generating the A-beta. The Gamma-Secretase is a multiprotein complex composed of Presenilin, Nicastrin, Aph-1, and Pen-2 and that all four proteins are necessary for proteolytic activity of APP. However, recent work indicates that enzymatically active gamma-secretase complexes have a mass (650 kDa) that is much greater than the predicted mass for a 1:1:1:1 stoichiometric complex. Here, to clarify whether there are novel components of gamma-secretase complexes, we are performing immunoprecipitation study for presenilin (and other gamma-secretase comlex related protein)-interacting proteins using in vivo crosslinking method.

## P1-h31 Early onset of microglial dystrophy in SAMP10 mice

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Microglia in normal aging human brains undergo morphological alterations referred to as 'microglial dystrophy'. However, dystrophic alterations in microglia have not been well documented in aging mice. We investigated potential changes in microglial morphology using SAMP10 strain as a mouse model of accelerated aging with cerebral degeneration in comparison with normal control SAMR1. Brain sections from SAMP10 (1, 3, 8, and 14 months) and SAMR1 (1, 3, 14 and 24 months) mice were stained with anti-Iba-1 antibody for labeling microglia. Microglia of SAMP10 had dystrophic morphology such as shortened processes, deramification, fragmentation and spheroidal bulbs at 3 months, which became more remarkable with aging. Ramification of microglia in SAMR1 was much better preserved throughout life. The SAMP10-specific alterations in microglial morphology might be related to functional loss in tissue surveillance activity, resulting in earlier onset of neuronal degeneration in SAMP 10.

KAKENHI(18590396)

## P1-h32 Accelerated release of exosome-associated GM1 ganglioside by endocytic pathway abnormality

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Endocytic pathway abnormalities were previously observed in neurons of brains affected with Alzheimer's disease (AD). In this study, we treated PC12 cells with chloroquine, an acidotropic agent, to clarify the relevance of these abnormalities to amyloid $\beta$-protein (A $\beta$) assemblies. Chloroquine treatment induced GM1 accumulation in the endosomes. Furthermore, KCl-induced depolarization following chloroquine treatment induced an increase in GM1 level in cell medium. Sucrose density gradient fractionation revealed that GM1 in the medium was recovered in fractions of exosomes, membranous vesicles that are secreted from cells. Notably, A $\beta$ assembly was accelerated in the presence of the exosomes. The co-incubation with an antibody against GM1-bound A $\beta$, a seed for AD amyloid, suppressed the A $\beta$ assembly. These results implicate novel significance of endosomal abnormality in GM1-induced A $\beta$ fibrillogenesis of AD brain.

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## P1-h33 Effects of Syntaxin5 isoforms on the accumulation of intracellular $\beta$APP and the secretion of A $\beta$ peptides

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Syntaxins (Syx) are involved in trafficking of various membrane proteins. Among them, Syx5 has another isoform containing extra N-terminal sequence (designated as Syx5L). We have previously shown that, Syx5 and Syx5L specifically interact with presenilin (PS) holoproteins. Syx5 overexpression resulted in the intracellular accumulation of $\beta$-amyloid precursor protein ($\beta$APP) and a reduction in the secretion of $\beta$-amyloid peptide (A $\beta$). In order to examine the difference in the effect of Syx5 isoforms on the processing $\beta$APP, we have constructed a series of Syx5L mutants. When Syx5L was overexpressed, the extent of accumulation of intracellular $\beta$APP holoprotein was lower than those of Syx5. While overexpression of wild type Syx5L did not inhibit the secretion of A $\beta$, Syx5L deletion mutants inhibited the secretion of the peptides as in Syx5. We present here the difference in the action between Syx5 isoforms and discuss the molecular bases for the modification of $\beta$APP processing.

KAKENHI (16700327)

## P1-h34 Reduced expression of the 6B4-epitope in perineuronal nets of the cerebral cortex of SAMP10 mice

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The accelerated senescence-prone SAMP10 strain of mice is a model for age-dependent neurodegeneration, which is characterized by brain atrophy and deterioration in performance of learning and memory tasks. Since perineuronal net is supposed to play important roles in synaptic plasticity, we examined perineuronal net-components in SAMP10 mouse brain. Immunohistochemical staining using a monoclonal antibody 6B4 (MAb 6B4) showed distinct staining of perineuronal nets surrounding neurons in the cortex of control mice, whereas there was quite faint immunostaining in the cortex of SAMP10 mice. Western blot analysis revealed that expression of the 6B4-epitope on aggrecan core protein was markedly reduced in SAMP10 mice. The biological significance of 6B4-epitope on brain function and its relationship to neurodegeneration in SAMP10 mice remain to be elucidated.

## P1-h35 Implication of matrix metalloprotease-9 in cognitive impairment induced by i.c.v. injection of A $\beta$ in mice

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In Alzheimer disease, the expression of matrix metalloproteases (MMPs), which are capable of degrading extracellular matrix proteins, is increased in the brain. Previous studies have demonstrated that amyloid beta protein (A $\beta$) can induce MMPs expression in cultured glial cells. In the present study, we investigated the role of MMP-9 in cognitive impairment induced by i.c.v. injection of A $\beta$ in mice. The i.c.v. injection of A $\beta$ 25-35 and A $\beta$ 1-40, but not A $\beta$ 40-1, increased MMP-9 activity and protein expression in the hippocampus. The A $\beta$-induced cognitive impairment was significantly alleviated in MMP-9 homozygous knockout mice compared with wild-type mice. These results suggest that the increase in MMP-9 expression in the hippocampus may be involved in the development of cognitive impairment induced by A $\beta$ 1-40.

## P1-h36 Unique GM1-ganglioside-enriched microdomain in synaptosomes prepared from aged mouse brain

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Aging is a strong risk factor for the development of Alzheimer's disease (AD). However, its pathological roles remain to be clarified. In the process of AD development, the conversion of the nontoxic amyloid $\beta$-protein (A $\beta$) monomer to its toxic assemblies is a fundamental process. We have recently reported that a toxic soluble A $\beta$ assembly is formed in the presence of synaptosomes (SPSs) prepared from aged mouse brains in a GM1 ganglioside (GM1)-dependent manner. In the present study, we report that the methyl-$\beta$-cyclodextrin-insensitive GM1-enriched microdomains are formed with age in SPSs. We also show that A $\beta$ assembly is markedly accelerated in the presence of SPSs prepared from the aged mouse brain. These results suggest that aging accelerates A $\beta$ assembly in the brain through formation of unique GM1-enriched microdomains in synaptic plasma membranes.

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## P1-h37 Somatostatin-immunoreactive degenerating structures in prefrontal cortex of aged tree shrews (Tupaia) and macaque monkeys

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In our previous study, expression of somatostatin (SS) mRNA was decreased in the cerebral cortex of aged macaques (Hayashi et al., 1997). To investigate changes in SS neurons, we applied immunohistochemistry of SS in the prefrontal cortex of aged tree shrews and macaques. We observed degenerating SS-immunoreactive (-ir) cells and SS-ir tangle-like structures in area 8 of macaques where beta-amyloid plaque existed (Hayashi et al., 2006), although there were no SS-ir tangle-like structures in areas 46, 9, 10, 6 and 24 of macaques and tree shrews. SS-ir tangle-like structures may be related to beta-amyloid plaque and impairment of cognitive functions during aging process.

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## P1-h38 In vivo detection of amyloid deposits in Alzheimer disease patients using [$^{11}$C]BF-227

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In vivo detection of amyloid deposits using PET would thus prove useful for early diagnosis of Alzheimer disease (AD) and tracking disease progression. The purpose of this study is to evaluate the diagnostic utility of a novel PET imaging probe, a benzoxazole derivative (BF-227), in the patients with AD. The clinical PET study using [$^{11}$C]BF-227 demonstrated the retention of this tracer in cerebral cortices of AD patients, but not in those of normal subjects. All AD patients were clearly distinguishable from normal individuals using temporal SUV ratio. Cortical BF-227 retention in MCI subjects appeared either AD-like or control-like. Patient with frontotemporal dementia (FTD) showed no cortical retention of BF-227. These findings suggest that [$^{11}$C]BF-227 is a promising PET probe for in vivo imaging dense amyloid deposits in AD patients.

Kouseikakenhi (H18-nano-11)

## P1-h39 Impact of proteasomal inhibition and formation of ubiquitinated inclusions on cultured neurons from SAMP10 mice

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SAMP10 mouse (P10) is a model for age-associated neurodegeneration. We have reported an age-related increase in neuronal cytoplasmic ubiquitin-positive inclusions (UPIs) and concomitant decrease in proteasomal activity in the limbic-related structures of P10 brain. To study the effect of decreased proteasomal activity and formation of UPIs on neuronal survival and morphology, we performed in vitro proteasome inhibition experiment using cultured cortical neurons from P10 and normal aging control SAMR1 mice (R1). Significant cell death occured 24 h after the addition of 1-10 μM MG115, a potent proteasome inhibitor, with no strain difference. UPIs, formed by exposing cells to 3 μM MG115 for 24 h, were more frequently observed in P10 neurons than in R1 neurons. Morphometric analysis revealed that UPI-bearing neurons had fewer long (>50 μm) neurites when compared to those without UPIs. UPIs may exert adverse effects on the maintenance of dendritic arbors.

## P1-h40 Change of N-glycan structures of amyloid precursor protein in familial Alzheimer's disease

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The N-glycans of human amyloid precursor protein (APP) and two APPs with familial mutations produced by mouse C17 cells were released quantitatively as oligosaccharides by hydrazinolysis. After being reduced with 2-aminobenzamide, their structures were analyzed comparatively. The results demonstrated that APP has bi- and tri-antennary complex type N-glycans with fucosylated and nonfucosylated trimannosyl cores. In comparison with normal human APP, both mutants showed higher contents of bisecting N-acetylglucosamine and core-fucose residues. These results suggest that change of glycosylation occurs in familial mutant APPs, and the processing of APP may be affected via the structure of its N-glycans. It is also possible that change of glycosylation of APP induces the altered metabolism of APP in sporadic Alzheimer's disease patients.

## P1-h41 Vulnerability to excitotoxic insults and defective microglial response in SAMP10 mouse

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Microglia may play a pivotal role for neuroprotection. We studied glial responses to excitotoxic insults in SAMP10 mice (P10), a model of age-related cerebral degeneration and normal control SAMR1 mice (R1). Three-month-old P10 and R1 received kainate (KA) intraperitoneally. Three days later, brains were stained with cresyl violet and immunohistochemically (Iba1). Hippocampal damages were graded as Grade 3 (widespread neuronal death), 2 (focal neuronal death) and 1 (no cell death but only glial response). Of 7 KA-treated P10, one, five and one had Grade 1, 2 and 3 lesions, respectively. All 10 KA-treated R1 had Grade 1. Many microglia had hypertrophic somata and bushy processes in Grade 1 lesions of R1. The cell-to-cell contact with neurons was enhanced in these hypertrophic microglia. Microglia in Grade 1 and 2 lesions of P10 lacked hypertrophy or bushy processes. The defective response of microglia may be causally related to neuronal vulnerability in P10.

KAKENHI18590396

## P1-h42 Effect of Semi-fluid diet breeding on the hippocampal dopamine release

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Using the intracerebral microdialysis technique, we examined whether the semi-fluid diet breeding is involved in the dopaminergic neuronal systems in the rat hippocampus. The hippocampal dopamine release under the high $K^+$ condition was analyzed in four groups: 1) Normal group fed with a hard food, 2) Normal group fed with soft food, 3) AD group (infusing the Beta-amyloid protein) fed with hard food, 4) AD group fed with soft food. In all groups, increases in the extracellular levels of dopamine (DA) were seen; however, the DA levels of the normal and AD groups with soft food were significantly lower than those of hard food groups. The data suggest the link between hardness of food and hippocampal DA processes.

## P1-h43 Aprataxin, the causative gene product for EAOH/AOA1, repairs damaged 3'-ends of DNA single strand breaks

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BACKGROUND: Aprataxin, the causative gene product for EAOH/AOA1, is a member of the histidine triad family of nucleotide hydrolases. Although aprataxin has been proposed to be involved in DNA single-strand break repair, its physiological role remains elucidated. METHODS: We produced recombinant human aprataxin and reacted various DNA substrates with aprataxin. The products were analyzed by denaturing 20% PAGE. Next, we quantified DNA damage induced by DNA-damaging agents in cerebellar neurons from wild-type and APTX-/- mice, by using comet assay. RESULTS: Aprataxin removed damaged 3'-ends including phosphate and phosphoglycolate, and disease-associated mutant forms of aprataxin lost the activity. After exposing to H2O2 or bleomycin, DNA damage in APTX-/- cells was significantly more increased than that in wild-type cells. These data indicate that aprataxin removes the damaged 3'-ends of SSBs and repairs SSBs induced by H2O2 or bleomycin in neurons.

## P1-h44 A drosophila PQBP-1 mutant is a novel model for mental retardation

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Recently, it has been reported that mutations of polyglutamine tract binding protein-1 (PQBP-1) are responsible for mental retardation. We found a piggyBack inserted fly is hypomorph mutant of PQBP-1. We examined whether the PQBP-1 mutant shows a defect in learning and memory and other phenotypes. Memory acquisition but not maintenance was disrupted during a classical olfactory conditioning in a PQBP-1 mutant fly. We observed loss of projection neurons which are related to early phase memory. Life-span of the mutant was shorter than wild type. The mutant fly showed enhanced courtship behavior, and a little disorder of circadian rhythms. Moreover, expression of exogenous PQBP-1 rescued the learning defect. Then, we conclude that the mutant is a novel mental retardation model. Next, we screened chemicals to recover the phenotypes of the mutant. We report results of the chemical test.

## P1-h45 Effects of mutant γPKC found in SCA14 on the nature of primary cultured Purkinje cells

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Recently, missense mutations have been identified in protein kinase Cγ (γPKC) gene in several families of spinocerebellar ataxia type 14 (SCA14), an autosomal dominant neurodegenerative disorder. We have previously demonstrated that these mutant γPKC is susceptible to cytoplasmic aggregation and causes cell death in CHO cells, indicating that this property is involved in the etiology of SCA14. In the present study, we examined how mutant γPKC affect the nature of Purkinje cells (PCs), which are degenerated in SCA14 patients, in mouse cerebellar primary culture. Mutant γPKC-GFP frequently formed aggregates in PCs. These aggregates were ubiquitin-positive and colocalized with proteasome. Moreover, mutant γPKC-GFPs significantly reduced the dendritic areas and the density of dendritic spines of PCs. These results indicate that mutant γPKC not only forms aggregates but also affects the dendritic morphology and the synaptic formation in PCs.

KAKENHI17700351

## P1-h46 Possible roles of LC3, ubiquitin, and p62 in the accumulation of lysosomes in neurons deficient in cathepsin D

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We have shown that autophagy contributes to the accumulation of lysosomal structures such as granular osmiophilic deposits (GRODs) in CNS tissue of cathepsin cathepsin D deficient (CD-/-) mice, a mouse model of neuronal ceroid lipofuscinosis (Koike et al., 2000, 2005). Using immunoelectron microscopy we found that ubiquitin, together with p62/A170/SQSTM1, a polyubiquitin binding protein, was localized on the surface membrane of GRODs in CD-/- neurons. Since P62 has been shown to interact also with LC3, a marker protein of autophagosome (Bjorkoy et al., 2005), we confirmed the co-existence of these three molecules. Based upon these findings we propose a model whereby p62 interacts with ubiquitin, as aggregates in the cytosol, on the membrane of abnormal organelles such as GRODs, and further interact with LC3 to degrade them by forming autophagosomes.

KAKENHI (17790141, 18021019)

## P1-h47 Development of a mouse model of sporadic ALS by deficient RNA editing

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Deficient RNA editing of GluR2 mRNA at the Q/R site has been proposed to be highly relevant to neuronal death in sporadic ALS. RNA editing at this site is specifically catalyzed by adenosine deaminase acting on RNA type 2 (ADAR2). To elucidate whether loss of ADAR2 induces neuronal death, we developed genetically modified mice that had both floxed ADAR2 gene and Cre gene with a motor neuron selective promoter. The resultant mutant mice developed progressive motor decline with degeneration of motor neurons, indicating that ADAR2 underactivity is likely the cause of GluR2 underediting in motor neurons of sporadic ALS.

KAKENHI17390251

## P1-h48 Global analysis of gene expression in glial cell transfected with mutant *GFAP*

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We have reported that mutant *GFAP* could induce functional abnormalities of astrocytes. To reveal the candidate for factors that modify aggregation of GFAP, we investigate gene expression of glial cell transfected with mutant *GFAP* using DNA microarray analysis. Total RNA was extracted from astrocytoma-derived cells transfected with three different mutant *GFAP* (Val87Gly, Arg88Cys, Arg416Trp) as well as wild-type *GFAP*, and gene expression profiles were investigated using an oligonucleotide microarray. Using hierarchical clustering analysis, we picked up 23 and 22 probe sets for which expression had increased and decreased, respectively, in mutant GFAP cells compared with wild-type GFAP. Among the clusters, interestingly, the expression of neprilysin was decreased in mutant GFAP cells. Neprilysin, which could be concerned with axonal transport or affect catabolism of some peptides, might be a potential candidate for the factors that modify aggregation of GFAP.

## P1-j01 Establishment of Hela cell lines suitable for the measurement of GluR2 RNA editing efficiency using Tet-on system

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We have recently demonstrated that deficient RNA editing of GluR2 mRNA at the Q/R site due to reduced activity of its catalytic enzyme adenosine deaminase acting on RNA type 2 (ADAR2) is the most likely cause of death of spinal motor neurons in sporadic ALS. Therefore, it seems likely that an enhancement of ADAR2 activity may inhibit neuronal death induced by deficient GluR2 RNA editing, and hence the progression of sporadic ALS. We could test substances that have such potency if we had cell lines that stably express half-edited GluR2 mRNA. However, as we could not find any such cell lines, we developed double-stable Hela cell lines that express a part of human GluR2 gene using Tet-on system. These cell lines express a stable GluR2 editing efficiency of 40-60%, hence may be useful for our purpose.

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## P1-j02 Inhibition of mutant huntingtin aggregate formation by Cdk5/p35

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Polyglutamine diseases are caused by expanded CAG repeats encoding polyglutamine residues in the affected proteins and one of the proteins is huntingtin (htt). Mutant huntingtin (mhtt) forms detergent insoluble cellular aggregates, a pathological feature of the disease. Questions are how aggregates form and how their formation is suppressed. It was recently reported that Cdk5/p35, a neuron specific proline-directed Ser/Thr kinase, inhibited aggregate formation of mhtt by phosphorylation. Using exon-1 portion of mhtt, we found aggregate formation was inhibited independent of phosphorylation. The inhibitory effect was also observed with atrophin-1, another affected protein. We searched the common inhibitory mechanism underlying aggregate formation. We will provide evidences indicating that the disruption of the microtubule network mediates the inhibition of aggregate formation by Cdk5/p35.

## P1-j03 Oxidative damage in nucleic acids in Huntington's disease model

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3-Nitropropionic acid (3NPA), which inactivates mitochondrial succinate dehydrogenase, has been used for studying the Huntington's disease (HD) pathology. The common chorea seen in HD patients may result from the loss of enkephalin- and substance P-containing, medium-sized spiny striatal projection neurons. Ingestion of 3NPA by eating contaminated sugar cane results in basal ganglia lesions and a marked dystonia in children. Similar behavioral and neuropathological changes were found in rodents after systemic administration of 3NPA. Although the striatal damage produced by 3NPA in rodents has been previously described, the mechanism of action of 3NPA is still unclear. In the present study, we found that chronic administration of 3NPA increases oxidative damage in nucleic acids in the spiny striatal neurons in mice, and mutant mice lacking two genes for 8-oxoguanine DNA glycosylase (OGG1) and oxidized purine nucleoside triphosphatase (MTH1) exhibit an increased susceptibility to 3NPA-induced neurodegeneration.

JSPS

## P1-j04 BACE1 cleavage mediates neurite morphology induced by sodium channel β4 subunit

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β-cleavage of amyloid precursor protein (APP) by β-site APP cleaving enzyme 1 (BACE1) is crucial for pathogenesis of Alzheimer disease (AD). Recently, we identified sodium channel β4 (β4) that is significantly downregulated in the striatum of Huntington disease (HD) model mice and patients and found that β4 is a substrate for BACE1. β4 is a member of the IgCAM superfamily and serves as an auxiliary subunit of the voltage-gated sodium channel. To examine the functional roles of β4 processing, Neuro2a cells were transfected with β4 and BACE1. Overexpression of β4 caused neurite extension and increased number of filopodia-like protrusions. Coexpression of β4 and BACE1 extended neurite and decreased number of filopodia-like protrusions compared with β4 expressing cells. These results suggest that BACE1 cleavage regulates β4-mediated neurite outgrowth activity. Our findings may provide new insights into the underlying pathology of HD as well as AD.

## P1-j05 Dysregulation of β4 gene transcription in the striatum of Huntington Disease transgenic mice

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Sodium channel β4 subunit (β4) was significantly downregulated in the striatum of Huntington Disease (HD) model mice. In situ hybridization with β4 probe, followed by immunohistochemistry using anti preproenkephalin (PPE) or anti preprotachykinin A (PPTA) indicated that β4 mRNA is expressed in two groups of striatal neurons projecting to globus pallidus (GP)(marker protein: PPE) and substantia nigra (SN)(marker: PPTA). TaqMan RT-PCR analysis indicated that both β4 and PPE mRNAs are preferentially decreased in striatum at a presymptomatic stage of HD mice, while PPTA mRNA is unaltered even at the symptomatic stage. These results indicate that there is a difference in downregulation of mRNA and its product among striatal projection neuron proteins and suggest that loss of β4 in HD transgenic mice is due to dysregulation of β4 gene transcription.

KAKENHI (18500284 )

## P1-j06 Role of Cys111 in an oxidative damage of human Cu/Zn-superoxide dismutase

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Although human Cu/Zn-superoxide dismutase (SOD1) protects cells against oxidative stress, SOD1 itself may be a major target of oxidative damage and may own pathogenicity in various neurodegenerative diseases including familial ALS. The role of Cys111, reactive free cysteine residue, in the oxidative damage of the SOD1 protein, by comparing the oxidative susceptibility of recombinant human SOD1 modified with 2-mercaptoethanol at Cys111 (2-ME-SOD1) to wild-type SOD1, was investigated. Wild-type SOD1 was more sensitive to oxidation generating more fragments and oligomers compared with 2-ME-SOD1. We further demonstrated that Cys111 is selectively oxidized to cysteine sulfinic acid and to cysteine sulfonic acid. Studies of the role of Cys111 and oxidized SOD1 in the pathophysiology are currently underway.

## P1-j07 Chorein deficiency leads to increase of DARPP-32 protein exprssion.

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Chorea-acanthocytosis(ChAc) is a hereditary neurodegenerative disorder caused by loss of function mutations in the VPS13A gene encoding chorein. Recently, we produced a ChAc model mouse using a gene targeting technique to delete exons 60-61 that corresponds to a human disease mutation. Selective neuronal degeneration was observed in the striatum of the ChAc-model mouse. In the present study, to understand protein expression changes mediated by a chorein deficiency, we used a parallel proteomic analysis of stiatal protein expression in the ChAc-model mouse. We found an increased protein level of DARPP-32 which is a key actor of integraion of neurotransmitter and neuromodulator signals in the striatum. Consistently, immunohistochemical analysis showed that DARPP-32 immunoreactivity was higher in the striatum. Our results suggest that DARPP-32 in the striatum is related to the brain pathology of ChAc.

## P1-j08 Characterization of ubiquitin and polyubiquitin conjugates in brains of Niemann-Pick C disease mouse

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Niemann-Pick type C disease (NPC) is a lipid storage disease characterized by progressive neuronal loss in central nervous system, accompanied by accumulation of ubiquitin-positive materials. To investigate the pathophysiologic processes, we characterized ubiquitin and polyubiquitin-conjugates in NPC brain. Levels of free ubiquitin and polyubiquitin-conjugates in Tris-buffer extracts of brains were not different between NPC and wild-type mice at 4 and 9 weeks of age. However, amounts of polyubiquitin-conjugates in 2% SDS-Tris buffer extracts of brains from the NPC mice were significantly greater than those from the wild-type mice. In addition, these conjugates were not completely solubilized by other detergents including Triton X-100 (1%) and CHAPS (1%) or RIPA buffer, suggesting involvement of abnormal/denatured proteins in their formation. Compositional similarity of the ubiquitin-conjugates in other neurodegenerative diseases will be discussed.

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## P1-j09 Effect of Charcot-Marie-Tooth mutations on filament assembly of neurofilament-L protein

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Charcot-Marie-Tooth disease (CMT) is the most common inherited peripheral nerve disorder. CMT is classified into two types on the basis of nerve conduction velocity, demyelinating CMT1 and axonal CMT2. NF-L gene has been identified as the causative gene for CMT2E. Neurofilaments (NFs) are the most abundant cytoskeletal element in axons and NF-L is the basic component of NFs. We investigated assembly properies of CMT2E mutants such as P8L, P22T and N97S in vitro. We observed the assembled filaments by negative staining and low-angle rotary-shadowing electron microscopy. P8L and N97S mutants formed large irregular aggregate as well as P22T. These results suggest that CMT mutations result in the filament assembly defect that would be a direct cause of the diseases.

## P1-j10 Reciprocal phosphorylation of tyrosine and serine residues of c-Met/HGF receptor in motoneurons implicates HGF-dependent disease retardation of a transgenic mouse model of ALS.

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ALS is a fatal disease characterized by progressive degeneration of motoneurons. We have demonstrated that HGF attenuates loss of both spinal and brainstem motoneurons of ALS model mice expressing mutated SOD1-G93A (G93A). Using double transgenic mice expressing HGF and mutated SOD1-G93A (G93A/HGF), we showed that tyrosine phosphorylation of c-Met, thereby its activation, was evident slightly in G93A and highly in G93A/HGF (but absent in HGF-Tg and WT), while serine phosphorylation of c-Met, negative regulatory residue of its activation, was evident in WT and HGF-Tg. Protein phosphatase 2A (PP2A), that is capable of dephosphorylating phopho-serine of c-Met, is upregulated in motoneurons of WT, HGF-Tg compared with G93A and G93A/HGF. Thus, c-Met activation is reciprocally regulated by phosphorylation between tyrosine and serine residues of c-Met via PP2A induction in the presence or absence of ALS-toxicity, and HGF functions more efficiently when ALS-toxicity is present.

## P1-j11 CHIP over-expression reduces mutant AR protein and ameliorates phenotypes of the SBMA transgenic mouse model

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Spinal and bulbar muscular atrophy (SBMA) is an inherited motor neuron disease caused by the expansion of a polyglutamine tract within the androgen receptor (AR). CHIP (carboxyl terminus of Hsc70-interacting protein), a U-box type E3 ubiquitin ligase, has been shown to interact with Hsp90 or Hsp70 and ubiquitylates unfolded proteins trapped by molecular chaperones and degrades them. Here, we demonstrate that transient over-expression of CHIP in a neuronal cell model reduces the monomeric mutant AR more effectively than it does the wild-type, suggesting that the mutant AR is more sensitive to CHIP than is the wild-type. High expression of CHIP in an SBMA transgenic mouse model also ameliorated motor symptoms and inhibited neuronal nuclear accumulation of the mutant AR. These findings suggest that CHIP over-expression ameliorates SBMA phenotypes in mice by reducing nuclear-localized mutant AR.

KAKENHI (17025020) from the Ministry of Education, Culture, Sports, Science and Technology, Japan

## P1-j12 Comprehensive cross-platform gene expression profiling analysis of dentatorubral-pallidoluysian atrophy (DRPLA) transgenic mice

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To reveal the transcriptional dysregulations in polylglutamine diseases, expression profiling was conducted using two platforms (Affymetrix and Agilent) on three transgenic mice lines (Q76, Q113, Q129) with each harboring a full-length human mutant DRPLA gene carrying various length of CAG repeats (76, 113, 129 repeats, respectively) at the same integration site and controls at three different ages (4, 8, 12 weeks). In Q129 mice cerebrum, the correlation coefficients of common signature genes between the two platforms were 0.83, 0.79 and 0.79 at 4, 8 and 12W, respectively. The number of correlated genes were 814, 695 and 910 at 4, 8 and 12W, respectively, while those of anticorrelated genes were only 11, 21 and 11. Thus cross-platform analysis should be a robust and comprehensive method for confirmation of microarray data.

## P1-j13 SUMO-1 modification increases human SOD1 stability and aggregation

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Approximately 20% cases of familial amyotrophic lateral sclerosis (FALS), which is characterized by selective loss of motor neurons, are caused by the mutations in the gene encoding copper-zinc superoxide dismutase (SOD1) through a toxic gain of function. The hallmark of FALS is the presence of inclusions formed by mutant SOD1. However, the precise mechanisms remain unclear. Here we for the first time report that human SOD1 is modified by SUMO-1 at lysine 75. We further show that the modification of wild type and mutant SOD1 by SUMO-1 increases SOD1 steady state level and aggregation. Moreover, SUMO-1 is recruited to the aggregates formed by SOD1. These findings imply that SUMO-1 modification at lysine 75 may participate in regulating SOD1 stability and its aggregation process. Taken together, our findings suggest that sumoylation of SOD1 may be involved in the pathogenesis of FALS associated with mutant SOD1.

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## P1-j14 Role of satellite III repeats in stress response and neurodegeneration

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Nuclear stress granules (NSG) are formed in all mammalian cells under the condition of heat shock. In humans, the heat shock transcription factor 1, activates the transcription of satellite III repeats (S3R) into stable RNAs that eventually form the NSG. The S3R non-coding transcripts associate with several RNA binding proteins, and thereby possibly protect them during stress conditions. We have blocked or over-expressed S3R transcripts under various stress conditions in a cellular model and evaluated the effect on cell survival and functions. Interestingly, besides the heat shock, the S3R transcription is induced by proteasomal and osmotic stress. While S3R knockdown leads to apoptosis in heat-shocked cells, its overexpression do not appear to have any adverse effect on cell survival. A functional homologue of S3R in Drosophila is a potent modifier of Polyglutamine toxicity. In the present study, we intend to examine the role of S3R in protein-conformation disorders. We will present and discuss the potential role of S3R transcripts in neurodegenerative disorders.

DBT,India

## P1-j15 p45 targets the polyglutamine disease protein ataxin-3 to the proteasome

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Machado-Joseph disease is an autosomal dominant neurodegenerative disorder. It has been widely accepted that MJD1 gene product, which has a polyglutamine tract near the C-terminus, is related to this disorder. Ataxin-3 was previously shown to undergo proteasome-dependent degradation, however, the control of the ataxin-3 is unknown. Using in vivo and in vitro assays, we show that ataxin-3 has direct links to p45, an ATPase subunit of the 19S proteasome. This interaction occurs through the N-terminus of ataxin-3. Moreover, p45 can stimulate the degradation of ataxin-3 by the proteasome and the regulation is functionally specific to both normal and expanded ataxin-3. Other ATPases of the 19S proteasome, MSS1, p48, p56 have no effect on ataxin-3 degradation. We also identified the UIM is essential in the degradation. These data provide evidence that p45 plays an important role in regulating ataxin-3 degradation by the proteasome.

## P1-j16 Bone marrow-derived microglia control inherited neurovascular degeneration of the retina

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The role of microglia in neurodegeneration is currently a matter of great debate and controversy. Here we show that bone marrow (BM)-derived microglia plays a neuro- and vaso-protective role in the models of retinitis pigmentosa (RP), a hereditary progressive neurovascular degenerative disease of the retina. BM-derived cells are recruited to the degenerating retina and home to the degenerative vessels and neurons where they differentiated into three types of microglia, perivascular, amoeboid and ramified. Inhibition of stromal derived factor -1 reduced the number of recruited microglia and accelerated neurovascular degeneration. In addition, systemic depletion of circulating microglial precursors also exhibits severe accelerated neural and vascular degeneration. Surprisingly, human RP patients had functional impairments of circulating BM-derived stem cells in CFU-GM compared to control subjects. These data suggest that BM-derived microglia may play an important role in the progression of RP.

KAKENHI

## P1-j17 Functional analysis of XPA using in utero electroporation method

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Xeroderma Pigmentosum (XP) is an autosomal recessive disorder characterized by sun sensitivity, high frequency of skin cancer and a variety of neurological symptoms. Approximately 20% of all XP patients show progressive neurodegeneration. XP patients (group A to G) are defective in the capacity of nucleotide excision repair. In contrast to the cutaneous symptoms that are caused by unrepaired UV photoproducts, etiology of neurological symptoms remains unresolved. XP group A (XPA) patients show the severe neurological symptoms characteristic of atrophic neurodegeneration. To address the important issue about the etiology of XPA, we adopted in utero electroporation method in the developmental stage of neocortex to examine radial migration and proliferation in XPA-knockdown neuronal cells. At the 3rd days after transfection, XPA-knockdown cells arrested in intermediate zone while control cells reached at the surface of cortex. In addition, proliferation of XPA-knockdown cells was promoted. The phenotypes suggest that XPA has a novel function besides nucleotide excision repair.

## P1-j18 Molecular mechanism of Dysbindin:a Schizophrenia-related factor

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Dysbindin is a Schizophrenia-related factor. Dysbindin is located at chromosome 6p22.3,one of the most susceptibility loci in schizophrenia. Recently Dysbindin has proven the association with schizophrenia by SNP analysis in many papers. Dysbindin is identified as a dystrobrevin binding protein. In the brain,dysbindin is expressed in axon bundles and especially in certain axon terminals of the cerebellum and hippocampus,and also concentrated in the nucleus of transfected cells. To determine the functional role of the dysbindin in nucleus, we have performed a yeast two-hybrid screening and DNA microarray. In this report, we describe the interaction between dysbindin and a transcription factor, and mechanism of regulation of gene expression.

## P1-j19 A system dynamics theory of the hypodopaminergic instability of the prefrontal cortex in schizophrenia

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Although not yet definitive, many studies have suggested hypodopaminergic transmission in the PFC in association with cognitive and negative symptoms. The so-called inverted-U shaped profile of the dependence of working memory performance on the D1 receptor activation level illustrates how the cognitive function declines with the deviation of the DA level in the PFC from the optimum point. A recent computational study, however, suggests that the PFC circuit is not always stable along the inverted-U shaped curve (Tanaka, 2006). The stability of the PFC circuit depends on the DA tone in the PFC, and may have more direct links to schizophrenia than the dopaminergic transmission itself. The closed-loop circuitry of the prefronto-mesoprefrontal system can work as a "regulator" of the PFC activity for working memory if the system is stable (Tanaka, 2006). Under hypodopaminergic conditions, however, the system becomes unstable and no longer works as a regulator of the PFC activity. This is because the "switchover" from a negative feedback system to a positive feedback system occurs as the cortical DA level decreases.

## P1-j20 Chandelier neurons control prefrontal cortical hyperactivation with hyperdopaminergic transmission

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Dysfunctions of chandelier cells have been associated with psychiatric and neurological diseases, such as schizophrenia and epilepsy. Our recent computational study on prefrontal cortical (PFC) circuit dynamics suggested that the PFC shows hyperactivity under a hyperdopaminergic condition. This violates the inverted-U shaped dopaminergic modulation principle of PFC activity, and would be associated with psychotic states. In the present study, we incorporated chandelier cells into our PFC circuit model to investigate how the cortical dynamics would change accordingly. The result of our computer simulation with this model shows that normally functioning chandelier cells effectively suppress such hyperactive states of the PFC. In contrast, reduced inhibition by the chandelier cells cannot suppress the hyperactivity. We, therefore, propose that chandelier cells are potent to control PFC activation under hyperdopaminergic conditions. This has an important clinical implication for a pharmacological treatment, targeting chandelier cells, of psychosis as well as epilepsy.

## P1-j21 Background activity affects sustained firing of prefrontal neurons engaged in working memory - a computational study -

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Recent studies showed the noise increase in the prefrontal cortex (PFC) correlated with the disturbance in the information processing, including working memory (WM), of schizophrenics. We constructed a neuronal circuit model of the PFC, which represented inverted-U dopamine D1 receptor action, according to the cellular basis and examined the influence of the background activity (BA), or "noise". Low levels of the BA expand the scope of cue-coded activity sustaining and help the activity change in response to the changes of the cue input on the a certain D1 receptor activity range, whereas high levels precipitate unexpectedly high neuronal activity, prevent the neural circuit from switching its own activity, and oversharpen the contrast resolution of cue input. These results support the presence of functional stochastic resonance in the PFC and suggest the possibility of the noise increase causing the WM disturbance of schizophrenics.

## P1-j22 Effects of systemic Phencyclidine on firing activity of amygdala neurons in freely moving rats

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Phencyclidine (PCP) -administered animals are now considered to be a pharmacological model of schizophrenia. In schizophrenic patients the negative symptoms, such as social withdrawal and blunted affect, usually persist for a long period, which makes rehabilitating patients difficult. Recent studies reported the close relation between the amygdala function and social behaviors. Disturbed amygdala function is also reported in schizophrenic patients. In this study we recorded the unit activity of amygdala neurons while rats were exposed to a novel environment and engaged in social behaviors. After identifying the response pattern of recorded neurons, we then recorded unit activity in the same neurons with systemic PCP. Some neurons exhibited a temporal increase of the spontaneous discharge immediate after exposure to the novel environment, and also exhibited long-lasting activation during the social interaction. The systemic PCP induced a long-lasting activation in most of these neurons.

## P1-j23 Implication of the pallidotegmental neurons in methamphetamine- and MK-801-induced impairment of prepulse inhibition of the startle reflex in mice

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Our previous study indicated that neural circuits containing pallidotegmental GABAergic neurons play a crucial role in prepulse inhibition (PPI) of the acoustic startle reflex in mice. In the present study, we examined whether PPI disruption caused by methamphetamine (METH) and MK-801 is associated with dysfunction of the pallidotegmental neurons in mice. Acute treatment with METH and MK-801 dose-dependently impaired PPI of the startle reflex. Repeated, but not acute, METH treatment resulted in a lasting impairment of PPI after the withdrawal. c-Fos immunohistochemistry revealed that lateral globus pallidus (LGP) was activated while caudal pontine reticular nucleus (PnC) was inhibited after the PPI test in saline-treated mice. In contrast, LGP was inhibited while PnC was activated in the PPI-disrupted mice pretreated with METH or MK-801. These results suggest that dysfunction of the pallidotegmental neurons is involved in PPI disruption caused by METH and MK-801 in mice.

## P1-j24 Inhibition of activin signaling in the brain causes a schizophrenia-like behavior

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We previously isolated activin $\beta$A as one of the genes whose expression is regulated by neural activity in the hippocampus. Activin $\beta$A, a member of transforming growth factor-$\beta$ (TGF-$\beta$) superfamily, plays an important part in development. However, the functional role of activin in the adult brain is still unknown. We recently found that follistatin, which antagonizes activin signal, inhibited late phase LTP formation. Furthermore, the activin in the brain modulates anxiety-related behavior. In this study, we further investigated roles that activin signaling plays in the brain. To this end, we analyzed performance of forebrain-specific follistatin transgenic mice (FSM) on Morris water-maze test, and found an impairment in working memory. Moreover, FSM showed a deficit in prepulse inhibition (PPI), indicating that activin is involved in the regulation of sensori-motor gating. It is known that Schizophrenic patients show an impairment of working memory and sensori-motor gating. Our study suggests that FSM may be a new and useful animal model of Schizophrenia.

## P1-j25 Neonatal hypomyelination in the ventral hippocampus induces behavioral changes in adulthood

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We reported hypomyelination in the neonatal hippocampus, but not in the cerebral cortex, in a mouse model for schizophrenia, which was made by the maternal injection of poly I:C. Another schizophrenia model has been produced by destruction of the neonatal rat ventral hippocampal tissue with ibotenic acid. In the latter model, the neonatal hippocampal damage may result in abnormal neuronal development in other parts of the brain, e.g. prefrontal cortex, which could cause abnormal behaviors later in adulthood. Taken these two models into account, we assume that only hypomyelination without neuronal damage in the ventral hippocampus may lead to schizophrenic behaviors. In the present study, we produced hypomyelination in the neonatal ventral hippocampus of rats by injecting 1% lysolecithin and analyzed behavior in adulthood. The rats showed decreased prepulse inhibition, increased anxiety, and increased sensitivity to methamphetamine.

## P1-j26 Effect of emodin for cognitive impairment in model of schizophrenia

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Most of current antipsychotic drugs have been developed, targeting monoamine receptors such as dopamine D2 receptor. A significant proportion of schizophrenia patients fail to respond to the conventional drugs, however. In the present research, we aimed at developing a new class of antipsychotic drugs using the novel schizophrenia animal models that have been established with neonatal EGF treatment and hippocampal lesion. These animal models are known to exhibit dopamine D2 hypersensitivity as well as behavioral deficits including lower prepulse inhibition scores. We examined the antipsychotic actions of emodin and its derivatives, which inhibit a tyrosine kinase activity of EGF receptor subunit (ErbB2). Subchronic p.o. administration of emodin (50 mg/kg) for a week significantly ameliorated the PPI deficits of both animal models. Emodin failed to affect locomotor activity, however, suggesting no apparent side effects. These results suggest that anthraquinone compounds may be a new class of antipsychotic drugs targeting EGF signaling.

## P1-j27 Phencyclidine rapidly induced glutamate release and increased BDNF expression in cultured cortical neurons

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Phencyclidine (PCP) is used as a dissociative, anesthetic agent and chronic administration of PCP generates positive and negative symptoms like schizophrenia in human and rodents. However, less is known about the effect of PCP on neuronal function in cellular level. Here, we monitored the neuronal response of cultured cortical neurons after PCP exposure. We found that an acute PCP treatment rapidly elicited a great deal of glutamate release within 5 min. This increase in glutamate release was transient and returned to a basal level within 30 min. Interestingly, the expression of BDNF was robustly increased 3 hours after PCP application, suggesting that an administration of PCP evokes glutamate release and the increased glutamate induces the BDNF up-regulation. It is possible that this abnormal glutamate release and BDNF expression may influence the synaptic function and neuronal survival.

## P1-j28 The role of the interaction between DISC1 and Kendrin in the nervous system

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*Disrupted-In-Schizophrenia 1 (DISC1)* was identified as a novel gene disrupted by a (1;11)(q42.1;q14.3) translocation that segregated with schizophrenia, bipolar disorder and other major mental illnesses in a Scottish family. We previously identified a giant coiled-coil centrosomal protein Kendrin as an interacting partner of DISC1. In this study, we demonstrate that carboxy-terminus of DISC1 enhances the interaction of DISC1 with Kendrin and is required for the centrosomal localization of DISC1. Furthermore, we show that overexpression of DISC1-binding region of Kendrin impairs the microtubule organization. These results suggest that the physiological role of DISC1 should be closely related to centrosomal functions by binding to Kendrin.

## P1-j29 Characteristics of emotional related cognitive function in schizophrenia evaluating by exploratory eye movements, and fMRI: comparison with healthy subjects.

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The present study compared the effects of facial affective stimuli on exploratory eye movements, fMRI in 12 schizophrenic patients and age match healthy subjects using the crying baby and the smiling baby. The Ethics Committee of Kurume University approved this study. Using the exploratory eye movements, the total scanning length (TESL) when viewing the smiling baby was longer than that when viewing the crying for controls. However, the TESL for the smiling baby was shorter than that for the crying baby for patients. There was significant difference observed between TESL in controls and patients for the smiling baby, but no difference obtained between two groups for the crying baby. Activation of fMRI around amygdale area was observed in controls when viewing a crying baby, but not when viewing the smiling baby. However, in patients the smiling baby activated amygdale area, but not the crying baby.

## P1-j30 Methamphetamine sensitization parallels decrease in dopamine axon density and length reduaction of the GABA axon terminal in the medial prefrontal cortex

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Rats were daily exposed to methamphetamine (MAP) (2 mg/kg, i.p.) for 12days (d1 -d12) and then withdrawn for 28 days. Motor response sensitization developed daily during the exposure period and remained at the level of d12 during the withdrawal. Tyrosine hydroxylase-positive axons daily decreased in density, up to 90 % of the control on d12; it was constant during withdrawal. GABAergic neurons (Parvalbumin-positive) form en passant synapses called "candles", which contact the initial segments of pyramidal neurons. Repeated MAP administration decreased total candle length, leading to the reduction in population of the candles with the oroginal spans. The daily morphological alterations were inversely related to the duration of the development and retention of motor response sensitization to MAP, showing a mirror image; this implicates enduring disinhibitory modulations of the dopaminergic and GABAergic morphological changes in the medial prefrontal cortex in psychostimulant sensitization.

## P1-j31 Overexpression of DISC1 impairs associative learning and arousal in *Drosophila*

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The *disrupted-in-Schizophrenia-1 (DISC1)* gene, originally identified at the breakpoint of a chromosome (1;11)(q42.1; q14.3) translocation in a Scottish pedigree, is a strong susceptibility gene for schizophrenia and affective disorder in large populations of patients. To address *DISC1* functions *in vivo*, we expressed the full-length (FL) and truncated (TR) human *DISC1* genes in *Drosophila*, where FL localized punctately in nuclei while TR diffusely in both nuclei and cytoplasm. Whereas neither axonal nor neurodegenerative abnormality was detected, overexpression of DISC1 abolished olfactory associative learning. Moreover, FL but not TR DISC1 impairs sleep-arousal state in flies. These results suggest potential interaction of DISC1 with cAMP-signaling cascade and demonstrate the importance of DISC1 nuclear function in the brain.

## P1-j32 Identification of novel ribonucleoproteins as DISC1-interacting molecules

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Disrupted-In-Schizophrenia 1 (DISC1) is a candidate gene for susceptibility to schizophrenia. However, the functions of DISC1 remain largely unknown. We here found that DISC1 was associated with the components of messenger ribonucleoproteins (mRNPs) including hnRNP-U, Pur $\alpha$, and RACK1. DISC1 was colocalized with mRNPs in dendrites of hippocampal neurons. mRNPs regulate the localization of mRNAs and local protein synthesis for synaptic formation and plasticity. Since mRNPs formed a large complex with RNAs, ribonuclease A treatment was used to verify the direct interaction of DISC1 with mRNPs. To identify the direct partner of DISC1 in the mRNP complex, we performed a modified DISC1-affinity column chromatography using rat brain lysate treated with ribonuclease A. As the result, we found putative direct partners of DISC1 among mRNPs. The physiological meaning of the interaction between DISC1 and its direct partners will be discussed.

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## P1-j33 The functional analysis of DISC1 and Dysbindin

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Schizophrenia is a multifuctorial disorder influenced by genetic,neurodevelopm ental and social factors. DISC1 and Dysbindin are potential susceptibility genes for major psychiatric disorders. We previously identified FEZ1(fasciculation and elongation protein zeta-1, Kendrin and DBZ(DISC1 Binding Zincfinger protein) as DISC1-interacting protein. Furthermore we showed that the expression level of Dysbindin affects secretion of Dopamine. Here, we searched the new function of DISC1 and Dysbindin and their partners.

## P1-j34 Alterations in Bax and Bcl-2 mRNA expression levels in hippocampus of olanzapine administrated rats.

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It has been hypothesized that apoptotic mechanism is related to the therapeutic effect of second generation antipsychotics. Olanzapine is one of the second generation antipsychotics. To test this hypothesis, we measured the pro-apoptotic protein Bax and anti-apoptotic protein Bcl-2 mRNA levels in hippocampus of the rat brain after acute administration with olanzapine by use of the quantitative RT-PCR methods. Each rat was intraperitoneally injected with olanzapine or vehicle control at 10:00 h daily for 7 days. Increase Bcl-2, no change Bax and decrease Bax/Bcl-2 mRNA expression ratio were seen in hippocampus following the treatment with olanzapine. We infer that an anti-apoptotic effect such as decrease Bax/Bcl-2 mRNA expression ratio in hippocampus is related to therapeutic effects of olanzapine.

## P1-j35 Functional analysis of Dysbindin, one of schizophrenia susceptibility genes

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Dysbindin has been reported as one of the most promising susceptibility genes for schizophrenia in linkage studies. Recent research indicates that the expression level of the dysbindin gene is reduced in the brains of patients with schizophrenia. To investigate the role of Dysbindin in brains, we performed a yeast two-hybrid screening and found candidate proteins binding to dysbindin protein in brains. Some of the candidates binding to dysbindin proteins are associated with axonal transport and cytoskeleton. Here we report the further analysis of those interactors and discuss about the pathological roles of dysbinbdin and those candidates.

## P1-j36 Dual effects of aripiprazole on prefrontal dopamine release

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This study examined whether the novel antipsychotic aripiprazole affects or not dopamine (DA) release in the medial prefrontal cortex (mPFC) when it is given locally into DA neurons of the ventral tegmental area (VTA), using microdialysis with dual probe implantation of awake, freely moving rats. Aripiprazole locally applied into the VTA significantly decreased extracellular DA levels in the mPFC. The effect of aripiprazole resembled that of quinpirole, a full $D_2$ receptor agonist, but not of raclopride, a $D_2$ receptor antagonist. When applied directly into the mPFC, aripiprazole increased prefrontal DA levels. This effect was completely abolished by pretreatment with WAY100,635, a 5-$HT_{1A}$ receptor antagonist. This is the first study to provide in vivo evidence for an agonistic action of aripiprazole on the VTA DA neurons. Moreover, aripiprazole may enhance DA function of the mPFC local circuits via activation of 5-$HT_{1A}$ receptors.

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## P1-j37 Characteristics of emotional charged cognitive function in patients with schizophrenia: P300 and LORETA analyses

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We analyzed the P300 component of the visual event-related potential in 24 patients with schizophrenia and age-mach 24 healthy controls, and also performed LORETA analysis. The Ethics Committee of Kurume University approved this study. The P300 amplitude for the crying face was significantly smaller in patients than in controls. In controls, the P300 amplitude was significantly larger for the crying face than for the laughing face, while in patients, there was no significant difference in the P300 amplitude between the 2 faces. LORETA analysis demonstrated that there were significant differences in the activity of amygdale viewing crying face. While in patients, there was significant activity difference in amygdale only viewing the laughing face. These results indicated that the cognitive function was influenced by affective stimulus and different emotional cognition is occurred in schizophrenic patients.

## P1-j38 The effect of psychotropic drugs on microglial activation induced by interferon-γ

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An accumulating body of evidences point to the significance of neuroinflammation and immunogenetics in schizophrenia or depression. In the central nervous system (CNS), the microglial cells are the major immunocompetent cells which release pro-inflammatory cytokines, nitric oxide (NO) and reactive oxygen species to mediate the inflammatory process. In the present study, we investigated whether or not psychotropic drugs such as antipsychotics and antidepressants have anti-inflammatory effects on the activated microglia induced by interferon-γ. Most psychotropic drugs examined significantly inhibibited NO generation or cytokine release from activated microglia. Psychotropic drugs may have an anti-inflammatory effect via the inhibition of microglial activation, which is not only directly toxic to neurons but also has an inhibitory effect on neurogenesis and oligodendrogenesis, both of which have been reported to play a crucial role in the pathology of schizophrenia or depression.

## P1-j39 Functional analysis of neuronal primary cilia using pericentrin mutant mice

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Pericentrin is a mammalian centrosomal protein involved in the formation of immotile primary cilia, which singly extend like antennae into the environment surrounding the cell and transduce sensory stimuli to the cell body. We previously identified pericentrin as an interactor of the product of *DISC1*, a candidate gene for mood disorders and schizophrenia. In this study we generated and characterized a hypomorphic mutation of the mouse pericentrin gene named opcd. The opcd homozygous mice revealed growth retardation and a decreased density of primary cilia in several tissues including the central nervous system. We compared the ciliary localization of cilia-related proteins such as IFT88, somatostatin receptor type 3 and adenylyl cyclase III in some brain regions between wild type and opcd mutant mice. Interestingly, these mutant mice displayed a markedly shortened time of immobility in the forced swimming test and the tail suspension test, suggesting biological roles for neuronal primary cilia in mediating mood or emotion.

## P1-j40 Expression analysis of neurotrophic factors in patients with mood disorders.

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Neurotrophic factors have key roles in the regulations of cell differentiation, survival, and synaptic plasticity. Although postmortem brain studies have indicated the change in the expressions of neurotrophic factors in mood disorder patients, it is unclear that these changes are state- or trait-dependent effects. In this study, we examined the expression levels of BDNF, NGF, NT-3, NT-4, and GDNF family (GDNF, ARTN, NRTN, and PSPN) mRNAs by using quantitative real-time PCR method in the peripheral white blood cells of patients with major depressive and bipolar disorders in both current depressive and remissive states. We found that expression levels of GDNF, ARTN, and NT-3 mRNAs were lowered in patients with major depressive disorder in current depressive state, but not in remissive state. Our results suggest that the changes in the expression levels of GDNF, ARTN, and NT-3 mRNAs would be a state-dependent effect, and are associated with the pathophysiology of major depression.

## P1-j41 Expression analysis of adhesion molecule CD44 isoform mRNA in the patients with mood disorders

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Dysfunction of glucocorticoid receptor (GR) has been thought to be involved in the pathophysiology of mood disorders. Previously, we reported that the reduced GR $\alpha$ mRNA level in the patients with both bipolar and major depressive disorders, while GR $\beta$ mRNA level was not altered. Also, we found the elevated SRp20 splicing factor mRNA level in bipolar disorder patients. These results suggest that dysregulation of alternative splicing play an important role in the pathophysiology of mood disorders. In this study, we evaluated the adhesion molecule CD44 isoform mRNA expressions in the peripheral white blood cells of mood disorder patients. The expression level of CD44H isoform mRNA was reduced in the patients with both the depressed bipolar and major depressive disorders compared to healthy controls. In contrast, the expression level of CD44 exon v9 isoform mRNA was not altered. These results suggest the aberrant expression of CD44 isoform produced by SRp20-mediated alternative splicing in the pathophysiology of mood disorders.

## P1-j42 Altered levels of whole blood sigma-1 receptor protein in patients with mood disorders

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Sigma-1 receptors are unique proteins that have high affinities for psychotrophic drugs such as antidepressants and for neurosteroids such as progesterone and dehydroepiandrosterone. We tried to examine whether sigma-1 receptors are involved in mood disorders. Fifty-six Japanese patients with mood disorders and fifty-six race- and gender-matched healthy control subjects were recruited. All patients were under partial or full remitted state and were treated with medication including antidepressants. The whole blood levels of sigma-1 receptors proteins were significantly decreased in only male patients with mood disorders when compared to male healthy control subjects, although the levels were not different between the female patients and the female control subjects. There was no association between dose of antidepressants and the levels of sigma-1 receptors. Whole blood levels of sigma-1 receptors could be a trait marker for male patients with mood disorders.

## P1-j43 Antidepressants-induced GDNF production in rat C6 glioma cells involves activation of FGF receptors.

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Recently, post-mortem studies provide evidence for glial reduction in different brain areas in mood disorders. We previously reported that antidepressants increased glial cell line-derived neurotrophic factor (GDNF) production through monoamine-independent PTK and ERK activation (Hisaoka et al., JPET 321: 148-157, 2007). In this study, we showed that (1) the antidepressant-induced ERK activation and GDNF production were completely inhibited by FGF receptor tyrosine kinase inhibitors, (2) antidepressant acutely increased phosphorylation of FGF receptors, (3) the antidepressant-induced ERK activation was significantly inhibited by heparin, bFGF neutralize antibody, and a matrix metalloproteinase inhibitor. These results suggest that antidepressant acutely induces a shedding of FGF receptor ligands such as bFGF and activates ERK via FGF receptors, finally resulting in GDNF production.

## P1-j44 Valproic acid, a drug for bipolar disorder, acts on both $IP_3$- and DAG- regulated behaviours in *C. elegans*

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Valproate (VPA) is widely used in the treatment of bipolar disorder, however, the mechanism of VPA action is unclear. We aim to understand how the mood-stabilizers work in brain by studying their effects on the nervous system of the model organism *C. elegans*. We show that in *C. elegans* VPA inhibits both inositol phosphate and diacylglycerol (DAG) signaling. VPA disrupted two behaviors regulated by the inositol phosphate $IP_3$: growth and defecation. VPA also inhibited behaviors regulated by DAG signaling: acetylcholine (ACh) release, egg-laying, and the expulsion step of defecation. The effects of VPA on DAG signaling are relieved by phorbol esters, a DAG analog, suggesting that VPA acts to inhibit DAG production and not DAG signaling. . DAG is a major regulator of mammalian neurotransmission and our results suggest that VPA may act to relieve bipolar disorder by modulating neurotransmitter release via DAG pathway.

## P1-j45 The effects of IL1-$\beta$ on behavioral responses and the density of monoamine axons

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Recent studies demonstrate that morphological changes in monoamine neurons are involved in the pathophysiology of depression. It has also been reported that depression is associated with the activation of immune systems. In this study, we examined the effects of intracerebroventricular infusion of IL1-$\beta$ for 2 weeks using osmotic minipumps (100 ng/24h) on behavioral responses (open field test, OFT and forced swim test, FST) and the density of serotonin (5-HT) and noradrenaline (NA) axons. Treatment with IL1-$\beta$ significantly decreased the number of rearing and activity in the central area in the OFT, and increased the duration of floating in the FST. The density of 5-HT and NA axons visualized by immunohistochemistry was assessed in the medial prefrontal cortex (mPFC), dentate gyrus (DG), and basolateral amygdala (BLA). In the IL1-$\beta$ treated rats, the density of 5-HT axons significantly decreased in the mPFC and DG, while the density of NA axons decreased in the DG and BLA. These results support the possibility that depression is associated with morphological changes in monoamine axons.

## P1-j46 Ameliorative effect of dental treatments on unexplained symptoms

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Many dentists have occasionally experienced that either adequate occlusal reconstruction or splint therapy was effective for improvement of unexplained physical and/or psychological complains, e.g. headache, stiff shoulder, and giddiness, tinnitus, or somatoform disorders. Since the link between these pathological phenomena and the cerebral limbic system has been suggested, we used an fMRI to prove this hypothesis. The patients, under the condition of inadequate occlusion and with the above abnormalities, showed a marked activation in the amygdala, when they clenched at habitual inter cuspal position; however, this activation was attenuated by set of the splint. The result suggests that dental treatments may help to ameliorate unexplained complains through neural processes in the amygdala.

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## P1-j47 Serotonergic modulation on the human attentional system: an fMRI study

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Investigating modulatory effects of psychopharmacological agents in the human brain allows for not only functional characterization of neurotransmitter systems involved in the human cognition, but also better understanding of pathophysiology of psychiatric disorders. Here we present an fMRI study to assess the effect of a selective serotonin reuptake inhibitor (fluvoxamine) on the human attentional system. Upon a single, acute administration of fluvoxamine, thirteen healthy adults performed a Stroop-like cognitive interference task during an fMRI session. We found that, in the absence of serotonergic modulation, a wide area of brain regions, including the cingulomotor area, the limbic area, and the cerebellum, exhibited significant activations. Under the fluvoxamine treatment, on the other hand, a part of the network, most prominently the limbic structures, exhibited decreased activity. We discuss the implication of the decreased activity in terms of clinical therapeutics.

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## P1-j48 The effect of subconvulsive electrical stimulation of the nucleus accumbens on depressive behavior

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Our aim was to test whether subconvulsive electrical stimulation (SCES) of the nucleus accumbens (NaC) can induce antidepressant effects. We used the chronic mild stress (CMS) model in rats and we measured the sucrose preference, exploration, and Morris water maze test and the level of brain-derived neurotrophic factor (BDNF). While CMS induced a reduction in sucrose preference, the number of mounts in the sexual behavior test, decreased rearing behavior and the number of the visits in the central area, which was normalized by SCES of the NAc. Learning in the Morris water maze was not affected by the SCES treatment. Finally, BDNF levels were increased in the dorsal dentate gyrus and in the striatum following SCES treatment of the NAc in CSM animals, suggesting a potential therapeutic mechanism. These results suggest that SCES is as effective as ECT without its side effect and point to the important role of the NaC in the pathology of depression.

HELENA RUBINSTEIN POSTDOCTORAL FELLOWSHIP

## P1-k01 Activation of interleukin receptor of nociceptive tigeminal ganglion neurons via satellite glial interleukin-1 $\beta$ contributes to the hyperalgesia following peripheral inflammation

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The present study was investigated whether inflammation alters the excitability of trigeminal ganglion (TRG) neuronal activity via cytokine. A; δ-TRG neurons responding to electrical stimulation of the whisker pad were recorded in anesthetized rats. The firing rates of their spontaneous activity in the TRG neurons were current-dependently decreased by local iontophoretic application of a IL-receptor type I (IL-1RI) receptor antagonist (IL-ra) in inflamed, but not non-inflamed rats. Their spontaneous activities were current-dependently increased by application of interleukin 1 $\beta$ (IL-1 $\beta$) in control and inflamed rats. There were no significant differences in the mechanical stimulation threshold between control and inflamed rats after iontophoretic application of IL-ra. These results suggest that inflammation potentiates the excitability of nociceptive-TRG neurons innervating the facial skin via IL-1 $\beta$ paracrine mechanism within trigeminal ganglia and this contribute to hyperalgesia.

KAKENHI

## P1-k02 Activation of prostaglandin receptor EP3 inhibits the bradykinin-induced B2 receptor sequestration

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We have previously reported that the activation of prostaglandin receptor EP3 attenuates the desensitization of bradykinin (BK)-induced increase of intracellular calcium in a PTX-sensitive manner in Chinese hamster ovary (CHO) cells expressing both EP3 and BK B2 receptor (B2R). The purpose of this study is to elucidate whether receptor sequestration is involved in the desensitization of BK response. B2R fused with green fluorescent protein (GFP) was transiently expressed in CHO cells stably expressing EP3, and its subcellular localization in living cells was chased after BK administration by confocal microscope. BK administration induced the gradual decrease in GFP fluorescence at the cell membrane. Treatment of the cells with EP3 agonist, ONO-AE-248, or a specific inhibitor of protein kinase A, H-89, tended to prevent the decrease of fluorescence intensity. It is thus suggested that EP3 activation attenuates the desensitization of BK response via Gi by inhibiting B2R sequestration.

## P1-k03 Investigation of pain-producing substances in skeletal muscles after downhill running.

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Although delayed onset muscle soreness (DOMS) is known to occur after strenuous eccentric muscle contraction, its mechanism is remained to be elucidated. We previously reported that the number of c-Fos$^+$ neurons in the spinal dorsal horn was increased following downhill running (DHr). In this study, to determine whether DHr can induce DOMS, rats performed DHr on treadmill for 90 min, and changes of the number of c-Fos$^+$ neurons, withdrawal threshold and exercised muscles were examined at optimal time after DHr. Maximum number of c-Fos$^+$ neurons and most decreased level of withdrawal threshold were detected at 12 hours post-DHr, while necrosis of myofibers and inflammatory reactions in soleus muscles peaked at 24-48 post-DHr. These results determined that peak of DOMS is not only induced at 12 hours post-DHr, but also DOMS is resulted in prior to remarkable development of necrotic myofibers and inflammatory reactions in exercised muscles. Further researches will be attempted to explore pain-producing substances in muscular fasciae following DHr.

## P1-k04 Changes in sensitivity of glutamate receptors at the peripheral endings of the nociceptive fivers following nerve injury

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In this study, to clarify changes of the sensitivity of glutamate receptors (GluRs) in the peripheral nerve endings following neuropathic pain, we tested the effect of s.c. injection of drugs associated with GluRs, and also capsaicin-induced Glu levels released in s.c. perfusate using microdialysis catheter. Thermal hyperalgesia induced by ligating the sciatic nerve in a rat (CCI) was examined using plantar test. In CCI rats, iGluR and mGluR agonists did not show significant effect on nerve injury-evoked hyperalgesia, while iGluR antagonists significantly increased withdrawal latency. mGluR antagonists did not show any effect. The capsaicin-induced Glu level was lower than that of normal rats. These results suggest that the alteration in sensitivity of GluRs in the peripheral endings of the nociceptive fivers contribute to development and/or maintenance of nerve injury-evoked neuropathic pain.

## P1-k05 Glia-mediated facilitation of neuronal excitation in the spinal dorsal horn in the arthritic rat

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Sensitization in the spinal dorsal horn is believed to be a cellular mechanism for enhanced pain sensitivity. In this study, by using optical imaging with voltage-sensitive dye, we measured the neuronal excitation in spinal cord slices from normal rats and Complete Freund's Adjuvant (CFA)-induced arthritis rats. The amplitude of evoked-neuronal excitation in the spinal dorsal horn in slices of arthritis rats was bigger than normal rats. The evoked-neuronal excitation in the dorsal horn of arthritis rats was suppressed in the presence of a glial metabolism inhibitor, monofluoro-acetic acid, a NMDA-receptor antagonist, D-AP5, and P2X-receptor antagonists, PPADS and TNP-ATP. In contrast, these inhibitor and antagonists did not show any affect on the evoked-neuronal excitation in the dorsal horn of normal rats. These results suggest that facilitation of neuronal excitation mediated by glial cells via NMDA-receptor and P2X-receptor in the spinal dorsal horn is underling mechanism for induction of mechanical hyperalgesia in arthritis rats.

## P1-k06 The role of NOX1/NADPH oxidase on hyperalgesia

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NADPH oxidase is a major superoxide-generating enzyme in the nervous system. Using genetically modified mice, we investigated the role of NOX1, a catalytic subunit of NADPH oxidase, in the pain signaling. No difference in nociceptive responses to heat or mechanical stimuli was observed between Nox1-deficient (KO) and wild-type (WT) mice. Reduced hyperalgesia was demonstrated in KO following the injection of carrageenan. In KO DRG, superoxide production was decreased, while levels of other Nox isoforms were unchanged. When TRPV1 function was evaluated in DRG neurons, no difference in capsaicin-induced calcium increase was observed between two genotypes. Pre-treatment with bradykinin or TPA augmented the capsaicin-induced calcium increase in WT DRG, whereas it was significantly attenuated in KO DRG. Translocation of PKCε induced by TPA was markedly disturbed in KO DRG, or in the presence of antioxidants. These results suggest the involvement of reactive oxygen species derived from NOX1/NADPH oxidase in the development of hyperalgesia, possibly by regulating the translocation of PKCε.

KAKENHI (17790172)

## P1-k07 Spinal HCN channels contribute to neuropathic pain

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We addressed the role of spinal HCN channels in the modulation of chronic pain in the experimental model of neuropathic pain. Intrathecally administered ZD7288, a specific blocker of HCN channels, reduced thermal and mechanical hypersensitivity in mice neuropathic pain condition that was induced by partial ligation of the sciatic nerve, while no analgesic effect was observed in nonligated animals. In visually identified substantia gelatinosa neurons of an adult mouse spinal cord slice with an attached dorsal root, Aδ- and/or C-fiber-mediated monosynaptic excitatory postsynaptic currents (EPSCs) were evoked by electrical stimulation of L4 or L5 dorsal root using a suction electrode. Bath applied ZD7288 preferentially reduced C-fiber-mediated monosynaptic EPSCs in slices prepared from mice after peripheral nerve injury. The present results obtained in behavioral and electrophysiological studies demonstrate clearly that spinal HCN channels contribute largely to hyperexcitability of the spinal dorsal horn that is crucial in the development and/or maintenance of neuropathic pain.

KAKENHI (18590150)

## P1-k08 An activation of cerebral oxygenation in prefrontal cortex and analgesic effect during gum chewing

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We previously examined the gum chewing (GC) suppresses nociceptive responses via the 5-HT descending inhibitory pathway. In the present study, we examined the effects of GC on cerebral oxygenation in prefrontal cortex (PFC). Subjects performed GC for 20 minutes. Cerebral oxygenation in PFC was assessed by concentration changes in oxygenated hemoglobin (oxyHb) and deoxygenated hemoglobin (deoxyHb) using multi channel near-infrared spectroscopy (NIRS). We found increases in right ventromedial and dorsolateral PFC and a decrese in right ventrolateral PFC. OxyHb level in PFC began to increase at ~5 minutes after the onset of GC. To evaluate analgesic effect by GC, we recorded nociceptive flexion reflex before and after GC. NFR level was lower after GC. Since the blood 5-HT levels increased after GC, we suggest that augmentation of brain 5-HT system may be triggered by activation of PFC and raise the pain threshold during GC.

## P1-k09 High frequency stimulation delivered to the amygdala produced analgesia.

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We have been studying how emotional condition modulates pain sensation. We studied the effect of high frequency stimuli (HFS, 100Hz, 20μA, 30s) delivered to the basolateral nucleus of the amygdala (BL) on pain behaviors in the formalin test and the plantar test. HFS delivered to the BL resulted in the reduction of the formalin-induced pain responses, especially licking. HFS in the BL induced the reduction of the pain score in the both the early and late phases with no change on spontaneous motor activities. However, in plantar test, HFS delivered to the BL had no effect on pain related behaviors. In formalin test, HFS in the BL induced the reduction of the pain score following i.p injection of naloxone. We previously reported that HFS delivered to the BL induced prolonged depression of the nociceptive specific response in the rat prefrontal cortex (PFC). All together, the results indicate that amygdala activation modulates pain sensation.

## P1-k10 Changes in the chloride homeostasis-related genes in trigeminal nuclei of the adult rats with nerve injury

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Chronic pain syndromes involves a trans-synaptic reduction in the expression of KCC2 (Coull. et al. Nature 2003). We have prepared a rat model of trigeminal neuropathic pain produced by a chronic constriction injury to the infraorbital nerve. By means of in situ and immunostaining, we found that KCC2 mRNA and protein were downregulated in injury side of Sp5 and the downregulation was correlated with the decrement in pain threshold and increase pain response. The NKCC1 mRNA was upregulated in the injured side of the TG. NKCC1 upregulation in primary neuron (TG) may induce excessive transmitter release by presynaptic GABAergic depolarization. KCC2 downregulation in secondary neuron (Sp5) may result in disinhibition by impairing GABA inhibition which could be crucial for the analgesic action at the Sp5. Such Cl- homeostatic disruption may increase transmission of pain sensation and this may underlie the pathophysiology of the trigeminal neuralgia.

## P1-k11 High frequency stimulation delivered to the hippocampal subiculum modified nociceptive responses recorded in the prefrontal cortex.

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We examined modifing effects of hippocampal subiculum pathways on nociceptive responses recorded in the prefrontal cortex (PFC). Extracellular unit discharges evoked by mechanical noxious stimulation delivered to the rat-tail were recorded in the PFC. Field potentials evoked by subiculum stimulation were also measured. High frequency stimulation (HFS) delivered to the subiculum areas to investigate the effects on the nociceptive responses. HFS delivered to the subiculum increased discharge rate in 78% (21/27cases) of nociceptive neurons recorded in PFC. And 22% (6/27cases) of PFC neurons inversely decreased responses. These plastic changes in discharge rates of PFC neurons prolonged and recovered to the control revel within 120 min. Changes in field potentials, which showed LTP, were correlated with unit discharges alternation. The results of this study indicate that Subiculum-PFC pathways modulate nociceptive response in PFC.

## P1-k12 Possible involvement of macrophage on vincristine-induced allodynia in mice

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The clinical use of vincristine (VCR) is limited its adverse effect, neuropathy. VCR-induced neuropathy includes allodynia, and the mechanisms are still unknown. Recently, some evidences suggest that peripheral invaded macrophage is involved in the development of and the maintenance of neuropathy. In this study, we examined the involvement of macrophage on VCR-induced allodynia. Mice were treated with VCR sulfate (0.1 mg/kg, i.p., once a day) for 14 days. Mechanical allodynia following VCR treatment was observed, showing maximum response on day 14, using von Frey filaments. On the other hand, immunoreactivity (IR) of macrophage markedly increased in surroundings of dorsal root ganglion (DRG) following VCR treatment; a significant increase in the IR was observed on day 7 and was continued until day 14. Moreover, some of the increased macrophage was co-localized with interleukin (IL) -6. These results suggest that VCR-induced mechanical allodynia may be elicited by the invaded macrophage in DRG, being mediated by the release of cytokines, such as IL-6.

## P1-k13 Propagation of neuronal excitation in the rat central nucleus of amygdala revealed by the optical imaging

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The central nucleus of amygdala (CeA), which is output of amygdaloid nucleus, is divided into four subdivisions and the interconnections between the different subdivisions of the CeA have been elucidated by anatomical studies. However, the propagation of neuronal signals between these connections is unknown. In this study, we measured the propagation of neuronal excitation in the CeA by using the optical imaging with the voltage-sensitive dye. Single-pulse stimulation to the dorsomedial part of the CeA induced fast propagation of optical response in the medial part and then slow response propagated in the lateral part of the CeA. Stimulation to the lateral part of the CeA induced the propagation of response mainly in the lateral part of the CeA. These propagations of neuronal excitation were strongly inhibited in $Ca^{2+}$-free solution and non-NMDA glutamate receptor antagonist, CNQX, suggesting that the optical response in this study mainly represent the postsynaptic elements of neuronal excitation via non-NMDA receptors.

## P1-k14 Antidepressant-like effect of green odor in mice

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Inhalation of hexenol and hexenal, which when mixed together produces the so-called 'green odor' (GO), has been reported to result in a release from the physiological and behavioral responses to stress in mammalians, including humans. The present study was undertaken to investigate the antidepressant-like effect of GO in a predictive model of depression, the mouse forced swimming test. The mice placed in GO-scented water exhibited a reduction of the immobility time and an increase of the swimming time, which were similar behavioral responses to that of selective serotonin reuptake inhibitors. Serotonin depletion, induced by p-chlorophenylalanine treatment, completely blocked these effects. GO inhalation did not affect general locomotor activity. These results suggest that GO has an antidepressant-like effect, which is likely mediated by the serotonergic system in mice.

## P1-k15 In vivo effects of a monoamionergic activity enhancer

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(-)-1-(Benzofuran-2-yl)-2-propylaminopentane [(-)-BPAP], a catecholaminergic and serotoninergic activities enhancer, has been shown to improve motor deficits in animal models of Parkinson's disease (PD). Here, we elucidated the possible mechanism of actions of (-)-BPAP and evaluated the synergistic interaction between (-)-BPAP and other anti-Parkinsonian agents. (-)-BPAP improved the catalepsy induced by reserpine alone, but not $\alpha$-methyl-*p*-tyrosine plus reserpine. In addition, (-)-BPAP induced ipsilateral rotations in the unilateral 6-hydroxydopamine-lesioned rats. These results indicate that (-)-BPAP acts to presynaptic site of catecholaminergic nerve terminal. Furthermore, co-administration of (-)-BPAP and L-Dopa, dopamine receptor agonists bromocriptine or talipexole, at subthreshold doses, improved catalepsy in reserpinized mice. Our results suggest that (-)-BPAP ameliorates the akinesia in PD, and it could be a useful treatment for PD not only as monotherapy but also in combination with dopaminergic drugs.

## P1-k16 Serotonin 1A ($5\text{-HT}_{1A}$) agonist decreases locomotor activity and prefrontal neuronal activation in amphetamine treated mice

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We recently demonstrated that co-administration of amphetamine (AMPH) and $5\text{-HT}_{1A}$ agonist 8-OH-DPAT produced an antihyperkinetic effect in mice. In this study, to define the role of $5\text{-HT}_{1A}$ signaling in this effect, we evaluated the effect of another $5\text{-HT}_{1A}$ agonist osemozotan, and examined c-Fos expression pattern in medial prefrontal cortex (mPFC). The results demonstrated that co-administration of osemozotan and AMPH exerted an antihyperkinetic effect, and pretreatment of 8-OH-DPAT suppressed the number of c-Fos-positive neurons in AMPH-treated mice. These results suggest that $5\text{-HT}_{1A}$ signaling regulating mPFC neurons might be involved in the antihyperkinetic effect of AMPH, providing insights into therapeutic mechanisms of psychostimulants in ADHD.

## P1-k17 Deletion of HPC-1/syntaxin1A gene causes autistic behavior in mice

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Previously, we have reported that consolidation and extinction of conditioned fear memory was impaired in HPC-1/syntaxin1A (STX1A) knockout mice. In this study, we have examined if STX1A knockout mice exhibited abnormal behavioral profiles. Homozygous mutant (KO) showed abnormal behaviors in novel object exploration test, social interaction test and latent inhibition (LI) test. The attenuation of LI in KO mice was recovered by administration of SSRI, implying serotonergic disturbance. Interestingly, these behavioral abnormalities were also observed in heterozygous mutant (HT), although the abnormalities in KO mice tend to be more prominent than that in HT mice, indicating gene dosage effect. These results suggested that the content of STX1A affect to neuropsychological feature. In addition, STX1A knockout mice revealed autistic-behavior and could be a model animal of autism.

KAKENHI18700368

## P1-k18 Cytological changes underlying the behavioral alterations after the Gamma Knife irradiation on the rat striatum

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We have reported the behavioral changes aroused by the Gamma Knife irradiation onto unilateral basal ganglia of the rat brain. The substantial changes underlying those behavioral changes were surveyed with immmunohistochemical detection of the functional proteins. The most remarkable cytological changes were observed on the astrocytes. Astrocytes showing the GFAP-LI were swollen in the irradiated sides, and sometimes showed unusual immunoreactivity, such as D2 type dopamine receptor-LI. D2DR-LI was generally enhanced in the regions intercalating the fascicles of Caudate Putamen in the irradiated side. It was not possible, however, to specify particular immunoreactive structures in the light microscopy level. The enkephaline-LI was also increased in the irradiated side, without specifiable structures. The participation of the enkephalinergic spiny neurons of the indirect pathway was suggested.

## P1-k19 Effects of copper metabolism on neurologigal functions in Wistar and LEC rats

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Sensorimotor functions of Wistar and LEC rats, Wilson disease animal model, were compared by measuring various behavioral tests with or without oral supplementation with copper (Cu) or Cu chelator. All of the LEC rats, irrespective of the treatment, exhibited higher locomotor activity, a decreased habituation to startle response or a lower prepulse inhibition, compared with Wistar rats. Besides, in the region of the striatum and the nucleus accumbens of the LEC rats, lower contents of norepinephrine (NE), and higher contents of dopamine and serotonin were observed compared with Wistar rats. Cu supplementation decreased PPI and the NE content of the frontal cortex in both LEC and Wistar rats. These findings suggest that an imbalance in Cu homeostasis affects monoamine metabolism and behavioral functions.

## P1-k20 A study on impulsivity and choice between delayed reinforcers in T-maze

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Impulsivity is often expressed as aggressive behaviour. It is associated with poor self control which can be a major portion of many diseases if it reaches pathological levels. It can be emerged in several forms such as rapid decision making, inability to tolerate a delay of enjoyment and tendency to terminate chains of responses prematurely. Tolerance to delay of reward has been proposed as self control. We investigated the involvement of delay duration and the amount of reinforcement in a T-maze based impulsivity task. After acclimatization, rats were trained to choose between a single food pellet delivered immediately and 4 or 8 pellets delivered after delay. The percent of animals that chose the arm baited with large reward were signified as response and evaluated compared to the development and sort of trials. The results show an enhancement of responses by enlargement in size of reward in animals. The responses were slowly declined with increasing delay time, this was significant in 10 to 30s. To clarify the details more studies can be conducted by using of this animal model.

## P1-k21 BOLD signal reflects temporal patterns of movement

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Due to slow time constant of the BOLD signal, it is assumed that fMRI technique is not useful to analyze brain activities for fine temporal patterns. Ogawa et al. (2000), however, demonstrated that intervals between two sensory stimuli can be detected as suppression of the BOLD signal change for the second stimulus. Therefore it might be possible to detect BOLD responses that closely reflect putative neuronal activity pattern. So we analyzed brain activities for different temporal patterns of movement. We used two temporal patterns which are so slowly that difference of BOLD response occurs. In 3T fMRI, participants executed tapping of the two patterns with or without model tones, or listened to the model tones without tapping. We compared time series of BOLD signal of two patterns for each voxel, and identified important sites for temporal control. Moreover we discuss function at different sites in relation to variation of BOLD responses.

KAKENHI18700429

## P1-k22 Web-based MRI Brain image database system.

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It is important in the field of neuroscience research and education to visualize brain structure three-dimensionally. Especially, it is useful to observe structural changes during development and aging within an individual, or to compare individual and species differences for understanding relationship between brain structure and functions. We have developed web-based applications for MR image database system, which enables both registration of MR images and viewing reconstructed 3D or 2D images by using ordinary web browsers. MR images are registered only by drag & drop on the web browser. Registered data are automatically reconstructed three-dimensionally, and can be searched by experimental number, date, name, or additional comments. Intuitive mouse operation enables us to view slices and rendered surface image of the brain from arbitrary angles, and to measure the position of arbitrary brain structure on standard anatomical coordinates. This database system can be run on several platforms such as Windows, Mac OSX, and Linux, as long as MySQL, Apache, and PHP are installed.

RIO-DB, AIST

## P1-k23 Prediction for finger force from cortical activity measured by near-infrared spectroscopy(NIRS)

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Near-infrared spectroscopy (NIRS) is one of the non-invasive neuroimaging methods.This method has advantages such as easy-use and portability. It may be possible to apply in both clinical use and daily life.However, it is unknown how NIRS signal reflects cortical activity.To make sure validity for NIRS signal, we measured cortical activity by NIRS when subjects generated force by their fingers. NIRS signal correlated with force level.Then we tried to predict actual force level from NIRS signal by using basic methods to examine how much NIRS has cortical information.This result suggests NIRS may be useful for brain-computer interface.

## P1-k24 Taste preferences and activation in the association cortex in humans.

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Recent studies have shown that the orbitofrontal cortex, insular/operculum, or anterior cingulate cortex are activated by giving taste stimulus. However, little is known about the relationships between taste preferences and brain regional activations. In this study, we used an fMRI in humans to evaluate this neural interaction. When the subjects received the foods whose taste are either pleasant or unpleasant, various areas in the association cortex in addition to the above regions were significantly activated, showing different activation patterns in each subject. The data suggest that taste preferences may be related to corticocortical integration processes in the association cortex.

## P1-k25 Activation of prefrontal area by attention task (Kana Pick-out Test)

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The Kana Pick-out Test is well thought, as a suitable test requiring attention for screening persons with mild cognitive impairment (MCI) in Japan. However at present, little quantitative data is known about brain regions that are utilized during this test. In this study, we assessed brain regional activities during the computerized Kana Pick-out Test using an fMRI. This test resulted in significant increase of blood oxygenation level-dependent signals in the prefrontal area (PFA), sensorimotor area, parietal association area and visual cortex. Activation of PFA by Kana Pick-out Test was greater than that when the story was only read. This study using an fMRI proved the hypothesis that Kana Pick-out Test is reflected PFA function.

KAKENHI (18791456)

## P1-k26 Pre-surgical mapping of the brain with brain tumor

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To determine the location of central sulcus (CS) and dominant hemisphere of the language is important for presurgical mapping. We use fMRI for the patient who needs a surgery. We considered the efficacy of fMRI in our institute, retrospectively.55 patients were examined. We use 5 tasks, that is, tactile stimulation to bilateral palm, finger tapping, verb generation, news listening, picture naming, and select suitable tasks with the clinical condition and the location with brain tumor. CS could be identified 96% and 85%, representatively by tactile stimulation and finger tapping in the affected hemisphere. The laterality of dominant hemisphere of language could be identified 78%, 59%, 58% by verb generation, news listening, picture naming, representatively.

## P1-k27 Nonlinear local neurovascular coupling in the cerebral cortex

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We will present a biophysical model of how electrical and hemodynamic brain signals could be generated within a cortical microcolumn. The model has three components. The first is the neural mass model of three neuronal populations that responds to incoming excitatory inputs. The second and third components model the generation of measured electrical and hemodynamic meso-states, respectively. We reformulate the model in the context of the continuous-discrete state space theory in order to make inferences about the unknown parameters and the unobservable states (i.e. membrane potentials, ionic currents, NO, flow-inducing signal, CBF, CBV, dHb) from the local linearization filter and the innovation approach. Inference on this model is performed from concurrent EEG/fMRI signals obtained during a visual experiment in which a subject observes a radial checkerboard with pattern reversal at 4 Hz.

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## P1-k28 Suppression of neural activity and hemodynamic responses during hypercapnic challenges in rats

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To investigate the effect of hypercapnia on the neural and hemodynamic responses, we measured electrophysiological signals (local field potentials, LFP<100 Hz; multiunit activity, MUA>300 Hz), optical intrinsic signals (586 and 605 nm, indicators of CBV and oxygenation) and spectral changes in reflected light. Rats were anesthetized with $\alpha$-chloralose and artificially ventilated with muscle relaxants. Hypercapnic challenge was performed by 10 % $CO_2$ inhalation. We recorded these signals while delivering 2-s electrical pulses to the hindpaw. We observed that hemodynamic responses (CBF, CBV) were suppressed more strongly than neural responses (LFP, MUA), and prolonged deoxyhemoglobin increases in the early phase with attenuation of deoxyhemoglobin decreases in the late phase (i.e., disruption of hyperoxygenation responses). These results suggested that transfer functions from neural to hemodynamic responses may be different between normo- and hypercapnia, and imaging of early dip optical signals and positive BOLD fMRI signals may not accurately reflect neural responses during respiratory depression.

## P1-k29 The application of pharmacological MRI in manganese enhancement of brain

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Manganese(Mn)-enhanced MRI (MEMRI) studies have been carried out extensively to visualize brain in vivo. In this paper, we observed much lower contrast enhancement in MEMRI in rats anesthetized with ketamine, compared with other anesthetics (urethane, pentobarbital, isoflurane). Ketamine is well known to be the noncompetitive antagonist for the NMDA receptors (NMDAR). Therefore, the contrast enhancement in MEMRI might be activity-dependently controlled by the NMDAR. To confirm these effects, the effects of both NMDAR and AMPAR antagonists, MK-801, and NBQX, respectively, on MEMRI were examined. When rats were treated with MK-801 remarkably inhibited the signal enhancement by $Mn^{2+}$ as well as ketamine. In contrast, treatment of rats with NBQX failed to inhibit the enhancement of the signal intensity in MEMRI. These MEMRI results suggest that the signal enhancement in MEMRI in rat brains is affected by NMDAR, but not AMPAR.

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## P2-a01 Actin-dependent regulation of Shank1 dynamics in Purkinje cell dendrites

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Purkinje cell dendrites possess the highest number of spines among CNS neurons. Though critical in cerebellar circuit formation, the molecular mechanisms underlying the formation and functional maturation of Purkinje cell spines are yet poorly understood. Ongoing studies suggest the existence of a tight control mechanism by which actin can efficiently accumulate in Purkinje cell spines. To gain further insights on the role of actin cytoskeleton in the organization of protein assembly at the postsynaptic density (PSD) of Purkinje cells, we studied the distribution and the re-distribution of several PSD molecules present in the Purkinje cell spines upon treatment with various actin cytoskeleton-modifying reagents. Interestingly, coincidence of actin depolymerization with a prolonged 55 mM-KCl depolarizing stimulation caused a robust mobilization of a key PSD scaffold molecule, Shank1, from the synapses to the dendritic shaft. Thus, neuronal activity and actin cytoskeletal reorganization may critically regulate Purkinje cell PSD assembly via control of Shank dynamics.

KAKENHI 17023010

## P2-a02 Existence of specific binding sites for Cbln1 in the hippocampus

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Cbln1 is a newly identified antegrade transneuronal modulator. It is released from cerebellar granule cells and regulates synaptic integrity at parallel fiber-Purkinje cell synapses in the cerebellum. Indeed, we previously reported that recombinant Cbln1 specifically bound to Purkinje cell dendrites. Although Cbln1 mRNA is also highly expressed in the entorhinal cortex, its function in the hippocampus has remained uncharacterized. As a first step to address this issue, here we performed binding assays using hippocampal slice preparations. Although Cbln1 mRNA is not expressed in the hippocampus, we found that recombinant Cbln1 bound preferentially to the dentate molecular layer and the stratum lacunosum-moleculare in the hippocampus. Thus, like Cbln1 in the cerebellum, Cbln1 may serve as a new transsynaptic modulator released from presynaptic neurons in the entorhinal cortex, bind to the specific binding sites in the postsynaptic neurons in the hippocampus, and possibly regulates synapse formation and synaptic plasticity.

## P2-a03 Characterization of the excitatory synaptic transmission between granule cells and interneurons in rat cerebellar cortex

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Repetitive activation of parallel fibers, biforked axons of granule cells (GCs), at short intervals elicits a well-known short-term presynaptic plasticity, a paired-pulse facilitation of the amplitude of excitatory postsynaptic currents (EPSCs) recorded from Purkinje cells. GCs innervate not only onto Purkinje cells but also to GABAergic interneurons. We here report that repetitive activation of GC axons at short intervals (30-100 ms) caused a novel presynaptic plasticity, a paired-pulse facilitation of the decay-time constant ($PPF_{decay}$) of the EPSCs recorded from the interneurons. The $PPF_{decay}$ was significantly suppressed by the low-affinity competitive antagonist of AMPA receptors, but unchanged if postsynaptic AMPA receptors were prevented from the saturation by pharmacological reduction of the glutamate contents in the presynaptic vesicles. The result suggests that the $PPF_{decay}$ is due to increase in the sequential mobilization of the multiple vesicles upon repetitive activation of the GC axon terminal.

## P2-a04 CNQX/AP5-resistant seizure-related oscillations in rat hippocampal slices

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We have previously shown that glutamatergic and GABAergic transmissions are required for neuronal synchronization in seizure-like oscillations induced by tetanus stimulation in rat hippocampal slices. Here, we found that pyramidal cells received rhythmic synaptic input following the tetanus even in the presence of the ionotropic glutamate receptor antagonists, CNQX and AP5. The CNQX/AP5-resistant rhythmic input was detected as depolarizing responses when membrane potentials were recorded through patch-clamp electrodes filled with high (40 mM) $Cl^-$. The reversal potentials of CNQX/AP5-resistant rhythmic input were -73.9±9.5 mV (n=7) corresponding to $GABA_A$ responses in voltage-clamp recordings through normal (4 mM) $Cl^-$ electrodes. We also observed that fast-spiking interneurons in the stratum pyramidale exhibited tetanus-induced, CNQX/AP5-resistant oscillations with robust firing. These results suggest that the interneuron network per se may generate the glutamate-independent oscillations related to seizure-like activity.

## P2-a05 Dendro- and synapto-architecture of the rat hippocampus CA3

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We analyzed the dendro-architecture of CA3 pyramidal neurons using intracellular injection of HRP, and the synapto-architecture on a montage electron micrograph. In total 3,353 synapses were analyzed to quantify the inputs to CA3 pyramidal neurons as follows. The spine densities of the dendrite of CA3 pyramidal neuron were 5.3, 14.7, 14.8 per 10 μm in the strata lacunosum-moleculare, radiatum, and oriens, respectively. The size of the spines and the related axonal terminals were closely correlated, but the size of the dendritic shaft and the related axonal terminals only showed a small correlation. Based on these results, we will present summary diagrams that depict 1) the relationship between CA3 pyramidal neurons and their inputs, and 2) the input strength of all kinds of excitatory and inhibitory synapses in each stratum.

## P2-a06 High potassium-induced facilitation of transmitter release in the absence of extracellular calcium

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The high potassium-induced potentiation of spontaneous glycine release in extracellular $Ca^{2+}$-free conditions was studied in mechanically dissociated rat spinal dorsal horn neurons using whole-cell patch-clamp technique. Elevating extracellular $K^+$ reversibly increased the frequency of spontaneous IPSCs in the absence of extracellular $Ca^{2+}$. The high potassium-induced increase in IPSC frequency was also observed in the absence of extracellular $Na^+$, although the recovery back to baseline levels of release was prolonged under these conditions. The action of high potassium solution on glycine release was prevented by BAPTA-AM, by thapsigargin and by the phospholipase C inhibitor U-73122. The results suggest that the elevated extracellular $K^+$ concentration causes $Ca^{2+}$ release from internal stores, and may reveal a novel mechanism by which the potassium-induced depolarization of presynaptic nerve terminals can regulate intracellular $Ca^{2+}$ concentration and exocytosis.

KAKENHI(18021027, 18500311)

## P2-a07 Dendritic filopodia formation is mediated by the interaction between telencephalin and ERM proteins

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Dendritic filopodia are long, thin, and dynamic protrusions that play a critical role as a precursor of spines. A telencepalon-specific cell adhesion molecule, telencephalin (TLCN), is expressed in dendritic filopodia, facilitates the filopodia formation, and slows spine maturation. Here we demonstrate that TLCN cytoplasmic region binds ERM family proteins that link membrane proteins to actin cytoskeleton. In cultured hippocampal neurons, phosphorylated ERM proteins were co-localized with TLCN in dendritic filopodia. Expression of constitutively active ezrin induced dendritic filopodia formation, whereas knockdown of ERM proteins decreased filopodia density and accelerated spine maturation. These results indicate the important role of TLCN-ERM interaction in the formation of dendritic filopodia.

KAKENHI 16044249, 17024063; CREST

## P2-a08 Characterization of slow outward current observed in mouse cerebellar Purkinje neurons of lobule 10 evoked by tetanic parallel fibers stimulation in slice

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Cerebellum consists of ten lobules in mice and some functional differences between lobules have been reported. In this study we recorded from Purkinje neurons (PNs) and focused on the slow outward current evoked by tetanic parallel fibers (PFs) stimulation in lobule 10. Recordings were obtained by whole-cell patch clamp at -70 mV in the presence of blockers of AMPA and $GABA_A$ receptors in sagittal section of postnatal 14-18 days mice. It is well known that slow inward current (mGluR current) is observed in PN by tetanic PFs stimulation. In lobule 10 but not lobule 3 or 9, we observed a faster transient outward current by tetanic PFs stimulation in addition to the transient inward current. The outward current was not inhibited by mGluR1 antagonist, SK and BK channel blockers, intracellular $Cs^+$, or 10 mM BAPTA, but was inhibited by TTX. This may be a novel inhibitory mechanism of Purkinje neurons by PFs.

## P2-a09 Analysis of subcellular localization of synapses in *C. elgans* RIA interneuron

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Neural signal is transmitted via synapses, and thus subcellular localization of synapses is important for neural function. RIA interneuron essential for thermotaxis in *C. elegans* has a single neurite, but pre and post synaptic region are clearly devided within it, which is a suitable model for the analysis of subcellular localization of synapses. We conducted a screen for the mutants defective in localization of synapses in RIA by observing the presynapses using synaptic vesicle protein SNB-1 fused to fluorescent protein. From this screen we isolated seven mutants, although the penetrances of their phenotypes are low. Recently, we reported that *ttx-7* mutant lacking Inositol Monophosphatase (IMPase) shows abnormal localization of synaptic proteins in RIA, which leads to thermotaxis defect (Tanizawa *et al.*, Gens Dev, 2006). We are now attempting to isolate suppressors for *ttx-7*. The screen has utilized the thermotaxis assay, which enables us to screen more genomes than screen with direct observation of synapses. We will then examine suppressor candidates as to whether the localization defect of synaptic proteins are also suppressed.

## P2-a10 Mechanisms of $GABA_B$ receptor-mediated inhibition of burst activities in mouse superior colliculus

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We examined the role of $GABA_B$ receptors ($GABA_B$Rs) for burst activities of neurons in the superior colliculus (SC), which may be related to saccade initiation, using slice preparations obtained from GAD67-GFP knock-in mice. Single electrical stimulation of the superficial gray layer (SGS) evoked burst EPSCs in non-GABAergic intermediate gray layer (SGI) neurons in the presence of a $GABA_A$R antagonist. Addition of $GABA_B$R antagonist CGP52432 (CGP) greatly increased the duration of the burst EPSCs, indicating that synaptically released GABA activates $GABA_B$Rs. Similar effects were observed when CGP was locally applied to the SGS but not to the SGI, suggesting $GABA_B$Rs localization in the SGS. Based on the results from a series of experiments, we concluded that both pre- and postsynaptic $GABA_B$Rs localized in the SGS can be activated by synaptically released GABA during burst activities and limit the duration of burst activities in the SC.

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## P2-a11 Involvement of Homer1a in the regulation of distribution of postsynaptic proteins

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Homer1a is induced by neural activities such as LTP or seizure. In cultured rat hippocampal neurons, we examined the changes in the distribution of synaptic proteins such as PSD95, Homer1c, or F-actin, after neuronal stimulations. When the cells were stimulated with glutamate, the numbers of the clusters of these proteins in dendrites decreased transiently for 1 h, then recovered, and several hours later reached the levels higher than the initial level before stimulation. The time courses of the biphasic changes in the distribution of these proteins are indistinguishable from each other. These changes were suppressed when the cellular Homer1a was knocked down by RNAi before the stimulation. It is suggested that the distribution changes may represent remodeling of the synapses through biphasic de-clustering and re-formation processes, and that Homer1a may play important roles in these processes.

## P2-a12 Activity-dependent regulation of glutamate receptor localization in *Drosophila* neuromuscular synapses

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It is well known that neural activities can modulate synaptic responses. However, effects of neural activities on localization of neurotransmitter receptors have not been fully understood. Here, we investigated how neural activities modulate the content of two major components of glutamate receptor (GluR), GluRIIA and IIB, in *Drosophila* larval neuromuscular synapses. In *eag-shaker* mutants, that display greatly enhanced nerve activity, the content of both GluRIIA and IIB in synapses was enhanced. On the other hand, it was decreased in *para*$^{ts1}$ mutants if the synaptic activity was prevented at elevated temperatures. These results were a contrast to our previous findings that postsynaptic activation of CaMKII decreased GluRIIA but increased IIB content. Taken together, these results suggest that the neural activity modulates GluR content of synapses through CaMKII-independent way and there are several mechanisms that regulate the localization of GluR in synapses.

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## P2-a13 Origins of the spontaneous depolarization wave in the embryonic rat CNS revealed by optical imaging with a voltage-sensitive dye

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We previously reported that a widely-propagating correlated neuronal activity (depolarization wave) is evoked by various sensory inputs in the embryonic CNS. Here, we examined the spontaneous depolarization wave and its origins. In E15-E16 rat embryos, spontaneous signals appeared in association with the rhythmic discharges of cranial MNs, and propagated widely. At E15, the spontaneous wave mostly originated in the cervical-upper lumbar cords. At E16, the wave was predominantly generated in the lumbosacral cord, although a wave associated with the second oscillatory burst was initiated in the rostral cord. At E16, a few waves also originated in the rostral ventrolateral medulla and the dorsomedial pons. When the influence of the caudal cord was removed, the contribution of the medulla and pons became more significant. These results show that the depolarization wave can be triggered by the spontaneous activity of multiple neuronal populations.

## P2-a14 An F-BAR/EFC protein Rapostlin regulates dendritic spine development

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Dendritic spines are tiny protrusions that receive most excitatory synaptic inputs. Dendritic spines show structural plasticity during development and in response to synaptic activity. However, the regulation mechanism of membrane trafficking for spine formation is poorly understood. Here we studied neuronal function of Rapostlin that has an F-BAR/EFC domain with a membrane invaginating property. Rapostlin is strongly expressed in developing brain including hippocampus and cerebral cortex in late developmental stages. Knockdown of Rapostlin by siRNA in primary cultured rat hippocampal neurons reduces spine density and size. These results suggest the implication of Rapostlin in spine development through the regulation of membrane trafficking. We are now further dissecting the detailed function of Rapostlin in spine formation.

KAKENHI(17079003)

## P2-a15 SAD kinase is a presynaptic component of mouse neuromuscular junctions.

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The serine/threonine kinase SAD regulates several synaptic functions such as synapse development, axon/dendrite polarization, and neurotransmitter release. In mammalian central nervous system (CNS), SAD is localized on synaptic vesicles and at the active zone in nerve terminals where SAD appears to phosphorylate the active zone protein RIM1 implicated in neurotransmitter release. However, expression and localization of SAD in peripheral nervous system (PNS) is currently unknown. Here we have attempted to examine its expression and localization at synapses of neuromuscular junctions (NMJs). In the mouse diaphragm, quadriceps femoris muscle, and lumbrical muscle, the immunoreactivity for SAD is colocalized with that for $\alpha$-bungarotoxin, a major marker for NMJs, which binds to acetylcholine receptors. These results suggest that SAD is localized at NMJs and a presynaptic component at the PNS as well as the CNS.

## P2-a16 GLAST is essential for cytological differentiation of Bergmann glia and climbing fiber monoinnervation by suppressing local ectopic innervation

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Glutamate released to the synaptic cleft has to be removed by glutamate transporter. In the cerebellum, glutamate transporter GLAST is richly expressed in Bergmann glial cells (BG) that enwrap synapses, dendrites and somata of Purkinje cells (PCs). In this study, anatomical analysis was applied to GLAST-deficient mice. In the mutant mice, lamellate processes from BG fibers became atrophic, resulting in incomplete sealing of synaptic cleft. Furthermore, regressive and abberant CF innervation was revealed. In light and electronmicroscopic analysis, local and ectopic innervation of ascending axons or transverse collaterals caused multiple CF innervation. These results suggest that GLAST is essential for cytological differentiation of BGs and for the establishment of CF mono-innervation by suppressing local and latreral innervation of CFs.

## P2-b01 Two types of synchronous timing in a somatosensory thalamocortical circuit

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Sensory (visual, auditory, somatosensory) signals are transferred to the neocortex, by way of the thalamus. Different types of synchronous firings have been reported in the thalamus, but it is still unknown whether these different types of synchronous firings have same or different impacts on recipient cortical neurons. To address this issue, we prepared brain slices where the thalamus and the neocortex are synaptically connected, and studied how two types of synchronous inputs (coincident and time-lagged inputs) from the thalamus are received by regular-spiking (RS) cells, principal cells in layer 4 of the neocortex. When thalamocortical inputs were not coupled to feedforward inhibition, cortical RS cells were strongly excited by synchronous but time-lagged thalamic inputs. Conversely, when thalamocortical inputs were coupled to feedforward inhibition, RS cells were strongly excited by coincident thalamic inputs. Thus, the two types of synchronous timing have different impacts on cortical RS cells.

## P2-b02 Making the knock-in mice that lack the interaction between CaMKII and syntaxin-1A

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Exocytotic synaptic vesicle recycling is regulated at several steps by different concentrations of $Ca^{2+}$. Particularly, submicromolar $Ca^{2+}$ is believed to be important for the vesicle translocation/tethering and docking, however, the molecular mechanisms underlying these processes are poorly understood. We previously found that the autophosphorylated CaMKII interacts with syntaxin-1A (Stx1A) in the presence of submicromolar $Ca^{2+}$, and demonstrated that, at cellular level, this binding modulates the frequency of exocytosis. This interaction is strongly dependent on R151 of Stx1A, a highly conserved residue in the linker domain of Stx1A, and R151G Stx1A completely lost the activities both binding to CaMKII and modulating exocytosis. To understand the significance of this interaction more in detail, we have generated knock-in mice having R151G Stx1A. So far, the homozygous mutants are developing without large defect, and we are now investigating the mice from the biochemical aspects.

KAKENHI(1723019)

## P2-b03 CO1305 encoded calcium (Cec) channels participate to active zone endocytosis and maintain synaptic transmission at low demand of vesicles.

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At synapse exocytosis of synaptic vesicles (SVs) is followed by endocytosis. We proposed that there were active zone and non active zone endocytosis in presynaptic boutons of Drosophila. Extents of exocytosis and endocytosis of SVs were determined by the amounts of FM1 43 released from and loaded in boutons, respectively. Bay K increased Ca in boutons. When Bay K was exposed to non treated boutons, no release and no load of FM1 43 were observed. When Bay K was exposed after high K in the presence of La which had caused exocytosis but not endocytosis, FM1 43 was loaded in boutons. This result suggests that Bay K activates Ca channels for endocytosis. In CG1305 mutant, beta subunit Ca channel mutant, Bay K caused no increase in Ca and no FM1-43 load. CG1305 antibodies stained active zone areas in boutons of wild type. CG1305 mutant showed irregular synaptic potentials evoked at low frofequency, and depression in the early period at high frequency

## P2-b04 Glutamate receptor δ2 subunit induces presynaptic differentiation of a cerebellar granule neuron

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The glutamate receptor δ2 subunit (δ2) is selectively expressed on the postsynaptic membrane at synapses between granule and Purkinje neurons (GN and PN). Implication of δ2 in formation or maintenance of the synapse as well as cerebellar long-term depression has been demonstrated. However, little is known about how δ2 is involved in synapse formation. Immunofluorescent staining showed that the presynaptic terminals were smaller in size and lower in density in the cerebellar culture prepared from δ2-knockout mice. Accumulation of GN presynaptic terminals on the δ2-transfected HEK cells in a co-culture was observed. The N-terminal domain of δ2 was sufficient for the accumulation. Further, the HEK cells transfected with both δ2 and ionotropic glutamate receptor GluR1 subunits in the co-culture exhibited glutamatergic EPSC-like transient inward currents. These results suggest that δ2 is capable to induce differentiation of presynaptic terminals of GNs.

## P2-b05 Imaging glutamate spillover from synaptic clefts using the fluorescent glutamate indicator

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Accumulating evidence suggests that glutamate released from presynaptic terminals escapes from synaptic clefts to activate receptors at the extrasynaptic site and the neighboring synapses. This phenomenon, which is known as glutamate spillover, has been indirectly assessed by the measurement of excitatory postsynaptic currents. We recently developed a novel fluorescent indicator of glutamate (glutamate (E) optical sensor, EOS) making use of the glutamate-binding domain of GluR2. In this study, we applied EOS to a direct measurement of glutamate around parallel fiber (PF)-Purkinje cell synapses. We succeeded in imaging the increase in glutamate concentration in extrasynaptic site upon PF stimulation. In addition, increased number of PF stimulation induced long-distance and wave-like glutamate transients that resulted in synaptic cross talk. These direct measurement of extrasynaptic glutamate would provide us deeper understanding of glutamate spillover.

## P2-b06 Functional development of synaptic connections in the chick auditory system: Optical imaging with a voltage-sensitive dye

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An optical survey of auditory network formation was performed in the E5.5-E8 chick N. VIII-brainstem preparations. In the E7-E8 medulla, we identified 4 response areas; the ipsilateral Nucleus magnocellularis (NM) and Nucleus angularis (NA), and the ipsi- and contralateral Nucleus laminaris (NL). In NM, NA and NL, the EPSP-related slow signals were detected from some E6 and all E7 and E8 preparations, indicating that functional synaptic connectivity arises by the E7 stage. In the E7-E8 pons, we identified 2 additional response areas; the ipsi- and contralateral Nucleus lemnisci lateralis (NLL). Furthermore, we detected responses from the contralateral cerebellum, which possibly correspond to transient projections observed only during embryogenesis. The present study demonstrates that the basic functional circuits related to auditory function are established in the chicken embryo at earlier stages than previously reported.

## P2-b07 Information content of synapses with realistic short-term dynamics

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Short-term synaptic plasticity is a common phenomenon for central synapses. However,the functional role of short-term plasticity is not fully understood. Here we apply information theoretic measurements to evaluate the mutual information between the inter-spike intervals (ISI) of the presynaptic spike trains and the membrane potential of postsynaptic responses(PSR). For each type of dynamics, there exists an optimal frequency range. Comparing the optimal information transmission frequency range with physiological effective input frequencies, we suggest the optimal parameters for the model synapse. We show that the activity-dependent recovery extends the optimal frequency range of the information transmission. We also measure the mutual information between PSR and its preceding spikes and observe the increase of the information about the preceding 2 or 3 spikes in an activity-dependent recovery synapse compared to a synapse with a constant recovery rate. Our study suggests that the activity-dependent recovery plays an important role in optimizing the information transmission via synapses.

## P2-b08 GABAB receptor-mediated modulation of glycine release in rat substantia gelatinosa neurons

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$GABA_B$ receptor-mediated presynaptic inhibition of glycinergic transmission was investigated in immature rat spinal cord using a whole-cell patch clamp technique. Action potential-dependent glycinergic inhibitory postsynaptic currents (eIPSCs) were recorded from substantia gelatinosa neurons. Baclofen greatly reduced the amplitude of glycinergic eIPSCs and increased the paired-pulse ratio. The baclofen-induced inhibition of eIPSCs was partially attenuated by forskolin, an adenylyl cyclase (AC) activator, but not affected by SQ22536, an AC inhibitor. The partial blockade of baclofen action on IPSCs by forskolin was reversed by KT5720, a selective PKA inhibitor. The baclofen-induced inhibition of eIPSCs was greatly attenuated by $Ca^{2+}$ channel blockers. The results suggest that presynaptic $GABA_B$ receptors inhibit action potential-dependent glycinergic transmission via the selective blockade of presynaptic $Ca^{2+}$ channels, without affecting the AC/cAMP signal transduction pathway.

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## P2-b09 Requirement of tonic and phasic dopamine signals in long-term potentiation of prefrontal synapses

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Prefrontal cortex (PFC) is involved in certain types of long-term memory. Using rat PFC slices, we study long-term plasticity of PFC glutamatergic synapses and its modulation by dopamine (DA). We have previously shown that bath-application of DA (100 microM, 12.5 min) facilitates the induction of long-term depression (LTD). This LTD converted to long-term potentiation (LTP) when background/tonic DA was provided prior to the conditioning. Here we characterized this effect of tonic DA on LTP as well as the role of phasic release of DA in LTP. First, we revealed that to facilitate LTP, the concentration of tonic DA has to be within optimal range (between 1 and 10 microM). Second, the tonic DA signal has to be accompanied by synaptic inputs presumably acting on NMDA receptors. Third, for LTP induction, phasic release of endogenous DA during conditioning stimuli is necessary. Fourth, tonic DA slowly activates MAP kinases, and these kinase activations are causally related to LTP. Based on these results, we propose cellular models for physiological and pathological cognitive processes in the PFC.

## P2-b10 Short-term plasticity defines postsynaptic excitation pattern in the solitary complex neurons of the rat

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To understand how visceral afferent information is converted into neuronal activities in the brain network, we have been analyzing the short-term plasticity of the synapse between the primary afferents and the second-order neurons in the solitary complex (SC). We have previously demonstrated the short-term plasticity is the primary determinant of the characteristics of the frequency-dependent filtering at these synapses under the voltage-clamp recording condition devoid of inhibitory transmission. To examine whether these frequency-filtering properties affect the postsynaptic firing patterns in the presence of inhibitory transmission, postsynaptic potential and action potentials were recoded from neurons in the SC. The results indicate that, even in the presence of inhibitory connections, the properties are still under strong influence of the presynaptic release mechanisms, suggesting that release machinery of each afferent neuron primarily defines its frequency-dependent influence to the secondary network.

KAKENHI(17023042)

## P2-b11 Maturation of thalamocortical transmission to GABAergic neurons and its possible relevance to plasticity in the barrel cortex

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Thalamic inputs to layer IV cortical cells are often followed by a rapid inhibition. For such feedforward inhibition to work, thalamic inputs to GABAergic neurons must be substantially earlier to arrive. We investigated latencies of multiple EPSPs simultaneously recorded from a single barrel elicited by thalamus in the thalamocortical slices. We found that latencies for GABAergic cells were always shorter than those for excitatory cells, irrespective of location of the cells. This was achieved by the differences in conduction velocities. Our results suggested that axons innervating GABA cells had larger diameter. Relevance to spike timing dependent plasticity in the barrel cortex will be discussed.

KAKENHI18021022

## P2-b12 Effect of X-irradiation on dendritic spine morphology of hippocampal neurons

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We previously reported that distinct radiosensitivity for the cell viability between mature and immature neurons. In the present study, we analyzed the effect of X-irradiation on dendritic spine-resident proteins immunocytochemically. We prepared primary neuronal cultures with Banker method. X-irradiation (30 Gy) was performed on the neurons at 21 days in vitro (DIV). After X-irradiation, the cells were immediately fixed and immunostained with antibody against to a presynaptic marker Synapsin I, and a postsynaptic actin-cytoskeleton regulator drebrin. After X-irradiation, drebrin cluster density along dendrites was significantly decreased, but that of Synapsin I was not. These findings show that X-irradiation induces the change in the postsynaptic actin-cytoskeleton, and suggest that it induces the synaptic dysfunction related to the abnormal spine morphology.

21st Century COE from JSPS

## P2-b13 Early postnatal stress alters the prefrontal synaptic response during extinction of contextual fear-related behavior in adult rats

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The effect of early postnatal stress on the neuronal regulation in the medial prefrontal cortex (mPFC) during context-dependent fear extinction was examined under consciousness. Adult rats exposed to footshock (FS) at the postnatal period of the third week (3W-FS) indicated the impaired extinction of contextual fear-related behavior, freezing. In the extinction session, the restoration of the inhibitory synaptic response in the mPFC observed in Non-FS controls was attenuated in 3W-FS. The neuronal and behavioral deficits observed in 3W-FS were prevented by single treatment with the 5-HT1A receptor agonist tandospirone (1 mg/kg, i.p.). These results suggest that early postnatal stress affects the neural circuit responsible for the extinction of context-dependent fear memory, which may be involved in serotonergic mechanism(s) mediated via 5-HT1A receptors.

## P2-b14 Neural mechanism of extracting behaviorally-relevant features of stimulus by short-term synaptic plasticity in electrosensory perception

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To investigate the neural mechanism of extracting behaviorally-relevant features of stimulus by short-term synaptic plasticity, we studied the functional roles of short-term synaptic plasticity in electrosensory perception under competing signals of electrolocation and electrocommunication. Weakly electric fish can detect object nearby the fish, so called electrolocation, and communication signals from a neighboring electric fish ,so called electrocommunication, using amplitude modulations of their self-generated electric organ discharge (EOD). We developed a model of fish body by which we calculate numerically the spatiotemporal patterns of electric field around the fish body, and made the model of electrosensory system which consists of receptors, hindbrain, and midbrain. In this paper, we show that the short-term synapses of hindbrain neurons are used in order to efficiently extract the spatiotemporal features required for electrolocation and communication from the competing signals.

## P2-b15 Phosphorylation of the δ2 glutamate receptor is not required for induction of long-term depression in cerebellar Purkinje cells

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Although cerebellar long-term depression (LTD) has a lot in common with LTD in other regions of the brain, there is a crucial difference-requirement of the δ2 glutamate receptor (GluRδ2). Nevertheless, the mechanisms by which GluRδ2 functions have been elusive. We showed that a protein kinase C activator induced postsynaptic LTD and phosphorylation of a serine residue at position 945 in the C-terminus of GluRδ2. To investigate the causal relationship between this phosphorylation and LTD, we prepared a Sindbis virus vector encoding a mutant GluRδ2, in which serine was replaced with alanine (S945A), and infected to GluRδ2-null Purkinje cells. Electrophysiological experiments in acute slice preparations revealed that like wild-type GluRδ2, GluRδ2-S945A could rescue the impaired LTD of GluRδ2-null Purkinje cells. Although the phosphorylation at S945 of GluRδ2 is reported to enhance its affinity to MAGI-2, our results indicate that it is not required for induction of LTD.

## P2-b16 Rapid actions of sex steroids in hippocampal dendritic spines

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It is believed that sex hormones are synthesized in the gonads and reach the brain via the blood brain circulation. In contrast with this view, we demonstrated that sex hormones are also synthesized locally in the hippocampus and that these steroids act rapidly to modulate neuronal synaptic plasticity. Dendritic spines of hippocampus are the sites of excitatory synaptic inputs. Here, we demonstrate that estrogens and androgens rapidly increased the spine density and morphology in the stratum radiatum of the CA1 region. These effects are probably mediated by synaptic steroid receptors. The involvement of MAPKs will be discussed.

## P2-b17 Locus coeruleus modulates hippocampal CA3 synaptic transmission

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Locus coeruleus (LC), a nucleus of norepinephrine (NE) neurons, gives the major NE projections to hippocampus. In this study, we isolated perforant path (PP)-CA3 monosynaptic responses and found that bidirectional synaptic plasticity, LTP and LTD, can be induced in this pathway in anesthetized SD rats. Injection of glutamate (Glu) to LC resulted in expression of c-Fos in LC and induced a transient potentiation at PP-CA3 synaptic responses without effecting on subsequent LTP and LTD induction. Repeated Glu injection to LC induced reproducible PP-CA3 potentiation. Potentiation of PP-CA3 synapses induced by glutamatergic activation of LC was inhibited by high dose of prapranolol. These results suggested that LC modulates the function of hippocampal CA3.

## P2-b18 Impaired delay, but normal trace classical eyeblink conditioning in the Cbln1 mutant mice

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Cbln1 is a member of the C1q/TNF superfamily and is predominantly produced in cerebellar granule cells. Recently, we have shown that the Cbln1 knockout mice display severe motor discoordination and impaired long-term depression in cerebellar Purkinje cells, suggesting that secreted Cbln1 plays crucial roles in synaptic plasticity and synaptic integrity of at the parallel fiber-Purkinje cell synapses. In order to investigate the function of Cbln1 at the behavior level, we performed classical eyeblink conditioning in the Cbln1 knockout mice. The Cbln1 knockout mice showed the impaired eyeblink conditioned responses in the delay paradigm, while the trace eyeblink conditioned responses were normal. There was no significant difference in the acoustic startle responses and the pain threshold between the Cbln1 knockout mice and the control. These results suggest that Cbln1 plays important roles in motor learning.

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## P2-b19 Accelerated maturation of visual response properties in telencephalin knockout mice

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Receptive field properties, such as orientation selectivity (ORI) and ocular dominance (OD), are shaped independently by experience through NR2A-PSD95 signaling and structural modification of dendritic spines, respectively (Fagiolini et al, 2003; Mataga et al, 2004). The adhesion molecule telencephalin (TLCN) regulates the transition from dendritic filopodia to mature spines. Here, we examined the role of TLCN in the emergence of ORI and OD. Mice lacking TLCN (KO) already exhibited high PSD95 content and ORI at eye-opening similar to adult values. Moreover, dark-rearing had no detrimental effect on ORI in KO mice. OD plasticity was also prematurely activated and saturated more rapidly upon brief monocular deprivation, consistent with early transition to mature spines in the KO mouse. Thus, TLCN acts as a 'brake' to ensure that functional maturation of selected visual response properties occurs with appropriate timing and sensory input. *equal contribution

RIKEN BSI & JST

## P2-b20 Glucagon-like pepetide-1 modulates synaptic transmission and long-term depression in the CA1 area of hippocampal slices

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Glucagon-like peptide-1 (GLP-1) is a proglucagon-derived peptide-hormone in the intestine and the brain. We reported that GLP-1 reduced the frequency of the θ wave of the rat's hippocampus, and improved amnesia in continuously β-amyloid-infused rats. However, it is unclear that how GLP-1 affects hippocampal synaptic transmission. In this study, we investigated the effects of GLP-1 on the synaptic function in acute hippocampal slices using electrophysiological technique. Hippocampal slices (400 μm) were prepared from Wistar rats (3-4 weeks). Field excitatory postsynaptic potentials (fEPSP) in the CA1 region were evoked by stimulation with bipolar tungsten electrodes, placed in the Schaffer collaterals. A high dose of GLP-1 (1000 nM) facilitated the slope of fEPSP. In contrast, the lower dose of GLP-1 (10 nM) had no effect on the fEPSP slope, paired-pulse ratio or the long-term potentiaion, but facilitated the long-term depression. These results suggest that GLP-1 modulates basal synaptic transmission and synaptic plasticity in the hippocampus.

## P2-b21 Critical time window of protein synthesis required for LOSS, a long-lasting decrease in synaptic strength accompanied by synapse elimination.

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Long-lasting synaptic plasticity is considered as the cellular basis of long-term memory. We previously found in the cultured rat hippocampal slices that the induction of LTD, only when repeated 3 times, caused a long-lasting (>3w) decrease in the synaptic strength, which was accompanied by the elimination of synapses. This phenomenon, called LOSS (LTD-repetition-Operated Synaptic Suppression) to discriminate it from conventional single LTD, is expected to serve as a model of long-lasting synaptic plasticity. Although the induction of LTD by an application of DHPG (a Group I mGluR agonist) was not affected by a translation inhibitor anisomycin, the development of LOSS was blocked by it. By varying the timing of anisomycin application, we found that the drug's blocking effect had a time window, which was within 6h after each LTD induction. These results indicate that the development of LOSS requires protein(s) synthesized within 6h after the induction of LTD.

## P2-b22 Genetic analyses of mechanisms for memory erasing in *Caenorhabditis elegans*

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An acquired short-term memory is usually erased within hours, although it is sometimes transformed into a stable long-term memory. However, the mechanism of the memory erasing processes remains largely unknown. Since *C. elegans* possesses a simple nervous system and their memories usually do not persist more than hours, it is suitable to study the mechanisms of memory erasing at the molecular level. To elucidate the processes, we used two types of behavioral plasticity as models for learning, the olfactory adaptation and the salt chemotaxis learning, in *C. elegans* . By using an improved method of Mos1-transposon-mediated mutagenesis, we isolated mutants defective in memory erasing. These mutants show prolonged expression of adaptation to diacetyl or the salt chemotaxis learning, although they exhibit normal onset of the adaptation and the learning. The analyses of the genes responsible for these mutants will reveal molecular mechanisms of the memory erasing and disclose the regulation of the memory storage in neural networks.

## P2-b23 Kinetic simulation analysis of signaling cascades regulating inhibitory synaptic plasticity in a cerebellar Purkinje neuron

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At inhibitory synapses on a cerebellar Purkinje neuron (PN), postsynaptic depolarization induces long-lasting potentiation of the $GABA_A$ receptor ($GABA_AR$) responsiveness (rebound potentiation: RP). Whether RP is induced or not is determined by the pattern of pre- and postsynaptic neuronal activities through alteration of activities of protein kinases (CaMKII and PKA) and phosphatases (PP-1 and calcineurin). To understand systematically how different input patterns modulate the activity balance of kinases and phosphatases, we developed a kinetic simulation model of complex biochemical pathways regulating RP. Different stimulation patterns resulted in bistable states of CaMKII activity, presumably corresponding to whether RP is induced or not. The systematic property inherent in the molecular network was analyzed, and several experimental predictions were obtained. Some predictions were examined using cultered PNs prepared from the rat cerebellum.

## P2-b24 Acute ethanol effects on GAD65 KO mouse

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The relation between ethanol intoxication and GABA is already reported, however, lack decisive evidences. Therefore, we investigate the acute ethanol effect on behavior and hypothalamic c-fos expression, and compare with glutamic acid decalboxylase (GAD65) knock out mice and wild type mice. In this study, we performed i.p. injection of 0.8g/kg - 1.6g/kg b.w. ethanol for 8-10 weeks old male mice. Blood and brain ethanol concentration after ethanol injection was not different significantly between KO mice and WT mice. Brain GABA concentration was not also changes by ethanol injection. However, latency to fall of KO mice after ethanol injection was significantly lower than ethanol injected wild type, and in open field test, WT mice behavior was not altered by 1.2mg/kg ethanol injection, but moving distance of KO mice after 1.2mg/kg ethanol injection was significantly longer than saline injection. Moreover, c-fos expression in paraventricular nuclei occurred by more low dose ethanol in KO mice than WT mice. Taken together, GABA deficit is thought to alter the threshold dose of ethanol.

## P2-b25 Effects of enkephaline on $K^+$ currents of oculomotor neurons in brainstem slices from rats

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We examined effects of methionine enkephalin (Met-Enk) on voltage dependent slowly inactivating $K^+$ currents (IKs) in the oculomotor neurons (OMNs) of rats brainstem slices using whole-cell patch clamp techniques. IKs were evoked with depolarizing commands to between -80 to 50 mV with a conditioning pulse to -50mV in 4-aminopyridine (5mM)-and tetrodotoxin (1μM)-containing $Ca^{2+}$-free solution and were sensitive to 20mM tetraethyl ammonium. Reversal potentials of tail currents of IKs were around -100mV. Met-Enk dissolved in extracellular solution reduced IKs in dose-dependent and reversible manners. Percentage of Met-Enk-induced IKs reduction to control IKs was around 40% (100μM Met-Enk), 30% (75μM), 20% (50μM) and 10% (25μM). The reduction of IKs by Met-Enk was antagonized by opiate μ receptor antagonist, d-PHE-CYS-TYR-d-TRP-ORN-THR-PEN-THR amide (CTOP, 1μM). We conclude that Met-Enk modulated voltage dependent, TEA-sensitive and slowly inactivating $K^+$ currents in OMNs through activation of opiate μ receptors.

## P2-b26 Analysis of Glutamate-mediated control of head motor neurons through thermotaxis neural circuit

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Despite various ongoing researches, how Glutamate controls neural signals in the neural circuit of live animals still remains to be understood. We found that Vesicular Glutamate Transporter functions in thermosensory neuron AFD and its downstream interneuron RIA for theimotaxis in *C. elegans*, which suggest that thermal signals from AFD or RIA to their postsynaptic neurons are transmitted by Glutamate. Now we focus on postsynaptic neurons of RIA. Interestingly, about 90% of RIA presynapses connect to SMD or RMD motor neurons controlling the head-swing muscles. To investigate the importance of head-swing control by SMD or RMD for thermotaxis, we are monitoring the head-swing during thermotaxis and monitoring the physiological responses of SMD and RMD to thermal stimuli through Ca++ imaging. Also, we are trying to detect Glutamate receptors function in SMD or RIA for thermotaxis. Through our analysis, we hope to reveal the Glutamate-mediated signals from sensory input to behavioral motor output in thermotaxis neural circuit.

## P2-b27 Increase in the frequency of spontaneous $GABA_A$-PSC by dopamine in neonatal mouse hippocampus

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The abnormality of neonatal hippocampus may lead to the impairment of prefrontal cortex as observed in schizophrenia. Dopamine is related to such disorder. Recently the dysfunction of GABAergic interneuron is suggested as the pathophysiology of schizophrenia. We have reported that dopamine profoundly suppresses GABAergic transmission in neonatal rat hippocampus. However, effect of dopamine on spontaneous activities of GABAergic interneurons in neonatal hippocampus is unknown. In this study, we studied the effect of dopamine on GABAergic interneurons in neonatal mouse hippocampus by whole-cell patch clamp recording from CA1 pyramidal cells. The amplitudes of evoked and spontaneous $GABA_A$-PSC were decreased by dopamine, whereas the frequency of spontaneous $GABA_A$-PSC was increased by dopamine in postnatal day 0-3 mouse. These complex effects of dopamine on GABAergic transmission may be related to the pathophysiology of schizophrenia.

## P2-b28 Roles of phosholipase C-related inactive protein (PRIP) in modulating $GABA_A$ IPSCs

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Phospholipase C (PLC)-related inactive protein-1/2 (PRIP-1/2) has recently been shown to play critical roles in $GABA_A$ receptor trafficking. We aimed to study the role of PRIP-1/2 in the modulation of GABA IPSCs in layer II/III pyramidal neurons (PNs) of the barrel cortex using PRIP-1/2 double knockout (DKO) mice. The half duration of miniature IPSCs was significantly shorter in DKO than in wild-type (WT) PNs, while the opposite was the case for IPSCs evoked by stimulation of layer II/III. On the other hand, the desensitization in response to puff application (2 sec duration) of GABA (200 μM) was significantly greater in DKO than in WT PNs, while only DKO PNs displayed hump-like responses at the offset of the puff application. These results suggest that PRIP-1/2 may play essential roles in the desensitization and resensitization in $GABA_A$ receptors, as reflected in the differences in the half-durations of miniature and evoked IPSCs between DKO and WT mice.

## P2-b29 The differential effects of neuromodulators on slow component of afterhyperpolalization of layer V pyramidal cells in the rat medial prefrontal cortex

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We investigated the effects 5-hydroxytryptamine(5-HT), noradrenaline(NA), dopamine(DA) and muscarinic receptor agonist carbachol on afterhyperpolaliza tion(AHP) of layer V pyramidal cells in the rat medial prefrontal cortex. Whole-cell recordings were performed on rat brain slices maintained *in vitro* in order to measure AHP and the currents underlying AHP and spike adaptations. AHPs consist of three components, fast AHP, medium AHP and slow AHP in current clamp configuration. The currents underlying slow AHP($_sI_{AHP}$) were investigated in voltage clamp mode. We found that carbachol, 5-HT and NA reduced the slow AHP and $_sI_{AHP}$ and spike-frequency adaptation. However DA did not show the reduction in spike-frequency adaptation, although DA showed small decrease in the slow AHP and $_sI_{AHP}$. These findings suggest that neuromodulators have differential effects on pyramidal cell in rat medial prefrontal cortex and play distinct psysiological roles in regulating the excitability of these output cells.

## P2-b30 N-terminus of tyrosine hydroxylase regulates the intracellular stability of the enzyme

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We recently reported that a PEST motif ($Met^1$-$Lys^{12}$) of human tyrosine hydroxylase type 1 (hTH1) might affect the intracellular stability of the enzyme. However, it is not clear whether only the PEST motif affects the stability. Wild-type hTH1 and the 5 mutants (del-12, del-22 and del-42, the first 12, 22 and 42 amino acid residues deleted; del-Pro2 and del-Ala11, the $Pro^2$ and $Ala^{11}$ residues deleted) were expressed in AtT-20 cells. The western blot analyses revealed that the mutants del-Ala11, del-22 and del-42 were much more stable than wild-type. However, the increased stability was observed in neither del-Pro2 nor del-12, although these two mutants and del-Ala11 were used to extinguish the PEST motif. The results indicate that the stability of hTH1 depends on the sequence around $Ala^{11}$ but not the PEST motif itself.

KAKENHI(18500301)

## P2-b31 Role for synaptotagmin VII in fusion pore dynamics revealed by total internal reflection fluorescence microscopy

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Synaptotagmin (Syt) VII has been suggested to be a vesicular $Ca^{2+}$ sensor for exocytosis. However, the molecular mechanisms underlying enhancement of exocytosis by Syt VII are unclear. Here, we established a PC12 cell line that stably expresses Syt VII-GFP or its $Ca^{2+}$-binding-site-deficient mutant (Syt VII-DN-GFP), and examined the effect of Syt VII-GFP expression on the kinetics of dense-core vesicle exocytosis by total internal reflection fluorescence microscopy. Expression of Syt VII-GFP enhanced the number of dense-core vesicle exocytosis, whereas expression of Syt VII-DN-GFP attenuated the number of exocytosis. Monitoring individual CFP-tagged tissue plasminogen activator release events revealed that full release events are increased in Syt VII-GFP-expressing cells, but not in Syt VII-DN-GFP-expressing cells. Taken together, Syt VII modulates the kinetics of $Ca^{2+}$-dependent dense-core vesicle exocytosis in PC12 cells, possibly by modulating fusion pore opening.

KAKENHI (18689008)

## P2-b32 Enkephalin gene expression is much weaker in the patch compartment than in the matrix compartment of the striatum: Analysis with transgenic mice

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Preproenkephalin (PPE) is known to be expressed by the striatal neurons projecting to the external globus pallidus. To visualize PPE-producing neurons, we generated transgenic mice with a bacterial artificial chromosome which contained a modified PPE gene to express green fluorescent protein (GFP). In the neostriatum of the best transgenic mouse line, almost all neurons expressing GFP were immunoreactive for PPE, indicating sufficient specificity of the line. The neostriatum of the mice possessed amorphous regions with very weak GFP fluorescence, and the regions were revealed to correspond to the patch comparment characterized by strong mu-opioid receptor immunoreactivity. These results indicate the heterogeneity of PPE expression in association with patch-matrix mosaic organization of the neostriatum.

## P2-b33 CA1-projecting median raphe neurons expressing 5HT1A receptors are serotonergic

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It has been reported that activation of serotonergic receptors(5HTIA-R)induces membrane hyperpolarization in serotonergic median raphe neurons(MRNs), and if so, it would be possible to identify the sertonergic neurons expressing 5HTIA-R by observing reduction in firing rate in extracellular recordings after application of 5HT1A-R agonist. In the present study, we examined whether all MRNs expressing 5HTIA-R contain 5HT or not using immunohistological and retrograde labeling techniques. The percentage of MRNs reacted with anti-5HT antibodies to those with anti-5HT1A-R antibodies was no more than 73%. As MRNs project to CA1, true blue was injected into CA1. The ratio of serotonergic MRNs out of 5HT1A-R positive CA1 projecting MRNs was about 87% in the whole median raphe nucleus. However, the value was close to 100% in the region confined to 300 μm from the caudal pole of the nucleus. The present result showed that MRNs expressing 5HT1A-R and projecting to CA1 were serotonergic in the caudal part of the median raphe nucleus.

## P2-b34 The serine racemase mRNA locates predominantly in neurons of rat brain

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D-Serine is an endogenous and obligatory coagonist for the glycine site of N-methyl-D-aspartate receptor in the mammalian brain. D-Serine is synthesized from L-serine by serine racemase. Serine racemase and D-serine have long been believed to occur predominantly in astrocytes by immunohistochemical studies. Here we report the application of in situ hybridization based on tyramide signal amplification for detection of serine racemase mRNA in rat brain. Serine racemase mRNA could be demonstrated in a large number of neurons throughout the brain, especially forebrain such as cerebral cortex, striatum and hippocampus. These results suggest that the neuron-derived D-serine could modulate the N-methyl-D-aspartate receptor mediated neurotransmission via the glycine site.

## P2-b35 Stereoscopic study of corticotropin-releasing factor positive climbing fiber projection in mouse cerebellum by whole mount immunostaining

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This study examined spatial organization of CRF positive climbing fiber projection in the mouse cerebellum by whole mount immunostaining. A striking parasagittal pattern of CRF staining was definite in the mouse cerebellum. Cryosections of whole mount CRF stained cerebellum demonstrated the appearance of CRF staining in climbing fiber subsets but not mossy fiber subsets. An array of CRF stripes was quite similar to that of Zebrin II stripes in the anterior vermis and hemispherical lobules. However, CRF defined parasagittal stripes and transverse zones in the posterior vermis and vestibulocerebellum were different from the Zebrin II compartment. The results suggest unique patterns of CRF positive climbing fiber innervation in the mouse cerebellum which is similar but not completely overlapped with the Zebrin II compartments.

## P2-b36 The role of inhibitory neurotransmission in ventral body wall formation

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Glutamate decarboxylases, GAD65 and GAD67 synthesize GABA from glutamate, while vesicular GABA transporter (VGAT) transports neurotransmitters, GABA and/or glycine from cytoplasm into synaptic vesicles. To study the role of inhibitory neurotransmission in vivo, we generated GAD65, GAD67 and VGAT knockout (KO) mice. All VGAT KO mice died in the perinatal period, and displayed omphalocele, a defect in ventral body wall closure. On the other hand, about a half of GAD67 KO fetuses showed omphalocele, and it was less severe than that of VGAT KO fetuses. These results indicate that GABAergic transmission plays critical roles in abdominal wall formation, but it remains unclear whether GAD65's contribution and/or glycinergic contribution are also required for the development of ventral body wall formation or not. In order to answer the question, we generated GAD65/GAD67 double KO mice to compare their phenotype with that of GAD67 or VGAT KO mice.

MEXT

## P2-b37 Immunohistochemical analysis of GABA neurons in the brain of the common marmoset

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The distribution pattern of γ-aminobutyric acid (GABA) neurons in the marmoset was determined on the basis of comparative morphology by immunohistochemistry with an anti-GAD antibody (Ab). GAD-immunoreactive (IR) neurons were observed in the cerebellar cortex, caudate nucleus, amygdala, thalamic nuclei, hippocampus, substantia nigra and locus ceruleus. On the other hand, GAD-IR neurons, which were observed in the nuclei below the midbrain, such as the superior or inferior colliculi, periaqueductal gray matter,interpeduncular nucleus and dorsal tegmental nucleus in rats, were essentially absent in these regions in the marmoset. Moreover, stellateand basket cell in the cerebellum were not immunostained by the anti-GAD Ab.We would like to determine whether the results above are the characteristic findings from the marmoset by examining the anti-GAD Ab used in this experiment and carrying out further study of GABA-IR neurons using an anti-GABA Ab.

## P2-b38 Immunohistochemical and biochemical determination of D-serine in the cultured astrocytes and neurons from the rat neocortex

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A co-agonist of the N-methyl-D-aspartate type glutamate receptor, D-serine, naturally occurs at high contents in the brain. To get further insight into the cellular and molecular mechanisms of the brain D-serine system, we examined the D-serine distribution in the cultured type 1 and 2 astrocytes and neurons from the rat neocortex by using an immunocytochemical technique and HPLC with fluorometric detection. Immunocytochemical staining with D-serine antibodies revealed the weak to moderate D-serine-like immunoreactivity in the type 1 and 2 astrocytes and neurons. The neurons showed higher immunoreactivity and contents of D-serine than the type 1 and 2 astrocytes. Our data suggest the neuron-preferable distribution of D-serine in the neocortex and support the possible physiological roles of the neurons in the regulation of the D-serine metabolism and functions.

## P2-b39 The genomic structure and the variants of nitric oxide synthase gene in the terrestrial slug Limax valentianus

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In the terrestrial slug Limax, NO is necessary for the synchronous oscillation of the local field potential in the procerebrum, which is thought to be involved in the odor discrimination and/or odor aversion learning. But there is no description about the genomic structure of the molluscan NO synthase, nor has the evolutionary origin of neuronal NO synthase (NOS) of mammals been clarified. Here we identified two types of NOS mRNAs of the slug, which show differential expression patterns within the brain. One is expressed broadly in the brain but in the low level (limNOS1), whereas, interestingly, the other is almost exclusively in the procerebrum (limNOS2). We also determined the whole genomic structure for limNOS1. It is composed of as many as 32 exons comparable to human nNOS gene, and has very similar exon-intron structure to that of human nNOS. Our results indicated that Limax NOS and human nNOS share the prototypical gene structure of NOS, and that their organization is highly conserved during the evolutionary history.

## P2-b40 Delayed reduction of the protein level in the striatum after genetic ablation of the tyrosine hydroxylase gene in the substantia nigra

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We generated floxed *TH* mice to disrupt the *TH* gene using adeno-associated viral vector expressing Cre recombinase (AAV-Cre). Injection of AAV-Cre into the substantia nigra pars compacta (SNc) of the floxed *TH* mice induced recombination of the *TH* gene, followed by the loss of TH protein in the SNc. A decrease of TH protein in the striatum was much slower than that in the SNc suggesting that the turnover rate of the TH protein would be different between the cell body and the termianal.

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## P2-b41 Activation of MAP kinase by GnRH via activation of CaM Kinase II in cultured hypothalamic neurons

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Gonadotropin-releasing hormone (GnRH) is secreted from hypothalamic neurons (GnRH neurons). We found that CaM kinase IIδ2 and synapsin I were expressed in the immortalized GnRH neurons (GT1-7 cells). Treatment of the cells with GnRH increased phosphorylation of synapsin I at serine 603, suggesting that GnRH treatment activated CaM kinase IIδ2. In addition, GnRH treatment activated MAP kinase. It was interesting that KN93, an inhibitor of CaM kinases, inhibited both phosphorylation of synapsin I and activation of MAP kinase by GnRH. Furthermore, stably overexpression of CaM kinase IIδ2 enhanced the activation of MAP kinase by GnRH. These results suggested that the activation of CaM kinase IIδ2 by GnRH induced the activation of MAP kinase in the cells.

The Naito Foundation

## P2-b43 N-glycans govern the innate immune response in neurocysticercosis

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*Taenia solium* neurocysticercosis is the most common parasitic infection of the CNS and accounts for onequarter epilepsy cases in South India. Seizures result when cysts degenerate in the brain releasing inflammatory cytokines. Therefore, it would be useful to identify cellular mediators that govern parasitic destruction and clear infection. The aim of this study was to evaluate protective innate immune responses in *T. solium* infection. Peripheral blood mononuclear cells from healthy volunteers (obtained with informed consent) were stimulated for 24 hours with 6 infection specific *T. solium* glycoproteins, their peptides or N-glycan components. We identified nitric oxide and IL-12 in culture supernatants and HSP-70 in cells, as mediators of protective innate immunity. Neither *T. solium* glycoproteins nor their peptide components elicited a response from the cells. However *T. solium* N-glycans were strong inducers of nitric oxide in macrophages leading to IL-12 upregulation and induction of HSP-70 that was cytoprotective. The role of *T. solium* N-glycans as mediators of protective innate immunity in neurocysticercosis will be discussed.

ICMR, New Delhi

## P2-b44 Calcium-dependent mechanisms underlying propagation of γPKC translocation along the dendrites of cerebellar Purkinje cell

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Regardless of the importance of protein kinase C (PKC) targeting, little is known concerning PKC translocation in CNS neurons. Especially, it remains unclear how, when and where PKC is targeted in response to $G_{q/11}$-coupled receptor activations. To address this issue, we have generated brain-region specific and inducible γPKC-GFP transgenic mice using Tet-regulated system, and found that in cerebellar Purkinje cells electrical stimulation of parallel fibers evoked not only local translocation but also propagation of the translocation along dendritic arbor. Calcium imaging studies have shown that the parallel fiber stimulation also induces slowly propagating calcium wave, which could lead propagation of PKC translocation. To understand mechanism underlying the PKC wave, we constructed a compartmental model of Purkinje cell dendrites. Computer simulations suggest interplay between γPKC, $Ca^{2+}$ channels and $K^+$ channels on the plasma membrane of dendrites.

## P2-b45 Mechanisms of nicotinic inhibition of neurogenesis. -Studies with use of HC2S2 cells.

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In our previous studies, we demonstrated that nicotine markedly inhibited neurogenesis in the rat dentate gyrus (Shingo and Kito; 2005). The present study was undertaken to examine how nicotine inhibits neurogenesis in the hippocampus from viewpoints of intracellular signal transduction.

Used materials were HC2S2, immortalized neuronal progenital cells originated from the adult rat hippocampus and their differentiated neuronal cells. In culture media of these undifferentiated as well as differentiated neuronal cells, nicotine and/or estradiol were added. Then, PKC, Akt, Erk1/2, pCREB, PSA-NCAM, NeuN, nicotinic receptor subtypes and IGF-1 were examined through techniques such as in situ hybridization, immunocytochemistry, real-time PCR assay and Western blotting.

It was concluded that estradiol inhibited nicotine-induced alpha7 mRNA up-regulation, while nicotine inhibited estradiol-induced IGF-1 mRNA expression. It was also noticed that addition of nicotine in the culture media had increased cell death with progress of time.

## P2-b46 Identification of creatine kinase B as a $Ca^{2+}$-dependent hippocalcin-associated protein in rat brain

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Hippocalcin (Hpca) is a member of the neuronal calcium sensor family predominantly expressed in the hippocampal neurons and acts as a multifunctional modulator in the $Ca^{2+}$-signaling underlying memory formation. Here we found several Hpca-associated proteins (HAPs) in the rat brain by filter overlay method using [$^{35}$S]Hpca. HAP-48, which has a molecular weight of 48kDa showing a $Ca^{2+}$-dependent Hpca-association property, was purified from the cytosolic fraction. Partial amino acid sequences of HAP-48 showed it to be identical to creatine kinase (CK)-B. Myristoylation of Hpca was not required for its association property. [$^{35}$S]Hpca did not associate with CK-M. CK activity of the postmicrosomal fraction from the hippocampus prepared under $Ca^{2+}$-free conditions, was higher than that prepared under $Ca^{2+}$-loaded conditions. The $Ca^{2+}$-dependent translocation of CK activity in the hippocampus was abolished by adding anti-Hpca antibody. These results indicate the possibility that Hpca associates with CK-BB isozyme and transfers it to the plasma membrane in a $Ca^{2+}$-dependent manner.

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## P2-b47 The characterization of protein arginine N-methyltransferases (PRMTs)-depleted PC12 cells.

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Protein arginine methylation is a post-translational modification whose influence on cell functions is becoming widely appreciated. The modification is catalyzed by a family of protein arginine methyltransferases (PRMTs), of which there are at least nine members, including CARM1. Tremendous efforts have been made over the last years to decipher the physiological and pathophysiological roles of CARM1 on the molecular level. Arginine methylation has been implicated in the regulation of transcription, translation and DNA repair (Bedford and Richard, 2005). Gene targeting of CARM1 (also referred to as PRMT4) in mice has been performed, and knockout mice, which are smaller than their wild-type littermates, die just after birth (Yadav et al., 2003). However, despite the progress made in recent years, no complete picture for the physiological role of the enzymatic activity of CARM1 has yet emerged.

## P2-b48 Biochemical comparisons of brains from $Ca^{2+}$/calmodulin-dependent protein kinase II α knock-out mice and inactivated knock-in mice

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$Ca^{2+}$/calmodulin-dependent protein kinase II (CaMKII) is one of the major protein kinases in the central nervous system and plays important roles in neuronal functions. We engineered inactivated CaMKII α knock-in (K/I) mice and have been characterizing functional significance of CaMKII activity in the brain. In such studies, comparison of phenotypes between inactivated K/I mice and knock-out (K/O) mice would be helpful, but basic biochemical data of brains from K/O mice are scanty. In this study, we made biochemical comparisons of brains from CaMKII α K/O mice and inactivated CaMKII α K/I mice. In heterozygous K/O mice, CaMKII α protein level was reduced to about a half of wild type control level in the forebrain and cerebellum, while no such decrease was observed in heterozygous K/I mice. On the other hand, the reduction in CaMKII activity was in a similar range between heterozygous K/O and K/I mice. Such comparisons are useful for correct interpretation of phenotypes of K/O and K/I mice.

KAKENHI (#17500218)

## P2-c01 Inhibitory effects of thiamylal on excitation propagation in brain slices

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Inhibitory effects of thiamylal (a barbital derivative) on excitation propagation in the cortical and hippocampal slices were characterized by optical imaging techniques. Brain slices of the C57BL6J mouse stained with a voltage sensitive dye RH414 and changes in membrane potential were recorded as video images. In cortical slices, excitation evoked by stimulation to layer V was propagated to layer II-III, and the response was widely extended and prolonged by application of strychnine (20 μmol/l). By a successive application of thiamylal (200 μmol/l), the initial excitation was evoked but following responses rapidly diminished and disappeared. In hippocampal slices, excitation propagation elicited by stimulation to the CA1 stratum radiatum was also enhanced and prolonged by strychnine. This enhanced and prolonged excitation remained unchanged even in the presence of thiamylal. The results suggest that thiamylal makes excited neurons calm down by enhancing $GABA_A$ receptor activity in the cerebral cortex.

## P2-c02 Facilitation of dopamine-induced $Na^+$ current response by Rho GTPase and phospholipase D in *Aplysia* neurons

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Stimulation of $D_1$ type of dopamine (DA)-receptor induces a slow $Na^+$ current response in identified *Aplysia* neurons under voltage clamp. We previously reported that the $Na^+$ current response to DA is facilitated by the activation of Rho GTPase. In the present study, we investigated possible effector molecules located at the downstream of the Rho. The $Na^+$ current response to DA was depressed after intracellular injection of $\alpha$ -synuclein, an inhibitory protein for phospholipase D (PLD). Furthermore, application of 1-butanol, which is known to inhibit PLD activity, significantly depressed the DA-induced current response, whereas 2-butanol, an inactive isomer of 1-butanol, had no significant effect on the response appreciably. In addition, dancylcadaverine, which is known to block receptor internalization, increased the DA-induced current response. These results suggest that activation of Rho and subsequent PLD may regulate the expression of the DA receptors on the surface membrane of these cells.

## P2-c03 The appearance of aprataxin in rat cultured neuron and glia

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The mutation of APTX gene, encoding aprataxin protein, was caused Ataxia-occulomotor apraxia 1 (AOA1). AOA1 are central and peripheral nervous system disease. Aprataxin includes a histidine triad motif, forming nucleotide-binding site related with DNA repair. APTX gene makes two splicing variants, long form and short form aprataxin. Long form aprataxin localized in nucleus and short form localized ubiquitously cytoplasm and nucleus. But, there is no histological report about the localization of aprataxin in neuron or glia. Therefore, we investigated that aprataxin localization in rat primary cultured neuron and glia by immunocytochemical technique. The appearance of aprataxin was obtained both nucleus and cytoplasm, however, the expression pattern were difference between neuron and glia. The localization of aprataxin was more detectable on cytoplasm in neuron and on nucleus in glia. These differences might suggest the diversity functions of aprataxin.

## P2-c04 The effect of thimerosal on bullfrog sympathetic ganglion cells

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Thimerosal is a thiol-oxidizing reagent and is known to trigger calcium oscillation in mouse eggs, and enhance an activity of ryanodine receptors. We have applied various concentrations of thimerosal to cultured bullfrog sympathetic ganglion cells and responses of cytoplasmic or intraluminal calcium concentrations to thimerosal have been tested using fluorescent Ca indicator, Fura-2 or Furaptra. Although thimerosal could not induce calcium oscillation unlike caffeine, it caused the slow increase in cytoplasmic Ca concentration followed by a sustained elevation. The addition of caffeine under the presence of thimerosal caused the duration of Ca oscillation broad.

## P2-c05 The involvement of PTEN and FAK in RGMa signaling

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Repulsive guidance molecule (RGM) is a potent inhibitor of axon regeneration in the central nervous system. Here we report that RGMa induces Rac inactivation depends on PTEN activity, PTEN activity is required for RGMa-induced neurite outgrowth inhibition, and Focal adhesion kinase (FAK) may involve in this signaling. When Cos7 cells and Entorhinal cortical (EC) neurons were stimulated with RGMa, Rac activity was decreased. In contrast, RGMa did not affect Rac activity in Cos7 cells expressing dominant negative PTEN C124S. Furthermore, overexpression of PTEN C124S neutralized the effect of RGMa-induced neurite outgrowth inhibition in EC neurons. In addition, RGMa stimulation decreased FAK activity in Cos7 cells and EC neurons. Because FAK can activate Rac through Cas-Crk-DOCK180-ELMO pathway, and PTEN inactivates FAK by dephosphorylation, it is suggested that RGMa decreases Rac activity through FAK inactivation by PTEN, and this signaling pathway is required for RGMa-induced neurite outgrowth inhibition.

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## P2-c06 Distribution of theSerum inducible kinase (Snk) mRNA in the rat nervous system; its up-regulation in lateral and basolateral amygdala after cued fear conditioning

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Snk is a member of the Polo-like kinases (Plks) that are conserved in organisms from yeasts to human. The expression of Snk in the central nervous system has not been fully described. Here, we report the detalied expression analysis of rat Snk mRNA in the series of coronal sections using in situ hybridization. The expression of Snk drastically commersed postnataly. In the adult rats, Snk mRNA was predominantly observed in the neurons of limbic system. By using the cued fear conditioning of C57BL/6J mice, Snk was transcriptionally up-regulated in the lateral and basolateral nucleus of amygdale. These results suggest that Snk may play an important role in the regulation of fear memories.

## P2-c07 Proteomics of unilateral irradiated and contralateral striata after gamma knife surgery

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Gamma knife surgery (GKS) is used for treatment of various brain disorders. Biological effects of GKS on targeted or surrounding areas in the brain are not well known. We analyzed unilateral irradiated (60 Gy) and contralateral striata of rat at 16 h post-irradiation by two-dimensional gel electrophoresis detecting a total of 17 differentially expressed protein spots in bilateral striata. Their subsequent analysis by tandem mass spectrometry resulted in the identification of 13 non-redundant proteins. Interestingly, out of these 13 proteins, 3 proteins were also detected in the contralateral striatum, such as protein disulfide isomerase A3 and peroxiredoxin 2. Our results indicate unilateral irradiation during GKS trigger molecular changes in the bilateral striata.

## P2-c08 Regulation of alpha CaMKII activation by its autophosphorylation and change in $Ca^{2+}$ level in living cells

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Previous studies suggest that in response to increase in $Ca^{2+}$ concentration, $\alpha$ CaMKII is activated by interaction with $Ca^{2+}$/CaM (CaM-interaction) and that prolonged increase in $Ca^{2+}$ level maintains the activation of $\alpha$ CaMKII through the conformational change by autophosphorylation at T286. To understand the molecular dynamics of $\alpha$ CaMKII activation in vivo, we have investigated CaM-interaction and conformational change of $\alpha$ CaMKII in living cells using FRET. From comparisons of results using several cell lines, we found that change in $Ca^{2+}$ concentration and autophosphorylation of $\alpha$ CaMKII critically affects the dynamics of $\alpha$ CaMKII activation; high-level of $Ca^{2+}$ induces continuous CaM-interaction and the conformational change. In contrast, low-level of $Ca^{2+}$ induces only transient CaM-interaction, but the conformational change of $\alpha$ CaMKII was still maintained following the termination of CaM-interaction. In addition, cell-specific regulation of autophospholyration at T305/306 determines the duration of CaM-intreaction.

## P2-c09 The isolation of bdif-1 derived from the adult brain, and its function as Rab-GAP

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The molecular machinery underlying intracellular protein delivery in neuronal cell has recently been explored in detail, and several signal molecules, which function in membrane trafficking or neuronal cell polarity formation at early developmental stage or pathological condition, such as phosphatidylinositol 3-kinase (PI3K) and Rho GTPases have been identified. However, the molecular mechanism involved in protein trafficking, especially in the central nervous system (CNS) of the adult has not been totally understood yet. In our present study, we report our newly identified gene derived from the adult rat brain, brain derived ito factor-1 (bdif-1), which encodes the protein containing TBC, SH3 and RUN conserved domains. Northern blotting analysis revealed that the mRNA was vigorously expressed in the brain, liver, testis and moderately expressed in the heart and kidney. Bdif-1 was showed to function as a GTPase-activating protein (GAP) and interacts with small GTPase Rabs. Bdif-1 was proposed to play a crucial role in protein trafficking in the adult CNS.

## P2-c10 Map-based model of neural activity with chaotic spike-bursting oscillations

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We propose a discrete time system as a phenomenological model of excitable and bursting neurons. The model is a discontinuous two-dimensional map. It is shown that this type of models simplifies nervous system study using both numerical and analytical approach. It is shown that in spite of the simple structure, our model is a very flexible instrument for nervous system investigation because it allows simulating a large quantity of modes of neural activities. We have investigated dynamics of the model using methods of the nonlinear oscillation theory and found dynamical structures providing simulations of the regimes of the neural activity. We have found the conditions on the parameters under which chaotic and regular attractors exist. Chaotic attractor provides simulating of spike-bursting oscillations. Regular attractors provide simulation of other neural oscillatory activity such as subthreshold oscillation and periodic spiking. It is shown also that this model is applicable for simulations of the threshold excitability property of the neurons.

## P2-c11 Interaction between N-terminal 126 amino acids of MAP1B and tubulin

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Previously we reported overexpression of MAP1B containing N-terminal 126 amino acids promoted neuronal death. We searched for the proteins that interact with N-terminal of MAP1B by two-hybrid system. $\alpha$ -tubulin(fragment 44-294aa of 451aa) was found to interact with MAP1B N-terminal and their in vitro interaction was proved with pull-down assay. The interaction of tubulin and MAP1B N-terminal has not yet been reported. $\beta$- and $\beta$III-tubulin fragment was also found to interact with MAP1B N-terminal. All three tubulins were detected in GST pull-down assay with rat brain homogenates. When MAP1B lacking some of 3 binding regions were transfected to neurons, N-terminal induced neuronal death . but lacking the region containing KKEE(I/V) motif induced much more neuronal death. Lacking C-terminal region showed little effect.There were much proportion of round formed COS7 cells in N-terminal containing MAP1B transfected cells than in N-terminal lacking MAP1B transfected cells. There might be some interference in interaction between MAP1B and tubulin in cells express MAP1B containing N-terminal.

## P2-c12 Loss of a novel regulator for protein trafficking results in progressive degeneration in Drosophila

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We identified the Drosophila Yata protein as a regulator of the intracellular trafficking of Hikaru genki (HIG) protein, which displays distinct stage-dependent localization in neurons. Both loss-of-function and gain-of-function studies of yata suggested its involvement in promoting trafficking for HIG. Yata mutant flies exhibited progressive degeneration and premature lethality. Exogenous expression of yata in neurons showed remarkable rescuing effect for the early death phenotype. We are now trying to identify other proteins whose trafficking is regulated by Yata and reveal their role for maintenance of neurons.

Program for Improvement of Research Environment for Young Researchers, Center of Excellence Grant, KAKENHI

## P2-c13 Relation between dendritic spine size and its drebrin A level in adult mouse brain

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We explore the relationship between levels of drebrin A, the NR2A subunit of N-methyl-D-aspartate receptors, and the size of spines in the perirhinal cortex of adult mouse brains, using the electron microscopic immunocytochemistry. In contrast to the membranous localization within neonatal spines, the majority of immunogold particles for drebrin A were localized to the cytoplasmic core region of spines in mature spines. Drebrin A immuno-positive (DIP) spines exhibited larger spine head areas and longer postsynaptic densities (PSDs) than drebrin A immuno-negative (DIN) spines. Further, spine head area and PSD lengths correlated positively with drebrin A levels. The number of synaptic NR2A-immunolabels was also higher in DIP spines than in DIN spines while their densities per unit lengths of PSD were not significantly different. These differences between the DIP and DIN spines indicate that spine sizes and synaptic protein composition of mature brains are regulated, at least in part, by drebrin A levels.

KAKENHI18700306

## P2-c14 Identification of VAMP2 as a binding partner for a brain enriched septin, Sept8

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The septins are members of a conserved family of cytoskeletal GTPases that associate with cellular membrane and the actin and microtubule cytoskeletons. Using western blot analysis, we found Sept8 was preferentially expressed in rat brain tissues and dramatically increased during postnatal development. We performed yeast two-hybrid screening and identified the soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) protein, VAMP2, as a binding partner for Sept8. Immunofluorescence studies revealed that Sept8 is well co-localized with VAMP2, in primary rat hippocampal neurons. It is known that synaptophysin binds and controls the targeting of VAMP2 to synaptic vesicles. The binding of synaptophysin and VAMP2 was suppressed by expression of Sept8. These results suggest that Sept8 may be involved in the targeting of VAMP2 to synaptic vesicle and regulate the synaptic function in neuronal cells.

KAKENHI17790088

## P2-c15 Neural activity modifies microtubules by tubulin polyglutamylation

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Neuronal activity has been implicated as an extrinsic cue that regulates various neuronal functions including neuronal morphology. While microtubular cytoskeletons are critical to form neuronal processes as well as for the polarized transport, it is remain to be elucidated by which mechanisms microtubules are regulated by neuronal activity. It has been reported that in neurons, tubulins are highly modified by polyglutamylation that modulates binding of MAPs and KIFs to the microtubules. Recent studies revealed that tubulin tyrosine ligase like (TTLL) families catalyse polyglutamylation. Here we report that neuronal activity enhances the polyglutamylation of tubulin. In membrane-depolarized cerebellar granule neurons, we showed that calcium entry from voltage dependent calcium channel (VDCC) and susequent activation of calcium/ calmodulin-dependent protein kinase (CaMK) are required for the induction of polyglutamylation. These results reveal the novel linkage between neuronal activity and microtubule functions via post-translational modification of tubulin.

## P2-c16 CLP36 is a new member of growth cone-associated proteins that regulates neurite formation through the interaction with alpha-actinin

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CLP36 is a member of enigma family proteins that contain a PDZ domain at N-terminal and LIM domains at C-terminal. We previously showed that CLP36 forms a complex with alpha-actinin in growth cones and its mRNA is upregulated after peripheral nerve injuries. To investigate the functions of this protein, we generated PC12 cell lines in which CLP36 expression is stably suppressed and examined morphological alterations in the knockdown cells. We found that down-regulation of CLP36 promoted neurite elongation and increased the number of neurites. In addition, the development of membrane extension caused flattered morphology, accompanied by spreading of the cell body. Such phenotype of the knockdown cells could be restored by addition of exogenous CLP36 and deletion mutants that include the middle (MID) portion of CLP36. Since alpha-actinin binds to CLP36 through the MID portion, these findings suggest that CLP36 plays an important role in neurite formation by defining the sites of actin dynamics through the interaction with alpha-actinin.

## P2-c17 Possible involvement of a microtubule-associated protein, doublecortin-like kinase 1, in postsynaptic function

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Doublecortin(DCX) is a microtubule-associated protein(MAP) required for neuronal migration in the cerebral cortex. Doublecortin-like kinase1(DCLK1) consists of an N-terminal domain 75% homologous to DCX, and a C-terminal domain similar to Ca2+/calmodulin dependent protein kinase. The DCX domain of DCLK1 binds to microtubules(MTs) and stimulates MT polymerization. Although the detailed functions of DCLK1 are not clear, expression profile of DCLK1 is distinct from DCX, suggesting their distinct functions. To elucidate the functions of DCLK1, I investigated localization and dynamics of DCLK1 in hippocampal neuronal culture. DCLK1 is localized along dendritic MTs with its highest concentration at the distal tip. However, unlike other MAPs, DCLK1 not only associates with MTs but also is present within dendritic spines and affects expression of a postsynaptic density(PSD) protein PSD-95. Furthermore, DCLK1 localization is sensitive to neuronal activity. Thus DCLK1 may function as an activity-dependent modulator of MTs and PSDs by its dynamic translocation between these two dendritic compartments.

## P2-c18 Spikar distributed in the cytoplasm rescues impairment of spine formation induced by Spikar siRNA

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Spikar is a drebrin-binding protein, which is localized at nucleus and dendritic spines. We have previously shown that Spikar knockdown (KD) using siRNA decreased the dendritic spine density of cultured hippocampal neurons by immunocytochemistry. In the present study, we first showed a significantly longer inter-event interval of miniature excitatory postsynaptic currents (mEPSCs) in Spikar-KD neurons without affecting the mEPSC amplitude. Then to examine whether Spikar functions for spine formation in nuclei or at spines, we performed rescue experiments of Spikar-KD neurons with siRNA-resistent Spikar (rSpikar). Either myc-tagged rSpikar or myc-tagged rSpikar with mutated NLS, which was exclusively located in the cytoplasm, inhibited the decrease of the spine density induced in Spikar-KD neurons. This suggests that Spikar in the cytoplasm is effective for spine formation, although we cannot rule out the possibility that Spikar also works in nucleus to regulate spine formation.

KAKENHI18700306, KAKENHI18021004

## P2-c19 Biochemical and cell biological analyses of Sept12 in the rat brain

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Septins are a family of conserved cytoskeletal GTPases implicated in a variety of cellular functions such as cell polarity formation and vesicle trafficking. Here we performed biochemical and cell biological analyses of a yet uncharacterized mammalian septin, Sept12. Based on western blotting, Sept12 was most likely to undergo complex splicing and distributed ubiquitously in tissue- and brain region-specific manners. Immunohistochemical analyses revealed that Sept12 is highly expressed in Purkinje cells in the cerebellum and neurons in the cerebral cortex. To gain some insight into the function of Sept12 in the brain, we performed yeast two-hybrid screening and identified synaptophysin as a binding partner for Sept12. These results suggest that Sept12 plays a role in neuronal functions such as neurotransmitter release.

KAKENHI18057031

## P2-c20 Induction of stanniocalcin1 expression by histone deacetylase in mouse neuroblastoma cells

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Stanniocalcin-1 (STC1) is a mammalian glycoprotein hormone and appears to be a regulator of neuronal function. A detailed mapping of STC1 mRNA in various developmental stages of mouse brains revealed that the transcripts were detectable on day 12 and peaked on day 16 of gestation and then declined slowly. For spatial distribution study, the results indicated a differential expression of STC1 in the regions; the highest level was detected in striatum, followed by hippocampus, parietal cortex, hypothalamus, frontal cortex, cerebellum and brain stem. The function of STC1 has remained elusive. Using mouse neuroblastoma cells, we have shown that the induction of STC1 mRNA and protein expression is integral to differentiation. We aimed to elucidate if HDACIs, such as trichostatin could regulate the expression of STC1. Our data indicated that HDACI induced cell differentiation that accompanied with the activation of STC1 expression. The induction of STC1 mRNA was transcriptionally regulated as revealed by Act D treatment. The protein factors involved in the regulation are investigated.

FRG/HKBU

## P2-c21 GeneChip analysis of the neocortical and hippocampal gene expression in kainate- and nicotine-injected mice

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Severe, repetitive, and chronic seizures during childhood could thereafter result in brain dysfunctions such as ataxia, mental retardation, and cognitive impairment. To examine irreversible changes in the developing brain following the seizures, juvenile inbred mice were intraperitoneally injected with semi-lethal doses of kainate and nicotine. Neocortices and hippocampi were isolated 7 days post-injection, and gene expression profiles were analyzed with a GeneChip microarray. Comparative analyses using stringent parameters revealed the marked differential expression in a region-specific and a drug-specific manner. This systematic approach to examining brain regional responses to different inducers of seizures may provide a better understanding of the molecular basis of the impediments caused by seizures during childhood that would culminate in dysfunctions of the adult brain.

Takeda Science Foundation

## P2-c22 Deletion analysis of cis-elements regulating GSK-3 $\beta$ gene promoter

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Glycogen Synthase Kinase-3 $\beta$ (GSK-3 $\beta$) is one of the key proteins involved in Alzheimer's disease. Level of GSK-3 $\beta$ gene products in neuronal cells is higher than in non-neuronal ones. To identify cis-elements contributing to this neuronal predominance in the expression, we studied with the deletion mutants of promoter, Δ-290/-41, Δ-40/+3, Δ+2/+29, Δ+30/+187, Δ+206/+389, Δ+390/+428 directing the luciferase gene. These mutants were introduced into neuronal cells (SH-SY5Y) and non-neuronal cells (HEK293). Although Δ+2/+29 did not affect to the luciferase activity, Δ+30/+187 enhanced it up to approximately 125% of the control promoter. On the contrary, four other mutants reduced the promoter activity down to 50% of the control. There may be the important sequence other than core promoter (-171 ~ +29). They also may control synergistically the promoter. On the other hands, no difference in these features between neuron and non-neuron was observed. Neuron specific transcription factor should be involved in the neuronal predominance.

## P2-c23 Gene expression analysis of motor-related areas and prefrontal area of the monkey neocortex

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Motor-related areas of cerebral neocortex are highly differentiated into several subareas from both functional and cytoarchitectural aspects in the higher primates. To assess the molecular basis of such differentiation, we investigated the gene expression profiles of primary motor area, premotor area (dorsal and ventral) and prefrontal area, as an outgroup, in the rhesus monkey (n = 3) by DNA microarray method. Although there were no global differences, some genes had differential expression patterns among areas. The expression profiles of dorsal and ventral premotor areas are similar to each other compared to primary motor area. The relationships between transcriptional regulation of these differentially expressed genes and the functional roles of individual areas will be discussed.

## P2-c24 An oligodendrocyte enhancer in a phylogenetically conserved intron region of the mammalian myelin gene *Opalin*

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Opalin is a transmembrane protein detected specifically in mammalian oligodendrocytes. Opalin homologs are found only in mammals and not in the genome sequences of other animal classes. We first determined the nucleotide sequences of Opalin orthologs and their flanking regions derived from four primitive primates. A conserved region was found in the first intron of Opalin. When the conserved domain was assayed for its enhancer activity in transgenic mice, oligodendrocyte-directed expression was observed. The conserved domain is composed of eight subdomains, some of which contain binding sites for Myt1 and CREB. Deletion analysis and cotransfection experiments revealed that the subdomains have critical roles in Opalin gene expression. LIF, cAMP signaling cascades, and Myt1 were found to play significant roles in the differentiation of oligodendrocytes through their action on the Opalin oligodendrocyte enhancer.

## P2-c25 Integration of neuronal activity into chromatin on the BDNF gene promoter I against the repressive transcriptional activity of REST/NRSF in neuron

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BDNF gene promoter I (BDNF-PI) is activated by intracellular $Ca^{2+}$ signals through, at least in part, CRE but negatively controlled by repressor element-1 (RE1, also called NRSE). Using rat cortical neuronal cells in culture, we investigated how activity-dependent activation of BDNF-PI is achieved against the silencing effect of BDNF-RE1. A marked increase in the expression of BDNF exon I by trichostatin A revealed that BDNF-PI is highly repressed in chromatin. We found that RE1 repressed BDNF-PI depending upon the expression of REST. The phosphorylated CREB bound to CRE but REST dissociated from BDNF-RE1 upon depolarization. Even after membrane depolarization was removed, the level of BDNF exon I expression remained elevated. Thus, BDNF-PI is constitutively repressed through REST but can be activated by neuronal activity, the activated level of which could competitively be determined by the strength of neuronal activity and the level of REST expression and be maintained without neuronal activity.

## P2-c26 Monoallelic gene regulation and combinatorial expression of *Protocadherin-β* family genes confer neuronal diversity in the brain.

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*Protocadherin-β (Pcdh-β)* family is a subfamily of the clustered protocadherins that consists of three subfamilies and over 50 protocadherins. Compared with the *Pcdh-α* and *-γ* gene clusters, the *Pcdh-β* gene cluster does not contain constant region sequences. Here we examined gene regulation of the *Pcdh-β* genes in the mouse brain. Using *in situ* hybridization, we found that *Pcdh-β* genes were expressed in almost all neurons. Next, we developed the split single-cell 3'RACE (rapid amplification of cDNA 3' end) method to reveal the allelic gene regulation of one-exon gene, each *Pcdh-β* isoform. This method revealed that *Pcdh-β* isoforms were occurred monoallelic, differential and combinatorial expression profiles in single Purkinje cells. Furthermore, their differential expression patterns were found by *in situ* hybridization not only in Purkinje cells, but also at least in hippocampal pyramidal neurons. Our results suggest that *Pcdh-β* family provides the diversified identity code on each neuron, which is important for neuronal circuit formation.

KAKENHI17024034

## P2-c27 Estradiol regulates alternative splicing of ER-α mRNA in the differentiated NG108-15 neuronal cells

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The actions of 17β-estradiol ($E_2$) require binding to estrogen receptor (ER) - α or - β. With regard to ER-α, pituitary-specific alternative spliced form was identified. However, alternative splicing of ER-α mRNA in central nervous system has not yet been reported. We attempted to determine alternative spliced form of ER-α using NG108-15 cells and examined the expression profile of its form by $E_2$. Cells were induced neuronal differentiation with retinoic acid for 3-4 days, and treated with serial doses of $E_2$ for 48 h. Different sites of ER-α gene were PCR-amplified by specific primers designed for each site and analyzed quantitatively. The PCR-product amplified by exon 3-4 or 4-5 primer was approximately one third to those by exon 6-7 or 7-8 primer. The ratio of exon 6-7 PCR-product to exon 4-5 PCR-product decreased with $E_2$ in dose-dependently. The antiestrogen ICI 182,780 suppressed $E_2$ stimulation. The present study demonstrated the existence of alternative spliced form of ER-α mRNA in neuronal cells, and confirmed that the appearance of its form is self-regulated by own ligand, $E_2$.

## P2-c28 Possible functional expression of bone-related genes in mouse brain

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We have previously shown the functional expression of different machineries required for glutamatergic signaling in bone and cartilage. This study focused on the expression of bone-related genes in the brain *vice versa*. Total RNA was extracted for semi-quantitative RT-PCR analysis. Runx2, osterix and Sox9 were invariably expressed in embryonic and adult mouse whole brains, while mRNA expression was not seen with RANK and RANKL in the murine brain. Sox9 mRNA was rather ubiquitously distributed in the mouse brain, while Runx2 mRNA expression was lower in both cerebellum and medulla-pons than in striatum, hippocampus and neocortex. By contrast, Osterix mRNA expression was lower in hippocampus compared to other discrete brain regions. Both Runx2 and Sox9 were expressed by cultured neurons, astrocytes and microglia, moreover, while Osterix was expressed by neurons and astrocytes but not by microglia. These results suggest that particular bone-related genes could be constitutively expressed by different cells required for orchestration of the normal architecture in the mouse brain.

## P2-c29 Exchange of genome structure in *Protocadherin-α* gene cluster alters their neuronal expression pattern

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*Protocadherin-α* (*Pcdhα*) gene cluster codes for diverse transmembrane proteins and is expressed in the brain. The genome structure of mouse *Pcdhα* is composed of 14 tandem first exons in the variable region and 3 commonly used exons in the constant region. One of the variable first exons is spliced to the constant exons. *Pcdhα* isoforms are differentially expressed from each gene-cluster allele in an individual Purkinje cell. To address the allelic gene regulation, we generated mutant mice. In the mice, number of variable exons was exchanged by interchromosomal Cre-mediated recombination between different targeted alleles inserted *loxP* sequences in the variable regions. The expression patterns of the *Pcdhα* isoforms were influenced by their exchanged genome structures. The results indicated that expression of the *Pcdhα* isoform at individual neurons is significantly regulated by their genome structure.

KAKENHI 17024034

## P2-c30 Distribution of MKL1/2, actin-binding coactivators for SRF, in developing rodent brain

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MKL(megakaryoblastic leukemia) family members, which are able to bind to G-actin, function as an SRF(serum response factor) coactivator. We previously showed that MKL1 promotes SRF-mediated gene expression and plays an important role in dendritic morphology in rat cortical neurons. However, little is known about brain and tissue distribution of MKL1/2 mRNA and the expression pattern in developing brain. We investigated the gene expression with *in situ* hybridization and RT-PCR method in developing mouse brain. These analyses revealed that the expression level of them in hippocampus and cortex is highly expressed and increased in developing mouse brain. Additionally, we are looking over the expression of MKL1 splice variants in rat brain. These studies would define the relationship between the changes of neuronal morphology via the MKL1/2 expression and their localization, and the effect on the brain development.

KAKENHI(17790056), Takeda Science Foundation, Narishige Neuroscience Research Foundation, Uehara Memorial Foundation

## P2-c31 Functional analysis of MKL1/2, actin-binding coactivators for SRF, in neuronal morphology and gene expression

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The dynamic change of neuronal morphology is involved in the normal neuronal network, while gene expression is indispensable, as well. MKL(megakaryoblastic leukemia) family members function as SRF (serum response factor) co-activators, as well as actin-binding proteins. Previous studies have shown that MKL1 plays a key role in formation of neuronal dendrites in mature neurons. In this study, we have focused upon MKL2, another member of MKL family, and investigated the role in neurons. Like MKL1, MKL2 is able to move into a nucleus and activates SRF-mediated transcriptional responses in cortical neurons. Furthermore, overexpression of MKL2 siRNA resulted in a decrease of SRF transcriptional response. Taken together, these results indicate that MKL2 as well as MKL1 plays an important role in regulating SRF-mediated transcription in neurons. Ongoing studies aimed at investigating the role of MKL2 in dendritic morphology have been performed.

KAKENHI(17790056), Hayashi Memorial Foundation For Female Natural Scientists

## P2-c32 Molecules regulated by patterns of electrical activity

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To decode specific key patterns of neuronal activities that relate to higher order functions, a new method was taken namely "reverse electrophysiological" approach whereby neurologically inactive neurons are electrically stimulated to reveal relationships between neuronal activity patterns and molecular functions, and the fundamental mechanism on information processing in the brain. By using this method, we stumbled across "neuregulin", a schizophrenia susceptibility gene that was cloned from cerebellum. In addition, other genes were analyzed focusing various patterns of activity (delta, theta, beta, gamma oscillations and high frequency) in gene expression. We are trying to find out some rules from the bases of experimental data to control molecular behaviors, finally higher order functions.

## P2-c33 Stem cell transplantation in rat model cognitive dysfunction: Neurobehavioral, Neurochemical and Immunohistochemical assessment

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Neural Progenitor cells can be expanded in culture can be differentiated into desired neuronal phenotype through specific mitogenic exposure. In the present study progenitor cells derived from of ED 11 rat fetuses was differentiated into specific cholinergic type by mitogenic support through FGF2 and EGF. These cells expressing immunoreactivity for ChAT, AChE and AChR were transplanted in kainic acid lesioned rat. Neurobehavioral, neurochemical and Immunohistochemical assessment were made four weeks post lesioning. Scores on Y Maze showed better ($p>0.005$) learning and memory recovery as compared to lesioned animals. 70% recovery in cholinergic receptor binding was evident in transplanted animals . This was accompained by higher ChAT expression in transplanted animals . Our results suggest, differentiated stem cells transplantation has recovery in cognitive dysfunction rat model, which is also applicable in various neurodegenerative diseases.

## P2-c34 Colocalization of neuronal markers and cell-cycle markers in GABAergic neuron progenitors in the mouse spinal cord

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It has been suggested that the main source of GABAergic neurons in the rodent spinal cord is in the ventricular zone. However, we observed the production of GABAergic neurons in the mouse spinal cord and realized that origin of the spinal GABAergic neurons still remains controversial. To have insight on these issues, we investigated the characteristics of the GABAergic neuron progenitors at single cell level by using GAD67-GFP knock-in mouse. We found a small fraction of GFP-positive cells in the mantle layer of the spinal cord at E17 expressed cell cycle markers. Thus, both in vivo and in vitro data together indicate that cell cycle markers are colocalized with neuronal markers in GAD67-GFP-positive cells. These cells may be the GABAergic neuron progenitors that contribute to the production of spinal GABAergic neurons at late embryonic stages.

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## P2-c35 Retardation of P19 neuronal differentiation by ifenprodil

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The pluripotent P19 cells can be differentiated into neurons after treatment with 1 μM retinoic acid for 4 days. Mild trypsinization of P19 neurospheres with subsequent plating of dissociated single cells onto polylysine treated glass cover slips led to the formation of large star-like neuronal clusters communicating via thick rope-like neurite fascicles. Ifenprodil (IF) severely retarded the development of these P19 neuronal clusters at 1 μM, associated with thinning and shortening of the neurite fascicles. The retardation by IF was dose dependent and reproducible only in differentiating P19 neurons, which express NR2B subunit of NMDA receptors. Effect was not seen when IF was added at 10 μM during neurosphere induction with subsequent removal from the medium of the differentiating P19 neurons. Either spermidine or spermine at 50 μM was effective in fully preventing the retardation by 1 μM IF. Moreover, spermine exerted a positive effect on the neuronal cluster size. These results suggest that IF may negatively modulate neuronal differentiation through the polyamine site of NR2B subunit on NMDA receptors in P19 cells.

## P2-c36 Lysophosphatidic acid stimulates neuronal differentiation through $LPA_1$

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Lysophosphatidic acid (LPA) is an extracellular lipid mediator that regulates cortical development. We have previously reported that LPA stimulates neuronal differentiation of cortical neuroblasts in a neurosphere culture system. Cortical neuroblasts are known to primarily express $lpa_1$, a subtype of LPA receptor family coupled to $G_i$. Therefore, we here explored the involvement of $LPA_1$ in cortical neuronal differentiation. When neurospheres were pretreated with Ki16425, an $LPA_1$-preferring antagonist, or pertussis toxin, the effects of LPA on both neuronal differentiation were significantly inhibited. To further confirm the involvement of $LPA_1$ in LPA-dependent neuronal differentiation, neurospheres were prepared from $lpa_1$-null embryos and the effects of LPA were examined. LPA-induced enhancement of neuronal differentiation was not observed in neurospheres derived from $lpa_1$-null mice. These results suggest that LPA promotes the commitment of neuroblasts to the neural lineage through the $LPA_1$-$G_{i/o}$ pathway.

## P2-c37 Searching for mechanisms that diversify the olfactory receptor neurons in *Drosophila*

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In the *Drosophila* olfactory sensory system, there are more than 50 different classes of olfactory receptor neurons (ORNs), each of which expresses one or two specific odorant receptors and innervates a specific glomerulus in the primary olfactory center of the brain, forming a spatial sensory map. Endo et al. (2007) recently reported that the ORN class is diversified by the differential activation of Notch signaling during the ORN development. ORNs form clusters of one to four cells that are derived from a single sensory organ precursor, and within each cluster, ORNs are differentiated into two classes depending on the Notch activity. However, this mechanism alone cannot determine the whole 50 classes of ORNs, and there must be some other mechanisms acting either in the sensory organ precursors to assign them different identities in advance, or in the ORNs that are already differentiated by the Notch-dependent mechanism to further diversify their identities. We are currently investigating such mechanisms to understand the whole ORN differentiation and the resulting glomerulus-specific innervation.

## P2-c38 IGFBPL-1 tends to strongly be expressed where newly born neurons are located in the developing brain

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To explore the cell and molecular mechanisms that control the process of cortical development, we have searched for the ventricular side-enriched secretory or transmembrane molecules (S/TMs) whose expression was regulated developmentally in the mouse cerebral cortex using DNA chip analysis and physicochemical algorithms. One of the early-stage specific S/TMs was insulin-like growth factor binding protein-like 1 (IGFBPL-1). IGFBPL-1 has a domain structure that resembles one in the insulin-like growth factor binding proteins, which is a domain known to be bound by insulin-like growth factors with high affinity and to thereby affect cell growth and metabolism. However, the distribution and functions of IGFBPL-1 in the brain are not known. Thus, in this study, we have investigated the expression patterns of IGFBPL-1 in developing mouse brains using in situ hybridization. The results indicated that IGFBPL-1 tends to strongly be expressed where newly born neurons are located.

## P2-c39 Induction of GDNF receptor, GFR $\alpha$1, expression by bHLH transcriptional factors

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While we have studied the gene expression patterns of glial cell line-derived neurotrophic factor receptors (GFR $\alpha$) in the chick embryo at early developmental stages, we realized that one of three avian GFR $\alpha$ species, GFR $\alpha$ 1, exhibited quite a similar distribution pattern of expressing cells along the dorso-ventral axis of the spinal cord to those of proneural basic helix-loop-helix (bHLH) transcriptional factors including Neurogenin1 (Ngn1), Neurogenin2 (Ngn2), and NeuroM. This led us to test the roles of these bHLH transcriptional factors in the regulation of the GFR $\alpha$ 1 expression. To address this possibility, we overexpressed Ngn1, Ngn2, and NeuroM in neural tubes of chick embryos using *in ovo* electroporation. Overexpression of these bHLH proteins spatially and quantitatively increased GFR $\alpha$ 1-expressing cells, largely without affecting the expression patterns of the other two GFR $\alpha$ (e.g. GFR $\alpha$ 2 and 4). These results show that bHLH proteins may be involved in the regulation of a partial repertoire of neuronal characteristics such as a neurotrophic factor receptor expression (GFR $\alpha$ 1) during the neurogenesis in the spinal cord.

## P2-c40 Effect of docosahexaenoic acid on the neurogenesis of neural stem cell

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We demonstrated that docosahexaenoic acid (DHA) promotes neurogenesis from neural stem/progenitor cells (NPCs). However, the molecular mechanisms are not completely understood. Basic helix-loop-helix (bHLH) transcriptional regulators play important roles in the control differentiation and proliferation of NPCs. In this study, the effects of DHA on the expression of bHLH transcriptional factors of cultured NPCs were investigated. NPCs from E14.5 rat were cultured as neurospheres in N2 medium containing basic fibroblast growth factor (bFGF). For the differentiation experiments, the cells were plated on the dish and cultured in the bFGF free medium containing with or without DHA for the several duration. Total RNA was prepared from each culture, and was performed RT-PCR (Hes1, Hes5, neurogenin1, Mash1 or NeuroD). Treatment of NPCs with DHA decreased the expression of Hes1 mRNA compared with that of control. The present study suggests that DHA stimulates neuronal differentiation by modulating expression levels of bHLH transcription factors.

## P2-c41 Clock genes regulate neurogenic transcription factors and the neuronal differentiation of adult neural stem/progenitor cells

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Circadian clock plays multiple roles in our bodies, and clock genes are expressed in various brain regions, including the subventricular zone (SVZ) where neural stem/progenitor cells (NSPCs) persist and postnatal neurogenesis continues. However, the functions of clock genes in adult NSPCs are not well understood. We investigated the expression of clock genes in the SVZ and verified how the expression levels of clock genes changed during differentiation of cultured adult NSPCs. Next, we used RNAi to observe the effects of clock genes on neuronal differentiation. Our results revealed that Clock and Bmal1 were expressed in the SVZ. In cultured adult NSPCs, the clock genes changed expression patterns during differentiation. Moreover, gene silencing of Clock or Bmal1 decreased the percentages of Map2-positive cells and expression of NeuroD1. These findings suggest that clock genes are involved in the neuronal differentiation of adult NSPCs.

## P2-c42 Meis2 activities in the control of three distinct developmental steps during the corticogenesis

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Previous studies on the cerebral cortical development suggested that there are at least five distinct developmental programs operated for the control of cortical projection neuron generation: neuronal differentiation, radial migration, cell cycle regulation, laminar and regional specifications. Meis2 is a homeodomain-containing transcription factor among TALE family proteins. Our analyses on the potential roles of Meis2 in the control of cortical projection neuron generation using electroporation-mediated gene transfer approach showed that Meis2 activities are critical for the control of rate of progression from mitosis to differentiation in cortical progenitors, temporal switch between neuronal versus glial differentiation, and proper radial migration of cortical neurons in nearly the entire developmental period of corticogenesis. Our results therefore suggest that TALE family proteins play fundamental roles in the control of cerebral cortical development.

KAKENHI(17390086)

## P2-c43 Developmental expression pattern of Foxp1 in the mouse spinal cord

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A member of forkhead transcription factors, Foxp1, is expressed in the developing CNS, including the striatum and spinal cord (SC). To get insight into the role of Foxp1 in the development of SC, we investigated detailed expression pattern of Foxp1 in the developing SC. From embryonic day (E) 9.5 to E17.5, Foxp1 mRNA and protein were detected in the ventral SC where motor neurons (MNs) and four classes of interneurons exist. Foxp1 was expressed in the ventral horn of brachial and lumbar levels of the SC, but not in cervical and thoracic levels. The ventral horn of SC contains postmitotic MNs that are divided into lateral motor column (LMC) and medial motor column neurons by their muscle innervation. LMC neurons that innervate limb muscles are present only at brachial and lumbar levels. Thus, Foxp1-expressing cells in the ventral horn may be LMC neurons. Foxp1 was also expressed in interneuron-like cells at all levels of the SC. Unique expression pattern of Foxp1 within the ventral SC suggests involvement of Foxp1 in the development of LMC neurons and interneurons.

## P2-c44 Direct/indirect mechanism of neuroprotection by neural stem cell against NMDA antagonist-induced damage

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Brain morphological change has been emerged and discussed as the important pathophysiology of schizophrenia. Neuroimaging studies have revealed the progressive volume change in the course of schizophrenia, and different effects of antipsychotics on brain structure. Previously, we demonstrated the different effects of typical/atypical antipsychotics on differentiation of neural stem cells (NSCs) and survival of neurons. In the present study, we investigate the effect of transplanted NSCs on neuronal survival in vitro, to clarify the possibility of stem cell therapy for schizophrenia.Primary cultures of neurons were prepared from E18 rat embryo. Neuronal survival change was assessed by MTT and LDH assay against MK801-induced neuronal damage.The results indicated the neuroprotective effect of NSCs transplanted onto the damaged neural network by MK801. The direct/indirect mechanism of neuroprotection by NSCs and effects of antipsychotics on the NSC neuroprotection will be discussed.

## P2-c45 Region-specific degeneration of NG2+ cells in the cerebral cortex of aged rats

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It has been known that NG2+ cells are broadly present as progenitor cells in the adult mammalian brain. It is still unclear whether aging affects number and morphological feature of NG2+ cells. In the adult (8-10weeks) rats, NG2+ cells were abundantly observed throughout the layer I-VI in the cerebral cortex and showed well-developed multiple radial processes. In the aged (96-100 weeks) rats, the number of NG2+ cells decreased and the cells showed the retracted processes in the parietal cortex. In the piriform cortex, however, such changes were not observed. These observations indicate region-specific degeneration of cortical NG2+ cells in aging

## P2-c46 Differential expression of a particular gene responsible for astroglial differentiation in neural progenitor cells of adult mouse hippocampus

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Hippocampi were isolated from brains of adult and fetal mice, followed by the collection of neural progenitors and subsequent culturing with epidermal growth factor (EGF) for 12 days. Marked round spheres were similarly formed from clustered cells within 2 to 4 days in adult and embryonic mouse hippocampal cultures. Immunocytochemical analysis revealed similarly high expression of nestin in neurospheres from both adult and fetal mouse hippocampi, without expression of MAP2 or GFAP. However, removal of EGF led to marked expression of both MAP2 and GFAP in cells from embryonic mouse hippocampus, but GFAP only in those from adult mouse hippocampus. Differential display analysis revealed higher expression of a particular gene in progenitors of matured than embryonic hippocampi. These results suggest that spontaneous differentiation would undergo into astroglia rather than neurons through higher constitutive expression of a particular gene in hippocampus of matured than developing brains.

## P2-c47 Time-table for transcription factor expression in daughter cells generated at the ventricular surface of the developing neocortex

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Asymmetric cell divisions contribute to cellular diversification in the developing mammalian brain. Models propose that cell-fate determinants are segregated asymmetrically during mitosis of each progenitor cell at the ventricular surface and daughter cells pair-generated from the progenitor cell can already be distinct from each other at the beginning of their lives. But, such models have not been accompanied by cell-fate tracking at the single cell level. To specify timing of the expression of Neurogenin2 in a daughter cell in relation to that of other transcription factors including Tbr2 immunostained daughter cells ($n > 200$) that were time-lapse monitored for various durations from their pair-generation by singly DiI-labeled progenitor cells ($n > 100$) in three-dimensional slice culture.

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## P2-c48 Ngn2 acts upstream of Tbr2 for neuron supply in the developing neocortex

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Loss- and gain-of-function studies have shown that Neurogenin2 (Ngn2) is important for neocortical progenitor cells to supply pyramidal neurons Ngn2 promotes progenitor cells to move to the subventricular zone (SVZ) for non-surface mitosis. These non-surface dividing cells have been shown to express Tbr2, a T-domain transcription factor, although the relationship between Ngn2 and Tbr2 has never been directly examined. Coexpression of these two factors was detected in many cells in the upper ventricular zone (VZ) and SVZ. To determine as clearly as possible whether Ngn2 induces Tbr2 in VZ cells, we then introduced Ngn2 by electroporation into the VZ of the ganglionic eminence that normally lacks both Ngn2 and Tbr2. We found that the ectopically introduced Ngn2 induced Tbr2 expression and observed Ngn2-induced transactivation of Tbr2 promoter containing E-box sequences, bHLH transcription factor binding motif. These results suggest that Ngn2 works upstream of Tbr2 for the commitment of progenitor cells to the neuronal lineage.

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## P2-d01 Non-uniform expression of Rac1 on the ventricular surface of developing neocortex

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Rac1 is known to be important for cortical neuronal migration. We now ask whether Rac1 also helps progenitor cells that play multiple histogenetic roles (i.e. cell production, interkinetic nuclear migration, and forming junctional meshwork). Anti-Rac1 immunostaining of coronal sections of developing cerebral walls showed line-like reactivity at the ventricular surface. Whole-mount anti-Rac1 and anti-$\alpha$E-catenin double stain followed by en face observation revealed that Rac1 reactivity is detected in a non-uniform pattern. Only about 20% of the ventricular endfeet were positive for Rac1. This was quite contrast with a diffuse surface staining pattern by anti-Cdc42. Similar non-uniform endfoot staining patterns were obtained when we used antibodies against GEFs for Rac1. Reactions to detect the activation of Rac1 (in situ Rac activity assay) further revealed positive signals in the Rac1-immunoreactive endfeet. These data suggest that this non-uniform Rac1 immunoreactivity is associated with physiological functions of Rac1 in progenitor cells.

## P2-d02 Axonal regressive events in rat corticospinal slice coculture studied with time-lapse confocal imaging of single axons

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Corticospinal projections in the gray matter of the rat spinal cord has the phase of axonal regression during postnatal development. In corticospinal slice coculture, we labeled cortical neurons with pCAGGS-EYFP and made daily confocal imaging from 5 DIV to 12 DIV. We observed, (1) gradual shortening, (2) fragmentation of the axon terminals, (3) amputation at branching points followed by shrinkage of the distal parts, and (4) abrupt disappearance of the total length of axons within an observation field in a day. We then tried to capture the event of abrupt disappearance by making higher time-resolution time-lapse imaging of 30-60 min interval for more than 8 hours. This enabled us to detect single axons showing degenerative changes. The event started with formation of beaded structure over considerable length followed by rapid disappearance. The rapid most case showed elimination of 1mm length within 2.5 hours. This may explain the long branch elimination of corticospinal axons during early postnatal period.

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## P2-d03 NGF-dependent formation of ruffles in PC12D cells required a different pathway from that for neurite outgrowth

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After the addition of NGF, broad ruffles were observed around the periphery of PC12 cells prior to neurite growth. We studied the signaling pathways of NGF-induced formation of ruffles and neurite growth in PC12D cells. Wortmannin and inhibitors for Akt and GSK-3 $\beta$ suppressed the neurite growth in NGF-treated cells. The neurite growth was inhibited with the mutant Ras. But upon the expression of dominant negative Rac1, cells extended protrusions, lacking F-actin. These results suggest that NGF-dependent neurite growth occurs via a mechanism involving activation of the Ras/PI-3K/Akt/GSK-3 $\beta$ pathway. However, inhibitors for GSK-3 $\beta$ and PI-3k did not suppress the NGF-dependent formation of ruffles. In addition, the formation of ruffles was not inhibited by the mutant Ras. But, it was suppressed by the dominant negative Rac1 or Cdc42. These results suggest that the NGF-induced ruffling requires activation of Rac1 and Cdc42, but does not require Ras, PI-3k, Akt and GSK-3 $\beta$, and these pathways might contribute to the formation of intact neurites due to combined actions including Rac1.

## P2-d04 Heparanase upregulation in astrocytes and macrophages recruited to the injured spinal cord

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Mammalian heparanase (Hpa1) is a heparan sulfate-cleaving endo-glucuronidase exploited by inflammatory and cancer cells to invade connective tissues. We hypothesize Hpa1 involvement also in the recruitment of reactive astrocytes and macrophages to the injured spinal cord. To test this, spinal cords of adult rats were hemisected at T8. Q-PCR for Hpa1 mRNA in the cord at T7-9 indicated initial (3 dpi) decrease followed by progressive increase to levels in excess of that in the normal cord by 14 dpi. Combined *in situ* hybridization for Hpa1 mRNA and immunocytochemistry for cell markers indicated increased Hpa1 expression in GFAP$^+$ astrocytes and ED-1$^+$ macrophages in the lesion by 7 - 14 dpi. Upregulated Hpa1 expression was also observed in astrocytes that invaded the scratched area in an *in vitro* model. Taken together, our results indicated upregulated expression of Hpa1 in astrocytes and macrophages that were recruited to the injury.

CRCG10204997&10205634 to DKYS

## P2-d05 Wnt5 signaling for glomerular patterning in the *Drosophila* olfactory system

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In *Drosophila*, odor specificity is represented as a spatial map of activated glomeruli in the first olfactory relay center called antennal lobe (AL). About 50 glomeruli, each formed by specific synaptic connections between olfactory receptor neurons (ORNs) and projection neurons (PNs), are stereotypically organized by multiple regulatory processes during olfactory development. Here we reveal that glomerular patterning is controlled by Wnt5 signaling transmitted in part from ORNs to PNs. In the *Wnt5* mutant, several glomeruli shifted to ectopic positions, and this phenotype was rescued by *Wnt5* expression in ORNs. Previous studies in *Drosophila* embryos have shown that Wnt5 repels the axons expressing Derailed (Drl). In the AL, however, our analyses indicate that Drl, localized in subsets of PN dendrites, has an antagonistic function in Wnt5 signaling. These data suggest that Wnt5 secreted from ORN axons differentially repels pre-patterned PN dendrites expressing varying amounts of Drl and a second receptor to establish the stereotyped glomerular pattern.

## P2-d06 Change in the microtubule-anchoring proteins at the centrosome during the differentiation of mouse neurons

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Neurons are rich in non-centrosomal microtubules. These microtubules are thought to be important for axon elongation. In general, microtubules are nucleated at centrosome with γ-tubulin and they are anchored there by microtubule-anchoring protein, ninine. In this study, we generated antibodies against ninein and studied the distribution of ninein, as well as those of γ-tubulin and pericentrin (centrosome marker) during the development of the mouse cerebral cortex. In radial cells at the ventricular zone, γ-tubulin and ninein are localized at the centrosome. In migrating neurons in E11 cortical plate, γ-tubulin was still detected at the centrosome, but ninein was not detected there. In differentiated cortical neurons of young adult mice, γ-tubulin and ninein was not detected at the centrosome. Pericentrin-antibody clearly stained centrosomes of all neurons. These observations suggest the change in the microtuble-anchoring function of centrosomes during the differentiation of neurons that might be responsible for the generation of non-centrosomal microtubles.

KAKENHI(16027207, 16500193)

## P2-d07 Function of draxin in the migration of neural crest cells

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Neural crests cells originate from dorsal neural tube and migrate out through anterior half of the somite to form diverse derivatives. Several repulsive axon guidance molecules, such as ephrin-B, semaphorin and slit have been shown to play important roles in neural crest migration. Here we show a novel molecule named draxin, which is expressed in roof plate and functions as a repulsive guidance molecule for spinal cord commissural axons. Neural crest cell migration from the neural tube explants was inhibited by draxin. After over expression of draxin in early chick embryos by electroporation at stage 12, many neural crest cells migrated ectopically at stage 18 along the dorsal-lateral pathway which is a normal pathway for later born cells start at stage 20. These studies suggest that draxin, a new repulsive axon guidance molecule, can also repel neural crest cells in migration out from chick neural tube.

## P2-d08 Analysis of a novel guidance molecule, draxin, in chick embryonic tectobulbar axon projection.

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Tectobulbar tract in chick embryo is the first long distance projecting fiber pathway to appear during the development of the avian optic tectum. It starts from E3 where fibers proceeding downward from dorsal to ventral side on the lateral wall of the optic tectum and join the medial longitudinal fasciculus of the same side or cross the floor of the optic tectum and descend on the opposite side and this tract does not cross the dorsal midline of the tectum. Here we report a molecule named draxin, which plays a role in spinal cord commissural axon guidance. Draxin is expressed in dorsal high to ventral low gradient in chick optic tectum. Draxin protein is deposited in the most dorsal part of the tectum. In vitro experiments show that this molecule inhibits neurite outgrowth from tectum explants and in vivo over expression induced misrouted or stopped axon outgrowth in the tectum. So this molecule may be an important member of the tectobulbar tract formation.

## P2-d09 Regulation of cortical laminar formation by cell-cell contacts

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Cerebral cortical neurons are sequentially generated in the ventricular/subventricular zones of the dorsal telencephalon, migrate to their destinations within the cortical plate, in which later-born neurons migrate over earlier-born deeper-layer neurons and occupy more superficial layers, and form a six-layered structure. Recently, behaviors of migrating neurons have been revealed from time-lapse imaging analyses and cell labeling experiments. They migrate along radial fibers separately and stay beneath the marginal zone (MZ) for about one day forming a relatively densely structure. These observations prompted us to hypothesize that birth-date-dependent cell adhesive specificity might regulate the birth-date-dependent cortical layer structure. To test this hypothesis, we screened for the adhesion-related molecules that showed a stage-dependent expression pattern beneath the MZ. Here we report the results of expression pattern and function of the identified molecule(s).

## P2-d10 The analyses of the role of DISC1 in the formation of the neocortex

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The recent advances based on genetic research have revealed candidate genes that cause schizophrenia. *Disrupted-In-Schizophrenia-1 (DISC1)* is one of the promising candidates. Recent linkage and association studies also indicate that DISC1 has a role in schizophrenia in general populations. We have shown that DISC1 plays important roles in the development of the cerebral cortex by utilizing the *in utero* gene transfer system. We next compared the effects of different reagents, and the affected neuronal migrations by the loss of DISC1 function were analyzed more in detail. We tried to elucidate the role of DISC1 in the neuronal radial migration as well as other steps of cortical development.

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## P2-d11 Functional analyses of the molecules that control the behavior of multipolar migration neurons in the developing cerebral neocortex

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In the developing cerebral neocortex, postmitotic neurons in the ventricular zone (VZ) do not directly migrate into the cortical plate (CP) but stay for some time in the subventricular zone (SVZ) with multipolar morphology. In this zone, they dynamically extend and retract multiple processes, moving as if they are seeking for some extracelluar signals. However, what molecules control this "multipolar migration" is still unknown. In this study, we identified receptor molecules that are preferentially expressed in the multipolar migration neurons in the SVZ. Although some of the ligand molecules for these receptors were not produced in the CP, these ligand proteins were detected in the intermediate zone or the VZ, facing to the SVZ. These coordinated distribution patterns of receptors and their ligands suggest that the behavior of multipolar migration neurons may be regulated by the adjacent structures through the ligand-receptor interactions.

## P2-d12 Molecular mechanism of enhancement of neurite outgrowth by Sema4D

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Semaphorin 4D (Sema4D) was identified as a transmembrane member of semaphorin family of vertebrate. Sema4D is enriched in the neuronal and immune tissue. Semaphorins were primarily found as repulsive molecules. However, recently it was revealed that these had also functions as attractive factors to grow axons depending on neuron types or developing stages. For example, we previously showed that Sema4D stimulated axonal outgrowth of embryonic dorsal root ganglion (DRG) neurons. Sema4D acted as an attractive cues in a nerve growth factor (NGF)-dependent manner in these neurons. In this study, we investigate whether Sema4D itself activates NGF receptor, TrkA or it stimulates NGF-triggered activation of TrkA. We found Sema4D accelated NGF-induced activation of TrkA when cells were transfected with trkA and plexinB1, but did not when cells were transfected with trkA alone. And we found that PlexinB1 is associated with TrkA by extracellular domains. Furthermore, we found that co-expression of PlexinB1 and TrkA regulated the Sema4D-induced activation of RhoA.

## P2-d13 Quantitative analysis of Shootin1 and neurite elongation.

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Shootin1 is known as a molecule which plays an important role in forming neuronal polarization. Active transportation from soma to neurite tip induces accumulation of Shootin1 during development. This accumulation is closely related to neurite elongation. However, the accumulation profile, especially how the accumulation attenuates, is still unknown. Although experiments show that the attenuation depends on the neurite length, it has not been quantified and analyzed. To evaluate the attenuation process, we developed the algorithm which quantifies neurite length and Shootin1 concentration at neurite tip. The quantified data shows that the decay time constant has negative relation to neurite length. Next, to clarify whether passive diffusion of Shootin1 results in this attenuation, we made biophysical analysis. Our result suggests that Shootin1 passively diffuses into neurite shaft. This result is helpful for modelingneuronal polarization by Shootin1 accumulation.

## P2-d14 A homeodomain transcriptional factor, Homothorax, controls dendritic and axonal targeting in the *Drosophila* brain

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The precise neuronal connectivity in the nervous system depends on specific axonal and dendritic targeting of individual neurons. Temporal and spatial regulation of gene expression by transcription factors (TFs) is thought to play seminal functions in determining wiring specificity. We are interested in the identification of the genes that control specification and connectivity of antennal lobe projection neurons (PNs), which convey olfactory information from antennal lobes, the primary olfactory centers in the fly brain, to higher order structures. Here, we show that Homothorax (Hth) is expressed in most of the PNs. MARCM mosaic analyses showed that *hth* mutations cause profound axonal and dendritic targeting defects. Intriguingly, *hth* is required for the expression of *extradenticle*, another homeodomain-containing TF, in PNs but seems to function independently of other TF such as *acj6* that have identified to control PNs. Based on these data, we will discuss the functional roles of hth in the dendritic and axonal specification in the *Drosophila* brain.

## P2-d15 Axonal calcium sweep: somatic remote control of axon elongation

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Local signal in axonal growth cones is believed to mediate their responses to guidance molecules, and little is known about whether other cellular compartments also remote-control axon outgrowth. In dispersed cultures of dentate granule neurons, we found that local application of glutamate to the perisomatic area, but not an axon terminal, induced a retraction of the axon terminal. This application initiated a wave-like propagation of calcium signal from the soma to the axon terminal, which was followed by axon retraction. Axon retraction and axonal calcium sweep were both inhibited by an L-type voltage-sensitive calcium channel blocker, thapsigargin, and 2-aminoethoxydiphenyl borate. Thus, even the perisomatic compartments of neurons are capable of controlling the dynamics of axon terminals through distant intracellular signal communication.

KAKENHI17689004, KAKENHI18021008, KAKENHI18890052

## P2-d16 Analysis of thalamocortical projection in *crmp1* deficient mice

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CRMPs (Collapsin Response Mediator Proteins) are predominantly expressed in the nervous system and possibly play important roles in neural development. However, *in vivo* function of CRMPs in axon guidance remains unclear. We analyzed the brains of newborn *crmp1*$^{-/-}$ mice to clarify the role of CRMPs in the formation of cortical circuit. We first examined immunostaining of neurofilament and L1 to evaluate axonal projections. In *crmp1*$^{-/-}$ mice, L1 immunoreactive thalamocortical axons diffusely distributed than those of *crmp1*$^{+/-}$ mice. Several neurofilament-positive fibers invaded marginal zone of cortical plate. Since CRMP1 mRNA was expressed in thalamic nuclei, we next examined thalamocortical projection by DiI-labeling. Several labeled axons from medial dorsal thalamus irregularly invaded the marginal zone in *crmp1*$^{-/-}$ but not in *crmp1*$^{+/-}$. These results suggest that CRMP1 may contribute to layer specific thalamocortical projection.

## P2-d17 The interpeduncular nucleus in zebrafish transmitted left-right information from the habenulae to the distinct targets in the hindbrain

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Although functional asymmetry is a conserved feature of the vertebrate brain, surprisingly little is known about the patterns of neuronal connectivity that account for it. We previously showed the asymmetric connectivity in the habenulo-interpeduncular projection in zebrafish, i.e. the stereotypic, topographic projection of left-sided habenular axons to the dorsal region of IPN and right-sided habenular axons to the ventral IPN. To identify the target region of IPN efferents, we labeled a small number of projection neurons in the dorsal and ventral IPN with lipophilic dyes under the guidance of GFP transgene which was expressed in the ventral IPN. The results revealed that the efferent fibers from the ventral IPN mainly projected to the raphe nuclei at the midline, while the efferent fibers from the dorsal IPN mainly projected to the periaqueductal gray. This suggested that the left and right habenulae could differentially influence the output pathway in the hindbrain through the midbrain interpeduncular nucleus.

KAKENHI18700331

## P2-d18 Sema4D/Plexin-B1 activates PTEN via R-Ras GAP activity,inducing growth cone collapse

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Plexins are receptors for repulsive axonal guidance molecules semaphorins, and semaphorin 4D (Sema4D) receptor Plexin-B1 induces growth cone collapse by functioning as an R-Ras GAP. We have recently reported that Sema4D/Plexin-B1 suppresses phosphatydylinositol-3-OH kinase (PI(3)K) signalling through the R-Ras GAP activity, inducing growth cone collapse. Phosphatydilinositol-3-phosphate (PIP3) level is critically balanced between PI(3)K and phosphatase and tensin homologue deleted on chromosome ten (PTEN). Here we examined the involvement of PTEN in the Plexin-B1 signalling of Sema4D. Phosphorylation of PTEN at Ser380 is supposed to suppress its phosphatase activity, and Sema4D induced the dephosphorylation of PTEN at Ser380 in hippocampal neurons. Furthermore, the R-Ras GAP activity was necessary for Plexin-B1-induced dephosphorylation of PTEN in COS-7 cells. Thus, we speculate that Sema4D concurrently induces PI(3)K inactivation and PTEN activation to control the PIP3 level through the R-Ras GAP activity of Plexin-B1, inducing growth cone collapse.

JSPS, KAKENHI

## P2-d19 Involvement of BDNF in mossy fiber fasciculation

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Hippocampal mossy fibers (MFs) are the axons of dentate granule cells and make excitatory synaptic contacts with CA3 pyramidal cells in the proximal portion of apical dendrites. To investigate the mechanism underlying layer-restricted projection of MFs in cultured slices, we established a novel experimental system to clearly visualize MF pathways by locally injecting GFP-expressing lentiviruses to the granule cell layer. Previously we have shown that application of K252a, an inhibitor of neurotrophin receptor, resulted in abnormal MF projections to the broad CA3 regions. In this study, we demonstrated that BDNF expression in granule cells using lentiviruses rescued the effect of K252a. We propose a crucial and novel role of BDNF in MF pathfindings.

## P2-d20 Functional regeneration of hippocampal neurons on multielectrode array dish controlled by femtosecond laser processing

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We have demonstrated the scission of a neuronal network by a focused femtosecond laser and analyzed the functional connections between neurons using multielectrode array. Cultured hippocampal neurons on multielectrode array dish were exposed to focused ~100 fs pulses of 800 nm wavelength. The spatiotemporal patterns of spontaneous action potentials were analyzed by a 64-site recording system for extracellular potentials of neurons. When the femtosecond laser was focused on neurons, the axons of neuronal cells were cut within range of a few μm due to multi-photon absorption process. After the laser irradiation, the synchronization of the spontaneous electric activities of neurons over the divided area drastically decreased, suggesting that the functional connections between neurons were divided because of the physical processing by the laser irradiation. Moreover, we found that spatiotemporal pattern in each divided region gradually re-synchronized within 10 days. The present results suggest that the neuronal network functionally regenerates with the development.

PRESTO, JST

## P2-d21 Neural cell recognition molecule NB-3 in the developing cerebellum

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NB-3 is a neural cell recognition molecule, which begins expression at the late stage of cerebellar development. We demonstrated previously that NB-3 deficient mice are impaired in motor coordination. To study the significance of NB-3 in cerebellar development and function, here we investigated the localization of NB-3 in the developing cerebellum in mouse. NB-3 immunoreactivity was overlapped with that of L1, which is localized on the surface of radially migrating granule cells and extending parallel fiber in the developing cerebellum between postnatal days 5 and 10. On the other hand, NB-3 immunoreactivity was not colocalized with that of TAG-1, which is expressed in the external granule cell layer at this developmental stage. Together, NB-3 may be implicated in the proper cell differentiation and migration of the cerebellar granule cells.

## P2-d22 Involvement of molecules in sub-areal expression of odorant receptor genes in the most ventro-lateral zone of the mouse olfactory epithelium

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Olfactory signals are received by odorant receptors (ORs) in the olfactory sensory neurons (OSNs) of the olfactory epithelium (OE) where expression areas of ORs are specific to each gene. We have so far analyzed a battery of OR genes in the most ventro-lateral zone (zone 4a and 4b), and found that the zone is divided further into at least three sub-areas, based on the expression and projection patterns for ORs. However, mechanisms, which specify the sub-areal expression of ORs and control the region-specific projection of OSNs to the olfactory bulb, are unknown. *Msx1*, *RALDH2* and *Neuropilin-2* are known to be expressed in ventro-lateral high to dorso-medial low gradients. Moreover, a subset of OSNs in the most ventro-lateral region of the OE specifically express cGMP-signaling components. Here, we have analyzed the detail expression patterns of these molecules and compared their expression areas with the sub-areal expression of ORs in zone 4a and 4b.

## P2-d23 Isolation of mutants that have abnormal localization of Netrin/UNC-6 or its receptor UNC-5 in C. elegans.

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Axon guidance molecules provide information that causes neurons to elongate their axons in appropriate direction. Thus, the proper localization of the axon guidance molecules and their receptors are required for the construction of the complex nervous system. Netrin is an evolutionarily conserved axon guidance molecule that has both axonal attraction and repulsion activities. In C. elegans, Netrin/UNC-6 is secreted from ventral cells, and it is thought to form a ventral-to-dorsal concentration gradient. UNC-5, a cell surface receptor of immunoglobulin superfamily, is implicated in mediating repulsion from Netrin/UNC-6 sources. However, their localization mechanisms are largely unknown. In order to elucidate the mechanism, we isolated many mutants in which the localization of Netrin/UNC-6 or UNC-5 was abnormal. We are now mapping the mutations and cloning the genes. We will discuss the possible mechanism of the localization of Netrin/UNC-6 or UNC-5.

## P2-d24 Downstream targets of Arx and Fez transcription factors regulating the development of mouse olfactory system

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During the mouse embryonic development, zinc-finger gene *Fez* and homeobox gene *Arx* are expressed in olfactory sensory neurons and in olfactory bulb interneurons/radial glia, respectively. In spite of the differential expression patterns, *Fez*- and *Arx*-deficient mice show similar phenotypes including the impaired innervation of olfactory axons and the abnormal morphogenesis of the olfactory bulb. To identify downstream targets of Fez and Arx, we performed microarray analyses between the wild-type and *Fez*-deficient olfactory epithelium, and between the wild-type and *Arx*-deficient telencephalon. For genes showing the changed expression level, these expression patterns were confirmed by *in situ* hybridization. By both analyses, we attempt to clarify the candidate genes that regulate the olfactory neural circuit formation.

KAKENHI16700300

## P2-d25 Axonal netrin-Gs transneuronally determine lamina-specific subdendritic segments

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Distinct populations of axons from multiple brain areas make contact with target dendrites in clearly-segregated and laminated patterns, thereby forming functional units for processing multiple inputs of information. Whether and how dendrites acquire the lamina-specific properties corresponding to each pathway is unknown. We report that vertebrate-specific membrane-anchored members of the netrin family organize the differentiation of subdendritic segments. Netrin-G1 and netrin-G2 are distributed on axons of different pathways. Their specific receptors, NGL-1 and NGL-2, are localized to distinct subdendritic segments of single neurons, corresponding to the lamina-specific termination of netrin-G1- and netrin-G2-positive axons, respectively. In netrin-G1 KO and netrin-G2 KO mice, the segmental distribution of NGL-1 and NGL-2 within dendrites is selectively abolished. These findings suggest that presynaptic innervation has an instructive role in dendritic segmentation. We are investigating a role for specific interactions of netrin-G/NGL in local synaptic functions.

KAKENHI (18700345)

## P2-d26 Activity-dependent nucleocytoplasmic translocation of HDAC9 is required for gene expression and dendritic growth in cortical development

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Chromatin remodeling in neuronal differentiation is an intriguing issue. Our recent study has shown that histone deacetylase 9 (HDAC9) is translocated from nucleus to cytoplasm in developing cortical neurons in response to neuronal activity. In this study, we investigated gene expression and morphological changes which the HDAC9 translocation induces by introducing an expression vector of HDAC9 mutant (S218A, S448A) or shRNA vector into dissociated cortical cell cultures. The result demonstrated that the mutant HDAC9 disrupted the translocation and suppressed expression of c-fos, an immediate early gene. Moreover, dendrite morphology became simple in the mutant transfected cells. In contrast, inhibition of endogenous HDAC9 by shRNA enhanced dendritic growth. These findings indicate that the HDAC9 translocation is required for activity-dependent gene expression and dendritic growth in cortical development.

KAKENHI 16700286, 18300105

## P2-d27 *In vivo* time-lapse imaging of migration of cortical interneurons during postnatal development

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Most GABAergic interneurons of the rodent cortex originate from the ganglionic eminences(GEs).During perinatal days, they migrate tangentially through the marginal zone(MZ) as well as intermediate zone/subventricular zone(IZ/SVZ) towards the cortex. After reaching the cortex, they migrate into the cortical plate and incorporate into the proper laminar position. To discuss the movement of the migration of interneurons in intact immature animal, we have developed *in vivo* time-lapse imaging with two-photon laser-scanning microscopy employed on immature transgenic mice expressing Venus-YFP in GABAergic interneurons of the cortex.

CREST,JST

## P2-d28 Netrin-1 regulates cell surface distribution of its receptor DCC in hamster primary cortical neurons

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DCC (deleted in colorectal cancer), a receptor for an axon guidance cue netrin-1, plays an important role in target innervation during development of central nervous system. In rat spinal commissural axons it is reported that intracellular vesicular pool of DCC exists and plasma membrane insertion of DCC occurs in response to netrin-1 / protein kinase A. In addition, rapid depletion of cell surface DCC after netrin-1 treatment is reported in retinal growth cones of *Xenopus laevis*, which is dependent on endocytosis.
In hamster cortical neurons netrin-1 is known to promote axon branching. In order to clarify whether translocation of DCC to, and/or endocytic removal of DCC from the plasma membrane occur by netrin-1 stimulation, we examined the changes in the level and distribution of DCC at the cell surface of hamster cortical neurons, employing total internal reflection fluorescence microscopy as well as cell-surface labeling techniques. A change in the distribution of cell surface DCC was observed in response to netrin-1 within 5 minutes.

## P2-d29 Identification of a novel zebrafish semaphorin

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Semaphorin gene family contains a large number of secreted or transmembrane proteins, and some of them function as the repulsive and attractive cues of axon guidance during development. Here we report a novel zebrafish semaphorin, semaphorin 6E (Sema6E). The ORF of Sema6E is 3192 bp (1064 aa). Zebrafish Sema6E protein showed a 45.1 % identity with zebrafish Sema6D protein. To clarify the expression pattern of Sema6E, in situ hybridization was performed. In the embryos, Sema6E is expressed in the lens and hindbrain. We also found that Sema6E induces the repulsion of the axons. These results indicate that Sema6E repels specific types of the axons and functions in the nervous system development.

KAKENHI on Priority Areas -Elucidation of neural network function in the brain- from the MEXT (18021031)

## P2-d30 Mechanism of activation of transforming growth factor-beta in the brain by exercise

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Transforming growth factor-beta (TGF-beta) is a well-known pleiotropic cytokine. We previously reported the increase of active TGF-beta in the cerebrospinal fluid (CSF) after exercise in rats. TGF-beta is secreted as a latent form which consists of latency associated protein (LAP) and mature TGF-beta itself. To reveal its activity, latent form should be activated by cleavage or removal of LAP. In this study we investigated the mechanism of activation of TGF-beta in the brain during exercise. Among the factors that increase during exercise in the blood, intraperitonial (i.p.) injection of lactate and beta-hydroxybutylic acid could reproduce the increase in active TGF-beta in the CSF. Matrix metalloproteinases and plasmin which degrade LAP, and thrombospondin-1 (TSP-1) which causes structural change to LAP, are reported to active TGF-beta. We found that intracisternal administration of LSKL peptide that inhibits the interaction between LAP and TSP-1 could only block the activation of TGF-beta by i.p. administration of lactate.

## P2-d31 The development of small nociceptive neurons in the dorsal root ganglia of oncostatin M receptor-deficient mice

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We have reported that oncostatin M receptor $\beta$ (OSMR $\beta$)-positive nociceptive neurons decrease in the dorsal root ganglia (DRGs) of the OSM-deficient mice. Recent studies have shown that interleukin-31 receptor A (IL-31RA) forms a functional receptor complex for IL-31 together with OSMR $\beta$. We have also demonstrated complete overlap of IL-31RA and OSMR $\beta$ in the small neurons of the adult DRGs, suggesting the redundant effects of OSM and IL-31 in the development of nociceptive neurons. In the present study, we investigated the development of nociceptive neurons of DRGs in OSMR $\beta$-deficient mice. Size frequency analysis revealed that IL-31RA-positive small-sized neurons decreased significantly in the DRGs of the OSMR $\beta$-deficient mice. In addition, TRPV1-positive, P2X3-positive, and TRPV1/P2X3-double positive neurons also decreased. Thus, signals through the OSMR $\beta$ is essential for the development of a subtype of nociceptive neurons in the DRGs.

## P2-d32 Neonatal heregulin / neuregulin signaling promotes development of cortical GABA neurons.

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Heregulin (HRG) is identical to neuregulin-1 and known to play crucial roles in the development of the central nervous system, such as glial differentiation, regulation of glutamate receptor expression, and neural migration. HRG receptor, ErbB4, is preferentially expressed in parvalbumin (PV)-positive GABAergic neurons. Here, we investigated the effect of HRG $\beta$ 1 on the development of cortical GABAergic neuron and inhibitory synaptic transmission. In dissociated cortical culture, chronic application of the HRG $\beta$ 1-EGF domain (25ng/ml/day for 7days) enhances the expression of PV, whereas it has no effect on GAD67 levels. Brain slices were prepared from the prefrontal cortex of the postnatal mice receiving the HRG $\beta$ 1-EGF domain and patch clump recording was performed in these slices. HRG $\beta$ 1 administration enhances both frequency and amplitudes of spontaneous IPSCs in pyramidal neurons. These results suggest that ErbB4 stimulation enhances the development of PV-positive GABAergic neurons and/or termini.

## P2-d33 Hypoxia inducible factor 1-$\alpha$ participate in dopaminergic differentiation from ES-derived neural stem cells

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To investigate how to induce DAergic differentiation from ES-derived neural stem cells (NSCs), low (3.5%) $O_2$ or cytokine cocktail (IL-1 $\beta$, IL-11, LIF, GDNF) was treated to NSCs. Treatment with cytokine cocktail or low $O_2$ increased the number of tyrosine hydroxylase (TH)-positive(+) cells as compared to controls (2.20 -fold or 1.83-fold respectively). Although major effect of cytokine cocktail was induced by LIF application alone, combination of low $O_2$ with cytokine cocktail did not show any additive effect. To investigate the mechanism of DAergic differentiation by cytokine cocktail, we focused on hypoxia inducible factor (HIF)-1 $\alpha$. As HIF-1 $\alpha$ expression was enhanced by LIF treatment or low $O_2$, antisense ODNs to HIF-1 $\alpha$ was treated to NSCs followed by TH immunnostaing, revealing that the number of TH (+) cells was significantly decreased in LIF-treated NSCs. Data suggest that enhanced differentiation to DAergic neurons from ES-derived NSCs by low $O_2$ or cytokines was mediated by increased expression of HIF-1 $\alpha$.

## P2-d34 Neonatal EGF challenge attenuates GABAergic development in the neocortex and hippocampus

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We found that administration of epidermal growth factor (EGF) to neonatal mice down-regulates expression of AMPA-type glutamate receptors in developing GABAergic neurons. In parallel, the strength of recurrent inhibitory activities is also decreased in the dentate gyrus (Neurosci. Res. (2005) 52 Suppl. S133). Here, we examined phenotypic expressions of GABAergic markers in the frontal cortex and hippocampus of EGF-treated postnatal mice. EGF administration suppressed protein-levels of parvalbumin (PV) in the brain regions. The expression of GABA transporter, GAT-1 was also decreased in the frontal cortex. Slice patch-clamp recordings from the dentate gyrus of the EGF-administered mice revealed that the frequency of spontaneous inhibitory postsynaptic currents was decreased in the granule cells. Thus, during early postnatal development, activation of EGF receptors (ErbBs) negatively influences the phenotypic and functional maturation of PV-positive GABAergic neurons.

## P2-d35 Analysis of regulatory mechanisms of LPS-induced GDNF gene expression in primary cultured glial cells

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GDNF is known as an important neurotrophic factor. However, the details of mechanisms that regulate the expression of GDNF gene are not well understood. In this study, we investigated regulatory mechanisms of LPS-induced GDNF gene expression in primary cultured astrocytes and microglia. First, we examined whether NF-κB is responsible for LPS-elicited expression of GDNF mRNA since its consensus sequence exists in intron 1 of GDNF gene. However, MG-132, a blocker of the NF-κB pathway, did not affect the GDNF mRNA induction, whereas it attenuated the up-regulation of iNOS and TNF-$\alpha$ mRNA elicited by LPS stimulation. Next, we estimated whether MAPK cascades are involved in LPS-induced GDNF mRNA expression. We found that SP600125, an inhibitor of JNK, blocked the GDNF mRNA induction in LPS-stimulated microglia but not astrocytes. U0126 (a MEK1/2 inhibitor) and SB202190 (a p38 inhibitor) showed no effect on this induction in both of the cells. Our data indicate that JNK participates in LPS-induced GDNF mRNA expression only in microglia.

## P2-d36 Roles of stem cell factor during mouse corticogenesis

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Stem cell factor (SCF) plays critical roles in proliferation and differentiation of various cells, mostly these derived from neural crest. Although SCF and its receptor c-kit are expressed in the developing cerebral cortex, their function in the corticogenesis is poorly understood. Therefore, we analyzed the effect of SCF injected into the ventricular space of mouse embryos at the embryonic days 13.5 in utero on the development of cerebral cortex. SCF formed heterotopia, which composed of neurons rather than astrocytes, in the anterior portion of lateral ventricule and/or in the brain parenchyma when observed at postnatal 6 days. Furthermore SCF stimulated proliferation of SOX2-positive cells (neural stem cells) in the ventricular zone, but did not that of Tbr2-positive cells (neuronal progenitor cells) in the subverntricular zone, demonstrating that SCF acts on neural stem cells rather than neuronal progenitor cells.

## P2-d37 Decreases in the expression of neurotrophins and the phosphorylation of MAP kinase in an animal model of depression

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Patients of depression are increasing in recent years with developing stressful society. Neurotrophins including nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF) and neurotrophin (NT)-3 are thought to be involved in pathogenesis or etiology of depression. Therefore, we examined mRNA expression of neurotrophins in an animal model for depression. Seven-week-old ddY male mice were forced to swim for 15 min, and kept in the leaning, drenching and rotated cages within 1-2 days, respectively. Mice given such stress showed typical behavior symptoms, including poor score in the burying glass marble test and tail suspension test. The levels of mRNAs of BDNF, NGF, or NT-3, and phosphorylation of mitogen-activated protein kinase (MAPK) were decreased in the brain including hippocampus of the stressed mice. As MAPK can be activated by all neurotrophins, the decreased neurotrophins might lower the MAPK activation, suggesting the involvement of neurotrophin production and action on MAPK signaling in pathogenesis of depression.

## P2-d38 Glucocorticoid reduced the BDNF-mediated synaptic maturation in developing hippocampal neurons

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Intensive glucocorticoid due to dysfunction of HPA axis may induce neuronal damage and cause depressive disorder. In contrast, the involvement of BDNF in the antidepressive effect has also been suggested. Thus, here, we investigated the effect of glucocorticoid (Dexamethasone, DEX) on synaptic maturation enhanced by BDNF in developing hippocampal neurons. In the immature stage, BDNF increased the outgrowth of dendrites and the expression of synaptic proteins including glutamate receptors and presynaptic proteins. As expected, DEX pretreatment significantly inhibited the BDNF-dependent up-regulation of dendritic outgrowth and synaptic proteins. Interestingly, DEX reduced the BDNF-activated MAPK pathway, a required intracellular signaling for the BDNF-increased synaptic proteins. Therefore, glucocorticoid may decrease the BDNF function via reducing of MAPK activation.

Japan Health Sciences Foundation

## P2-d39 Glucocorticoid depresses the BDNF-induced glutamate release via the reduction of TrkB-GR interaction

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Intensive level of glucocorticoid is supposed to contribute to the pathophysiology of depressive disorder. In contrast, BDNF is considered critical for the antidepressive effect, however, the influence of glucocorticoid on the action of BDNF has not been identified. Here, we found that pretreatment with glucocorticoid (dexamethasone, DEX) suppressed BDNF function including BDNF-induced glutamate release and intracellular Ca increase in cortical neurons. Interestingly, down-regulation of the glucocorticoid receptor (GR) was observed in DEX-treated cultures. Remarkably, GR interacted with TrkB and the GR-TrkB interaction was decreased after DEX exposure, suggesting that the down-regulation of GR caused by DEX results in reduction of the GR/TrkB complex and BDNF-dependent neuronal function.

Japan Foundation for Aging and Health and Ministry of Education, Culture, Sports, Science and Technology

## P2-d40 Involvement of NFAT in chemokine release from microglia induced by ATP

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A prominent feature of activated microglia is to produce and release proinflammatory cytokines or chemokines, which play crucial roles in pathological CNS inflammation. ATP leaked from damaged cells is a potent activator of glial cells including microglia, and ATP has been reported to release cytokines from microglia. However, little is known about the role of ATP in release of chemokines from microglia. Here we show that ATP evokes the release of MIP-1 $\alpha$ (a CC chemokine) from MG-5, a microglial cell line. The peak of the ATP-evoked MIP-1 $\alpha$ release was observed at 3 hr after ATP stimulation. ATP also caused the de-phosphorylation and the nuclear translocation of NFAT, a transcription factor involved in the production of MIP-1 $\alpha$, in MG-5. The ATP-evoked MIP-1 $\alpha$ release was prevented by BBG, an antagonist of $P2X_7$ receptor, and by a NFAT inhibitor.These results suggest that stimulating $P2X_7$ receptors by ATP may release MIP-1 $\alpha$ from microglia via NFAT activation.

## P2-d41 Protective effect of genipin on the typical ER stress-induced cell death

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We have previously indicated that genipin, a natural iridoid compound, induced neuronal differentiation in PC12h and Neuro2a cells and that the compound exerts protective effect on 6-hydroxydopamine-induced cell death with oxidative and/or ER stress in Neuro2a cells. In this study, we examined whether genipin is effective against the cell death induced by typical ER stress using its specific inducers in Neuro2a cells. Each ER stress inducer we used, *i.e.* A23187 ($Ca^{2+}$ ionophore), tunicamycin (an inhibitor of N-glycosylation in ER), brefeldin A (an inhibitor of ER-Golgi transport) or thapsigargin (a disruptor of $Ca^{2+}$ homeostasis), induced expression of GRP78 protein, a marker of ER stress. Genipin significantly suppressed the cytotoxicity induced by these inducers. These results suggest that genipin is an effective compound against ER stress-induced cell death. Thus, genipin may be useful in the treatment of several diseases resulted from ER stress.

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## P2-d42 Circadian changes of BDNF and NT-3 are important for cortical development of juvenile rats

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We have shown that the concentrations of cortical BDNF and NT-3 in 2-week-old male pups are low and high, respectively, in the light phase. Here we examined environmental factors influencing levels of cortical neurotrophins. In pups kept for one week from 6-day-old in a room with an eight-hour phase advance of the light-dark (LD) cycle, cortical BDNF in the light phase increased. Continuous light or dark for 24 h had no changes in levels of BDNF or NT-3. In sleep-deprived rats, BDNF increased whereas NT-3 decreased. In urethane-treated rats, opposite patterns for BDNF and NT-3 changes were observed. BDNF and NT-3 were localized in neurons of the cortical layers, V-VI and IV, respectively. Phase advance of the LD cycle caused low expression of synaptophysin in the layer IV, which was also found in BDNF gene-deficient mice. Thus a balance between BDNF and NT-3 levels, being influenced by sleep rhythm, appears important for functional development of the cortex.

## P2-d43 Distinct expression profiles of TrkB variants in the developing mouse nervous system

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TrkB is a brain-derived neurotrophic factor (BDNF) receptor and three spliced variants of TrkB, namely the tyrosine kinase domain (TK) intact receptor TK(+) and two TK(-) isoforms T1 and T2, have been characterized in rat. Truncated TK(-) forms have been assumed to play a dominant negative role on TK(+). Recently, others and we showed that T1 alone could activate a discrete signaling cascade in astrocytes, yet roles of T2 are uncertain. Here we extensively map mRNA expression patterns of TrkB variants and BDNF by using a sensitive in situ hybridization method further to gain insights in TK(-) specific functions during mouse development. As a result, we found that TK(-) mRNA expression did occur in TK(+) negative cell populations both at embryonic and postnatal stages. In addition, we realized differential expression profiles of T1 and T2 transcripts along the ventricular zone of embryonic and postnatal brains, implicating novel and separable roles of TK(-) forms in regulating cell division, migration and differentiation of ependymal cells.

## P2-d44 The nuclear translocation of p25/Cdk5 in cultured neurons undergoing cell death

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We reported recently that ER stress induced the calpain-dependent cleavage of p35 Cdk5 activator to the N-terminal truncated p25 in primary cultured cortical neurons, resulted in the translocation of p25/Cdk5 from cytoplasm to nucleus. Furthermore, ER stress-induced neuronal death was inhibited by Cdk5 inhibitor roscovitine, suggesting that p25/Cdk5 is a proapoptotic factor that promotes ER stress-induced neuronal death in nuclei. However, it is not known yet how p25/Cdk5 induces cell death in nuclei. To elucidate the mechanism of p25/Cdk5-induced neuronal death, we expressed p25 in primary cultured neurons using the adenovirus vector. Unexpectedly, however, the exogenously expressed p25 exhibited neither specific nuclear localization nor induction of cell death. p25 was distributed mainly in the cytoplasm. When the ER stressor, tunicamycin or thapsigargin, was applied to the p25 expressing neurons, the nuclear accumulation of p25 was detected. We are now investigating the mechanism of p25/Cdk5 accumulation in nuclei.

## P2-d45 The effect of TNF-$\alpha$ and IL-1$\beta$ against the A$\beta$25-35 induces neuronal cell death in mouse organotypic hippocampal slice cultures (OHSCs)

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Neuroinflammation is known key player in the pathology of Alzheimer's disease (AD) and cytokines such as TNF$\alpha$ and IL-1$\beta$ plays key role in the progression of inflammatory cascade. In this study, we investigate the effect of TNF$\alpha$ and IL-1$\beta$ in A$\beta$-induced toxicity using mouse OHSCs. Our result that the A$\beta$25-35 induces dose-dependent neuronal cell death. Additionally, we co-treated A$\beta$25-35 with TNF$\alpha$ or IL-1$\beta$ which observed neuronal cell death through propidium iodide and immunofluorescence staining. As treating of TNF-$\alpha$ or IL-1$\beta$, cell death induced by A$\beta$25-35 was reduced. So, we are studying about the role of NFκB in AD using transgenic mice. These results indicate that TNF$\alpha$ and IL-1$\beta$ have partial role in A$\beta$25-35-induced neurotoxicity.

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## P2-d46 Microglia and astroglia prevent oxidative neuronal cell death in patient with aceruloplasminemia.

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Glial predominant cells contribute more to iron(Fe) accumulation but are more resistant to oxidative stress than neuron(N). In this study, we showed that cell death occurred in N and microglia(MG) after Fe overload. However,astrocyte(AS) would rather proliferated under the same condition. AS had a low specific activity for transferrin-independent Fe uptake(Tf-IU), but MG had extremely high specific activity. N-MG co-cultures suppressed the Fe uptake and Fe-induced cell death. N-AS co-cultures increased the Fe uptake and completely suppressed the cell death. These results suggest that N-MG co-cultures suppress the Tf-IU and N-AS coculture takes up Fe for AS proliferation to protect N. This in viro model may explain the pathophysiological observations in the brains of patient with aceruloplasminemia.

## P2-d47 Importance of up-regulated Bim expression during tunicamycin-induced ER stress-mediated apoptosis in PC12 cells

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We have reported that tunicamycin (Tm) induces ER stress-mediated apoptosis via the activation of caspase cascade in PC12 cells. However, it is not clear whether there exist some different mechanisms between ER stress-induced apoptosis and ordinary apoptosis or not. We have focused on the expression of Bcl-2 family protein, Bim, which is known to be induced by ordinary apoptosis. In the present study, we show that Tm up-regulated Bim expression 24 hours after the treatment in PC12 cells. Tm-induced ER stress-mediated apoptosis was prevented by the addition of siRNA against Bim. The relationship between Bim expression and the activation of caspase-12 during ER stress-induced apoptosis, and the mechanism underlying the effect of nerve growth factor on Bim expression and the activation of caspase-12 are now under investigation.

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## P2-d48 Signaling pathway of caspase-4 in endoplasmic reticulum stress-induced apoptosis of familial Alzheimer disease

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Caspase-4 functions as an endoplasmic reticulum (ER) stress-specific caspase in humans and suggested to play a key role in pathogenesis of Alzheimer disease (AD). We found that cleavage of caspase-4 under ER stress is enhanced by familial AD (FAD)-linked mutation, showing that caspase-4 is the key caspase involved in the pathogenesis of AD. We also exhibited that overexpression of caspasae-4 induced the cleavage of caspase-9 and caspase-3 without any effect on effulux of cytochrome-c from mitochondria to cytosol. Thus present study established that caspase-3 and caspase-9, which are existed in the downstream of caspase-4, could be cleaved and activated directly by the active form of caspase-4 without cytochrome-c pathway under ER stresses.

## P2-e01 Survival-promoting effect of pituitary adenylate cyclase-activating polypeptide (PACAP) on rat motoneurons in culture

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Amyotrophic lateral sclerosis (ALS) is a progressive motoneuron disease, whose pathogenesis is yet to be clarified. PACAP has been shown to be neurotrophic or neuroprotective in many types of brain neurons in culture. PACAP was shown to be expressed in spinal motoneurons in vivo and its expression increased markedly after axotomy, suggesting a role of PACAP as a lesion-induced factor. However, the direct survival effect of PACAP on motoneurons has not been reported and, therefore, it was tested in this study using rat motoneurons in culture. In the presence of a phosphodiesterase inhibitor, IBMX, PACAP showed a significant survival effect. Previously we have shown that 5-HT as well as glutamate was excitotoxic to motoneurons. PACAP also showed the survival effect on motoneurons under these excitotoxic conditions. Taken together with previous reports, these results suggest that PACAP is a physiological neuroprotective factor acting on motoneurons in an autocrine manner.

Ministry of Health, Labour and Welfare, Research Contract 18-8

## P2-e02 The mechanism of neuronal death through the activation of caspase-9,-3 via caspase-4 in human neuroblastoma SH-SY5Y cells

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Recently, it has been reported that endoplasmic reticulum (ER) dysfunction is involved in neurodegenerative diseases. Several lines of evidence indicate that caspase-4, a member of caspase-1 subfamily, is localized to the ER membrane, and is activated when cells are treated with ER stress-inducing reagents. Tunicamycin is an agent to impairs the functions of ER. In this study, we examined the molecular mechanisms of ER stress-induced apoptosis via activation of caspase-4 by tunicamcyin in human neuroblastoma SH-SY5Y cells. ER stress-induced apoptosis was inhibited by a caspase-3 inhibitor DEVD-CHO, a caspase-9 inhibitor z-LEHD-fmk or a caspase-4 inhibitor LEVD-CHO. To examined whether caspase-4 can activate caspase-3 or caspase-9, the cell lysates were incubated with human recombinant active caspase-4 protein. Caspase-9 and caspase-3 were activated by human recombinant active caspase-4 protein. These results suggest that ER stress-induced apoptosis is mediated by caspase-3 or caspase-9 activation partly through the activation of caspase-4.

## P2-e03 Effects of proteasome inhibitor on tunicamycin-, thapsigargin-, and polyglutamine-induced ER stress and cell death in PC12 cells

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Treatments of tunicamycin (Tm) and thapsigargin (Tg) and expression of polyglutamine (Q81) induced ER stress and cell death in PC12 cells. PC12 cells transfected with Q81-EYFP-Myc constructs under the control of Tet-Off system were kindly provided by Dr Yoshitaka Nagai (Osaka University). Tm, Tg and Q81 induced up-regulation of Bip/GRP 78 and P-eIF2 $\alpha$ levels. Q81 led to protein ubiquitination, whereas Tm (1 μg/ml) and Tg (1 μM) caused no or only slight induction of the ubiquitination. Treatment of an proteasome inhibitor, lactacystin at 1 μM slightly promoted the decrease in the viability of the cells treated with Tm, Tg and Q81. Tm, Tg and Q81 induced the activation of caspase-3 and JNK, which was slightly promoted by lactacystin at 1 μM. Based on these results, involvement of ubiquitin-proteasome systems in Tm, Tg and polyglutamine-induced death of PC12 cells is discussed.

## P2-e04 A genomic screen for novel genes with a cell-death-related function in neurons

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Most of neurodegenerative disorder is frequently correlated with brain region-specific patterns of neuronal cell death. The mechanisms underlying neuronal cell death remain poorly understood. Drosophila is a powerful genetic model system, which has been well studied as a developmental system. Recently, Drosophila has been used to elucidate mechanisms of human neurodegenerative diseases, including Alzheimer's, Parkinson's, and Huntington's diseases. To identify previously unknown genes associated with neuronal cell death, we have carried out a genetic screen in Drosophila. We generated mutant animals by ethylmethane sulphonate treatment. In total, approximately 1000 mutants were screened. So far, we found out 3 mutant animals which have neurodegenerative phenotypes. One mutant causes age-dependent neuronal cell death, and other two mutants cause neurodegeneration during development. Now, we are trying to identify the candidate genes for these 3 mutants. Analyzing these mutants may provide us the new idea for the mechanism of neuronal cell death.

## P2-e05 Amyloid-like aggregate of human GAPDH is involved in neuronal cell death

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Glyceraldehydes-3-phosphate dehydrogenase (GAPDH) is involved in neuronal cell death triggered by oxidative stress, and is deposited in neurodegenerative diseases. Recently, we reported that oxidative stress induced the aggregation of rabbit GAPDH through disulfide bonds including Cys149 and Cys281. In the present study, we investigated whether oxidative stress induced the aggregation of human GAPDH and aggregates could cause neuronal cell death. We generated cystein-substituted mutants. Since human GAPDH lacked the cystein corresponding to Cys281, we also generated S283C mutant. An oxidant induced the aggregation of the wild type (WT) and S283C, and the aggregation level of S283C is much higher. In contrast, C151A failed to show the aggregation completely. Additionally, an exposure to oxidant resulted in formation of the amyloid-like fibrils of WT and S283C respectively. Then, we prepared stable cell lines of SH-SY5Y expressing WT, C151A, or S283C. Causal relationship between aberrant aggregation of human GAPDH and neurodegeneration is under investigation.

## P2-e06 Survival effect of cyclophosphamide in cultured cortical neurons

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Cyclophosphamide (CP) has been known as an antitumour agent or immunosuppressant in the clinic treatment, however, a neuronal role of CP has not been investigated. Here, we found that CP exerted a survival promoting effect on cultured cortical neurons prepared from rats cereberal cortex. Interestingly, activation of MAPK and up-regulation of Bcl-2, both function as an anti-apoptotic factor, were induced by CP exposure significantly. Furthermore, U0126, a MAPK pathway blocker, reduced the CP-dependent Bcl-2 up-regulation and cell survival, suggesting that CP has a survival promoting effect on CNS neurons via stimulating the MAPK/Bcl-2 pathway.

## P2-e07 Function of chondoroitin sulfate in ER stress exposed glial cells

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Chondoroitin sulfate (CS) is a long linear sugar chains constructed by repeating disaccharides units with or without sulfation. And CS is thought to be crucial for regulating cell survival, cell migration, cell maturation and differentiation signals. CS is known to be an axon growth inhibitory molecule in the glial scar. Astrocytic CS production is reported to be increased after injury and neurodegeneration by Amyloid-$\beta$. Therefore, CS expression by glial cells may be regulated under stress conditions in the brain. To demonstrate this hypothesis, we examined alteration of CS expression levels under ER stress condition and effect of CS on ER stress induced cell death in astrocytes and C6 glioma cells. ER stress by tunicamycin and thapsigargin altered CS expression level in C6 cells. Addition of CS reduced cell death induced by thapsigargin, but not by tunicamycin in C6 cells and astrocytes. While CS had no significant effect on ER stress induced neuronal cell death. Our data suggests that synthesis of glial CS in injured brain contribute to glial cell survival.

## P2-e08 Alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) rapidly induces necrosis and DNA fragmentation in hippocampal neurons

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Under the video enhanced contrast-differential interference contrast microscope, continuous exposure to AMPA dose-dependently showed the nuclear granulation and the cellular swelling in hippocampal neurons. Since the character of the nuclear granulation remains unclear, we investigated: 1) whether the process is necrosis or apoptosis; and 2) whether the nuclear granulation coressponds to DNA fragmentation. To detect the types of cell death, hippocampal neurons were stained with propidium iodide (PI) and fluorescein-4-isothiocyanate (FITC) conjugated annexin V (annexin V-FITC). DNA fragmentation was assessed using comet assay. The results showed that: 1) AMPA (10 μM-1 mM) dose-dependently induced necrosis; and 2) the treatment with AMPA (100 μM, 1 mM) for 1 h significantly increased the percentage of severer DNA damage. We conclude that AMPA application induced DNA fragmentation in the very early stage of necrosis.

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## P2-e09 Free intracellular high-mannose oligosaccharides protect cell death induced by endoplasmic reticulum (ER) stress

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Free high-mannose oligosaccharides (OS) are known to be released into the ER lumen during N-glycosylation process. However, the functions of free OS have not been focused in mammalian cells. In the present study, we found that treatment of the PC-12 cells with an $\alpha$-mannosidase inhibitor 1-deoxymannojirimycin (DMM) increased the concentration of intracellular free OS and suppressed ER stress (tunicamycin, thapsigargin, and amyloid $\beta$ 1-40) - induced cell death in PC-12 cells. These results suggest that increased free Man9GlcNAc2 by DMM treatment is involved in the suppression mechanism on ER stress induced cell death. Furthermore, in vitro amyloid formation assay by using thioflavin T, Man9GlcNAc2 oligosaccharide inhibited the amyloid formation of amyloid $\beta$ 1-42. Our findings suggest that free OS may act as a suppressor of neurodegenaration by reducing protein misfolding and/or aggregation, and be applicable to the diagnostic marker of the risk of neurodegenerative diseases.

## P2-e10 Neuroprotection of PDGF-B against Oxidative Stress via the PI3K/Akt signal

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The PDGFs are expressed in the mammalian CNS. At present, we analyzed neuroprotective effects of PDGF-B against hydrogen peroxide-induced cytotoxicity in cultured cortical neurons. The cytotoxicity was detected by in MTT assay and LDH cytotoxicity assay, and TUNEL staining. PDGF-B suppressed these radical-induced cytotoxicities. PDGF-B induced the phosphorylation of Akt, GSK3-b, BAD, and CREB in Western blot. Pretreatment with LY294002, a specific PI3K inhibitor, abolished these effects of PDGF-B. These results suggest that PDGF-B protects neurons from oxidative stress via the PI3K/Akt signal.

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## P2-e11 DNA demethylation is involved in ER stress induced cell death in mouse neuroblastoma Neuro2a cells

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Endoplasmic reticulum (ER) stress induced neuronal cell death is a common mechanism of neurodegeneration. And DNA demethylation has been reported to contribute cell death mainly in cancer cells. In Alzheimer disease, higher plasma concentration of homocystein which correlate with DNA hypomethylation has been reported. These reports suggest that DNA demethylation may be involved in neuronal cell death. To demonstrate this hypothesis, we studied correlation of DNA methylation ratio with ER stress induced cell death in Neuro2a cells. Neuro2a cells were induced cell death by treating with DNA methylation inhibitor 5-aza-deoxycytidine as well as ER stress inducing reagents tunicamycin (TM) and thapsigargin (TG). Pretreatment of the cells with methionine which is known to induce hypermethylation of DNA attenuated TM and TG toxicity. Moreover, we found that TM and TG reduced the ratio of methylated-cytidine in genomic DNA by quantitation analysis using HPLC. Our data suggests that DNA demethylation is involved in ER stress induced neuronal cell death.

## P2-e12 Activity patterns of diaphragm during voluntary movements in awake cats

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The diaphragm is the main mover of inspiratory muscles. It was reported that diaphragm activity was influenced by postural change and locomotion. In the passive change of body position in awake cats, the vestibular system was adjusting the activity of respiratory muscles. However, the activities of the diaphragm were not fully described when the posture was changed voluntarily. To investigate how the diaphragm acts at transient stage during voluntary movements, we analyzed the diaphragm EMG and video images of movements on 3 cats. The activity patterns of diaphragm were classified by the characteristics of movements. In the rest state inspiratory diaphragm activity was clearly observed, but in the postural changing state non respiratory diaphragm activity appeared. The duration of the diaphragm activity became to be irregular and the rhythmic respiratory activity pattern could not be observed.

## P2-e13 Neuronal activity in primary motor cortex during treadmill locomotion of the Japanese monkey

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To examine cortical mechanisms related to the control of primate locomotion, we recorded neuronal activity in primary motor cortex (M1) of a freely-moving Japanese monkey during quadrupedal locomotion on the treadmill. Tungsten microelectrodes were inserted into M1 hindlimb region using an electromotive micro-manipulator. We found that almost all neurons modulated their discharge phasically time-locked to the step cycle. We then tested effects of treadmill speed on the neuronal discharge pattern. As the speed increased, the monkey increased stride length and stepping frequency of the hindlimb. With such changes in the limb kinematics, significant proportion of the recorded neurons increased their discharge frequency. These results suggest that primary motor cortex in the macaque monkey directly and/or indirectly acts on spinal circuitries generating a basic pattern of rhythmic activity during simple locomotion in a manner different from that in subprimates.

KAKENHI(17075002)

## P2-e14 Juxtacellular/multiunit recordings from head-restraint rats performing forelimb movement task

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Juxtacellular recording, a novel electrophysiological technique to identify morphological subtypes of recorded neurons, has been applied to in vivo experiments using anesthetized or sleeping animals. However, no attempt at juxtacellular recording from task-performing animals has been reported despite its great advantages, because practically only one recorded neuron should be labeled in each animal trained to learn particular tasks in a head-restraint condition. Here, we developed an efficient task-training system for stereotaxic recording experiments using adult head-restraint rats performing a forelimb movement task. The training system made it possible that head-restraint rats noticed the relation between action (lever-pressing) and reward (a drop of water) in several days and learnt to perform the self-paced lever-pressing task within one or two weeks, allowing us to use at least two well-trained rats every week for juxtacellular and multiunit recordings from their motor-related brain areas.

KAKENHI18019041,18700386

## P2-e15 Postural movements induced by pressure stimulus with various velocities to the plantar sole under first metatarsal head

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We applied pressure stimuli to plantar soles under the first metatarsal head with various velocities, to elucidate a functional role of the foot pressure information in standing. Subjects were 11 healthy young adults. A device that has 2 stimulus square areas (20×20 mm), in which 9 columns with 5 mm diameter were arranged at 2 mm intervals, was used for the stimuli. Subjects stood with eyes closed on a force platform with the device, putting their first metatarsal heads in the middle position of the stimulus square areas. The used column amplitude was 1.1 mm (SD=0.6), and the column velocity was 0.125-2.0 mm/s in each subject. The whole body moved forward in slower velocity and backward in faster velocity. The critical stimulus velocity in forward and backward movement was 0.31mm/s (SD=0.16) and 0.42mm/s (SD=0.25), respectively. It is conceivable that foot pressure information has two roles to build the sensory reference frame of standing position and to respond to a disturbed balance.

KAKENHI(18650168)

## P2-e16 Abnormal activity of basal ganglia neurons in a DYT1 transgenic mouse model of early-onset generalized dystonia.

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Dystonia is a neurological disorder characterized by sustained or repetitive involuntary muscle contractions and abnormal postures. A major group of early-onset generalized dystonia arises from a mutation in the DYT1 gene, which encodes torsinA protein. We recorded activity of basal ganglia neurons in a mouse model of early-onset generalized dystonia, DYT1 transgenic mice overexpressing torsinA under awake conditions. Spontaneous firing rates of the entopeduncular nucleus (Ep) and globus pallidus (GP) neurons in the DYT1 transgenic mice were significantly decreased in comparison with those in the normal mice. Spontaneous firing patterns and responses to the motor cortical stimulation of the Ep and GP neurons in the DYT1 transgenic mice were also different from those in the normal mice. These results suggest that the decreased activity and abnormal firing patterns of the Ep and GP neurons may participate in the abnormal motor control in dystonia.

## P2-e17 Organization of motor cortical inputs in the globus pallidus via the subthalamic nucleus in the monkey

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To investigate motor cortical inputs in the external (GPe) and internal (GPi) segments of the globus pallidus via the subthalamic nucleus (STN), we examined the responses of the GPe/GPi neurons to the stimulation of the orofacial, forelimb and hindlimb regions of the primary motor cortex (M1) and the forelimb and hindlimb regions of the supplementary motor area (SMA) in a Taiwan monkey. Attention was paid to the early excitation evoked by cortical stimulation, because it is mediated by the cortico-STN projection. Neurons responding mainly to the M1 stimulation were located ventrally in the GPe/GPi, while neurons responding mainly to the SMA stimulation were found in the dorsal part of more rostral planes. In the M1 domain, GPe/GPi neurons responding to the stimulation of the hindlimb, forelimb and orofacial regions were represented along the dorsorostral-ventrocaudal axis. The present study has shown the somatotopically organized cortical inputs from the motor cortices in the GPe/GPi via the STN.

## P2-e18 Spinomuscular coherence in monkey performing a precision grip task

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To examine the coherence between spinal local field potentials (LFPs) and muscular activities (spinomuscular coherence), we trained one Japanese monkey to perform a precision grip task. We implanted a recording chamber to lower cervical vertebrae and EMG wires to 19 muscles, and recorded LFPs and EMGs simultaneously. Among 129 LFP-EMG pairs, significant coherence has been found in 28 pairs (22%) and this coherence could be classified into two groups. The first group (A) showed coherence selectively in 20-40Hz range, and the other (B) showed that over 10-100Hz (B). While A was recorded from large area of gray matter, B was recorded only from ventral half. Directional coherence analysis indicated that A was induced by mutual influence between spinal cord and muscles, and B may reflect the dominance of efferent influence. We propose that spinomuscular coherence may have two subtypes and both have a crucial role for sensorimotor integration in movement control.

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## P2-e19 Different pathways for Mauthner and Non-Mauthner escapes in zebrafish

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Three pairs of reticulospinal neurons in teleost hindbrain, the Mauthner (M) cell and two morphological homologs (MiD2cm and MiD3cm) called M-series, are thought to be involved in control of fast escape from sudden noxious stimulus. To investigate effective sensory inputs to initiate fast escape, we performed confocal $Ca^{2+}$ imaging of M-series during escape in larval zebrafish (> 5 dpf) with the rostral body embedded in agar. Water pulse applied to otic vesicle (OV, immature inner ear) evoked fast escape with short latency (< 6 ms), associated with $Ca^{2+}$ increase in M-cell, corresponding to an action potential (M-escape), and MiD3cm. Laser ablation of M-cell precluded the fast escape with short latency. Water pulse applied to head skin also elicited fast escape but with longer latency. In this case, M-cell did not fire (Non-M escape) whereas MiD3cm showed apparent $Ca^{2+}$ increase. The response of MiD3cm was suppressed when the M-cell fired. These data suggests that the M-cell firing is necessary for fast escape with minimum latency elicited by auditory stimulus and that MiD3cm is involved in Non-M escape driven by tactile stimulus.

KAKENHI (17023029, 18300134)

## P2-e20 Activity of globus pallidus neurons in behaving monkeys that receive inputs from cortical motor areas.

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Pallidal neurons receive excitatory input from the subthalamic nucleus (STN) and inhibitory input from the striatum. Cortical stimulation induces a triphasic response in pallidal neurons, consisting of early excitation, inhibition, and late excitation. Previous studies have shown that these excitations and inhibition are derived from the STN or the striatum, respectively. However, the amplitude of each component differs among neurons, suggesting the differential contributions of the STN and striatal inputs to pallidal neuron activity. On the other hand, pallidal neurons change their activity in relation to movements. Using Japanese monkeys, we recorded pallidal neuron activity during the performance of a reaching task, and analyzed the relationship between the neuronal activity and the responses to stimulation of the forelimb regions of the primary motor cortex and the supplementary motor area.

## P2-e21 Glutamatergic and GABAergic modulation of monkey pallidal neuron activity in relation to motor task.

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Pallidal neurons receive excitatory glutamatergic input from the subthalamic nucleus and inhibitory GABAergic input from the striatum. Using Japanese monkeys, we recorded pallidal neuron activity during the performance of a reaching task, and investigated the involvement of these glutamatergic and GABAergic inputs in pallidal neuron activity. Injections of CPP (NMDA receptor antagonist), NBQX (AMPA/kainate receptor antagonist), and/or gabazine ($GABA_A$ receptor antagonist) were made in the vicinity of recorded neurons. CPP/NBQX injection diminished an activity increase during the motor task, and occasionally induced even an activity decrease. Additional gabazine injection greatly diminished all responses. These results suggest that pallidal neuron activity in relation to the motor task is modulated by ionotropic glutamatergic and GABAergic inputs.

## P2-e22 Postural adjustment of decerebrate rats on the rotating platform

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It has been established that midbrain animals exhibiting various righting reflexes may stand with the four limbs, and right against small perturbations, but little is known about their adaptive capability. We analyzed postural movements in midbrain rats during quadrupedal standing on a vertically rotating platform. Wistar male adult rats were precollicularly decerebrated, and postural movements were video recorded after recovery from anesthesia. In some cases emg activities were recorded from hindlimb muscles. When the rats were put on a platform rolling slowly (ca. 0.2 Hz), they often stood without any support on the platform with four limbs. The hindquarter was moved in relation to the platform rotation. The phase relation between them could be modified as trials were repeated. At the beginning both movements were almost in phase; left side down of the platform induced left limb extension, vice versa. However, after 5-10 trials, the phase of the limb extension was advanced. It was shown that the midbrain rat can adapt standing movements to a cyclic perturbation.

## P2-e23 Eye movements during balance control of the inverted pendulum

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To keep an inverted pendulum upright, visual information is indispensable, but little is known about how human subjects utilize it. The present study investigated eye movements while humans stood an inverted pendulum constructed in a virtual space. The virtual inverted pendulum (VIP) presented on a computer screen was controlled by a computer mouse, of which movements were directly linked to transverse movements of the VIP bottom on the screen. The software of the VIP system recorded horizontal positions of the VIP top and bottom at every moment of calculation cycle (10 msec). Horizontal eye movements were recorded by electrooculography. During VIP control, smooth pursuit and saccadic eye movements were observed; the former almost following movements of the VIP top was interrupted by saccades. Saccades occurred at various timings of the VIP movement, and to the same or the inverse directions of on-going top movements. Two kinds of saccadic eye movements presumably take place, sensory-related and action-related ones.

## P2-e24 Modulation of μ opioid receptors on cholinergic interneurons in the striosomes/ matrix compartments of the striatum.

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We previously reported that activation of μ opioid receptor (MOR) suppressed GABAergic IPSCs in medium-spiny (MS) projection neurons in compartment-specific manner. In this study, we investigated the MOR-mediated effects on the excitability of cholinergic interneurons. To identify the striosomes, we used a TH-GFP transgenic mouse strain expressing eGFP under the control of promoter of tyrosine hydroxylase. Because dopaminergic neurons densely innervate the striosomes in early postnatal stage, a striosome is identified as a bright area under a microscope. Activation of MOR suppressed IPSCs in cholinergic interneurons located near the striosomes. By contrast, blockade of muscarinic acetylcholine receptors enhanced the MOR-mediated suppression of IPSCs, suggesting the interaction of the activity of cholinergic neurons with MOR signaling cascade. MOR-mediated effects on synaptic transmission may control the outputs of striatal compartments.

## P2-e25 Reference frame of action-related neuronal activity in the medial frontal motor areas

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To investigate the involvement of the supplementary motor area (SMA) and pre-supplementary motor area (pre-SMA) in organizing bimanual sequential movements, we recorded neuronal activity while two monkeys were performing a motor task consisting of pronation or supination of either arm, with an intervening delay. We found arm-use selective neurons and action selective neurons in the SMA and pre-SMA. In this report, we focus our analysis on whether neuronal activity reflects an intrinsic frame of reference (i.e. supination versus pronation) or extrinsic frame of reference (i.e. turning left versus turning right). Within action selective neurons, a majority of neurons reflected the intrinsic or body-centered coordinates, rather than the extrinsic or world-centered coordinates.

## P2-e26 Recovery of the finger dexterity after lesion of the corticospinal tract at cervical segments in monkey; Lesion at the C2 compared to the C5

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To examine the contribution of the indirect cortico-motoneuronal pathway via propriospinal neurons in the C3-C4 segments (C3-C4 PN) for the control of dexterous finger movements such as the precision grip, we compared to the recovery process of the precision grip after the corticospinal tract (CST) lesion at the C2 (N=2) and the C5 (N=3).

After lesion at the C5, success rates of the precision grip recovered within 2 months. But, after lesion at the C2, success rates of the precision grip didn't recover and independent finger movements disappeared for the total period of observation (4 months). These results suggested that indirect cortico-motoneuronal pathway via the C3-C4 PN contributes to the functional recovery of the finger dexterity after the CST lesion.

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## P2-e27 C-terminals in the spinal ventral horn originate in cholinergic neurons in the medial lamina VII

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C-terminals are large cholinergic boutons on the somata and proximal dendrites of $\alpha$-motoneurons. To determine the origin of C-terminals, we first confirmed that C-terminals were immunoreactive for high-affinity choline transporter (CHT) using immunoelectron microscopy. We next carried out anterograde axonal tracing using biotinylated dextran amine (BDA) combined with immunohistochemistry for CHT. BDA was visualized with Streptavidin-Cy2 while CHT was detected immunohistochemically using primary antibody against CHT and secondary antibody conjugated with Cy3. Following BDA injection in the medial lamina VII, double-labeled large boutons were observed in close apposition to the somata of putative $\alpha$-motoneurons. The double-labeled boutons were found within two spinal segments from the injection site. These data clearly show that C-terminals on ventral horn motoneurons originate in cholinergic neurons in the medial lamina VII.

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## P2-e28 Figure/ground segregation in natural images based on surround modulation in early vision

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We propose a robust network mechanism for figure/ground segregation, based on the cortical mechanisms underlying the determination of border ownership (BO) that indicates the side of figure with respect to a border. The model is based on the surround modulation reported in early vision, which comprises hundreds of model cells with their surround regions given randomly. Many model cells determined consistently BO for a set of pseudo-random stimuli that approximate all possible shapes, indicating high consistency among various shapes and invariance to shape complexity and occlusion. The simulation results with natural images showed correct BO determination for around 70% of the entire contours marked by human observers. The analysis of the results revealed that the model tends to follow Gestalt factors such as convexity, proximity and parallelism, suggesting that the surround modulation in early vision appears to detect inherently the Gestalt factors for figure/ground segregation.

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## P2-e29 Spatial specificity of V1/V2 responses to an occluded surface

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How does the brain represent a partially occluded object? We have previously reported the possibility that the human brain would topographically complete the occluded portion in V1 and V2. There remains, however, another possibility that the observed responses might reflect neural processing of not occlusion completion but local image features such as T-shaped junctions at the occluding borders. We therefore re-examined spatial specificity of the occlusion-related fMRI activity. Precisely-defined ROI analyses revealed robust fMRI responses even within the retinotopic subregions representing the middle position of the occluded surface, confirming the completion hypothesis. Our finding suggests that the visual system solves the occlusion problem utilizing topographic information at the very early stage of visual processing.

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## P2-e30 Relationship between color perception and neural activity in area TE of the monkey

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Previous studies suggested that area TE of the monkey plays an important role in color discrimination.To examine the correlation between responses of TE neurons and color perception, we recorded activities of color selective neurons from a small region in anterior TE during a color similarity judgment task in which monkey must judge the similarity of a sample color and 2 target colors. First, we computed neurometric function based on ROC analysis, which is an objective measure of how a neuron can discriminate the sample and target colors. Although neural sensitivity was on average lower than the behavioral sensitivity, we found a few neurons showed sensitivity comparable with that of the monkey. Second, we examined the choice probability that is the correlation between trial-to-trial variation of the monkey's color judgment and neural responses. We found most cells showed little correlations, but a few showed a significant correlation. These results suggest that linkage between neuron activities in this TE region and color perception is relatively weak.

## P2-e31 Explicit representation of 3D object parts in monkey inferotemporal cortex

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We used a novel adaptive stimulus strategy to demonstrate explicit neural signals for 3D object parts in awake monkey inferotemporal cortex (IT). A response-driven evolutionary algorithm guided initially random shapes through successive generations of morphed progeny, in effect letting the neuron design its own stimulus set from scratch. This procedure revealed that many IT neurons respond to any object containing a specific configuration of 3D surface fragments at specific object-relative 3D positions. These configurations correspond to familiar geometric motifs like curving torsos and projecting limbs. They show a strong bias toward high convex curvature, which may reflect theecological importance of protruding object parts. Our data indicate that,in addition to 2D image processing, the visual system generates an explicitrepresentation of 3D object structure, which may explain our tendency todescribe objects as spatial arrangements of simple 3D parts.

## P2-e32 Depth discrimination for transient and sustained images by the human stereoscopic system

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A few psychophysical studies propose that stereoscopic depth is processed by different mechanisms, depending on the temporal properties of visual inputs. We tested this hypothesis by considering the recent physiological findings that neurons in the dorsal and ventral pathways compute the disparity energy and the global-match solution to the stereo correspondence problem, respectively. Subjects discriminated the depth of dynamic random-dot stereograms (RDSs). The pattern refresh rate of RDSs was varied across blocks of trials. In a single block, we varied disparity sign and the ratio between binocularly correlated and anti-correlated dots. At a fast refresh rate (e.g., 43 Hz), subjects perceived reversed depth for anti-correlated RDSs (aRDSs), and no depth when the two groups of dots balanced. At a slow refresh rate (e.g., 5 Hz), the reversed depth for aRDSs was weak, and correct depth was perceived at the balanced point. These results suggest that the disparity-energy and binocular-matching computations underlie stereopsis for transient and sustained visual inputs, respectively.

## P2-e33 An evaluation of 3-d binocular receptive fields in the early visual cortex

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A binocular receptive field (RF) is inherently a 3-d entity defined as a function of visual direction (x,y) and distance z. However, binocular RFs have conventionally been measured only in 2-d, because bar stimuli of optimal orientation are used. Therefore, it is not known whether binocular RF profiles are uniform along the length axis (parallel to the preferred orientations). Stimulating neurons in the early visual cortex by 2-d dynamic dense noise stimuli presented dichoptically, we examined 3-d binocular RFs, which now have the length axis in addition to the left-eye and right-eye position axes. These analyses showed many cells with uniform profiles along the length axis. However, some neurons exhibited gradual changes in structure, indicating non-uniformity of the binocular RFs. Our new method allows evaluation of the true 3-d forms of the binocular RFs and their internal structures of visual cortical neurons.

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## P2-e34 Developmental changes in electrophysiological properties of layer III pyramidal neurons in macaque visual cortices

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We studied developmental changes of intrinsic membrane properties of layer III pyramidal neurons in the striate (V1) and inferior temporal (TE) cortices of macaque monkeys. Whole-cell patch-clamp techniques were applied to slices prepared from animals of different ages (10 days, 7-8 months, adult). The membrane time constant decreased with age in both V1 and TE. In V1, the value at 7-8 months was already close to that in adults, while in TE it gradually decreased up to adulthood. Inward rectification in response to hyperpolarization became stronger with age, and this change was more evident in V1 than in TE. In both areas, the maximal rate of firing in response to depolarizing current injection increased with age, and the duration of individual action potentials shortened. The results demonstrate that electrophysiological properties of cortical neurons in monkeys become matured over a prolonged period after birth, which may contribute to refinement of visual responsiveness during development.

CREST

## P2-e35 Approximate independent components of large natural scenes

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Linear multilayer ICA (LMICA) is an approximate algorithm forindependent component analysis (ICA). In LMICA, approximateindependent components are efficiently estimated by optimizing onlyhighly-dependent pairs of signals. In the former part of thispresentation, a new method named "recursive multidimensionalscaling (recursive MDS)" is proposed for the selection of pairsof highly-dependent signals. In recursive MDS, signals are sorted byone-dimensional MDS at first. Then, the sorted signals are dividedinto two sections and each of them is sorted by MDSrecursively. Because recursive MDS is based on adaptive PCA, it doesnot need the stepsize control and its global optimality isguaranteed. Numerical results verify the effectiveness of this improvement of LMICA. In the latter part, LMICA with recursive MDS isapplied to large natural scenes. It generates various interestingedge detectors (long edge ones, the ones with multiple edges, etc.).Besides, the characteristics of detectors vary according to the degree of approximation. This interesting phenomenon is analyzed by thestatistical properties of detectors.

## P2-e36 Neural dynamics involved in the 3-D object perception as aseessed by MEG/fMRI integrated imaging technique

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Recent neuroimaging studies suggest the involvement of the parieto-occipital (PO), the intraparietal (IP), and the inferior-temporal (IT) regions in the 3-D object perception from 2-D motion (3D-SFM: 3-D structure-from-motion), however, the neural dynamics underlying the 3D-SFM is not fully understood. Here, we used both MEG and fMRI measurements to visualize the brain dynamics during 3-D SFM. The coherence of the random-dot motion was parametrically controlled. The results of the fMRI analysis were used to impose plausible constraints on the MEG inverse calculation. IT, PO, and IP regions showed increased neural activity in the highly coherent motion conditions in which subjects perceived robust 3-D object structure at different latencies. These results are in agreement with the previous fMRI studies and add further insight into the temporal dynamics of the neural activities in the multiple brain regions along both the dorsal and the ventral visual processing streams during 3D-SFM.

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## P2-e37 Difference in coding efficiency between feedforward and feedback models of orientation selectivity

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Several neural mechanisms of orientation selectivity in visual cortex have been proposed in the past decade. These can be classified into two main groups: feedforward models and feedback models. In the feedforward models, the LGN afferences are the main source of orientation selectivity. In the feedback models, the cortical circuitry plays a much more central role in establishing orientation selectivity. We compare these models of orientation selectivity from the point of view of Fisher information. To calculate Fisher information, we need to know correlations in neuronal activities. Although it is generally very difficult to calculate the correlations, by using a simple neural network model with stochastic dynamics, we can analytically calculate the correlations. We show that the Fisher information of the feedback model is smaller than that of the feedforward models. This means that the pattern of correlations in the feedback model is inefficient for coding the stimulus orientation.

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## P2-e38 Dynamics of intrinsic optical signals in cat primary visual cortex

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Although optical imaging based on intrinsic signals has been widely used in visualizing the brain functional organization, its property is not well known. The purpose of this study was to investigate the temporal property and visualize the signal dynamics over the cortical surface. We applied the intrinsic optical imaging to cat V1. Reflection images were taken under the illumination light tuned to 605 nm at 500 ms interval. The time-course model of signal was created by averaging the optical intensity values at active regions. We calculated the correlation coefficient, at each pixel, between the time-series of the response evoked by the visual stimulation and the model. The value was compared to the correlation coefficients to the model shifted every 500 ms in time scale. Aligned by the shift time of model, we observed that the area with higher correlation coefficient gradually shifted from the cortical region showing functional mapping signals to the region covered by relatively large blood vessels. The results demonstrate the dynamics in cat V1 and suggest the validity of the proposed method in extracting localized functional signals.

## P2-e39 A new approach to estimating receptive fields from responses to natural stimuli

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The reasons for using natural stimuli to study sensory function are quickly mounting, but neurophysiologists have been slow to incorporate them, partly because of the difficulty of estimating receptive fields (RFs) from responses to stimuli with complex statistical properties. Natural stimuli typically contain strong correlations and often have skewed distributions of light intensity. These statistical complexities can bias the result of RF estimation when standard techniques such as spike-triggered averaging or reverse correlation are used. Here, we develop a method for RF estimation that accounts for both the correlations and the skewed intensity distributions. The method involves a transformation of the original stimulus to decrease correlations and skews, as well as an explicit correction for residual biases. The utility of the method is demonstrated by estimating RFs from the responses of retinal ganglion cells to natural stimuli and using these RFs to predict responses to novel stimuli.

## P2-e40 The origin of spreading burst activities in the local circuit of the superior colliculus

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Our previous studies have shown that when slices of the superior colliculus (SC) are exposed to a solution containing 10 μM bicuculline and low $Mg^{2+}$ (0.1 mM), most neurons in deeper layers (dSC) and some neurons in the superficial layers (sSC) exhibited spontaneous depolarization and burst firing. In present study, we used this condition to study the mechanism of the spread of burst activity in the mice SC with the field potential recording from 64 multi-planar electrodes and the whole cell patch clamp recording. The burst originally occurred from the ventral portion of the sSC and then spread to the dSC. The current injection to a wide-field vertical (WFV) cell, mainly located in the ventral sSC, could evoke the burst response, which spread from the sSC to the dSC. The WFV cells uniquely exhibit large $I_h$ currents among the SC cell groups and application of 50-100 μM ZD7288, a $I_h$ blocker, completely eliminated the spontaneous bursts. These findings supported the idea that WFV cells are the origin for the propagation of burst activity in the SC.

MEXT (No. 18200027)

## P2-e41 Visual cortical representation in cats reared under the deprivation of pattern vision

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Little is known about how the absence of pattern vision disrupts the development of orientation maps. Here, we report how the cortical maps develop in kittens persistently wearing goggles that show the animals a white or black uniform image. We reared kittens with these goggles from postnatal 3-4 weeks to 20 weeks, and repeated optical imaging of intrinsic signals in the visual cortex of identical kittens in the anesthetized condition. In the first several weeks of pattern deprivation, orientation responses were mainly non-selective and reliable orientation maps could not be reconstructed either for 0.15 and 0.5 cycles/degree gratings. A longer period of pattern deprivation allowed orientation maps to appear in these cats. However, horizontal orientation representation occupied relatively lager cortical territory. In addition, response strength for either eye tended to dominate significantly over that for the other eye. These results suggest that the deprivation of pattern vision leads to instability in the formation of balanced representation of orientation preference and ocular dominance.

## P2-e42 Critical contribution of slow-decay synaptic potential to complex-cell receptive field formation

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Position invariance in complex cell responses has been proposed to emerge from feed-forward integration of activities of simple cells having different spatial phases of receptive fields (RFs). However, only the Hebbian learning cannot account for such feed-forward connections. In this study, we attempted to explain how complex-cell RFs are formed in upper layers which receive afferent inputs from simple cells in layer 4, taking into account dynamics of two components of synaptic potential with fast and slow decay constants in addition to the Hebbian learning rule. Computer simulations demonstrated that when the decay constant of the slow component was set at 100ms or longer, upper layer cells exhibiting phase-invariant RFs were predominant over cells exhibiting phase-variant RFs. When the decay constant was set at 50ms or shorter, a large portion of upper layer cells showed phase-variant RFs. These results suggest that a slow-decay component of synaptic potential plays an important role for the activity-dependent formation of complex cell RFs.

## P2-e43 C38 enhances neuronal maturation of P19 embryonal carcinoma cells.

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Purpose: C38 is a neuron specific molecule that we have isolated from retina. We tried to determine the developmental expression pattern and molecular functions of C38 in neuronal cells. Methods: Pre- and postnatal rat retinas were immunostained with C38 antibody. Neuronal phenotypes were investigated in P19 embryonal carcinoma cells overexpressing C38. Results: During the development, C38 was first detected in the ganglion cell layer at E16, when doublecortin was abundantly expressed in the retina. In horizontal cells, C38 is first detected at P0. Overexpression of C38 did not promote neuronal differentiation of P19 cells. However, when neuronal differentiation of P19 was stimulated by retinoic acid, C38 enhanced neuronal maturation. Conclusion: In rat retina, horizontal cells complete their mitosis until E16 but their neuronal maturation starts postnatally. Taking together, C38 can enhance neuronal maturation of destined to neurons.

## P2-e44 Visual cortical depression induced by spatial sensory mismatching between visual and whisker inputs in strabismic mice

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Visual cortical plasticity induced by strabismus was investigated in mice. Divergent strabismus by 30 degree was produced using a monocular prism goggle, which was attached on the head of mice between P28 and P35. After removal of the goggle, cortical responses to LED stimuli were investigated in anesthetized mice using transcranial flavoprotein fluorescence imaging. The strabismic eye responses were significantly depressed compared to the normal eye responses. This depression was observed not only in the binocular region but also in the monocular region of the visual cortex, while ocular dominance plasticity induced by monocular deprivation was observed only in the binocular region. Surprisingly, a monocular prism goggle failed to produce any cortical depression in mice, in which whiskers were trimmed bilaterally. From these results, it is suggested that spatial sensory mismatching between visual and whisker inputs might produce the cortical depression found in the strabismic mice.

## P2-e45 Synaptic connections to layer 2/3 pyramidal neurons in rat visual cortex revealed by two-photon activation of caged glutamate

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We have improved two-photon photolysis of caged glutamate in order to induce an action potential at single neurons as well as a synaptic potential at single dendritic spines. This new method allowed us to systematically stimulate individual neurons in acute slices of rat visual cortex. Three-dimensional mapping of synaptic inputs indicated columnar distribution of excitatory and inhibitory neurons innervating to layer 2/3 pyramidal cells. This method would be a uniquely powerful tool to clarify functional connections and information processing in the cortical circuit even *in vivo*.

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## P2-e46 Time-dependent and exofocal alterations in the lateral geniculate nucleus following retinal damage in mice

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Intravitreal injection of N-methyl-D-aspartate (NMDA) into the left eye induced retinal damage at 1 day after the injection. At 7 days after NMDA injection, atrophy of the optic tract was observed on the contralateral side. In the contralateral side of the dorsal LGN (d-LGN) and contralateral ventral LGN-lateral (vLGN-l), neurons were decreased at 90 and 180 days, respectively after NMDA injection, and neuronal cell-size was decreased at 7 and 30 days, respectively after NMDA injection. Furthermore, during neuronal degeneration, expressions of glial fibrillary acid protein (GFAP) and brain-derived neurotrophic factor (BDNF) were increased in the contralateral side of the dLGN and vLGN-l. In addition, some GFAP-positive astroglial cells exhibited BDNF.

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## P2-e47 Function of CCK-A receptors for the visual signal transductions: Regulation of the circadian clock system

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Daily behavioral rhythms are entrained to environmental light-dark cycles using photic input pathways to the circadian clock. Although presence of cholecystokinin (CCK) peptides has been shown in retinal amacrine cells (ACs) of various vertebrate, their specific function was unknown. Here, we show that CCK in mouse retina functions for the circadian photo entrainment. Specific type of the ACs, including one that contains glycine transporter, were visualized by lacZ reporter genes linking to CCK-A receptor genes. The CCK-A agonist, CCK-8s, released Ca2+ from internal stores in the retinal cultures but not in the retino-recipient suprachiasmatic nucleus neurons. Since CCK-8s-induced Ca2+ response in the ACs was absence and light pulse-induced behavioral phase-shifts were significantly reduced in CCK-A receptor knockout mice, we suggest novel ocular signal transduction to control mammalian circadian clock systems.

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## P2-e48 A novel device improves myopic visual acuity through pupil constriction during far accommodation

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We discovered a special training method for myopia by moving a visual object (projected on a screen) in cycles from a near point to a far point and back, while keeping the retinal projection size and brightness of the object constant. After several months of training (for 2 minutes 3 times per week) an unexpected paradoxical pupillary constriction occurs during far accommodation in trained myopic children, which is not seen in normal subjects nor in untrained myopic children. This leads to an increased depth of focus and a decreased aberration resulting in a pronounced improvement of visual acuity.

## P2-f01 Auditory stream segregation induced by low-pass filtering characteristics of auditory cortical neurons

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This research aimed at investigating cortical mechanisms of auditory stream segregation occurring for an alternating sequence of high and low tones. Optical recordings (voltage-sensitive dye, RH795) showed spatio-temporal response patterns in the guinea pig auditory cortex. The strong response was observed for the onset of the alternating sequence of tones, while the responses to the following tones were suppressed relatively. This tendency was more clearly observed when the frequency separation of the high and low tones was smaller and when the tone repetition rate was higher. This result indicates that low-pass filtering characteristics, produced presumably by interactions between excitatory and inhibitory neurons, are capable of changing the response pattern for alternative tones into that observed for two single tones. These findings imply that such low-pass filtering characteristics of a population of neurons contribute to segregating alternative tones into two streams on the basis of the Gestalt principle of proximity.

## P2-f02 Characterization of belt regions caudal to the dorsocaudal field in guinea pig auditory cortex

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The auditory cortex of many mammals has a core area and surrounding belt regions. In guinea pigs, a core area dorsocaudal field (DC) is reported to be embraced by a single belt region in electrophysiological studies. In a previous optical imaging study, however, two fields in the caudal area adjacent to DC have been reported. To solve this discrepancy, here we recorded cortical responses to tone stimulations in DC and its caudal regions in anesthetized guinea pigs, using a optical imaging system. The caudal auditory cortex was exposed and stained with the voltage-sensitive dye RH-795. Early responses appeared at two areas caudal to DC, aligned roughly in a dorsoventral direction. The early responses in both areas exhibited tonotopy. At about 40 ms after the onset of tone stimulation, however, the two areas merged into one with expanded dimension exceeding 2 mm in the rostrocaudal direction. Our results suggest that the belt area caudal to DC is possibly composed of more than one area and that the dimension of these areas is much larger than previously thought.

## P2-f03 Expression of NB-2 in the superior olivary complex of the auditory system.

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NB-2 that we identified is a neural cell recognition molecule prominently expressed in the central auditory system in rat and mouse. We demonstrated previously that NB-2 knockout mice are impaired in the auditory function monitored by audiogenic seizure tests and c-fos expression after pure tone exposure. Thus, NB-2 plays important roles in the auditory system. Here we report the expression of NB-2 in the superior olivary complex (SOC) in rat.NB-2 was transiently expressed between embryonic day 19 and postnatal day (P) 10 with the maximum level at P7 in SOC. NB-2 was localized in each nucleus of SOC partially overlapping with VGLUT1, but not with GAD67 or GLYT2. This indicates that NB-2 is expressed in the subpopulation of glutamatergic neurons in SOC. NB-2 may play (an) essential role(s) in the development of glutamatergic neurons of the central auditory system.

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## P2-f04 Topography of thalamic reticular nucleus projections to the medial geniculate body

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Tonotopically comparable subfields of the primary and non-primary auditory areas in the rat cortex have similar topographies in the projection to the medial geniculate body (MG) but reverse topographies in the projection to the thalamic reticular nucleus (TRN). Corticofugal excitations from the primary and non-primary auditory areas are assumed to modulate thalamic cell activity in the same part of the MG, while it could be possible that corticofugal inhibitions via the TRN modulate cell activity in different parts. In this study, we determined features of TRN projections based on axonal trajectories of single auditory TRN cells, using juxtacellular labeling technique. Auditory TRN cells sent projections to limited parts of the MG, and the projections were topographic. The results suggest that tonotopically comparable subfields of the primary and non-primary auditory areas in the cortex provide inhibition to different parts of the MG. We propose that the primary and non-primary auditory areas in the rat cortex serve distinctive gating functions for auditory attention.

## P2-f05 Interaction between interaural time and intensity differences in the central auditory system: A psychophysical approach

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Physiological studies using animal models generally indicate that the auditory system has two segregated binaural mechanisms. The medial superior olive mainly represents low frequencies, and processes primarily interaural time difference (ITD) information with a coincident detector. The lateral superior olive over-represents high frequencies, and encodes interaural intensity difference (IID) information with contralateral inhibition. This study examined psychophysically whether the *human* auditory system has such segregated pathways in terms of cue type and operating frequency range. Hypothetical separate "channels" for ITD and IID processing were considered, and the degree of channel interaction was evaluated by comparing the detectability of individual ITD and IID changes with that of simultaneous changes. The results showed that the interaction was relatively large for high frequency signals, implying a common channel for ITD and IID, whereas it was small for low frequency signals, implying the existence of independent channels. Thus, different binaural mechanisms are possibly involved for low and high frequencies.

## P2-f06 Modulatory effects of environmental stimuli on the acoustic responsiveness of auditory cortical neurons

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Animals are continuously exposed to ever changing environmental stimuli (ES) of various modalities. The interpretation to acoustic signals depends on environmental conditions; for example, a sound approaching toward animal has different biological meanings from the one leaving away from it, even if acoustically almost identical.The purpose of the study is to see how neurons' responses in the primary auditory and belt fields are modulated in distinct environmental situations. Unit recording was made from unanesthetized and unconstrained guinea pigs while various acoustic stimuli, with/without presentation of ESs, were given. ES including turning on/off of room light, experimenter's approaching and leaving, his existence by the animal, etc modulated vigorously spontaneous and/or evoked firing rate. For example, tuning room light off induced sustained decrease in spontaneous firing rate if it had been high. Under this condition, firing evoked by appropriate acoustic signals became prominent. Such modulation was frequently observed in the belt fields.

## P2-f07 Developmental changes in cochlear spiral ligament fibrocytes of rat inner ear

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Cochlear spiral ligament (SL) has been known to play an important role in the homeostasis of the endolymph by recycling K+. SL fibrocytes (SLFs) are classified to 5 subtypes based on their morphological and functional properties. In this study, we investigated on differentiation process in various types of SLFs of rats. Inner ears of Wistar Imamichi rat on postnatal day 1-28 (P1-28) were subjected to an immunohistochemical study. At P10, S100 $\beta$ labeling expanded to almost entire region of the SL. At P14, SFLs had carbonic anhydrase II (CAII)+, S100 $\beta$ +, ATPase $\beta$ 1- cells, CAII+, S100 $\beta$ -, ATPase $\beta$ 1+ cells, and CAII+, S100 $\beta$ +, ATPase $\beta$ 1+ cells. Distribution of these proteins expressed on P14 was indistinguishable from those in the inner ear of adult at P28. These findings suggest that the expression of ATPase $\beta$ 1-positive cells is followed by those of S100 $\beta$ -positive cells in the SL. Our observation also raises the possibility that type II SLFs are derived from hypothetical S100 $\beta$ -positive progenitor cells within the SL.

## P2-f08 Acid-sensing ion channel-1b in the stereocilia of mammalian cochlear hair cells

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We investigated whether amiloride-blockable proton-gated cation channels ASIC1a (acid-sensing ion channel-1a) and ASIC1b are expressed in the stereocilia of mouse cochlear hair cells. *In situ* hybridization studies showed that ASIC1b transcripts, but not ASIC1a transcripts, were expressed in the inner and outer hair cells. Fluorescent immunohistochemical and immunogold electron microscopic analyses revealed that the ASIC1b channels were located at the insertions of the stereocilia into the hair cells. Our findings provide a novel molecular key to the understanding of cochlear physiology and pathophysiology.

KAKENHI (16390490)

## P2-f09 Multisynaptic pathways from CA1 of the hippocampus to the temporal auditory cortex in the rat

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Using retrograde transneuronal transport of rabies virus, the organization of mutisynaptic pathways from CA1 of the hippocampus to the temporal auditory cortex was examined in the rat. Depending on the sites of rabies injections in the auditory cortical areas, the differential distribution patterns of transsynaptic labeling were seen in CA1 pyramidal neurons as second- or third-order neurons. After injection into the primary auditory area receiving input from the ventral medial geniculate, dense labeling was observed at the temporal level of CA1, while only sparse labeling was found at the septal level. In contrast, injections into the association auditory areas receiving input from the dorsal medial geniculate, pyramidal neuron labeling occurred through all septo-temporal levels of CA1. These results indicate that the temporal CA1 region sends output extensively to both the primary and the association auditory cortical areas, whereas the septal CA1 region sends output specifically to the association areas.

## P2-f10 Effects of optic flow produced by virtual reality on ocular movement and locomotion

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It has been reported that postural control in patients with unilateral vestibular loss deviates towards the lesion side. Optic flow may recalibrate the sensory process, and be beneficial for use in rehabilitation of these patients. In this study, we examined the effects of optic flow produced by virtual reality on ocular movement and locomotion in 10 healthy subjects. During the 20-min period, the virtual environment moved to the right in combination with subjects' movement. The locomotion error towards the right and optokinetic nystagmus towards the right was consistently observed during the experiment. The degree of the both in the last phase of the experiment was significantly higher than that of the first phase. This study suggests this environment may recalibrate the sensory process in healthy subjects resulting in the deviation of ocular control and locomotion.

KAKENHI (18591897)

## P2-f11 The local circuits of mammalian inferior colliculus studied by juxtacellular staining and in vivo whole cell recording

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Inferior colliculus(IC) is a center of integration for auditory information, receiving inputs of ascending, descending and some intrinsic activities, and its local circuits are considered to play roles in shaping response properties of IC neurons to sound.

We performed in vivo patch recordings for detecting unit activities and for whole cell responsibility, trying to understand (1) the exact form of auditory signal processing in IC, and (2) the signal processing in terms of inhibitory and excitatory interaction. For these purposes we have developed (a) a technique for in vivo patch recordings, and (b) a juxtacellular staining technique to identify the neurons of which activities were recorded.

## P2-f12 Firing patterns of Purkinje cells in the cerebellar nodulus and uvula during vertical head rotation in awake cats.

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We recorded Purkinje cell activities in the cerebellar nodulus and uvula during vertical head rotation in awake cats. The simple-spike (SS) activities of 191 responded cells during sinusoidal vertical rotation were examined. Of 118 cells in which the four stimulus planes of pitch, roll, and two diagonal planes (orienting 45°and 135°from the sagittal plane) were tested to determine the best response plane, 61 cells responded most strongly during pitch plane rotation, 38 cells during roll plane rotation, and 19 cells during diagonal plane rotation. The phase and gain of SS responses were determined at stimulus frequencies of 0.125-1.0Hz in the best response plane. Based on the results, we classified the responding cells into two types: head-position and head-velocity types, suggesting that they seemed to primarily receive otolithic input and vertical semicircular canal input, respectively. Such information could be transmitted to the brainstem nuclei to control optokinetic responses under head-tilt conditions in each specific plane.

## P2-f13 Auditory space map: Comparison of the three midbrain nuclei of gerbils

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The midbrain pathway runs from the central nucleus of the inferior colliculus (ICc), through the external nucleus of the inferior colliculus (ICx), to the superior colliculus (SC). This pathway plays an important role in forming the auditory space map. This study compared the three nuclei of anesthetized gerbils as regards the rigorousness of the topographical representation of the auditory space. The stimuli were 50-ms wide-band noise bursts with various sound levels whose azimuths varied on the horizontal plane in a virtual acoustic space. The best azimuth, defined as that where the unit exhibits its maximum firing rate, was calculated for the units that exhibited significant spatial selectivity. There was no topographical arrangement of the best azimuths in the ICc. For a low stimulus level only, the best azimuths exhibited a topographical gradient in the ICx (rostral units preferred lateral to rear stimuli). A space map that is robust to stimulus level was found in the SC. The results indicate that in mammals such as gerbils, the formation of a rigorous auditory space map is incomplete until the SC level.

## P2-f14 Sustained responses to frequency modulated sound in cat primary auditory cortex

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Previous studies on frequency modulation (FM) reported that the primary auditory cortex (A1) neurons in anesthetized cats transiently responded to instantaneous FM-frequency (Fi) only at the boundary of cell's tonal frequency-receptive-field (FRF), and preferred fast FM-speeds. In the present study on awake cats, we found a group of A1 neurons that show sustained FM-responses and prefer slow FM-speeds. These cells showed sustained responses to pure tone stimuli. When the sweep-speed of FM stimuli slowly traversed a cell's FRF, the cell continuously responded to the FM tone until it passed FRF. The peak response was triggered by Fi corresponding to the frequency, which evoked the best response in pure tone stimuli. Cells preferring upward FM direction tended to have dominant high frequency inhibitory-FRF (defined by two tone stimuli), suggesting contribution of inhibitory-FRF to the direction selectivity. Thus, the sustained response cells may play a role in continuously encoding FM-sweep trajectories of cat's vocalizations.

## P2-f15 A role of the lemniscal pathway in the single-whisker directional discrimination in rats

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In the rat somatosensory pathway, information received with a whisker is conveyed to the barrel cortex via trigeminal and thalamic nucleus mainly by two parallel pathways, the lemniscal and paralemniscal. The former includes the nucleus of trigeminal Pr5 and thalamic VPm, and the latter includes trigeminal Sp5i and thalamic POm. Previously, we showed the specific involvement of the lemniscal pathway in the single-whisker directional discrimination using behavioral task and selective lesion or muscimol inactivation of each nucleus. In this study, to investigate the effect of muscimol inactivation of each nucleus to the single whisker-evoked activity in the rat somatosensory cortex, we used optical imaging with a voltage-sensitive dye and evaluated the responses before and after the inactivation. In the inactivation of Pr5 or VPm, the response in the somatosensory cortex disappeared completely. However in the inactivation of Sp5i or POm, similar response before inactivation was observed.

## P2-f16 Noradrenergic activation amplifies signal-to-noise ratios in the VPM thalamus

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The thalamus receives sensory signals via primary sensory afferents, cortical signals via corticothalamic afferents, and inhibitory signals via nucleus reticularis thalamic afferents. In addition, the thalamus receives neuromodulatory afferents that release, among others, acetylcholine and norepinephrine during arousal. Hence, different neuromodulators may set different thalamic modes of sensory information processing. In order to address this hypothesis, we studied the response properties of single-units in the ventroposterior medial nucleus (VPM) of the thalamus and in the barrel cortex of urethane-anesthetized rats during quiescent states and during thalamic activation produced by application of either cholinergic or noradrenergic agonists via microdialysis. We found that cholinergic activation increased the spontaneous firing and enlarged the receptive fields of VPM cells, while noradrenergic activation had the opposite effect. These thalamic changes significantly impacted the receptive fields and temporal responses of barrel cortex cells.

## P2-f17 Experience-dependent plasticity induced by whisker trimming in thalamocortical responses of slices obtained from the mouse barrel cortex

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We have reported that experience-dependent changes in flavoprotein fluorescence responses were induced by whisker trimming in the coronal slices of the rat barrel cortex. However, roles of thalamocortical synapses were not clear in these experiments. In the present study, we prepared slices of the mouse barrel cortex containing thalamocortical afferents. Flavoprotein fluorescence responses after repetitive thalamic stimulation at 20 Hz were observed around layer IV in the barrel cortex. We confirmed that the fluorescence responses elicited by intracortical stimulation were depressed after whisker trimming during a period of 5 weeks. The whisker trimming also suppressed the flavoprotein fluorescence responses in layer IV of the barrel cortex elicited by repetitive thalamic stimulation. These results strongly suggest that thalamocortical synapses are important for experience-dependent plasticity after whisker trimming in the mouse barrel cortex.

## P2-f18 *In vivo* patch clamp analysis of synaptic responses evoked in primary somatosensory cortex in inflamed rats

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Previous studies using extracellular recordings have shown that acute inflammation causes enhancement of the responsiveness of primary somatosensory (SI) cortex neurons to noxious stimulation. However, little is known about how plastic changes were occurred in SI under chronic inflammation. In this study, we analyzed synaptic responses evoked in SI cortex neurons of inflamed rats using *in vivo* patch-clamp tequniques. Whole-cell patch-clamp recordings were made from Layer III or V pyramidal neurons under urethane anesthesia. In chronic inflamed rats, the frequency of bursting activities of EPSCs was slowed and their duration was prolonged rather than those in normal rats. These bursting activities were completely inhibited by CNQX applied to the surface of the cortex. These findings together with our previous observations suggest that the inflammation causes the change in the synchronization of the outputs from the thalamus to SI neurons.

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## P2-f19 Sensitization of somatosensory cortical responses after blockade of spontaneous peripheral nerve firing in mice.

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Nerve injury induces neuropathic pain. However, the trigger mechanisms are unknown. In anesthetized mice, we observed that somatosensory cortical responses elicited by vibratory forepaw stimulation was gradually recovered within 3-4 h after cutting the median and ulnar nerves innervating the forepaw. Similar rapid recovery was induced by pharmacological conduction block of these nerves. The recovered responses were eliminated by cutting the remaining radial and muscle cutaneous nerves innervating the back of the forepaw. The recovery after nerve cutting was suppressed by electrical stimulation at 0.1 Hz applied to the central cut end of the nerves. GDNF applied to the central cut end also suppressed the recovery, and the effect of GDNF was abolished by adding TTX to GDNF. These results suggest that blockade of spontaneous peripheral nerve firing, which may be induced by endogenous substances such as GDNF, triggers sensitization of somatosensory cortical responses.

## P2-f20 Activation of Toll-like receptor 3 in spinal microglia contributes to tactile allodynia after nerve injury

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Neuropathic pain that occurs after peripheral nerve injury is poorly controlled by current therapies. Increasing evidence shows that glial cells in the spinal cord play an important role in the induction and maintenance of neuropathic pain. On the other hand, cells of the innate immune system, including monocytes/ macrophages, natural killer cells, neutrophils, and microglia recognize invariant molecular structures of pathogens by means of stable, genetically conserved, pattern-recognition receptors on the cell surface. The genes producing these receptors are homologous to the Toll gene in Drosophila and are therefore termed Toll-like receptors. We now show that nerve injury induces the activation of Toll-like receptor 3 (TLR3) in microglia in the spinal dorsal horn, and this TLR3 activation increases MAPK phosphorylation in spinal microglia and contributes to the development of tactile allodynia. Our findings point to the potential blockade of the TLR3/MAPK signaling pathway in spinal microglia as a new therapeutic strategy for pain caused by nerve injury.

## P2-f21 TAK1 upregulation in spinal astrocytes contributes to tactile allodynia after nerve injury

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Neuropathic pain that occurs after peripheral nerve injury is poorly controlled by current therapies. Increasing evidence shows that glial cells in the spinal cord play an important role in the induction and maintenance of neuropathic pain. On the other hand, transforming growth factor-activated kinase 1 (TAK1) is a member of the MAPKKK family and plays an essential role in tumor necrosis factor (TNF), interleukin 1 (IL-1), and Toll-like receptor (TLR) signaling pathways. TAK1 functions as a MAPKKK in these pathways and is indispensable for the activation of JNK and p38 MAPK. We now show that nerve injury increases the expression of TAK1 in astrocytes in the spinal dorsal horn, and this TAK1 upregulation increases MAPK phosphorylation in spinal astrocytes and contributes to the development of tactile allodynia. Our findings point to the potential blockade of the TAK1/MAPK signaling pathway in spinal astrocytes as a new therapeutic strategy for pain caused by nerve injury.

## P2-f22 Functional analyses of the prostaglandin (PGE2) receptors in the interneurons of the spinal dorsal horn

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The molecular mechanism underlying the modulation of pain sensation is one of the most complicated field in modern neuroscience. For example, various substances and interneurons interact to form the complex neural circuit. However the exact interrelationship among the interneuron, neurotransmitters and their receptors have not been established yet. In the current study we try to understand the mechanism by which the PGs exert their effect in the modultion of interneuron. Four subtypes of PGE2 receptors (EP1-4) have been reported, which are considered to take part in the modulation of pain sensation. By using the immunofluorescence technique, we obtained preliminary data on the specific localization of these receptors. Our results suggested that EP1 receptor is related to the inhibitory interneurons and EP2 and 4 are related to the excitatory interneurons. We are investigating the differential role of their interneurons with differnt EPs on the modulation of pain sensation. All these data will be presented to elucidate the function of interneurons in the dorsal horn.

## P2-f23 Phospho-NFkB is a suitable marker for activated primary and higher order sensory neurons.

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We examined if phosphorylated nuclear factor kappa B (pNFkB) is a suitable marker for activated primary and higher order sensory neurons. Male C57BL6 mice were injected with 20 μl of 5 % formalin subcutaneously in the right hind paw. Twenty minutes after the injection, they were perfused from the left cardiac ventricle with 4 % lidocaine. Frozen sections of DRG, spinal cord, and brain were immunostained for pNFkB and neurofilament 200 (a neuronal marker). PNFkB-positive neurons were found only the right side of DRG and superficial layer of dorsal horn corresponding to the L4-L5 levels. In the brain, pNFkB-positive neurons were more abundant in the left parabrachial nucleus than in the right one. These results indicate that pNFkB is a suitable marker for activated primary and higher order sensory neurons.

KAKENHI 16390059

## P2-f24 Is it enough for neuronal excitability of MI to produce force perception?

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Sensation of force is closely related to the motor command rather than the afferent input. We have recently showed with fMRI that the activity of the primary motor cortex (MI) is closely correlated with force perception. In the present study, we examined the perceived magnitude of handgrip force with the contralateral limb-matching method (McCloskey et al., 1974) in pre- and post-repetitive transcranial magnetic stimulation (rTMS) to elucidate a cause-and -effect relationship between neuronal activity of MI and force perception. The handgrip force exerted by indicator (left) hand significantly increased after the application of the rTMS with low-frequency (0.9 Hz) over individually determined left MI for 10 minutes (600 total pulses), in spite of the same handgrip force exerted by the reference (right) after the rTMS as that before. This result suggests that the neuronal activity in MI plays an important role in coding neuronal correlates of force perception.

KAKENHI (No.16500425)

## P2-f25 On selectivity of laser-heat irradiation in activation of cutaneous nociceptors

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The aim of this study is to clarify selective activation of laser heat irradiation in animal preparation. We used dorsal root compound action potential recording(CAP) to compare A $\beta$, A$\delta$ and C responses evoked by electrical stimulation of the sciatic nerve(ES_SN), the hindpaw(ES_paw), and laser stimulation of the hindpaw(LS_paw). At the same time, multiple single-unit recording of neurons in the ventroposterior lateral nucleus of the thalamus(VPL)was recorded with 16 channel Michigan Probe. LS_paw of the pentobarbital anesthetized rat evoked A $\beta$, A$\delta$ and C components, just as strong ES_SN. The C/A $\beta$ and A$\delta$/A $\beta$ ratios of the CAP evoked by LS_paw were 5 to 15 times higher than by ES_SN($p<0.05$). In the same preparation, ES_paw, no matter how strong, failed to elicit C response. Therefore, we concluded that the laser heat stimulus had significantly better selectivity. VPL ensemble neuronal activity showed pattern of A $\beta$, A$\delta$ and C responses corresponded to the dorsal root CAP patterns. We are in the process of using this technique to reassess the nociceptive processing of the thalamic nuclei.

NSC (95-2311-B-002-017-MY)

## P2-f26 c-fos expression in the catecholaminergic neurons of the brainstem after eccentric rotation of rats

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The expression of *c-fos* gene in catecholaminergic neurons within the brainstem was examined immunohistochemically. Conscious animals were subjected to eccentric rotation on the horizontal plane. Neuronal activation was defined by Fos protein expression. After stimulation, the Fos protein were mainly distributed in the vestibular nucleus complex (VNC), parabrachial nucleus (PBN), nucleus of the solitary tract (NTS), locus coeruleus (LC), substantia nigra (SN), caudo- or rostro-ventrolateral reticular formation(CVL, RVL),etc. Double immunofluorescent staining showed that most of catecholaminergic neurons in the NTS were activation (double labeled Fos/TH neurons) after eccentric rotation. Double labeled Fos/TH neurons were also observed in LC, CVL, RVL, and other regions related to sympathetic activities of the brainstem. However, there were no double labeled Fos/TH neurons in the SN. Our findings suggest that the catecholaminergic neurons in the brainstem may contribute to the changes of the sympathetic activities induced by the gravity-related spatial information during head movements.

## P2-f27 Neural activity of rats related to head elevation angle

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Head direction cells (HD cells) in rats are thought to be the "neural compass" in the horizontal plane. However, it is unknown how rats perceive their head direction in three-dimensional space. In this study, we monitored neural activities of freely moving rats using extracellular recordings. As a result, we found that the activities of some cells were highly correlated with the rat's head elevation. That is, these cells showed high firing rate bursts when the rat raised its head to a certain angle. The activities were independent of the rat's position or the head direction in the horizontal plane, suggesting that these cells are neither place cells nor HD cells. The burst activity did not cease when the lights were turned off. In addition, our preliminary results show that the burst activity is more correlated with the head elevation angle relative to the trunk than that relative to the experimental coordinate.

KAKENHI 17206022, Mobiligence Project

## P2-f28 Measurement of the size and number of the sensory fibers in three branches of the trigeminal nerve in the mouse

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The sensory part of trigeminal nerve is divided into three main branches, ophthalmic (V1), maxillary (V2) and mandibular nerves (V3). We investigated the number and size of the sensory fibers in the three branches in the mouse. The animals were perfused with 2% paraformaldehyde and 2% glutaraldehyde in 0.1M phosphate buffer (pH 7.4). Each branch was embedded in the epoxy resin, then semi-thin sections were cut. The microphotographs were taken in a computer and the cross-sectional areas and number of fibers were measured by a computer software (Image-Pro Plus, Planetron, Inc.). The cross-sectional areas of fibers were 6.11±0.11 $\mu m^2$ (mean ± SEM) for V1 fibers, 8.93±0.05 $\mu m^2$ for V2 fibers, and 6.01±0.06 $\mu m^2$ for V3 fibers. Total number of the sensory fibers was about 36,000 (per one side), of which 7% were V1, 82% were V2, and 11% were V3 fibers. The large size and number of V2 fibers may reflect well-developed vibrissae in the maxillary part and a wide area innervated by this branch.

## P2-f29 Population coding of tactile information of object in rat's whiskerring behavior

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Rats use their whiskers to navigate, to explore, to detect objects, and to determine the size, shape and texture of those objects. The 'whiskering' motion greatly expands the space that can be sampled, but it also complicates the calculations that the brain has to perform to identify the location of an object. Experimental studies have suggested that the nervous system extracts sufficient information from sparse spike trains to accurately encode complex stimuli. However, it is not yet clear by what kinds of features of the spike trains the tactile information is represented. To address this issue, we made a model of primary afferents, in which each afferent is arrayed in 2D space. We show here that the information of object distance and its size is represented by the combination of synchronous area of afferent spikes and latency of the first spikes. It is also shown that synaptic integration with short time constant well extracts the object features.

## P2-f30 Inhibitory input from the interpolar nucleus of the spinal trigeminal complex to the principal trigeminal nucleus

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The brainstem trigeminal complex is the first relay stations in the vibrissal system. The lemniscal pathway which arises from the principal trigeminal nucleus and the paralemniscal pathway which arises from interpolar nucleus of the spinal trigeminal complex convey information to the somatosensory cortices. In the principal nucleus neurons, discharges were evoked by the motion of one vibrissa (the principal whisker), while inhibition was induced by deflection of adjacent whiskers. Since very few GABAergic neurons are distributed in the principal nucleus, it is unlikely that the inhibition comes from local cells in the principal nucleus. Using tract tracing technique, we revealed that a subgroup of the interpolar nucleus neurons send GABAergic afferents to the principal nucleus. This suggest that those two parallel pathways coordinately act even in the brainstem.

KAKENHI 18019017, 18700342

## P2-f31 Orexin modulates neuronal activities in rat mesencephalic trigeminal sensory neurons

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Orexin is a regulatory peptide involved in the control of feeding, motivation and adaptive behaviors. To examine the role of orexin in mastication, whole cell patch-clamp recordings from mesencephalic trigeminal sensory neurons (Mes V), which are critical components of the circuits controlling oral-motor activity, were performed on brainstem slice preparations from Wistar rats aged between postnatal days (P) 0-17. By bath-application of orexin-A (100-500 nM), small membrane depolarizations < 3 mV with decreases of the input resistance were observed at the resting potential in the presence of tetrodotoxin (1 μM). In rats older than P7 or P8, it is known that the burst discharges are induced by a depolarizing step pulse at a holding potential of about -40 to -50 mV in Mes V neurons. Such conditional burst discharges in Mes V neurons were reduced to 61% of control by bath-application of orexin-A. Persistent sodium currents, which contribute to production of such conditional burst discharges, were also reduced by orexin-A. These results suggest that orexin modulates oral-motor behavior via Mes V neurons.

## P2-f32 Neurons that become active during hand and mouth movements in the hand region of the secondary somatosensory cortex of the macaque monkey

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Neuronal activities during a simple feeding task were investigated in the hand region of the secondary somatosensory cortex (SII) using an unanesthetized macaque monkey. The monkey was trained to pick up a small piece of food from the experimenter's hand and ate it. Among 704 isolated neurons from both sides of SII, 557 were active during retrieval of food. Most of them (54%) discharged when the ipsilateral as well as contralateral hand was used (bilateral activities). We found 61 neurons that were active during eating as well as retrieving food (hand/mouth neurons). Almost all of the hand/mouth neurons (92%) showed the bilateral activities suggesting that integration of information about the movements of the hand and mouth occurs among the neurons showing bilateral activities during hand movements. We believe that integrated information of these hand/mouth neurons contributes to the coordination of the hand and mouth movements.

KAKENHI (17500205)

## P2-f33 Behavioral inference based on hippocampal multi-neuronal activities of rats

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We tried to infer behaviors of rats from multi-neuronal spike data of the hippocampus. The rats were conditioned to poke the right (left) hole when they heard a high (low) tone. We recorded the neuronal activities from the hippocampus CA1 region of the rats, and we obtained the multi-neuronal spike activities applying the spike sorting technique to the data. Next, we measured a similarity among multi-neuronal spike activities. The similarity we used is defined by the kernel function termed by Spikernel. Performing the kernel k-means clustering and the kernel PCA with the similarity matrices, we classified the multi-neuronal spike activities into two groups, each of which is in good agreement with the discrimination behavior. Our results suggest that the sequence of spike activities plays a crucial role in the better inference.

## P2-f34 Prediction of multi-dimensional decision-making based on the single-trial activities of a population of prefrontal neurons

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To utilize the brain signals related to complex decisions for multi-dimensional sensorimotor responses, we have developed an algorithm that produces a post-hoc prediction of trial types based on the single-trial activity of 323 prefrontal neurons which were recorded individually while monkeys remembered one of 6 cue locations either to make or not to make a saccade. We used a template matching method to classify the activity into 12 trial types (6 locations by 2 responses) in the following two ways. The single-dimensional classification of 12 trial types at once made correct predictions on about 80% of trials during the late cue period. The multi-dimensional classification, which combined separate predictions of cue locations during the early cue period and behavioral responses during the late delay period, made correct predictions on more than 90% of trials. These results suggest that the multi-dimensional classification might have advantages in developing a better cognitive brain-machine interface.

KAKENHI (17700167,NM;18019048,RPH);NEI (RPH)

## P2-f35 Development of an electrodes array for a homogeneous sampling of orientation tuned cells in the visual cortex

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We recorded multiple single units simultaneously from the primary visual cortex of an anesthetized cat. We attempted to predict an orientation of the light bar stimulus based on the multineuron data of a single trial. For a better prediction, we need homogeneous samplings of differently orientation tuned cells. However, orientation selectivities were rather heterogeneous in our previous data recorded by 4-tetrodes array. To solve the problem, we developed a 8-single electrodes array. Since new array`s channels were more densely distributed, we expected that orientation selectivities were more homogeneous. By the circular variance, we compared the distribution of unit`s orientation selectivities between 8-single electrodes array and 4-tetrodes array. Some recordings suggested that 8-single electrodes array can record units with a larger variance.

## P2-f36 Modeling of extracellular multiple neuronal activities based on the multi-unit recording

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To obtain good measurement of extracellular multi-unit activities, adjustment of electrode parameters such as the size and spacing of electrodes is essential. However, these parameters have been decided according to the researches' experience. To optimize the electrode design, computer simulation system, we constructed a model of multi-unit recording in a three-dimensional neural tissue using multi-site electrodes. Computer simulations show that this model faithfully reproduces waveforms of multi-unit activities, amplitude distribution of extracellular potential and power spectral densities of the signals. These results suggest this system is useful for designing multi-site recording electrode.

## P2-f37 Study of attention assignment estimation by Auditory Steady State Response EEG

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Although a large number of studies have been made on the method of own motion imaging for detection the evoked potential for BMI, little is known about the method of auditory for detection the evoked potential. We studied the detecting method of using auditory stimulations to get the eliciting stimuli. The human brain shows the steady state response corresponding to the frequency modulation when listens the auditory stimuli by the sinusoidally amplituted-modulated tone ( SAM tone ) , and this response is known to change by the attention assignmnet. So, we presented two kinds of SAM tone with different frequency modulation from two speakers with separated to subjects at a time. And, we instruct subjects to attention to one sound at indicated side, then we got the EEG accepting the modulation when a subject focused his attention to the SAM tone indicated side. And then, we disucuss the possibility of the BMI using this approach.

## P2-f38 Development of on-line data processing platform for EEG-based BCI

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An EEG-based Brain-Computer Interface (BCI) platform using mu or beta oscillation during motor imagery task was developed. The platform consisted of several modules such as stimulus presentation, data acquisition, digital filter, command detection, and on-line feedback. This platform worked under Linux with Real-Time Application Interface (RTAI) extension. The data acquisition board with 16 input channels was connected to PCI-bus. COMEDI was used as device driver and Qt was chosen to implement Graphical User Interface. A BCI system for binary switching previously developed by authors was implemented on this platform, and was tested with healthy subjects. This system detected bursts of beta oscillation while the subject imagined hand or foot movements. A gauge bar with a length proportional to the band power of beta oscillation was presented to the subject through LCD monitor. The experiment showed that the system was suitable for developing an EEG-based BCI. The platform is generic, and easy to use. By adding new modules, custom BCI systems can be implemented easily.

## P2-f39 A brain-machine interface for predicting both arm-reaching movements and postures

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In the present study, we developed a brain-machine interface (BMI) system that can predict both arm-reaching movements and target-holding postures from neuronal activity in the primary motor area (MI) when a monkey is performing a motor task. Low-pass filtered electromyography (fEMG) signals were computed from the neuronal activity by a linear regression model. Arm-movement trajectories were calculated from the fEMG signals by combination of an artificial neural network and servo-mechanism. The estimated fEMG signals and trajectories were well correlated with actually recorded EMGs and trajectories during movements and posture maintenances. Positions not only of the arm tip (wrist joint) but also the middle joint (elbow joint) were accurately estimated. These results suggest that our BMI system could predict both the dynamics and kinematics of neuroprosthetic devices simultaneously.

JST CREST Program

## P2-f40 Role of nitric oxide synthase-containing hypothalamic paraventricular-spinal neurons in the CRF-induced activation of the central sympatho-adrenal outflow in rats

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Centrally administered corticotropin-releasing factor (CRF) has been shown to activate sympatho-adrenal outflow in rats, but the central mechanisms remain unclear. In this study, we examined the role of paraventricular hypothalamic nucleus (PVN) neurons, known as the autonomic center, activated by in centrally administered CRF in rats, with regard to nitric oxide synthase (NOS). After injection of retrograde tracer into the spinal cord, retrogradely labeled cells were detected in the dorsal cap, ventral and posterior part of the PVN. After administration of CRF, neurons co-expressing of inducible NOS (iNOS) and c-Fos were observed in these retrogradely labeled cells. The CRF had little effect on neuronal NOS. These data suggest that PVN-spinal neurons expressing iNOS play a role in the centrally administered CRF-induced activation of the sympatho-adrenal outflow in rats.

## P2-f41 Neurokinin 1 receptor plastically changes excitability of neurons in reticular formation of rat medulla oblongata

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Our previous experiments using rats suggested that several autonomic responses accompanying with emesis were controlled by neurons in reticular formation near the nucleus ambiguus (NA) of medulla oblongata, and induced via neurokinin 1 receptor (NK1R) activation. In this study, the plastic changes of activities in the neurons near NA were investigated. Whole-cell recording was applied to the neurons in coronal slice preparations of brainstem isolated from infant rat (6-12 days). In the several neurons, tetanus stimulation (100Hz, 2 sec.) induced the augmentation of amplitude of EPSCs that was maintained for a few minutes. However, these augmentations were canceled by perfusion of NK1R antagonist, sendide (1μM). These results suggest that the excitability of neurons plastically changes via NK1R activation and hence participates in generating long lasting autonomic responses accompanying with emesis.

## P2-f42 Effects of $CO_2$ inhalation on 5-HT release in the dorsomedial medulla oblongata in infant rats

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5-HT release in the dorsomedial medulla oblongata (DMM), especially in the hypoglossal nucleus, affects genioglossus muscle activity, which may be related to obstructive sleep apnea. We previously showed that 5-HT release in the DMM in adult mice is increased by $CO_2$ inhalation. Prematurity in medullary 5-HT neurons has been reported in sudden infant death syndrome. In the present study, 5-HT release in the DMM and the responses to $CO_2$ inhalation were investigated in infant rats. Infant rats were anesthetized with pentobarbital sodium i.p. for insertion of a microdialysis probe in the DMM. They were then placed in a double chamber plethysmograph to record respiratory variables. After recovery from anesthesia, extracellular fluid was collected at 1.2 μL/minute every 5 minutes. The 5-HT was analyzed with an ECD-HPLC. Infant rats were exposed to 0, 5, 7 and 9% $CO_2$ in $O_2$. 5-HT release in the DMM in infant rats was not dose-dependently increased by $CO_2$ inhalation. The result suggests that 5-HT release in the DMM does not play a dominant role in mediation of $CO_2$/pH sensitivity in infant rats.

## P2-f43 The role of corticotropin-releasing factor neurons on arousal/yawning response of rats

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Our previous studies have suggested that an activation of the corticotropin-releasing factor (CRF) neurons in the hypothalamic paraventricular nucleus (PVN) is involved in yawning response of rats. We assessed the role of CRF neurons on arousal/yawning response induced by the PVN stimulation in anesthetized, spontaneously breathing rats. We recorded an intercostal electromyogram as an index of inspiratory activity, blood pressure (BP) as an index of autonomic function and the electrocorticogram (ECoG) to evaluate arousal responses during yawning. Although the PVN stimulation induced a stereotyped yawning response, which was characterized by a single large inspiration, an initial fall in BP and an arousal shift in the ECoG, the arousal response was significantly supressed after intracerebroventricular injection of α helical CRF, a CRF antagonist. These results suggest that CRF neuron in the PVN is involved in the arousal/yawning response of rats, and especially responsible for the arousal response accompanied by yawning behavior.

## P2-f44 The effect of lingual nerve stimulation on the cardiac-related activity of the parvicellular reticular formation neurons, and their projections to the rostral ventrolateral medulla

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The rostral part of parvicellular reticular formation (rRFp) receives somatic and visceral afferents, and has efferent projections to rostral ventrolateral medulla (RVLM) which are involved in somato-visceral sensory integration as well as in controlling the cardiovascular system. We investigated the effect of lingual nerve stimulation on the cardiac related activity of the rRFp neurons, and their projections to the RVLM in urethane-chloralose anaesthetized rats. Out of 24 spontaneously active rRFp neurons that received cardiac inputs, 20 neurons altered firing rate following stimulation of lingual nerve. Among them, 7 neurons were antidromically activated by RVLM stimulation. Remaining 4 neurons did not alter their firing rate, and any of neurons was not activated by RVLM stimulation. In conclusion, lingual nerve inputs can modulate cardiac related activity of the rRFp neurons, and some of them mediate the integrated inputs to the RVLM.

## P2-f45 Effects of midline sagittal sectioning of medulla on pre-inspiratory and inspiratory neuron activity in vitro

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We examined effects of midline sagittal sectioning of the medulla on pre-inspiratory (Pre-I) and inspiratory (Insp) neuron activity in the brainstem-spinal cord preparation isolated from newborn (0-1 day-old) rats. We monitored the fourth cervical ventral root (C4) discharge for Insp neuron activity, and the facial nerve and the first lumbar ventral root (L1) discharges for Pre-I neuron activity. When the medulla was cut through the midline upper than the level of mid hypoglossal roots, all nerve root discharges ceased. Then, 0.5-1 μM DAMGO (a μ opioid agonist) was applied by superfusion. Rhythmic discharge of the facial nerve and L1 but not C4 appeared within 5 min after DAMGO application. The right (left) facial nerve activity synchronized with left (right) L1 activity, respectively. We detected rhythmic neuronal activity in the rostral ventrolateral medulla, using optical recordings and whole cell patch clamp recordings. The results suggested that interactions via commissural fibers are necessary for Insp burst generation but not for rhythmic Pre-I neuron activity.

KAKENHI(16500208)

## P2-f46 Ultrastructure of the myenteric ganglion of the subdiaphragmatic esophagus and its vagal efferent terminals from the dorsal motor nucleus of the vagus (DMV) of the rat

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Motility of the subdiaphragmatic esophagus (ES) is regulated by the vagus nerve, which project to the motor endplates or the myenteric ganglia. We have examined the ultrastructure of ES myenteric ganglia, and whether the ganglion received direct projections from DMV using WGA-HRP. The myenteric ganglion was composed of small (20.1X11.1μm) and medium-sized neurons (23.8X14.8μm). They had dark cytoplasm containing many ribosomes, mitochondria, and an oval nucleus, and received only a few axosomatic terminals. About 85% of the axodendritic terminals contained pleomorphic vesicles, while the rest contained round vesicles. All axon terminals formed asymmetric synaptic contacts. When WGA-HRP was injected into DMV, anterogradely labeled terminals were found in the myenteric ganglia. The labeled terminals were large (1.99μm), and contacted exclusively the dendrites. All labeled terminals contained round vesicles and small granular vesicles, but not large granular vesicles.

## P2-f47 Brain thromboxane $A_2$ is involved in the centrally administered NMDA-induced adrenomedullary outflow in rats

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Previously, we reported that the brain arachidonic acid (AA) cascade was involved in the central activation of sympatho-adrenomedullary outflow in rats. In the present study, we investigated the mechanisms involved in the centrally administered NMDA-induced elevation of plasma catecholamines in regard to the brain AA cascade in urethane-anesthetized rats. Intracerebroventricullary (i.c.v.) administered NMDA (1, 5 and 10 nmol/rats, i.c.v.) dose-dependently elevated plasma noradrenaline (NA) and adrenaline (Ad). Indomethacin (a cyclooxygenase inhibitor) dose-dependently (0.6 and 1.2 μmol/rat, i.c.v.) reduced the NMDA (5 nmol/rat, i.c.v.)-induced elevation of both catecholamines. The NMDA-induced response was also dose-dependently (0.9 and 1.8 μmol/rat, i.c.v.) attenuatted by furegrelate [a thromboxane $A_2$ ($TxA_2$) synthase inhibitor], and abolished by acute bilateral adrenalectomy. These results suggest that the elevation of both plasma NA and Ad reflects the brain $TxA_2$-mediated secretion from adrenal medulla in rats.

## P2-f48 Synaptic contacts between median preoptic neurons and subfornical organ neurons projecting to the paraventricular hypothalamic nucleus

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The subfornical organ (SFO) innervates the paraventricular hypothalamic nucleus (PVN) by at least two routes: directly to the PVN and indirectly relaying at the median preoptic nucleus (POMe). The POMe, in addition to projections to the PVN, projects to the SFO and makes synapses onto its neurons. However, the projection feature of these SFO neurons is not clear. We pursued the possibility that POMe neurons innervate SFO neurons projecting to the PVN, using combined anterograde and retrograde tracing techniques in rats. An anterograde tracer and a retrograde tracer were simultaneously injected in the POMe and PVN, respectively. After injections, anterogradely labeled varicose fibers and retrogradely labeled cell bodies were found frequently in the SFO. Electron microscopy revealed axo-somatic and axo-dendritic synapses between these neuronal elements labeled. The finding indicates a possible innervation of SFO neurons projecting to the PVN by POMe neurons.

## P2-g01 Glutamatergic pathways from the commissural subnucleus of the nucleus of the solitary tract to the phrenic nucleus in the rat

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We have shown that moderate numbers of axon terminals from the commissural subnucleus of the solitary tract (comNST) are distributed in the Kölliker-Fuse nucleus (KF) and some of them are in contiguity with dendrites and somata of the KF neurons projecting to the rostral ventral respiratory group (rVRG) and phrenic nucleus (PhN). In this study, we first show the general distribution of glutamatergic neurons in the dorsal tegmental field of the pons and medulla oblongata by using in situ hybridization technique (ISH) for mRNA encoding vesicular glutamate transporter 2 (VGLUT2). Then, using retrograde tracing technique combined with ISH for mRNA encoding VGLUT2 or glutamic acid decarboxylase 67 (GAD67), we demonstrate that large numbers of KF-projecting comNST neurons as well as rVRG- and PhN-projecting KF neurons express VGLUT2 mRNA but not GAD mRNA. These results suggest that the comNST-KF-PhN and comNST-KF-rVRG pathways are made up of glutamatergic neurons.

## P2-g02 Effects of 8-OH DPAT on firing rates of respiratory neurons in raphe nuclei

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The midline raphe nuclei may be involved in the control of respiration. The present study investigated whether the respiratory neurons in the medullary raphe nuclei are chemosensitive and whether they are serotonergic. The experiments were conducted on decerebrated, vagotomized, paralyzed and artificially ventirated rats. Extracellular recordings were obtained from neurons showing firing patterns related to phrenic nerve discharge in the midline medullary tegmentum. The recorded respiratory neurons were tested for responsiveness to hypercarbic ventilatory challenge, and examined for the effects of i.v. administration of 8-OH DPAT on their discharge rates. We recorded inspiratory (I) throughout and I-frequency modulated neurons. When end-tidal CO2 concentration was raised, two types of response of raphe respiratory neurons were shown; firing rate increased and no response. Dose of 8-OH DPAT had little effect on firing rates of both types of respiratory neurons. These results suggest that some respiratory neurons in the raphe nuclei are chemosensitive, but not serotonergic.

## P2-g03 Physiological role of sex hormones and biological clock in thermoregulation in rodents

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We assessed the roles of estrogen in thermoregulation in female rodents. Ovariectomy was done for female rats, and a telemeter for body temperature measurement was placed. Silicone tubes containing 17-beta estradiol were subcutaneously placed in the half of the rats ($E_2$(+)group). The tubes without estradiol were implanted in the rest of the rats ($E_2$(+)group). Rats were exposed to 5°C cold for 2h. The brown and white adipose tissues (BAT and WAT) were incised, and the total brain was excised. Total RNA was obtained from the BAT and WAT. Quantitative analysis of UCP1 mRNA was conducted for the BAT, and that of UCP2 mRNA for the BAT and WAT. Brain slices were made for cFos immunohstochemistry, and cFos positive neurons in the hypothalamic were counted. Body temperature decreased in the cold in both groups, however; the reduction was greater in $E_2$(-) group. The number of cFos positive neurons was greater in $E_2$(+) group, and UCP1 mRNA was also larger in $E_2$(+) group. Estrogen may be involved in the cold defence mechanism in female rodents.

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## P2-g04 Response of cortical cerebral blood flow during stimulating the basal forebrain in cats

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We have recently showed that regional cerebral blood flow (rCBF) responses due to noxious somatic stimulation of a forelimb in anesthetized cats was quite different from that reported in rats when blood pressure changes were prevented by section of the spinal cord at T1 level. The responses in rats were due, in part, to the activation of the basal forebrain-originating cholinergic vasodilator fibers projecting to the cerebral cortex. Therefore we examined whether stimulation of the basal forebrain produces cortical vasodilation in cats. In anesthetized cats with a transected spinal cord at T1 level, focal electrical stimulation of the unilateral basal forebrain increased rCBF of the ipsilateral primary somatosensory cortex that was increased by stimulation of the contralateral forepaw. The response was the largest when the tip of the electrode was located within the area known to contain the basal forebrain neurons projecting to the primary somatosensory cortex. Thus, cortical vasodilation seems to be a general function of the basal forebrain neurons projecting to the cerebral cortex.

## P2-g05 An investigation of the stress-responsive molecular system in the hippocampus

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An exposure to stressors precipitates or exacerbates psychiatric disorders. The stress response of the brain may involve a complex genetic one. We have previously reported that three genes (BTG2, CCN1 and Adamts1) in the cerebral cortex were up-regulated in the stress response. In the present study, we carried out a further investigation on the genetic response to stress in the hippocampus of young adult mice. Real-time PCR demonstrated a statistically significant increase in the mRNAs of the three genes one hour after an acute injection of FG 7142 (20 mg/kg, i.p.), a pharmacological stressor. In situ hybridization analysis revealed that the transcripts of the genes were markedly enhanced in the dentate gyrus one hour after the drug. Moreover, immobilization stress for one hour produced a statistically significant increase in the mRNAs of BTG2 and CCN1, but not of Adamts1, in the hippocampus. Thus, the present study indicates that the three genes also play an important role in the molecular stress-responsive system of hippocampus.

KAKENHI(17591196)

## P2-g06 Creative art activities regulate stress

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Very few studies have been conducted on the effects of creative art activities. The effects of creative art activity (piano performance, calligraphy, clay molding) on human stress were investigated in this study.Thirty healthy college students (15 male and 15 female) were recruited and 10 students (5 male and 5 female) participated in each 30-min session of one of the following creative activities: 1) playing the piano, 2) molding a piece of art using clay, 3) writing using a brush and ink and remaining silence (control). Cortisol (C) level and State-trait anxiety inventory (STAI-I) were measured. Post-session C levels were markedly decreased for calligraphy, clay molding, and music groups, indicating a reduction in stress due to participation in sessions of creative activity. Post-session STAI scores decreased significantly in all groups, indicating a reduction in anxiety induced by engaging in creative activities. No differences in C and STAI results were observed among sessions.The psychological and physiological stress reduction effects of creative activity were demonstrated.

Tokubetsu Kyouiku Kenkyu Keihi

## P2-g07 Unique response of extracelluar zinc in the ventral hippocampus against novelty stress

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To evaluate the role of hippocampal zinc in exploratory behavior, the response of extracellular zinc in the hippocampus was examined in novel environments. Rats were placed for 50 min in a novel environment once a day for 8 days. Extracellular glutamate in the hippocampus was increased significantly during exploratory behavior on day 1, whereas extracellular zinc was decreased significantly. Extracellular zinc returned to the basal level during exploratory behavior on day 8. When exploratory behavior was observed during perfusion with 1 mM CaEDTA, a membrane-impermeable zinc chelator, locomotor activity in the novel environment was decreased. The decrease in extracellular zinc and the increase in extracellular glutamate in exploratory period were abolished by perfusion with CaEDTA. These results suggest that zinc uptake by hippocampal cells is linked to exploratory activity and required for the activation of glutamatergic neurotransmitter system.

## P2-g08 Early postnatal stress alters the synaptic response in the cortico-limbic system underlying regulation of emotional expression in adult rats

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The effect of early postnatal stress on the synaptic response in the cortico-limbic system underlying emotional regulation was examined under consciousness in adult rats, which had been exposed to footshock (FS) at the postnatal period of the second (2wFS) or the third week (3wFS). During the retention of contextual fear conditioning (CFC), remarkable decreases in freezing behavior and attenuation of the inhibitory synaptic response in the hippocampal CA1 field was found in 2wFS. In the extinction session, the restoration of the inhibitory synaptic response in the medial prefrontal cortex with declination of freezing was attenuated in 3wFS. These findings suggest that early postnatal stress affects the neural circuit responsible for the different processes of emotional expression, depending on the postnatal period exposed to aversive stress.

## P2-g09 Neurobehavioral and neurochemical changes caused by dichlorvos: Role of forced swim stress

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Stress conditions when combined with chemical exposure can act as predisposing factor in modifying out come of exposure. Dichlorvos, an organophosphate pesticide exerts its toxic effects in target animals by inhibiting AChE. In the present study the effect of co-exposure of Dichlorvos and physical stress on neurobehavioral and neurochemical alteration was investigated. The rats were given swim stress for 20min/day for 14 days prior to along with Dichlorvos (30mg/kg/bw, i.p) and were compared with Dichlorvos alone treated or unstressed animals. Animal were assessed for locomotor activity, Grip strength and passive avoidance response. For neurochemical assessment AchR ($p<0.001$) in the hippocampal region was estimated. Neurobehavioral results show that dichlorvos+stressed group exhibited significant impairment in learning ability (42%) and locomotor activity (50%) as compared to dichlorvos alone or stress alone group. The physical stress play role in Dichlorvos induced neurotoxicity

## P2-g10 Effects of neonatal maternal separation (MS) on peri-pubertal onset of aggressive behavior in male mice

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We reported that estrogen receptor $\beta$ knockout ($\beta$ ERKO) male mice exhibit higher levels and earlier onset of aggression around peri-pubertal period compared to wild-type (WT) mice (Nomura et al, 2002). Recently, we found that neonatal MS influences development of social and emotional behaviors tested as adult (Tsuda et al, 2006). In the present study, using $\beta$ ERKO and WT male mice, we examined whether MS may affect peri-pubertal onset and/or expression of male aggressive behavior and there may be any genotype differences in the effects of MS on these endopoints. Pups were separated daily (4h) from their mother from postnatal day 1 to 14. At 4 wks of age, males were isolated and tested for intermale aggression at 5wks and 6wks of age. In the non-MS control group, $\beta$ ERKO mice were significantly more aggressive than WT mice as seen in our previous studies. In both genotypes, MS significantly decreased cumulative duration of aggressive behavior at 5wks, but not at 6wks. These results suggest that MS may affect peri-pubertal onset of male aggressive behavior regardless of genotype.

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## P2-g11 Chewing-related neuronal mechanism under stress condition

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To evaluate the chewing-related mechanism underlying stress relief, we examined the effect of chewing gum without any taste on brain activity under noisy stress condition using fMR, together with its effect on plasma levels of catecholamine and ACTH. The stress condition significantly increased BOLD signals in the amygdala and the frontal association cortex, and elevated plasma levels of NA, DA and ACTH. However, gum chewing suppressed the noisy sound-induced increase in BOLD signals in these regions, and plasma levels of NA, DA and ACTH, suggesting that chewing may be a useful therapy for reducing stress.

## P2-g12 Anxiolytic and anti-stress effects of coffee aroma in mice

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It has been reported that the aroma of coffee causes relaxation in humans. To clarify the relationship between coffee aroma and psychological stress, we investigated the effects of coffee aroma on mouse behavior in the elevated plus-maze test, which is a method for measuring anxiety in animals. In addition, we measured the stress-induced polyamine changes in the mouse brain following restraint stress. In the elevated plus-maze test, C57BL/6J mice were exposed to coffee aroma for 4 h and placed on the central square of the maze. Coffee aroma increased the time spent in and the number of entries into open arms, while it reduced the time spent in closed arms. In the polyamine assay, coffee aroma inhibited the elevation of the putrescine level in the mouse brain induced by restraint stress for 2 h. These results suggest that coffee aroma has anxiolytic and anti-stress effects under stressful conditions.

## P2-g13 Impaired reactivity to mental stress in premenstrual dysphoric disorder (PMDD) patients

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PMDD patients experience disruptions in mood or physical complaints during the menstrual cycle. The predominate symptoms are depression, anxiety, or irritability. It has been hypothesized that hormonal imbalance in response to stress is responsible for these symptoms. The present study was designed to examine this possibility in PMDD patients and normal healthy subjects. PMDD patients (diagnosed by DSM-IV) showed significantly higher trait ($P<0.001$) and state anxiety scores ($p<0.05$) as compared with control. Unexpectedly, plasma levels of the stress hormone, cortisol, were significantly reduced ($p<0.05$) in the PMDD patients. Peripheral-type benzodiazepine receptors (PBR) in platelets, a biological marker for stress, were reduced to some extent. The results indicate that reactivity to mental stress is impaired in PMDD patients.

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※Public Health Research Foundation

## P2-g14 The comparison of acute spontaneous and forced exercise on activation of several brain areas related to psychological responses

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Behavioral, endocrinal and immune responses are known to be related to psychological alterations during exercise. In the present study, we investigated neuronal activity in several brain areas related to psychological responses during 1hour spontaneous (SWR) and forced wheel running (FWR) using immunohistrogical methods in rats. The acute running distance for 1hour was broadly equal between SWR and FWR. FWR strongly activated corticotropin-releasing hormone neurons in the paraventricular nucleus of hypothalamus compared to SWR. The noradrenergic neurons in the locus coeruleus were significantly activated in both SWR and FWR, and no significant differences were found between SWR and FWR. SWR significantly activated of dopamine neurons in the ventral tegmental area and serotonin neurons in the dorsal raphe nucleus compared to FWR. These results suggest that characteristics of neuronal activity of brain areas related to psychological responses are different between SWR and FWR.

## P2-g15 Involvement of glucocorticoid response in hippocampal changes in aged SAMP8 mice with occlusal disharmony

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Our recent studies have shown that, in the aged SAMP8 mice, occlusal disharmony induces deficits in spatial memory, being due to pathological changes in the hippocampus, such as neuronal degeneration. In this study, we used SAMP8 mice under the bite-raising condition to examine the involvement of the glucocorticoid response in occlusal disharmony-induced hippocampal changes. The bite-raised condition caused a significant increase in plasma corticosterone levels compared with the control condition. Pretreatment of metyrapone prevent the bite-raised condition-induced increase in plasma corticosterone levels, reduction in CA3 pyramidal neuron numbers, and impairment of spatial learning. The results suggest a link between the bite-raised condition and glucocorticoid response, which might be involved in the spatial learning deficits and hippocampal neuronal death in aged SAMP8 mice.

## P2-g16 Chronic stress arrests the maturation of hippocampal neuronal progenitors in mice overexpressing an AD type presenilin 1 (PS1).

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Early studies have suggested that chronic stress lowers the hippocampal neurogenesis, thereby impinging on cognitive functions. However, the detailed machineries remain elusive. Here we analyze the effect of chronic stress on the hippocampal neurogenesis using a transgenic mouse line overexpressing human PS1 with an Alzheimer's disease (AD)-related mutation (Tg) to link the stress with cognitive dysfunctions in AD. Consequently, we found that the stress primarily reduced the production of Ki67/Pax6-positive (+) neural progenitors in the hippocampal granule cell layer (GCL) both in Tg and the wild type mice. In contrast, the number of DCX+ post-mitotic neuronal progenitors in GCL was increased only in Tg with the stress. Of note, DCX+ cells in the GCL of Tg under the stress were irregularly distributed in a pattern like those of aged rats. These data raise the possibility that chronic stress could perturb maturation of post-mitotic neuronal progenitors, which may accelerates cognitive dysfunctions in AD.

※KAKENHI (18790607)

## P2-g17 The combined DEX/CRH test as a marker of subjective stress in non-clinical population

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Hypothalamic-pituitary-adrenal (HPA) axis function, as measured by the response to the combined dexamethasone/corticotrophin-releasing hormone (DEX/CRH) test, has been reported to be abnormal in various stress-related diseases including major depression, but psychosocial stress among healthy people has not been well examined using this test. To clarify the possible association between subjectively perceived stress and the HPA axis function, we administered the DEX/CRH test and the Hopkins Symptom Checklist (HSCL), a self-reported questionnaire consisting of 54 items that assess recently perceived stress, to 35 healthy adults. The outcomes of the DEX/CRH test were the plasma cortisol and arginine vasopressin (AVP) just before and 1 h after the CRH challenge. The stress score was positively correlated with the cortisol and AVP levels, suggesting that DEX/CRH test may serve as a biological marker of subjectively rated stress symptoms among non-clinical population.

## P2-g18 Reversibility of behavior abnormalities induced by isolation housing in young mice

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In this study, male ddY mice at the 3 weeks age were reared either isolation or group housing for 2, 4 and 8 weeks and then subjected to a forced swimming test, novel object recognition test, locomotor activity, and aggression test. In the forced swimming test, significant decrease in the immobilizing time was observed by the social isolation for 2 and 4 weeks, but not for 8 weeks. However, the locomotor activity was significantly increased by the social isolation for 8 weeks, but not for 2 and 4 weeks. In addition, the social isolation led to cognitive impairment and aggressive behaviors within 2 weeks. The behavioral changes induced by 8-weeks isolation were abolished by subsequent group housing for 4 weeks. Taken together, a 2 weeks post-weaning period of the social isolation led to sociality deficiencies with cognitive dysfunction and aggression, which are restored by social interaction in group housing.

## P2-g19 Involvement of microRNA-mediated downregulation of glucocorticoid receptor in vulnerability to stress

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Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis is associated with vulnerability to depression and anxiety disorders. Daily repeated exposure to a stressor resulted in reduced HPA axis response to the same stressor, that is termed habituation. In this study, we established the animal model of vulnerability to repeated stress. We found that Fischer344 (F344) rats showed the deficit in habituation to repeated restraint stress such as suppressed inhibition of HPA axis and hypothalamic neuronal activity. In addition, F344 rats showed the decreased cell proliferation in hippocampus and increased anxiety behavior. Moreover, we found that microRNA (miR)-18a inhibited translation of glucocorticoid receptor (GR) mRNA in vitro and F344 rats showed the decreased expression of GR protein and increased expression of miR-18a in the hypothalamus. Thus, our results suggest that miR-18a-mediated downregulation of GR could be involved in vulnerability to stress.

## P2-g20 Loud white noise attenuates depressive response without affecting glucocorticoid levels in rats

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Although long-term exposure to loud sound has been shown to induce stress responses and hearing loss, some people tend to prefer harmful loud sound when they use portable music player suggesting its unknown appetitive effects other than aversive ones. To elucidate psycho-physiological actions of loud sound, we examined the effects of loud white noise (100dB, exposed for 30 min or 2 h) on serum corticosterone levels and emotional behaviors in the elevated plus maze test (EPM) and forced swimming test (FSW) using male Wistar rats. Loud sound exposed for 2h but not 30 min facilitated grooming responses in EPM and reduced immobility, an index of depressive response, in FSW without affecting corticosterone levels. Since 4 h exposure to loud noise has been shown to induce stress responses, adequate duration of exposure to loud sound may facilitate stress-coping function and reduce negative feeling like depression.

21st Century COE Program, KAKENHI(16209006)

## P2-g21 Electrostatic pulse stimulation may alter respiration pace by unconsciously weak startle reflex

Tatsuhiro Yasuda
*Free*

Some people may experience respiration pace alteration by subtle electrostatic stimuli without conscious shocking perception but with mild startle reflex accompanying phrenic contraction. To study the human electrostatics sensitivity from personal computer (PC) transaction, we experienced that phenomenon by using periodic temperature alteration measurement in which a thermocouple is put near human subjects' lips and by giving electrostatic stimuli generated with periodical dialog flashes transaction on PC. Degree of temperature change is proportional to respiration depth. We found the periodical altering flashes may alter some subject's respiration pace. Some subjects may have reception sensitivity on that electrostatic induction, others may not. Almost all subjects felt uncomfortable and fatigue after the experiments. These experiments indicate the sense of touch by static electricity through skin and tympanic membrane may generate neural signals which are sent to inspiration nucleus in the medulla, and inspiration is induced. Or continuance of subjects' subtle muscle contraction stiffness by weak electrostatic pulse may make them hypoxia state and gasp induction.

## P2-g22 Acute stress increased neuropsin mRNA level via glucocorticoid pathway in the hippocampus

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Stress has been shown to influence hippocampal plasticity such as long-term potentiation, spine morphology and behavioral memory. Recently many studies have shown that steroid hormones regulated the expression of kallikrein (KLK)-family serine proteases in cancer cell lines, and one of the KLK protease, named neuropsin (NP), localized in the limbic area where is responsible for stress. In this study, therefore, we monitored the expression level of NP mRNA after an acute restraint stress session in the hippocampus by semi-quantative RT-PCR. The acute stress extensively increased NP mRNA expression right after the stress session and it kept high level for more than 24 hrs. To explore a cascade from behavioral stress to upregulation of NP mRNA, we examined whether CORT regulated the expression of NP mRNA. Application of CORT onto cultured hippocampal neurons significantly increased the expression of NP mRNA. These results suggest that NP would be regulated via CORT derived by stress, and involved in the stress-induced neuronal plasticity.

21st century COE research

## P2-g23 Statistical estimation of phase response curves under the various types of noisy environments

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Phase response curve(PRC) is a convenient tool for understanding the properties of periodic activity of dynamical systems such as synchronized neurons. PRCs describe the effect of a perturbation on a dynamical system. Once PRCs are measured, the collective dynamics of the entire network, such as synchronization properties, can be analysed theoretically. For this purpose, many researchers have recently measured PRCs of neurons, which usually provide very noisy data owing to some external and intrinsic stochasticity. In this talk, we consider various types of noise under which PRCs are measured experimentally and try to estimate PRCs under the noise by using modern statistical methods, including Gaussian process regression.

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## P2-g24 Rhythmic control of AANAT translation by hnRNP Q in circadian melatonin production

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The circadian rhythm of pineal melatonin requires the nocturnal increment of serotonin N-acetyltransferase (arylalkylamine N-acetyltransferase [AANAT]) protein. To date, only limited information is available in the critical issue of how AANAT protein expression is up-regulated exclusively at night regardless of its species-specific mRNA profiles. Here we show that the circadian timing of AANAT protein expression is regulated by rhythmic translation of AANAT mRNA. This rhythmic control is mediated by both a highly conserved IRES (internal ribosome entry site) element within the AANAT 5'UTR and its partner hnRNP Q (heterogeneous nuclear ribonucleoprotein Q) with a peak in the middle of the night. Consistent with the enhancing role of hnRNP Q in AANAT IRES activities, knockdown of hnRNP Q level elicited a dramatic decrease of peak amplitude in AANAT protein profile parallel to reduced melatonin production in pinealocytes. This translational regulation of AANAT mRNA provides a novel aspect for achieving the circadian rhythmicity of vertebrate melatonin.

## P2-g25 Neuronal activity in the paraventricular nucleus during penile erection in unanaesthetized rats.

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The oxytocin neurons in the paraventricular nucleus (PVN) project directly to the sacral spinal chord and are involved in penile erection (PE). To assess the role of the PVN in PE, we recorded the neurons in and around the PVN. Rats were implanted with a telemetric transducer to measure corpus spongiosum of the penis (CSP) pressure, wires to measure neck and bulbospongiosum muscle activities and screws on their skull to record the EEG. With a glass micropipette electrode, neurons in and around the PVN were recorded under head restrained unanaesthetized conditions.PE is characterized by a slow increase in CSP pressure with riding CSP pressure peaks. Of 11 PVN neurons, 6 showed phasic firing in some relation with the sharp CSP pressure changes during PE, while 3 neurons decreased firing during PE. Two of five neurons dorsal to the PVN (reuniens nucleus) also phasic firing in relation with PE, while three of them decreased firing during PE. These results suggest that the PVN as well as its dorsal part have a crucial role in the generation of PE.

## P2-g26 Menstrual cycle affects thermal regulation and comfort

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We examined thermal regulation and comfort during a mild-cold exposure in women with regular menstrual cycle (n = 8). Experiments were conducted twice for each subject in the follicular (F) and the luteal (L) phases. A 120-min exposure at 23.5 C° fallowed a 40-min baseline at 29.5 C°. Body core temperature was higher ($P<0.05$; 36.9 ± 0.1 C° (mean SE) in F, 37.3 ± 0.1 C° in L.) in L than in F throughout the experiments. In addition body core temperature increased ($P<0.05$) in F and L. Both estradiol and progesterone were greater ($P<0.05$) in L than in F. Mean skin temperature, $VO_2$ and skin blood flow were not different between the two phases. Rating scores for cold discomfort and thermal sensation for the extremities were not difference between these groups,either. These results suggest that set point of body core temperature and thermal sensations change depending on phases in the menstrual cycle.

KAKENHI(17052023)

## P2-g27 Role of post-translational modification of BMAL1 in mammalian circadian clock

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Control of protein turn over via ubiquitination (Ub) is thought to involved in various biological processes. We showed that reversible inhibition of Ub-Proteasome by MG132 during dexamethazone (Dex)-pulse treatment-induced cellular clock resetting of NIH-3T3 cells significantly damped BMAL1:CLOCK-mediated circadian rhythm of luciferase-reporting Per2 gene expression (Per2-luc rhythm) and reduced clock proteins during circadian cycle after the release of MG132. Among clock proteins, endogenous Ub-BMAL1 accumulation with MG132 during Dex-pulse was significantly suppressed by U0126, ERK pathway inhibitor. Mouse BMAL1 contains a conserved ERK phosphorylation site. Ectopic expressed BMAL1 mutants lacking an ERK phosphorylation site in BMAL1-deficient mouse embryonic fibrobrasts during clock-resetting resulted in dramatically reduced Ub-BMAL1 level. We proposes that controlling protein turn over of BMAL1 via ERK phohorylation dependent ubiquitination has essential roles in circadian clock.

KAKENHI (18500300)

## P2-g28 Differential effect of nuclei Gigantocellularis and Magnocellularis stimulation on muscular activity under the absence of pontine regulation.

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Both pontine reticular formation (PIA) and the rostral medial medulla (RMM) are known to control muscle atonia. Microdialysis samples were collected during electrical stimulation in the RMM with and without lidocaine injection into the PIA using decerebrated cats. The muscle tone had an opposite response to the stimulation of the nuclei gigantocellularis (NGC) vs. magnocellularis (NMC) during the lidocaine injection. Stimulation of the NGC had no effect during the lidocaine inactivation of PIA. Whereas, effect of NMC stimulation was not altered. GABA release onto the motoneuron pool is increased and unaltered during NGC stimulation without (120.54% ± 11.1; $p = 0.03$) and with (98.96% ± 5.88; $p = 0.87$) lidocaine, respectively. In contrast, an increase in GABA release was found by either NMC stimulation alone (138.81% ± 22.09, $p = 0.02$) or with lidocaine (122.80% ± 10.55; $p = 0.04$).Our results indicate that the effect of NGC and NMC on muscle activity may be mediated through a different mechanism.

## P2-g29 Sleep health and sleep bruxism in the children of Japan

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The purpose of this study is to investigate into relationship sleep health and sleep bruxism in children(n=109,aged 6-12) with informed consent. The survey showed that feelings of sleep maintenance and sound sleep were significantly better in the children with severe bruxism than mild/non bruxism group. This study suggested that high frequent sleep bruxism in children may be caused by escaped control of cortex due to high appearance of deep sleep. In the next prospective pilot study, quantity/quality of sleep was examined by using actigraphy and heart rate variability in two boys(6 yrs.) with bruxism. Wake time after sleep onset (WASO) was 20 min. and HF of HRV was 1540 ms in a boy. High HF and few WASO were observed, sleep did not be disturbed.

## P2-g30 Intrinsic correlates of sleep state transition in EEG rhythms and cerebral hemodynamics

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Previous studies reported that cerebral hemodynamics changed in synchronization with the electroencephalography amplitude changes (EEGAC) during sleep. However, there was a concern that cerebral hemodynamics might reflect the change of the cardiovascular activity rather than EEGAC. The purpose of this study is to examine the intrinsic correlation between EEGAC and the cerebral hemodynamics by eliminating influences from the cardiovascular activity. We simultaneously measured EEG, cerebral hemoglobin concentration changes (HbCC), and the arterial blood pressure (ABP). The HbCC was measured at eighty-eight positions covering whole head of subject by optical topography probes. We extracted the intrinsic correlation between EEGAC and HbCC without ABP contributions by the cross-correlation and partial correlation analyses. As a result from sleep data of twelve adults, we found the intrinsic correlations between EEGAC and HbCC which were related in the specific sleep transitions.

JST CREST

## P2-g31 Role of arterial baroreceptor in circadian rhythm of renal sympathetic nerve activity in rats

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Role of arterial baroreceptors in circadian rhythm of renal sympathetic nerve activity (RSNA) was studied in rats. Wistar mal rats were instrumented chronically for the measurements of RSNA, electrocardiogram (ECG), and electromyogram (EMG) in sino-aortic denervated (SAD) and sham operated (SO) rats. At least 10 days after the recovery period, RSNA, ECG and EMG were measured continuously over three days in both SAD and SO rats. There was a robust circadian rhythm of RSNA in SO rats; it increased by 27% during dark period relative to light period. However, in SAD rats, RSNA increased by only 9% during dark period. Interestingly, there was also a robust circadian rhythm of heart rate, but the variation of heart rate in SO rats was similar to that in SAD rats. It is therefore suggested that the arterial baroreceptors play a significant role in causing the circadian rhythm of RSNA, but seem to have minor influence on the circadian rhythm of RSNA.

KAKENHI(17590203)

## P2-g32 Local cerebral blood flow in the hippocampus during REM sleep in rats

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Responses of regional cerebral blood flow in the hippocampus to changes in behavioural were studied in rats. Wistar males rats were instrumented chronically with laser-Doppler flow probe made with a pair of glass fiber (0.25mm outer diameter) in the CA1 region of the hippocampus, and also instrumented with electrode for the measurements of electroencephalogram, electrocardiogram and electromyogram, and with catheter for the measurements of systemic arterial and central venous pressure. The mean value of the local cerebral blood flow in the CA1 region of the hippocampus was the highest during REM sleep compared with the other behavioural states including non-REM sleep, quiet awake, moving and grooming. The vascular resistance in the CA1 region of the hippocampus decreased significantly during REM sleep. It is therefore suggested that neural activity in the hippocampus is the most active during REM sleep, causing active vasodilatation in rats.

KAKENHI(180190271)

## P2-g33 Behavioral state-dependent simultaneously change between the respiratory pattern and information processing mode in hippocampus

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Behavioral states control the information processing mode in hippocampus, which is reflected on the local EEG pattern. Hippocampus processes neocortical inputs and shows theta-band oscillatory activity during exploratory behavior, while it shows an irregular slow wave EEG pattern with sharp wave-ripple complexes during slow-wave sleep and awake non-exploratory behavior. Because the respiratory pattern has a close relationship to behavioral states, we examined whether the respiration pattern correlated with hippocampal EEG in diverse behavioral states in freely moving rats. The results showed an intimate correlation between the respiratory pattern and the hippocampal EEG pattern across diverse behavioral states, and the correlation was observed even in bilaterally bulbectomized rats. These results indicate that behavioral states coordinately control the respiration pattern and the information processing mode in hippocampus.

KAKENHI( 18100004, 17023012)

## P2-g34 Multi-neuronal dynamic in rat neocortex and hippocampus as a measure of natural sleep

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Natural sleep has been pictured as stepwise changes of the brain activity. In vivo recordings of the local field potential (LFP) from the sleeping brain typically exhibit cyclic alteration of brain activity characterized by changes in the power spectrum of LFP. With the recent development of the multiunit recording methods from a naturally sleeping animal, it is tempting to revisit the sleep stages that have been defined with LFP. We performed multiunit recording by tetrodes from the neocortex and hippocampus of the naturally sleeping rat without anesthesia and obtained the brain activity pattern that alternates between awake and sleep for several hours. Thus obtained data help us characterize awake, REM and non-REM sleeps from the view point of changes in neural assembly. Y.I and T.F thank financial support from the MEXT.
17022036, 18019041, 18700386

## P2-g35 PACAP deficient mice exhibit light parameter dependent abnormalities of nonvisual photoreception

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Pituitary adenylate cyclase-activating polypeptide (PACAP) is exclusively coexpressed with melanopsin in a small subset of retinal ganglion cells and is predicted to be involved extensively in the nonvisual photoreception such as entrainment of circadian clock, negative masking and pupillary light reflex (PLR). However, its functional role has not been well understood. Here we show that PACAP deficient mice exhibit severe dysfunctions of entrainment and masking, but not PLR. Under a light/dark cycle, PACAP deficient mice express an unusually early onset of activities, from approximately four hours before the dark period, without influencing the phase of the endogenous circadian clock. These results indicate that PACAP regulates particular nonvisual light responses and has a critical role in daily behavioral patterns.

## P2-g36 The glutamatergic receptors and gap junction involving the generation of carbachol-induced beta oscillation in rat hippocampal slices

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The glutamategic transmission and gap junction are important for the generation of the neuronal oscillation such as theta, and gamma rhythms. In the present study, the involvements of them in the generation of carbachol-induced beta oscillation were studied. Cholinergic agent carbachol (30 μM) induces beta oscillation which occurs like bursts intermittently in rat hippocampal slices. An AMPA receptor blocker CNQX suppressed the oscillation in a concentration-dependent manner. The suppression accompanied with the decrease in the frequency of the oscillation. NMDA receptor antagonists, both APV and MK801 suppressed the oscillation, too. They also changed the pattern of oscillation. A gap junction blocker carbenoxolone also suppressed the oscillation. Thus the glutamatergic transmission and gap junction should be related to the generation and modulation of the oscillation.

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## P2-g37 β-Endorphin regulates body temperature of Syrian hamster during maintenance phase of hibernation.

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The present study focused on the role of beta-endorphin on regulation of body temperature during maintenance phase of hibernation. Intracerebroventricular (icv) injection of naloxonazine (μ1 antagonist) was effective in arousing hamster from hibernation. In contrast, neither naltrindole (δ antagonist) nor nor-BNI (κ antagonist) interrupted the hibernation. The number of c-Fos positive neuron increased in arcuate nucleus (ARC) at 1 hr after hibernation onset compared with before hibernation. Furthermore, although the expression level of proopiomelanocortin (POMC) mRNA did not change, the endoprotease-2 mRNA increased in ARC. β-Endorphin-like immunoreactivity (β-End IR) was observed in ARC. With progress of hibernation, the β-End IR decreased with ARC and increased with PON. These results suggest that the β-endorphin was transported to PON from ARC by axonal flow and then regulates body temperature during maintenance phase of hibernation via μ1-opioid receptors.

KAKENHI(18500303)

## P2-g38 Histamine receptors genes knockout mice and their phenotypes

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Three histamine receptor subtypes (H1, H2, and H3) mediate these neuronal actions. There are many physiological roles of histamine in the brain such as sleep-awake cycle, locomotor activity, appetite and drinking, convulsion and learning/memory. The phenotypes of single gene knockout mice are characteristic of each histamine receptor. H1-KO mice significantly increased the ratio of ambulation during the light period to the total ambulation for 24 hr in an accustomed environment, and H3-KO mice showed a decrease in overall locomotion, wheel-running behavior, and body temperature during the dark phase but maintained normal circadian rhythm. H2-KO mice exhibited a marked hypertrophy with enlarged folds in gastric mucosa with the elevated level of serum gastrin. However, it is still unclear whether the deficiency of multiple histamine receptors has any effects on maintenance of life and fertility. Therefore we make multiple histamine receptors genes knockout mice to clarify the effects of multiple histamine receptors genes knockout on the phenotypes.

KAKENHI(18500286)

## P2-g39 Ameliorative effect of biting on the stress-attenuated hippocampal memory process: a Morris Water Maze study

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We examined the effect of biting on the stress-attenuated hippocampal activities, using the Morris water maze by which the hippocampus-related spatial memory has been evaluated in vivo. Ten-weeks old ddY mice were underwent three conditions, the restraint stress condition (stress group, ST), the biting condition during the restraint state (biting group, SB), and the control condition without any treatments (control group, CT). Both of restraint groups showed an impairment of spatial memory (escape latency; CT: 16.3 ± 3.2, ST: 28.8 ± 8.1, SB: 29.9 ± 8.0s) at 6 hours after the restraint; however, this impairment was recovered in biting group at 24 hour after the restraint (CT: 11.5 ± 3.2, ST: 19.9 ± 6.8, SB: 10.1 ± 2.9s). The results suggest that the biting might accelerate the relief from stress-induced hippocampal memory deficit.

## P2-g40 Ameliorative effect of biting on the stress-attenuated hippocampal memory process: a CA1-LTP study.

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To assess the ameliorative effect of biting on the stress-attenuated hippocampal activities, we measured a long-term potentiation (LTP) of field excitatory postsynaptic potential in the hippocampal CA1 region from the rats that underwent three conditions, the restraint stress condition (stress group), the biting condition during the restraint state (biting group), and the control condition without any treatments (control group). In the stress group, the LTP was reduced by about 48%, compared with that in the control group (182.8 ± 22%, n=5). However, in the biting group, stress-reduced LTP was recovered (191.9 ± 24%, n=10), attending to the control level. The data suggest the involvement of biting in the ameliorative mechanism underlying stress-induced attenuation of memory process in the hippocampus.

KAKENHI 18592254

## P2-g41 Increased serotonin efflux in the dorsal raphe nucleus of rats working for delayed rewards

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A low level of serotonin is often associated with impulsive behavior (Buhot, 1997; Robbins, 2000). We proposed that serotonin regulates the time scale of reward prediction (Doya, 2002). The aim of this research is to test this hypothesis by measuring the level of serotonin in the dorsal raphe nucleus (DRN) (N = 4) of free moving rats using in vivo microdialysis. In the sequential food-water navigation task, rat had to visit between two rewarding sites (site 1 food, site 2 water) alternately via a center circle in the open field (1.5 m diameter). Rats performed this task under three reward conditions, immediate reward, delayed reward (1 - 4 s) and reward omission. Significant increases in serotonin efflux were observed during delayed reward condition compared to immediate reward and reward omission conditions. These data suggest that the DRN serotonergic activity plays role in behaviors for delayed reward.

## P2-g42 Temporal window for consolidation of memory trace of adaptation of mouse optokinetic response eye movement

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Repetitions of trainings are necessary for acquisition and consolidation of motor memory. We reported that exposure of mice to 1h optokinetic stimuli induced a short-term adaptation in the gains of the horizontal optokinetic response that decay in 1 day, and succession of 1h of training for 4-5 days induced long-term adaptation that last for 1-2 week (Shutoh et al., 2006). Here, to reveal the time course for the conversion from short- to long-term adaptations, we compared adaptations among three groups of mice: one which received 1h training in 1 day, second which received 15 min of daily training successively for 4 days, and third which received 4 times of 15 min training at the interval of 1h in 1 day. Although the gain increases at the end of training were similar among the three groups, their decay time course differed markedly. Taken together with the effects of flocculus shutdowns, we suggest that the consolidation of memory trace starts within 1h after the end of the training.

## P2-g43 Effects of long term supplementation of PUFA on rat hippocampal neuron

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We previously demonstrated that dietary supplementation of Arachidonic acid (AA) longer than 3 mo for senescent rats could restore their water-maze behavior, the degree of long term potentiation (LTP), and the membrane fluidity in the CA1 neuron as those in young animals. The age-dependent deficiency in induction and maintenance of LTP are assumed to be caused by alteration in dominance of calcium entry path from NMDA receptor pathway to voltage dependent calcium channel (VDCC). This assumption was examined among 4 dietary groups; old control (OC), old AA supplemented (OA), old docosahexaenoic acid (DHA) supplemented (OD), and old AA & DHA mixture supplemented (OM) by pharmacological elimination of each calcium pathway. The degree of maintenance of LTP at 60 min after induction was followings; OC: 1.06±0.02; OA: 1.38±0.03; OD: 1.34±0.05; OM: 1.46±0.06 with statistic significance. Induction of LTP by $Ca^{2+}$ influx in OC was dominant through VDCC while that in OA was dominant through NMDA receptor as same as young animal.

## P2-g44 Activity of serotonergic neurons in the dorsal raphe nucleus of freely moving rats during reward and non-reward delay period

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Our working hypothesis is that the serotonergic system regulates the time scale of future reward prediction (Doya 2002). To test this hypothesis, we recorded neural activity chronically from the dorsal raphe nucleus (DRN) of behaving rats. Rats were trained in an open-field (1.5 m in diameter) to perform a sequential food-water navigation task. In the control condition, rats had to visit alternately between two rewarding sites (site 1 food, site 2 water) via a non-rewarding site 3, where only a tone (8 kHz) was presented upon entry. In the test condition, we introduced a delay period (1-4 s) between the insertion of the rat's nose into all sites and delivery of rewards or tone. The DRN neurons showed significantly increased firing during the reward delay period. Some of the neurons also increased firing during the tone delay period. Increased 5-HT neuron activity during reward and non-reward delay period may facilitate rat's waiting behavior in prospect of forthcoming reward.

## P2-g45 BESOM model consistent with the main anatomical characteristics of cerebral cortex

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The main anatomical characteristics of cerebral cortex are consistent with BESOM model proposed by the author.BESOM has the ability to acquire the efficient model of the world with unsupervised learning and to calculate Bayes estimate of the state of the world.The anatomical characteristics of cerebral cortex, six-layer structure, column structure and bidirectional connection rule between higher and lower areas, are very consistent with the neural network that calculates Bayes estimate.Horizontal connections between areas (such as V4 and MT) are understood as neural networks that execute independent component analysis, which is a part of the mechanism to acquire the generative model of the world.Areas that form cortico-basal ganglia loop are understood as tables of state-action pair, which is a part of the reinforcement learning mechanism. The author thinks BESOM model is plausible, from point of view of both neuroscience and computational theory. Computer simulation of this model may realize the main function of the brain.URL: http://staff.aist.go.jp/y-ichisugi/j-index.html

## P2-g46 Long-term measurement for spatiotemporal dynamics of cultured neural networks

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There have been few studies on the long-term measurement of spatiotemporal dynamics in neural networks during the developmental period. We developed a system of long-term measurement for spatiotemporal dynamics of cultured neural networks to investigate how spontaneous activity changes during the developmental period under non-invasive condition. The system consists of two subsystems. One is for temporal recording of neural firing on 8*8 = 64 multi-electrode arrays. Each array is a square of 50 microns. The A-D sampling rate is 20 kHz. The other is for spatial optical recording of the connectivity between neurons using time-lapse images. For the long-term measurement, a cell culture medium containing CO2 gas is supplied continuously using a syringe pump. A conventional incubator is not needed. We present the details of this novel system and provide examples of the spatiotemporal dynamics of the developmental changes in primary cultured neural networks of the rat cortical cortex measured over a period of more than one month.

## P2-g47 Effects of bilateral flocculus shutdown on the primate long-term adaptation of horizontal vestibulo-ocular reflex

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Adaptation of the horizontal vestibulo-ocular reflex (HVOR) induced by sustained visual-vestibular interaction is a prototype of cerebellum-dependent motor learning. We already reported that the memory trace of the adaptation of the HVOR induced by short-term (2h) training resides within the cerebellar flocculus by monkey experiments (Nagao and Kitazawa, 2003). Here, we examined the effects of flocculus shutdown on the long-term adaptation of the HVOR. Three rhesus monkeys were trained to wear the left-right reversing prism continuously for 3 days. They also received 2h vestibular training by 10deg-0.11Hz sinusoidal oscillation of the chair in light. We microinjected lidocaine solution into bilateral flocculi at the end of trainings. Although the reductions of the HVOR gains induced by 2h vestibular training were markedly affected by flocculus shutdown, those induced by 3-day prism-wearing were affected only partially, suggesting that the memory traces of short- and long-term adaptations reside in different neural areas.

## P2-g48 Neural mechanism of fluctuated recognition memory

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There is no assurance that once remembered memories are retrieved any time and each time we need. Those fluctuated memories either lack stability or some factors disturb them. We used the repetition retrieval paradigm to make "unstable" memory, and compared the neural mechanism with that of "stable" memory using rapid event-related fMRI. The right amygdala activation during encoding could predict the lack of retrieval stability and the activation of right middle temporal gyrus was associated with retrieval stability, regardless of memory accuracy. The amygdala activity suggests that it enhance interrupting or miss-leading cue association during encoding automatically. The latter suggests automatic or effortless retrieval without confirming details of memory. These results demonstrated that neural substrates of memory stability and fluctuation exist.

## P2-h01 Differences between single neuronal responses in anterior and posterior insula during multi-trial reward schedules

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The anterior (AI) and posterior insula (PI) have anatomically different neuronal connections with other brain areas, suggesting the functional subdivision between them. We previously showed that many of single neurons in monkey AI reacted to immediately forthcoming reward during multi-trial reward schedules. To examine whether PI neurons also show reward-expectancy-related responses, we recorded 38 PI neurons using the same task. Though 21/120 AI neurons reacted to possible reward, preliminary results show that none of 38 PI neurons reacted to it. On the other hand, none of 120 AI neurons reacted to reward solenoid apparatus activation whereas 8/38 PI neurons reacted to it (possibly its activation sound). These differences in information processing might come from different anatomical connections that AI and PI have dense connections with the areas responsive to reward and sound, respectively.

## P2-h02 Visualization of the convergence of entorhinal and hippocampal CA3 input onto CA1 neurons.

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The hippocampal area CA1 receives excitatory input from the entorhinal cortex through the perforant pathway, directly from layer III and indirectly from layer II through the dentate gyrus-CA3-Schaffer collateral circuit. Here we report anatomical evidence in support of such convergence in CA1 single neurons. In rats, anterograde tracers were injected into layer III of the entorhinal cortex and into CA3, to tag simultaneously the entorhinal fibers and the Schaffer collaterals with different Alexa Fluor ™ dyes (488 and 633). In 400 μm thick slices of fixed brains we intracellularly filled the Alexa Fluor hydrazide 555 in CA1 pyramidal cells and interneurons. Thin sections of these slices were scanned in a confocal laser scanning microscope and with the use of 3D reconstruction algorithms we confirmed that boutons on the labeled input fibers from both entorhinal cortex and CA3 were in contact with single CA1 interneurons as well as pyramidal cells.

## P2-h03 Establishment of *in vivo* gene transfer system into newly-hatched chick brain

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Newly-hatched domestic chick serves an ideal model for studies of neural basis of behavioral plasticity, particularly for understanding the mechanism of learning such as filial imprinting. In order to elucidate the molecular basis and gene functions involved in learning, we developed an *in vivo* gene transfer system in the brain of a living chick using electroporation. When green fluorescent protein-encoding plasmids were transfected to a chick brain, green fluorescence was clearly observed, and expression at the protein level was confirmed by immunoblotting. Most of the transfected brain cells exhibited dendrites and were immunostained using anti-NeuN antibodies, suggesting that these cells were neuronal cells. This neuron-selective electroporation system will facilitate the analysis of gene function in the living chick brain and provide further clues as to the molecular mechanisms of avian learning.

## P2-h04 Novel hydrolase AHO-3 is required for associative learning between cultivation temperature and feeding state in *C. elegans*

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Animals exhibit many types of behavioral plasticity. To elucidate the regulation mechanism of behavioral plasticity, we are using the temperature-food associative learning paradigm in *C. elegans*: well-fed animals migrate to and starved animals avoid from their cultivation temperature on a temperature gradient. The *aho-3* mutant has defect in this associative learning; *aho-3* mutants showed defect in starvation-induced cultivation temperature avoidance, while they showed normal response in food recognition. We found that *aho-3* gene encodes a predicted hydrolase that is a highly conserved protein throughout yeast to human. However, the molecular properties have not been characterized. *aho-3* was expressed in several neurons required for thermotaxis, and the learning defect of *aho-3* mutant was partially rescued by *aho-3* expression in thermotaxis neural circuit. Currently, we are trying to dissect the molecular and cellular analysis of *aho-3* gene further.

KAKENHI

## P2-h05 Context-dependent olfactory conditioning in cockroaches

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We demonstrated context-dependent olfactory learning by behavioral and electrophysiological methods. Cockroaches, *Periplaneta americana*, were trained with differential conditioning procedure in which the arrangement of conditioning stimuli (CS) and unconditioned stimuli (US) is the opposite between two light conditions (contexts); under context 1, odor A was paired with reward (US+) and odor B was paired with punishment (or presented without pairing, US-) and under context 2, the pairings were A-US- and B-US+. After training in the behavioral experiment, cockroaches selected the rewarded odor in each context. In the electrophysiological experiment, response of salivary neurons to rewarded odor was significantly greater than that to unrewarded odor after training in each context. We showed that (1) cockroaches are capable of selecting different odor according to the visual context and that (2) the contextual learning can be monitored by activities of salivary neurons.

## P2-h06 Nigrostriatal dopamine neurons form wide and highly dense axon terminals in the striatum of rat: single-cell study

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Dopamine (DA) neurons in the midbrain mediate a variety of brain functions, such as motor control, emotion, and reward. We studied the axonal arborization of DA neurons with Sindbis virus vectors that expressed membrane-targeted green fluorescent protein (GFP). After injections of the viral vectors into the rat substantia nigra compacta (SNc), infected single neurons were first examined their tyrosine hydroxylase immunoreactivity (TH-IR) and were then visualized completely by the inimmunoperoxidase method with an anti-GFP antibody. Of 70 infected neurons, 8 nigrostrital neurons showing TH-IR were reconstructed by camera lucida and formed wide and highly dense bushes in the striatum, amounted to hundreds of thousands μm in total length. The present results are considered to support the reinforcement-learning hypothesis of the basal ganglia.

## P2-h07 GABA receptors play roles in acquisition and retention of mouse eyeblink conditioning in the cerebellar nuclei.

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We examined the role of γ-aminobutyric acid receptors (GABA-R) in the deep cerebellar nuclei (DCN) during delay eyeblink conditioning and rotor rod tests in C57BL/6 mice by using GABA-R agonist (Muscimol: MSC) and antagonist (Picrotoxin: PTX). Bilateral injection of MSC and PTX into the DCN induced a severe motor deficient in rotor-rod tests, but the performance recovered 24h after drug injection. In the acquisition tests of eyeblink conditioning, both MSC- and PTX-injected mice showed a significant impairment in acquisition of conditioned responses (CR). In the retention tests after learning, the injection of MSC and PTX impaired the expression of CR. Furthermore, unilateral (ipsilateral and contralateral) injection of MSC and PTX also impaired the induction of CR, suggesting a possible crosstalk between left and right DCN in eyeblink conditioning. These results revealed that GABA-R play roles in acquisition and retention of mouse eyeblink conditioning in the DCN.

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## P2-h08 Proteolytic degradation of SCOP contributes to activation of ERK/MAPK and memory formation

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SCOP is a member of the leucine-rich repeat (LRR)-containing proteins. In addition to circadian expression in the suprachiasmatic nucleus, SCOP is constitutively expressed in neurons throughout the brain. SCOP interacts directly through its LRR domain with K-Ras, and negatively regulates K-Ras function and its down stream signaling, namely ERK/MAPK. We found that SCOP is proteolyzed by Calpain when hippocampal neurons are stimulated by BDNF or KCl. Moreover, training for novel object memory decreases SCOP in the hippocampus. To determin if hippocampus-dependent memory is influenced by SCOP in vivo, we generated a transgenic mouse strain for the inducible overexpression of SCOP in the forebrain. Overexpression of SCOP completely blocked long-term memory for novel objects. We conclude that degradation of SCOP by Calpain contributes to activation of ERK/MAPK during long-term memory formation.

## P2-h09 Intrinsic connections of the retrosplenial cortex in the rat

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The present study analyzed the connections between each area of the retrosplenial cortex (areas 29a-d) in the rat by use of retrograde transport of cholera toxin B subunit (CTb). Injection of CTb into area 29a retrogradely labeled cells in area 29b and caudal areas 29c and 29d. Injection into area 29b labeled cells in area 29a and caudal areas 29c and 29d. In case of injection into caudal area 29c, labeled cells were found in areas 29b, 29c, and 29d, whereas in case of injection into rostral area 29c, labeled cells were found in rostral area 29d. Injection into caudal area 29d labeled cells in area 29b and caudal areas 29c and 29d, whereas injection into rostral area 29d labeled cells in rostral area 29c. The results show that each area of the retrosplenial cortex projects massively to each other within a coronal plane. Furthermore, rostrocaudal projections within the retrosplenial cortex are more massive than caudorostral projections. These intrinsic connections may constitute pathways that transmit spatial and navigational information within the retrosplenial cortex.

## P2-h10 Improvement of memory in *Drosophila* by specific point mutations in PKA

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While many mutations have deleterious effects on memory, few have been shown to improve memory. To date, no single point mutations that increase memory retention have been identified.

Previously, we identified heterozygous mutations in *DC0*, the gene encoding the catalytic subunit of protein kinase A (PKA) as strong suppressors of age-related memory impairment (AMI). Heterozygous mutants which reduce PKA activity to 50% that of wild type can delay AMI two fold. While characterizing these mutants further, we found that heterozygotes of certain allele-specific point mutations in *DC0* also improve memory at young ages. Initial learning remains unchanged in our *DC0* mutants but memory retention is vastly improved, due to an increase in a specific consolidated memory phase, anesthesia-resistant memory.

Previous work in learning and memory has focused on increasing PKA activity as a method for improving memory. We demonstrate that PKA plays multiple roles in memory formation, and at least two of these roles are inhibitory. We are currently identifying PKA complexes that inhibit memory formation.

## P2-h11 Relationship between cocaine-induced conditioned place preference and hippocampal theta rhythm

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The hippocampus is thought to be important for spatial memory, however, the role in drug addiction has not been well known. In this study, the relationship between cocaine-induced conditioned place preference and hippocampal EEG, especially hippocampal theta rhythm was examined. Cocaine(15mg/kg, i.p.) was paired with one side of the three compartment apparatus and saline was paired with another side within a day for three consecutive days. The ratios in the power of T2(6.0-7.9Hz)/T1(4.0-5.9Hz) in the hippocampal theta were calculated by fast Fourier transformation. The results showed that the T2/T1 ratio during cocaine session was higher than that during saline session, and the T2/T1 ratio increased as the cocaine session proceed, but T2/T1 ratio didn't change as the saline session proceed. These results suggest that the information processing by the T2 theta band contributes to the development of association between cocaine and the environment.

## P2-h12 Electrophysiologicaly probed geometric correlation of E/I synapses and directional activation of the rat hippocampal CA3 area

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The area CA3 of the hippocampus is important in an associative memory function, and its recurrent network should be important for the coherent oscillation. It is thus interesting to ask how the recurrent network is formed in terms of synaptic order. We probed the order in rat hippocampal slice by recording synaptic currents from single cells while activating synaptic inputs with glass microelectrodes placed at the pyramidal cell layer. The stimulation caused a mixture of inhibitory (IPSC) and excitatory currents (EPSC). The IPSC amplitude decreased as the distance increased, while the rate of rise remained constant. The EPSC amplitude showed similar tendency, while the rate of rise decreased with distance. The functional synaptic arrangement in the CA3 area was further examined by the cell spike firing probability and optical recordings with voltage sensitive dye and could produce a sequential activation of CA3.

RIKEN BSI

## P2-h13 CASY-1, a homolog of Calsyntenin/Alcadein, is essential for multiple forms of learning in *C. elegans*

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Animals show dramatic behavioral changes depending on the environments. They show decrease of response or even avoidance of a favorable stimulus after they are treated with both the continuous stimulus and severe conditions such as starvation.

We isolated *casy-1(pe401)* mutant as a new mutant that is defective in salt chemotaxis learning. CASY-1 is the sole C. elegans homolog of human Calsyntenin/Alcadein family. Several *casy-1* deletion mutants show defects in multiple forms of behavioral plasticity and sensory processing. We found that CASY-1 acts in the ASER sensory neuron in the adult stage for salt chemotaxis learning.

The ectodomain of CASY-1 is released into the body cavity and then taken up by scavenger cells. Similar cleavages are reported for Calsyntenins. Moreover, we determined the molecular region that is important for learning. We will discuss the relationship between the proteolytic cleavage of CASY-1 and the regulation of learning.

## P2-h14 Memory recall of object-place associations in a hierarchical network self-organized by theta phase coding

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Object-place associative memory is known to be managed by the hippocampus, while its neural mechanism is an open question. The authors have proposed that theta phase coding in the hippocampus enables an associative memory network formation for object-scene associations in one time experience. Interestingly in their computer simulation, the resultant memory network represents hierarchical relations of spatial scales by asymmetric connections from scene units (large) to object units (small). However, the functional role of the hierarchical structure remains unclear. In this paper, we elucidated memory recall dynamics in the hierarchical network. Our analyses demonstrated that the recall is efficiently controlled by scene units with various spatial scales: Through large-to-small downstream propagation of the activities, relevant object-place association pairs are sequentially recalled. Our results indicate that episodic memory recall is controlled by hierarchical information flow in the hippocampal network.

## P2-h15 The leftward structural asymmetry in the inferior frontal gyrus as an indicator of acquiring syntax in second language: A VBM study

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Using MRI, a recent voxel-based morphometry (VBM) study reported the vocabulary-related neuroplasticity of the grey matter (GM) in bilingual brains, but the structural basis of the individual variability for acquiring syntax in a second language and the associated structural asymmetry remain unknown. In the present study with VBM, we calculated voxel-by-voxel asymmetry indices [AIs; 2×(L-R)/(L+R) in the left hemisphere] for the GM volume. To evaluate the acquisition of syntax for adolescents aged 13 to 17 (n = 78), we examined their performances of syntactic (Syn) and spelling (Spe) tasks on English sentences. In the left triangular part of the inferior frontal gyrus (F3t), the AIs were positively correlated with the Syn scores, whereas there was no significant effect for the Spe scores. These results suggest that the individual variation in the syntactic ability is associated with the magnitude of the leftward structural asymmetry in the F3t.

CREST, JST

## P2-h16 Molecular mechanisms of decision making in Caenorhabditis elegans

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The sensory neurons of *C. elegans* respond to a variety of environmental signals such as volatile and water soluble chemicals, temperature, etc. Thermotaxis is a well characterised behaviour in which after cultivation with food (*Escherichia coli*) at a certain temperature, the worms are able to associate it with the presence or absence of food. This is therefore an ideal scenario to investigate the molecular basis of sensory integration that leads to complex thoughts such as memory or learning.We are interested in how the assimilation of these sensory cues leads to memory, and how the memory can be modified by the appearance of a novel input which might cause a new decision to arise. To approach this issue we are currently looking for mutants that, once they have been conditioned to find *E. coli* at a certain temperature, do not, or take longer on leaving this temperature than wild type animals even after an unsuccessful search for food. Besides assaying some known mutants for this phenotype we have started performing a screening. After EMS mutagenesis, we isolated several putative mutants presenting the desired phenotype. These mutants are currently under characterisation.

MEC

## P2-h17 Correlation analysis of scalp EEG responses to spoken sentences modulated by M-sequence

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We have developed a new technique for assessment of verbal comprehension (Takeichi et al., 2007). EEGs were recorded from Japanese speakers while they were listening to a continuous speech sound. We generated an eleventh order m-sequence, an array of 2047 binary numbers: each *zero* was replaced with a 23 ms-long off (blank) window and each *one* was replaced with a 23 ms-long on window to modulate the sound in amplitude. The derivative of the m-sequence was generated by replacing the first *one* in a train of *ones* with *1* and the other *ones* with *0*, and the first *zero* in a train of *zeroes* with *-1* and the other *zeroes* with *0*. A circular cross correlation function between the EEGs and the derivative was analyzed using independent component analysis. In the present study, the EEGs were additionally correlated with a gap-end-marker, *1* at each speech resumption and *0* elsewhere, and a gap-start-marker, *1* at each speech cessation and *0* elsewhere. As a result, the peaks observed in the original correlation function were also observed when the gap-start-marker was used.

JST to SK

## P2-h18 Neural correlates of emotional voice recognition: an event-related functional magnetic resonance imaging study

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Understanding other's emotional states is essential for human communication. We know other's emotional states not only by their faces but also by the prosody of their voices. In the present study, an event-related functional magnetic resonance imaging (fMRI) was used to investigate the neural correlates involved in the processing of the emotional prosody. Thirteen subjects listened to semantically-neutral Japanese words (e.g., /nami/ and /arayuru/) with different emotional tones of voices (angry, glad, and neutral) and judged the prosody type in the three-forced choice task. We found different activations among the emotional tones (angry > neutral > glad). The activation sites were the medial prefrontal cortex (MPFC), the cingulate gyrus and the precuneus. Our findings suggest that a network centered on the MPFC is recruited when we understand other's emotional states, which is consistent with the previous fMRI studies using visual emotional stimuli (e.g., Ochsner et al., 2004).

## P2-h19 Study of long-term potentiation (LTP) in the song bird brain regions essential for vocal learning using multi-electrode dish (MED)

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Vocal learning in song birds provides a good model for human vocal learning, a well-defined behavior and discrete neural structure.The bird brain regions underlying vocal learning and production have been determined. As one neural mechanism mediating vocal learning, long- term potentiation (LTP) at the LMAN and area X in zebra finch forebrain has been demonstrated. The caudal medial nidopallium (NCM) in an area of the auditory hindbrain is important for a long lasting memory of tutor song. In order to study LTP in NCM, we applied multi-electrode dish ( MED ) system to acute brain slices of song birds (budgerigar, zebra finch, Bengalese finch). Brain slices, 350-400 μm thick, were prepared by a vibrating slicer. Bipolar constant current pulses (20-50 μA, 0.1-0.12 ms) were delivered on the brain slice. Evoked field potentials at eight sites in the NCM were recorded simultaneously by the MED system. LTP in budgerigar brain slices was induced by 100 Hz electrical stimulation (tetanus). We are trying to induce late LTP in NCM, LMAN and the area X.

## P2-h20 Behaveoral responses to frequency modulated sounds in Mongolian gerbils: upward FM vs. downward FM

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It is known that Mongolian gerbils (*Meriones unguicultus*) have communication calls that are mainly frequency modulated (FM) sounds. They vocalize downward FM (dFM) sounds more often than upward FM (uFM) sounds in low frequency range (<16kHz). We considered that gerbils could perceive small differences between the dFM rates, since they use different types of dFM sounds in daily life. We compared behavioral responses to dFM sounds with those to uFM sounds by using GO/NOGO discrimination tasks. Gerbils were trained to discriminate FM sounds (dFM sound and uFM sound) from constant frequency (CF) sounds. After training was completed, test stimuli with different FM rates were presented to gerbils. As a result, the go response decreased as the FM rate decreased in the cace of dFM sounds. However, gerbils did not appear to discriminate differences within uFM sounds.

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## P2-h21 Phonological and lexico-semantic processing during kanji word reading: An MEG study

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We measured neural activities including the effect of phonological information on the lexico-semantic process during silent reading of kanji homophone words by using MEG. Subjects were presented prime-target word pairs that consisted of two kanji characters. The primes were phonologically same as or different from the following target words, or pseudo-characters. The neural activation differed between conditions in the left posterior superior temporal and inferior parietal areas, and became weaker by the phonological repetition. Furthermore, activity was larger for different condition in the anterior temporal lobe, and for same condition in Broca's area, as compared with the other two conditions at 400-500 ms. Our results suggest that the phonological information influences the lexico-semantic process even under the strong constraint of orthographic information.

## P2-h22 Two distinct neural networks for semantic access during visual word recognition

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To investigate how Japanese efficiently access the meanings of written words in the brain, this study employed a two-by-two factorial design that manipulates both word types (Kanji vs. Kana) and task types (semantic vs. phonological). Two main findings emerged. First, differential semantic - phonological activation was significantly greater for Kanji than Kana in the left inferior temporal gyrus, left parahippocampal gyrus, left hippocampus, left caudate and right anterior fusiform gyrus. This finding suggests that these areas constitute the direct pathway from orthography to semantic system. Second, differential semantic - phonological activation was greater for Kana than Kanji in the left inferior occipital gyrus. This indicates that these areas are involved in reading through phonological mediation prior to accessing the semantic system.

## P2-h23 IFG activity in observing music performance

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Broca's area is involved in processing of hierarchical sensory and motor structures, and is activated when subjects are observing practiced actions. The current study was conducted on the hypothesis that the response of this region while viewing a structured action, such as music performance, would be influenced by the experience of observers. To this end, healthy right-handed adult participants were exposed to hand movements playing either "Twinkle, twinkle, little star" or "Mary had a little lamb" that was paired with its piano sounds either in a congruent or in an incongruent fashion. The subjects were asked to determine which music the hand was performing independent of the heard sounds. Cortical activity was measured using a near-infrared spectroscopy. Consequently, Broca's area of the expert subjects showed higher activity in the incongruent condition requiring a higher processing load although all the subjects answered the song names correctly. Thus, Broca's area is assumed to be involved in transforming a sequence of discrete actions into unified meaningful performance.

21c COE (A14), JST, Nakayama Fundation for Human Science

## P2-h24 Early GABA function regulates sensory critical period during birdsong learning.

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Neural circuits are shaped by experience during restricted critical periods in early life. In mouse visual cortex, this timing is triggered by the maturation of inhibitory function. Here, we tested whether the principle also applies to another widely studied system, birdsong learning. Male zebra finches were injected into the ventricle with the positive $GABA_A$ receptor modulator diazepam (DZ) or vehicle control for 4 consecutive days before the critical period (~20 days post-hatch). Birds in which inhibitory function was briefly accelerated suffered long-lasting learning deficits (>120 days) across several parameters: delayed singing onset, reduced song consistency or similarity to the tutors' songs. Rate of motor learning was not affected. Premature closure of the sensory acquisition phase was instead implicated using a sequential, dual-tutoring paradigm, as well as by neuroanatomical consequences of early DZ treatment. Taken together, inhibitory circuits may generally influence the timing of developmental critical periods.

CREST

## P2-h25 Flexible word order in Japanese and its effects on frontal activation: A NIRS study

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Japanese has flexible word order. In order to determine the basic word order in Japanese, oxy-hemoglobin concentration (oxyHb) changes in the frontal region were monitored by a 52-channel near-infrared spectroscopy (NIRS) in 32 right-handed healthy males during Subject (*whyS/Swhy*) and Object (*SOV/OSV*) Scrambling Conditions. The results have shown that while oxyHb changes in the sentences with Object Scrambling Condition (*OSV*) were significantly larger than that in the non-scrambled sentences (*SOV*) in the left frontal lobe, such effects cannot be observed in the case of Subject Scrambling Condition. These findings suggest that while *OSV* is derived from the basic order of *SOV*, there is no such derivational relationship between *whyS* and *Swhy*: Both of these constitute the basic order.

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## P2-h26 Neural mechanisms of the lexically ambiguity resolution - An MEG study

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We measured neuromagnetic signals during the semantic judgment of lexically ambiguous or disambiguous words following semantically related or unrelated words to clarify the neural mechanisms of the lexically ambiguity resolution. The activities in the left anterior inferior frontal cortex were larger for ambiguous words than for disambiguous ones at approximately 200 to 300 ms, suggesting that multiple meanings for ambiguous words are accessed independent of context. At around 400 ms, the activities in the left posterior inferior frontal cortex showed a clear context effect (unrelated > related) for disambiguous words but not for ambiguous ones, and were stronger for related ambiguous words than for related disambiguous ones. The results suggest that the left posterior inferior frontal cortex plays an important role in selecting an appropriate meaning as the contextual integration.

## P2-h27 Changes of the brain activity related to the differences in manner of articulation.

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Differences between consonants of stop and fricative were known from perspectives of language development and acquired language disorder. However, neural mechanisms underlying these were unclear. We measured brain activity for speech motor control for stop and fricative consonants using [t] and [s], respectively, by functional MRI. The results showed significantly higher activation in the supplementary motor area, bilateral operculum extended into insula and superior temporal gyrus, bilateral supramarginal gyrus and the bilateral cerebellum during speech of [s] compared with [t]. These brain regions may have important role for production of different speech sounds even using with the same articulator.

## P2-h28 Reanalysis in Japanese sentence comprehension: An fMRI study

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The aim of this study was to specify brain regions associated with reanalysis of Japanese sentences. In this experiment, four different types of sentences that is, coordination (normal), object relative (syntactically complex), reanalysis and filler sentences were visually presented to subjects and measured brain activation using fMRI. For each condition versus null condition, similar regions were activated. But, direct comparison between reanalysis versus object relative analysis showed significant activation in the medial aspect of left superior frontal gyrus and right intra parietal sulcus. Therefore, we demonstrated that there are neural substrates, medial aspect of left superior frontal gyrus and right intra parietal sulcus, specifically associated with reanalysis aside from syntactically complex sentence.

JST/RISTEX, JST/CREST, the 21st COE Program (Tohoku University), and KAKENHI (17652039)

## P2-h29 Difference in neural distributions between face-to-face and remote situations in a communicative game

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In order to understand the difference between face-to-face and remote communication, brain activation were compared using fMRI. Participants (n=11) were instructed to play the rock-scissors-paper game against an opponent in a scanner room in face-to-face situation and against another opponent in an operation room via a video system in remote situation. The fMRI observation revealed that the several brain regions including the right anterior cingulate cortex (ACC;BA10), the left superior occipital gyrus (BA31) and the bilateral inferior temporal gyrus (BA37), were activated during the game. In addition, the right ACC (BA10) and the bilateral occipital (BA37) areas showed larger activity in the face-to-face situation. The activation observed at ACC is known to relate to mentalizing process and the result suggests that the mentalizing process works stronger in the face-to-face situation.

## P2-h30 A multilayer SOM for category specific semantic memory impairments

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An implementation of category-specific impairments of object recognition and identification was built to explain the nature of semantic memory and its disorders. Neuropsychological evidence suggests that semantic memory in the brain may be organized along with categories such as animals, tools, fruits, etc. On the other hand, brain imaging studies do not always agree on the exact areas dealing with each category among patients. This may imply that the cortical map of meanings could emerge as a result of learning through a self-organization algorithm. Several neural network models have been proposed. But none of these models have proposed explanations for the arbitrariness of the location of each meaning and the category-specificity of semantic memory simultaneously. It is apparent that all the data cannot be accounted for in terms of a single information processing disorder for all patients. Therefore, a multilayer neural network architecture was introduced to describe different patients with different brain damage loci. The proposed model would be plausible when we consider consistency among neuropsychology, neural networking, and brain-imaging studies.

## P2-h31 A longitudinal fMRI study of neural plasticity in the second language lexical processing

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The present fMRI study investigated whether brain activities for their second language (L2) lexical processing changes when proficiency level of L2 learners increases. Firstly, we measured brain activities for L2 lexical decision task by six non-fluent Chinese learners of Japanese. After three months, we measured those by the same subjects using the same paradigm. Imaging data were analyzed using SPM2. While the second measurement showed greater activation in the left inferior frontal gyrus, but less activation in the left intra-parietal sulcus, insula, and the right cerebellum than the first measurement. These results indicate that when proficiency level of L2 learners increases, cortical representation for the lexical processing changes.

JST/RISTEX, JST/CREST, 21st COE Program

## P2-h32 Role of receptor for advanced glycation end products in neuronal uptake of amyloid β-peptide

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Alzheimer's disease (AD) is a progressive neurodegenerative disease that is characterized by senile plaques, in which amyloid β-peptide (Aβ) is a major constituent. We have recently found that intracellular Aβ causes mitochondrial dysfunction leading to neuronal apoptosis. However, the mechanism of membrane Aβ transport system is unclear. It has also been reported that receptor for advanced glycation end products (RAGE), a multiligand receptor of the immunoglobulin superfamily, is associated with Aβ transport in blood-brain barrier. In this study, we examined the role of RAGE in Aβ uptake in neurons. Treatment of an siRNA for RAGE or a RAGE antibody decreased the Aβ uptake and Aβ-mediated mitochondrial dysfunction in mouse neuron cultures. These data indicated that RAGE is involved in Aβ transport into cytosol and RAGE may be a new drug target of AD therapy.

## P2-h33 Dramatic decrease in new neurons in the dentate gyrus of learning attenuated aged monkeys

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Aging induces several cognitive impairments without any neurological disorders. However, pathogenic mechanisms of these cognitive impairments with aging remain unclear. In this study, we report learning deficits using visual pattern discrimination task, and several neurological differences with aged *Macaca fascicularis*. We counted the number of the cells, which express NeuroD (neuronal determination factor) and Doublecortin (migrating neuronal factor) with immunofluorescent staining. The number of NeuroD/ Doublecortin positive cells significantly decreases in dentate gyrus of aged monkeys compared with young monkeys. This indicates some type of immature neuron dominantly descends in dentate gyrus with aging. This dramatically decreasing of new neurons in aged monkeys may effects attenuation of learning performance with aging.

KAKENHI

## P2-h34 The role of C-terminus of amyloid precursor protein under endoplasmic reticulum stress

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Endoplasmic reticulum (ER) stress has been reported to be linked to neuronal death in various neurodegenerative diseases including Alzheimer's disease. Although Alzheimer's disease is characterized by the appearance of senile plaques which are composed of amyloid β peptide derived from amyloid precursor protein (APP), little is known about the relationship between APP and ER stress. We therefore investigated the role of APP under ER stress. When HEK293 cells were treated with tunicamycin to induce ER stress, the expression levels of APP mRNA and C83 protein, which is C-terminus of APP cleaved by α-secretase, were remarkably increased, and cell death was observed. When HEK293 cells were transfected with APP cDNA, cell death was observed whereas no cell death was observed in mock-transfected HEK293 cells. This cell death was attenuated by the treatment of TAPI1, α-secretase inhibitor, or DAPI, γ-secretase inhibitor. These results suggest the possibility that C-terminus of APP causes cell death under ER stress.

## P2-h35 Isoform dependent effect of apolipoprotein E on cholesterol efflux

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Apolipoprotein E (ApoE) is a major cholesterol (CH) transporter in the brain. We previously reported that the level of CH efflux induced by ApoE4 is lower than that by ApoE3, which is explained partially by domain interaction, and that the ability of 22-kDa fragment of ApoE (22-kDa-ApoE) to release CH is also ApoE isoform dependent. However, the molecular mechanism remained unknown. Here we report that 22-kDa-ApoE3 can form dimers by disulfide binding but 22-kDa-ApoE4 does not, because ApoE4 has no cysteine, and that 22-kDa-ApoE3 dimers induces greater CH efflux than monomeric 22-kDa-ApoE3 or 22-kDa-ApoE4. This is also that case for intact ApoE3 and ApoE4. The CH efflux induced by ApoE3 is mediated by ABCA1 protein. These findings suggest that cholesterol efflux induced by ApoE is regulated by two major factors: the presence or absence of inter-molecular dimer formation and the intra-molecular domain interaction.

NIBIO 05-27

## P2-h36 PI3K/AKT/GSK3β pathway regulates the expression of gamma secretase complex.

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Background: Presenilin 1(PS1) activates phosphatidylinositol 3 kinase(PI3K). Glycogen synthase kinase 3β (GSK3β), downstream of PI3K, phosphorylates PS1. Objective: The effect of phophorylation of PS1 on the metabolism of the entire γ-secreatase complex(GSC) was studied. Methods: The expression levels of the GSC component proteins on PI3K, AKT or GSK3β inhibiton were examined. The mRNA expression was also examined. The proteolytic system for GSC was determined using the proteasome or autophagy inhibitors. The ubiquitination of the PS1-containing immune complex was examined by immunoprecipitation. Results: Both PI3K and AKT inhibitors increased the expression of the GSC components. GSK3β inhibitors cancelled such increase. PI3K inhibition caused the PS1-containing immune complexs more multi-monoubiquitinated than polyubiquitinated. Conclusion: Inhibition of PI3K/AKT pathway leads to the GSC monoubiquitination and stabilization to proteasomal degradation.

## P2-h37 The role of microglia on the neurotoxicity of Aβ

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**Objective**: To study the difference of neurotoxicity induced by soluble or fibrillar amyloid beta (Aβ) and to clarify the role of microglia in Aβ-induced neurotoxicity.

**Background**: Aβ42, the main component of senile plaques, induces neurotoxicity. The precise role of microglia against Aβ-induced neurotoxicity is unclear.

**Methods**: Neuronal cell cultures were prepared from C57BL/6 mice. Neuronal damage by soluble or fibrillar Aβ was estimated by cell counting kit and cytoskeletal changes. The role of microglia was examined in neuron-microglial co-culture, which was damaged by soluble or fibrillar Aβ.

**Results**: Soluble Aβ showed more striking neurotoxicity than fibrillar Aβ. Unstimulated microglia did not show neuroprotective effect. However, activated microglia by toll like receptor ligands, peptidoglycan, lipopolysaccharide, or CpG DNA showed neuroprotection against Aβ toxicity.

**Conclusion**: Soluble Aβ is considered to involve in the pathogenesis of Alzheimer disease. Activated microglia may play a neuroprotective role in Aβ toxicity through toll like receptor signal.

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## P2-h38 Causative candidates for the dementing diseases analysed by SJLB mice

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Mutations in the tau gene were found causative for autosomal dominantly inherited frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17). SJLB mice, expressing human tau with N279K mutation show a behavioral phenotype mimicking features of human FTDP-17 and decreased in vivo LTP in hippocampus. In this study, we compared the proteome profilings of hippocampus comparing between SJLB mice and control non transgenic mice (Control mice). Several proteins including clathrins and neuron specific enolase showed different expression patterns. Semi-quantitative RT-PCR also showed different mRNA expressions. We also analyzed them by histochemistry. Though the differences in the proteins and mRNAs between SJLB and Control mice were slight, it assumed that they may be causative for the dementing disease.

KAKENHI 18500242

## P2-h39 DAPT and L-685,458, γ-secretase inhibitors perturb Wnt signaling

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To inhibit A $\beta$ production is a major therapeutic target for Alzheimer's disease. γ-Secretase inhibitors inhibit intramembrane proteolysis producing A $\beta$. However, γ-secretase blocking activity cause serious side-effects, since they usually inhibit sequential endoproteolysis of Notch receptors. Wnt, as well as Notch, is a crucial signaling pathway for differentiation. Both Notch and Wnt signaling cooperatively regulate differentiation of squamous cells in small intestine. Moreover, γ-inhibitor is known to affect the differentiation. Therefore, we asked direct effect of γ-inhibitor on Wnt signaling.

Wnt signaling was assessed by modified luciferase reporter genes. Surprisingly, DAPT and L-685,458 significantly reduced Wnt signaling in cultured cells. However, dominant negative form of presenilin did not inhibit Wnt signaling. These results suggest that the inhibitions of Wnt signaling were independent of γ-secretase activity of presenilin.

## P2-h40 A new membrane microdomain-dependent regulatory mechanism of amyloid precursor protein processing

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Cholesterol-dependent cleavage of amyloid precursor protein (APP) by BACE1 in lipid rafts is thought to be a critical regulatory event in generating amyloid-beta peptide, which plays a key role in the pathogenesis of Alzheimer's disease. To investigate the underlying molecular mechanisms, we analyzed APP-containing detergent-resistant membranes from mouse brains. APP was incorporated into cholesterol-dependent lipid-protein complexes linked through a network of protein interactions, which excluded BACE1. In copatching experiments, APP was preferentially associated with microdomains marked by prion protein and not BACE1-containing microdomains. Protein interactions were necessary for association with the prion-containing microdomains and mediated inhibitory effects on APP-BACE1 interaction and beta-cleavage. Thus we propose a novel regulatory mechanism of APP processing through differential association with distinct microdomains controlled by protein interactions.

KAKENHI 18500283

## P2-h41 Morphological analysis of human tau transgenic mice showing age-dependent learning disorders

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In this study, to understand the effects of human tau on phosphorylation and neurodegeneration, we morphologically examined transgenic (Tg) mice overexpressing wild-type or P301L mutant human tau under control of alpha CaMKII promoter, which showed the age-dependent impairment in spatial learning and context learning, respectively. Both wild-type and P301L tau Tg mice at 12 and 24 months of age expressed transgenes at similar levels. At 24 months of age, PHF-1 phosphorylation of tau was highly accumulated without neurofibrilarry tangles or neuronal death in the perirhinal and entorhinal cortices of wild-type tau Tg mice, whereas neuronal loss and astrogliosis was prominent in these regions of P301L tau Tg mice. These results suggest that different forms of human tau affect neurons in the distinct manner, leading to learning disorders in these Tg mice.

KAKENHI (18700353)

## P2-h42 White matter lesions in the brain with amyotrophic lateral sclerosis with dementia: TDP-43 immunopositive inclusion is not co-localized with ubiquitin but p62

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Amyotrophic lateral sclerosis (ALS), is sometimes accompanied by cognitive decline, which is referred to as ALS-D. In this study, we showed TDP-43 immunopositive glial and threads-like inclusions throughout cerebral white matter except of occipital lobe in ALS-D. These inclusions were also immunoreactive with p62 but not with ubiquitin (ub). The numbers of TDP-43 immunopositive inclusions were higher than those of p62 immunopositive inclusions. We conclude that white matter degeneration, characterized by TDP-43 and p62 aggregations without ub, which is differs in the neuronal degeneration characterized by ub. Our results also indicated that pathological change might take place in the white matter as well as in the gray matter in the brain with ALS-D.

## P2-h43 The linoleic acid derivative DCP-LA ameliorates impairment of learning and memory

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The newly synthesized linoleic acid derivative DCP-LA potentiated presynaptic $\alpha$7 nicotinic ACh receptor responses by directly and selectively activating PKC-ε, thereby stimulating glutamate release from presynaptic terminals in the hippocampus, and then leading to an LTP-like long-lasting hippocampal synaptic transmission. Those DCP-LA effects were proved with both the in vitro and in vivo systems. DCP-LA (1mg/kg, i.p.) significantly improved spatial learning and memory impairment in rats with continuous intraventricular injection with amyloid-$\beta_{1\text{-}40}$ peptide in the lateral ventricle. Furthermore, DCP-LA (1 mg/kg, i.p.) significantly ameliorated memory deficit in rats treated with scopolamine, with the advantage similar to or over galantamine, a modest inhibitor of acetylcholinesterase with nicotinic ACh receptor modulation. DCP-LA (1 mg/kg, i.p.) also improved memory disorders in senescence accelerated mice-prone series (SAMP8). DCP-LA, thus, could be developed as a promising anti-dementia drug.

## P2-h44 GABA-related acceleration of aging process in Alzheimer's model mouse

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AD is characterized by accelerated memory loss in aging. GABA can suppress synaptic plasticity essential for memory. To test if that might explain memory impairment in PSAPP mouse model of AD, we estimated effect of GABA on LTP by using GABA receptor antagonist. Large GABA effect observed in 'middle aged' PSAPP resulted in low LTP and memory impairment by MWM. In contrast, GABA effect was reciprocally diminished at 'old' age of PSAPP. Consistent with its high LTP, 'old' PSAPP seemed to remember about repeated experience even better than 'old' nonTG, which showed large GABA effect. But, 'old' PSAPP mouse simultaneously showed less accurate learning and lack of memory flexibility. Large GABA effect in 'old' nonTG mouse is consistent with increasing GABA activity with aging in human brain. Large GABA effect in 'middle aged' PSAPP in contrast to such effect in 'old' nonTG indicates that acceleration of aging process in AD may be related to changing GABA activity.

## P2-h45 HRD1 involved in ER-associated degradation (ERAD) suppresses amyloid precursor protein-induced apoptosis

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Endoplasmic reticulum-associated degradation (ERAD) is retrograde transport of proteins from the ER to the cytosol and subsequent degradation of the proteins by the ubiquitin-proteasome system. We previously identified HRD1, a human homolog of *S. cerevisiae* Hrd1p, which is an ER membrane protein that acts as a ubiquitin ligase (E3) in ERAD and protects against ER stress-induced apoptosis. We showed that HRD1 promoted degradation of overexpressed amyloid precursor protein (APP) depending on its ubiquitin ligase activity. Overexpression of APP in SH-SY5Y cells showed the accumulation of APP that forms aggresome-like structures. In this situation, suppression of HRD1 by RNAi induced disappearance of the aggresome and apoptosis with caspase-3 activation. These results suggest that HRD1 protects against APP-induced apoptosis by ERAD-mediated APP elimination from the ER.

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## P2-h46 A natural scavenger of peroxynitrites, rosmarinic acid, protects against impairment of memory induced by Abeta (25-35)

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Peroxynitrite ($ONOO^-$) - mediated damage is regarded to be responsible for the cognitive dysfunction induced by amyloid beta protein (Abeta) in Alzheimer disease (AD). In the present study, we examined the protective effects of rosmarinic acid (RA), a natural scavenger of $ONOO^-$, on the memory impairment in a mouse model induced by acute *i.c.v.* injection of Abeta (25-35). Mice daily received RA (*i.p.*) after the injection of Abeta (25-35). RA prevented Abeta (25-35)-induced nitration of proteins, an indirect indicator of $ONOO^-$ damage, in the hippocampus. RA prevented the memory impairments induced by Abeta (25-35) in the Y maze test and novel object recognition task. RA also prevented nitration of proteins in the hippocampus and impairment of recognition memory induced by $ONOO^-$ *-i.c.v.* -injection. These results demonstrated that daily consumption of RA may protect against memory impairments associated with Abeta in AD.

## P2-h47 Protective effect of pyruvate on survival of hippocampal slice culture and against beta-amyroid toxicity.

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[Background and Purpose] There is still controversial on the role of monocarboxylate (MCT) on brain function. Pyruvate is known to be protective against ischemic cell toxicity and beta-amyloid cell toxicity using primary cell culture system. We studied the effects of two MCTs, lactate and pyruvate, on the survival of hippocampal slice culture, and also their protective effects against beta-amyloid cell toxicity. [Method] Rat hippocampal slice culture was maintained 14 days in-vitro, and propidium iodide was used for evaluation of cell death after 24 and 48 hours after each treatment. [Results and Conclusion] MCT alone could not maintain slice culture after glucose deprivation. Pyruvate showed the similar protective effects on cell survival and beta-amyloid cell toxicity as vitamin E analogue and nicotinamide. Lactate did not show protective effects. NAD levels in the slices were preserved in the presence of pyruvate and glucose. The preservation of NAD levels as well as anti-oxidation is suggested as protective mechanism of pyruvate.

## P2-h48 Analysis of the mechanism of the physical interaction between reticulon 3 and BACE1

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$\beta$-secretase BACE1 is a membrane-bound aspartyl protease directly involved in the generation of $\beta$-amyloid protein (A$\beta$). BACE1 is one of the important therapeutic targets for Alzheimer's disease. Recently, we identified reticulon 3 (RTN3) and reticulon 4-B/C (RTN4-B/C or Nogo-B/C) as membrane proteins that interact with BACE1 and inhibit its ability to produce A$\beta$. In the present study, we investigated the mechanism of the interaction between reticulons and BACE1. RTN3 constructs with N-terminal truncation, C-terminal truncation or short deletions in the loop region were generated and used for the analysis. C. elegans RTN (ce RTN) was also included in the analysis. We found that these RTN3 mutants and ce RTN interacted with BACE1 as well as wild-type RTN3. Further, the RTN3 mutants as well as ce RTN retained ability to inhibit A$\beta$ secretion. These data suggest that the C- and N-terminal regions are not critical and the two transmembrane regions in the RTN homology domain are essential for the interaction with BACE1.

## P2-j01 Small-angle neutron scattering (SANS) and dynamic light scattering (DLS) analyses of the initial aggregation process of tau by oxidative stress

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We illustrated the conformational change of tau in the initial aggregation caused by oxidative stress by SANS and DLS. The hydrodynamic radius, Rh, for 4-repeat tau monomer (MW. 47-KDa, including two cysteine residues) and 3-repeat tau tau monomer (MW. 43-KDa, one cysteine) were 3.8 nm and 5.7 nm. The self-assembled cluster, Rh, was 60.6 nm (a long and thin rotating ellipsoid) for 4-repeat tau and 63.7 nm (random coils) for 3-repeat tau. The cluster size of 3-repeat tau increased to 120 nm by oxidative stress. Tau is stabilized by forming a huge self-assembled cluster, however, once intermolecular disulfide bonds are induced among the molecules, irreversible aggregation begins especially on 3-repeat tau.

## P2-j02 MPC, a newly synthesized compound, improves learning and memory deficits of neurodegenerative animal models and protects SH-SY5Y cells from neurotoxicities

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MPC was newly synthesized by the esterification of maltol and p-coumaric acid. Both structures exhibited antioxidant activities known as a therapeutic strategy of healing many neurodegenerative diseases, especially Alzheimer disease, characterized by serious cognitive deficits as a consequence of selective neuronal death. MPC significantly ameliorated the learning and memory impairment of scopolamine-induced rats, AD model rats induced by the Abeta peptide infusion into the lateral ventricle, and SAMP8 mice, using a passive avoidance and a water maze test. As a result, we infer that MPC can enhance memory and cognitive function in neurodegenerative animal models possibly by increasing ChAT activity and ACh contents. These activities of cognitive enhancement and neuroprotection of MPC may be effective therapeutic value in AD that is characterized by cholinergic neurodegeneration and progressive deterioration of learning and memory processes.

## P2-j03 Characterization of the processing of α-dystroglycan

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In several forms of congenital muscular dystrophy, glycosylation of α-dystroglycan (α-DG) is defective and its laminin binding activity is severely decreased. Recently, it was reported that α-DG is cleaved by protein convertase, furin, however, its implication in the physiological function of α-DG or the pathological disease processes remains to be determined. Here, we generated a set of antibodies against α-DG and demonstrated that N-terminal domain of α-DG is cleaved by furin and secreted into culture medium of C2C12 myotubes. The N-terminal domain was also detected in the medium of PC 12, RT4, COS, HEK293 or Hela cells. These results indicate that the cleavage of the N-terminal domain of α-DG is a fundamental event in wide variety of cell types or tissues. Currently, we are testing the tissue distribution and localization of the N-terminal domain *in vivo*.

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## P2-j04 Sept9 sequence alterations causing HNA are associated with altered interactions with Sept4/11 and resistance to Rho/Rhotekin signaling

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Sequence alterations in Sept9 are known to cause hereditary neuralgic amyotrophy (HNA) but precise cellular consequences have yet to be determined. Since Sept9 is thought to function through interaction with other septins and small GTPase Rho-mediated signaling, we analyzed the properties of HNA-assosiated Sept9 missense variants, Sept9F and Sept9W. We found both sequence variants, but not the wild-type, to form filaments with Sept4 along stress fibers in mesenchymal mouse mammary gland NmuMG cells. In the epithelial cells, the variants, but not the wild-type, were co-localized with Sept11 at cell-cell junctions. In addition, although septin filaments containing Sept9_v3 were disrupted by Rho/Rhotekin signaling, this was not the case with Sept9F and Sept9W. Sequence variations in Sept9 causing HNA are thus likely to alter modes of interaction with partner molecules in cells, and consequently contribute to the pathogenesis of HNA.

## P2-j05 Effects of low power laser irradiation on muscle contraction of the frog gastrocnemius

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Since laser irradiation could block the generation of anode-break-excitation in frog sciatic nerve, we examined effects of laser irradiation on contraction of the gastrocnemius. Nineteen frogs (Xenopus laevis) were used and their gastrocnemius muscle with sciatic nerve was prepared. Muscle tension continuously induced by a supramaximal stimulus to the sciatic nerve at 1/s for 12 min gradually attenuated. Direct irradiation of laser (Nd:$YVO_4$, 532 nm, 180 mW) to the muscle surface (28.3 $mm^2$) during the stimulating significantly retarded its attenuation ($p<0.01$). Half time defined as duration between maximum and half amplitude in tension showed a declining tendency with increasing of frog weight. Laser irradiation made its slope more gradually. When the rest period for 2 min was set between stimulating sessions and the laser irradiation was applied during the rest period, the attenuation was also retarded by laser irradiation ($p<0.05$). It is suggested that laser irradiation influences transmission at neuromuscular junction and excitation-contraction coupling.

MEXT HAITEKU (2004-08)

## P2-j06 Nitric oxide production results in disuse-induced muscle atrophy through dislocation of neuronal nitric oxide synthase

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To elucidate the mechanism of muscle atrophy, we analyzed the expression of dystrophin glycoprotein complex (DGC) members during tail suspension, a model of unloading-induced muscle atrophy. Neuronal nitric oxide synthase (nNOS) quickly disappeared from the sarcolemma to the cytoplasm and produce NO during tail suspension. nNOS-null mice showed much milder muscle atrophy than wild-type mice. Nuclear accumulation of de-phosphorylated Foxo3a was not evident in nNOS-null muscle, together with the lack of up-regulation of the muscle-specific E3-ubiquitin-ligases. We conclude that nNOS/NO mediates muscle atrophy via regulation of Foxo transcription factors.

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## P2-j07 PCP-induced abnormal gene expression pattern in NMDARε4 KO mice

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Phencyclidine (PCP) is a noncompetitive blocker of the N-methyl-D-aspartate receptor (NMDAR). To investigate effect of PCP on one of NMDAR subunits, ε4, we performed cDNA array analysis using mRNA derived from pooled striata of wild- or NMDARε4 knockout (KO) mice 2 h after injection of saline or PCP (10 mg/kg, s.c.). We observed that reduced expressions of several genes in saline-injected KO mice were well-consisting with those in PCP-injected wild mice. By contrast, gene expression pattern in PCP-injected KO mice was completely different from that in PCP-injected wild mice. Therefore, the observed abnormal gene expression pattern in NMDARε4 KO mice might be due to NMDA receptor dysfunction. These results suggest that NMDARε4 may play on important role for PCP effect via NMDAR.

## P2-j08 Role of the zinc-finger transcriptional factor Gli3 in determining striatal structure and striatal-based behavior

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The striatum is the largest component of the basal ganglia circuit and plays important roles in motor, cognitive, and emotional functions. The lateral and medial ganglionic eminence is a major source of striatal cells during development. Gli3 is a repressor of vertebrate Hedgehog signaling and is critical in determining the ventral-dorsal patterning of the telencephalon. In the homozygous embryos of Extra-toes mice, which carry a deletion in the Gli3 gene, the ventral telencephalon is expanded. We hypothesized that Gli3 may have an important role in the development of the striatum. We found abnormal striatal structure (increased volume and reduced number of cholinergic interneurons) and abnormal striatal-based behavior (enhanced locomotor response, sensitization and place preference to methamphetamine) in adult heterozygous Extra-toes mouse. These results indicate that Gli3 plays an important role in the development of the striatum and sheds light on the relationship between the morphology of striatum and sensitivity to drugs.

## P2-j09 Frontal lobe function in eating disorder:a multichannel near-infrared spectroscopy study

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Background: Functional neuroimaging has been employed for eating disorder (ED) only in several studies. Completely noninvasive nature and natural setting for measurement of near-infrared spectroscopy (NIRS) is suitable for neuroimaging analyses of ED pathophysiology. Materials and Method: Oxygenated hemoglobin concentration ([oxy-Hb]) changes were monitored during a verbal fluency task using a multichannel NIRS (Hitachi Medical, ETG-4000) in 18 ED patients, and their correlations with clinical symptoms assessed with the Eating Attitude Scale (EAT-26) were examined.Results: [oxy-Hb] increases were smaller in the ED group, especially in right fronto-temporal areas, and negatively correlated with dieting tendency scores of the EAT-26 in right fronto-temporal areas and with restriction of eating and binge eating scores in left frontal areas, but not with body mass index. Conclusion: Pathophysiology of ED is considered to consist of dieting tendency and eating behavior problems mediated by right frontal and left frontal cortex, respectively.

## P2-j10 A new model to evaluate affective states associated with reward-predicting cues in mice

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The main obstacle to treating addiction is relapse, i.e. restoration of reward intake. Relapse can be precipitated by environmental cues previously associated with reward. Little research has been performed on affective states resulting from presentation of cues predicting reward and their role in creating, maintaining and restoring addiction. To investigate this, we adapted to mice the taste reactivity paradigm; a procedure to assess affective states. We coupled this to a procedure for inducing anticipation of reward that consisted of repeatedly presenting food to mice in a testing cage 30 min after introduction. Taste reactivity was recorded at different phases of the development and extinction of anticipatory behavior. In food-deprived, but not food-sated mice, an anticipatory activity progressively developed that extinguished when food was no longer presented. Analysis of taste reactivity is in progress. This novel model should greatly aid in understanding how affective states change during the transition to addiction, and their underlying neurobiology.

## P2-j11 Catecholamine activity during drug conditioned place preference and aversion

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In conditioned place preference (CPP), distinct environments are paired independently with either vehicle, or a rewarding drug. After conditioning, animals will bias their exposure to the drug-paired environment when presented with both at once. CPP demonstrates the unique way that addictive drugs are able to influence learning because the drug CPP involves volitional control of behavior (i.e. choice) although animals do not learn any instrumental actions directed at obtaining drug. In this way, it may represent the most fundamental processes involved in drug seeking behavior during addiction. Still, the neural mechanisms underlying learning and memory during CPP remain poorly understood. To yield neurobiological data directly related to this behavior, we measured neurochemical activity via microdialysis during the acquisition and expression of CPP. Here we show extracellular levels of dopamine and serotonin within the nucleus accumbens during the acquisition and expression of morphine CPP and naloxone CPA (e.g. conditioned place aversion). The results will be discussed in light of the involvement of catecholamines in reward-seeking behavior and learning.

## P2-j12 Methamphetamine induced dopamine terminal toxicity involves endoplasmic reticulum stress

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We examined the methamphetamine (METH)-associated change of gene expression in CATH.a cells, a cell line derived from dopaminergic neuron. Because CATH.a cells undergo apoptosis if they are treated by either dopamine (DA) or METH, we administrated DA or METH to the cells and compared gene expression profiling with DNA microarrays. The gene expression profiles of DA or METH treated cells were distinctive and several genes were specifically regulated in the cells that undergo METH induced apoptosis. One of the genes was ddit3 gene for CHOP, a marker for endoplasmic reticulum stress. The expression of ddit3 gene was examined by real-time quantitative PCR.

## P2-j13 The expression of cocaine-induced behavioral sensitization is blocked by microinjection of CART peptide 55-102 into the nucleus accumbens.

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The role of CART 55-102 peptide in the nucleus accumbens (NAcc) in the expression of cocaine-induced behavioral sensitization was examined. Four groups of rats were either saline or cocaine pre-exposed (15 mg/kg, i.p., once daily for 7 days). After 3 weeks of withdrawal, rats were challenged by cocaine (10 mg/kg, i.p.) following microinjection into the NAcc of either saline or CART 55-102 (1.0, or 2.5 μg/0.5 μl/side). CART 55-102 peptide microinjected into the NAcc dose-dependently blocked the expression of locomotor sensitization produced by repeated cocaine pre-exposures. These results suggest that CART 55-102 peptide in the NAcc may play a compensatory inhibitory role in the expression of cocaine-induced behavioral sensitization. Supported by the Neurobiology Research Program grant from the Korea Ministry of Science and Technology (to J.H.K).

## P2-j14 Epinephrine inhibits analgesic tolerance to intrathecal morphine while increases the expression of CaM kinase II α

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Stress inhibits the development of morphine tolerance via activating Hypothalamic-Pituitary-Adrenal (HPA) axis. Also, the expression of CaMK II α is increased following chronic administration of morphine. We examined the effect of epinephrine, morphine tolerance and expression of CaMK II α. Tolerance was induced by i.t. injections of morphine 15 μg/kg, twice a day for 5 days. Eepinephrine was administrated 20 minutes before morphine injections. Analgesia was assessed using tail flick test. Gene expression assay was done using RT-PCR. Combined treatment of epinephrine (2, 5 or 10 μg/kg) and morphine for 5 days, inhibited the development of morphine tolerance. co-administration of epinephrine reduced the tolerance which was developed to morphine. Morphine, epinephrine and their combination increased the expression of CaMK II α. Results showed the ability of epinephrine to inhibit and reverse the tolerance to local administrated morphine. The expression of CaMK II α can be increased without tolerance development.

## P2-j15 Diamond microelectrodes for dopamine detection in the brain

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Voltammetric recording with carbon-fiber microelectrodes detects extracellular dopamine changes with subsecond temporal resolution. As well as carbon, recently developed boron-doped diamond (BDD) electrodes allow detection with lower noise. This fiber was applied for dopamine measurement in the mouse brain. BDD microelectrodes (10 um in diameter at the tip) were prepared on tungsten wires by chemical vapor deposition (CVD) system. Either BDD or carbon-fiber microelectrode was placed in the striatum of C57BL/6 mice. A stimulating electrode was placed on the medial forebrain bundle (MFB) to induce dopamine release. Both electrodes were able to detect transient elevation of electrochemical current following MFB stimulation. Since carbon and BDD electrodes have different selectivity for the molecules in the brain, combinational use of two kinds of microelectrodes will improve the chemical resolution.

MEXT, Mobiligence Project in Priority Areas Japan 17075002 .

## P2-j16 Sensitization of TRPA1 by PAR-2 through PLC/PIP2-dependent mechanisms

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The functional interaction of the proteinase-activated receptor-2 (PAR-2) and TRPA1, was investigated by electrophysiological analys in both heterologous expression system and in dorsal root ganglion (DRG) neurons. PAR-2 activation increased the TRPA1 currents evoked by allyl isothiocyanate (AITC) or cinnamaldehyde in HEK293 cells transfected with TRPA1. Phospholipase C (PLC) inhibitors suppressed this potentiation; however downstream effectors of PLC did not participate in the potentiation. Rather, intracellular PIP2 significantly inhibited the potentiation of AITC-induced currents by PAR-2. Application of a PIP2 antibody or a PIP2 scavenger mimicked the potentiation of TRPA1 activity by PAR-2. Thus, the increased TRPA1 sensitivity may be due to activation of PLC, which in sequence releases the inhibition of TRPA1 from plasma membrane PIP2. This functional interaction of PAR-2/TRPA1 and the responsible intracellular signal cascade were also observed in rat DRG neurons and confirmed at the behavioral level.

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## P2-j17 Potentiation of TRPA1 activity by Bradykinin

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Bradykinin (BK) is an inflammatory mediator that plays a pivotal role in pain and hyperalgesia in inflamed tissues by exciting and/or sensitizing nociceptors. TRPA1 is an important component of the transduction machinery through which environmental irritants and endogenous proalgesic agents depolarize nociceptors to elicit inflammatory pain. Here, using electrophysiological analyses, we show a functional interaction of these two inflammation-related molecules in primary sensory neurons. We found that BK increased the TRPA1 currents evoked by allyl isothiocyanate (AITC) or cinnamaldehyde in HEK293 cells expressing TRPA1. This potentiation was inhibited by PKA inhibitors, and mimicked by PKA activators. In contrast, PKC activation was not involved in the potentiation. The functional interaction was also observed in rat DRG neurons and confirmed at a behavioral level. These results represent a novel mechanism through which bradykinin released in response to tissue inflammation might trigger the sensation of pain by TRPA1 activation.

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## P2-j18 P2Y12 receptor expressed in spinal microglia contributes to neuropathic pain

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Spinal microglia is known to be activated by extracellular ATP, mediated by the intracellular signals of MAPK pathway and may contribute to the neuropathic pain. We have reported that the P2Y12 receptor was constitutively expressed in spinal microglia. In this study, we investigated whether P2Y12 receptor in microglia was involved in the pathogenesis of neuropathic pain following peripheral nerve injury.
Using partial sciatic nerve ligation (PSNL) model rat, we investigated the expression of P2Y12 mRNA in spinal cord. P2Y12 receptor mRNA showed the increase in activated microglia following PSNL and peaked at 3day after injury. Intraspinal administration of P2Y12 receptor antagonist (MRS2395) attenuated pain-related behavior and inhibited the phosphorylation of p38 MAPK in spinal microglia after PSNL. Our data suggest that the P2Y12 receptor in microglia could participate in the activation of MAPK pathway and play a crucial role in the generation of neuropathic pain.

## P2-j19 Involvement of ion channels in experimental models of muscle pain

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The roles of ion channels in muscle-mechanical hyperalgesia (M-MH) were examined in the two models of muscle pain. Rats were injected with carrageenan (CI) or subjected to eccentric exercise (ECC) in the gastrocnemius muscle (GM). Randall-Selitto test and von Frey test were performed at the calves to evaluate M-MH. Effects of antagonists on the MH were evaluated. The changes of expression for ion channels in muscle sensory neurons were examined. The MH was observed in both CI and ECC with Randall-Selitto test. Infiltrations of the inflammatory cells in the GM were seen in CI but not in ECC. Antagonists against ASICs and TRPV2, but not TRPV1 showed suppressive effect on the MH after both CI and ECC. Expressions of ASICs and TRPV2 in sensory neurons showed no changes. Although time course and underlying etiology might be different, ASICs and TRPV2 are important components of muscle MH in both CI and ECC model of muscle pain.

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## P2-j20 Effects of LPA injection into the cisterna magna, trigeminal ganglion and inferior alveolar nerve on the Von Frey test and c-Fos expression

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Lysophosphatidic acid (LPA) is released when nerve is injured and induces demyelination, which causes neuropathic pain. Here, we examined the pain appearance pattern after LPA injection into the cisterna magna(CM), trigeminal ganglion(TG) and inferior alveolar nerve(IAN). The nociceptive face avoidance threshold to mechanical stimulation was examined using Von Frey test. The c-Fos was stained immunohistochemically. After LPA injection into the CM, the lowering of threshold in Von Frey test was observed at 3 and 7 days at masseteric area. When injected LPA into the CM, the mean numbers of c-Fos-immunoreactive neurons increased compared with the control in the principal nucleus (Vp), in the oral nucleus (Vo) and in the Vi/caudal nucleus (Vc) transition zone. These results indicate that the release of LPA in the medullary cord induce neuronal hyperactivity and neuropathic pain.

## P2-j21 Tramadol produces nerve conduction block in a manner independent of opioid receptor activation and monoamine uptake inhibition

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Tramadol is thought to exhibit antinociception through various actions including opioid receptor activation and monoamine uptake inhibition. Although tramadol is also known to have a local anesthetic effect, this action has not been examined yet thoroughly. The present study examined the effects of tramadol and its metabolite mono-*O*-demethyl-tramadol (M1) on compound action potentials (CAPs) by applying the air-gap method to frog sciatic nerves. Tramadol reduced the peak amplitude of the CAP in a manner independent of naloxone. On the other hand, M1 did not affect CAPs. This tramadol action was not affected by a combination of noradrenaline (NA)-uptake inhibitor desipramine and 5-hydroxytryptamine (5-HT)-uptake inhibitor fluoxetine. It is concluded that the inhibitory action of tramadol is not due to opioid receptor activation and also to NA and 5-HT uptake inhibition. The methyl group present in tramadol but not M1 is suggested to play an important role in producing nerve conduction block.

## P2-j22 Studies on pain control system (Rept. 67): Opioids regulate sodium currents in rat cultured thalamic neurons

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Some opioids exert inhibitory effects on the generation and conduction of nerve action potentials in peripheral tissues. However, the specific contribution of opioids in the CNS to the inhibition of voltage-gated sodium channels remains unclear. The present study was designed to investigate the effects of opioids could affect the voltage-gated sodium channels in the rat thalamus. Whole-cell patch-clamp recordings were obtained from cultured thalamic neurons to study the activating sodium current. Bath application of morphine or oxycodone produced a reduction in the peak amplitude of sodium current evoked by a step from a holding potential of -70 mV to a command potential of -20 mV. The present data suggest the possibility that morphine and oxycodone may decrease the activity of neuronal sodium channels in the rat thalamus.

## P2-j23 Immunohistochemical changes of pain-related molecules in the spinal dorsal horn of the joint immobilized rat

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The casting (immobilization) may induce not only musculoskeletal motor dysfunction but also pain in the affected limb. It is hypothesized that immobilization may alter nociceptive function of the spinal cord. In order to characterize the casting model, the pain-related molecules were investigated in the spinal dorsal horn.We prepared a rat model of wrist immobilization and analyzed c-Fos, calcitonin gene-related peptide (CGRP), protein kinase C gamma (PKCγ), and glial fibrillary acidic protein (GFAP) expression in the spinal dorsal horn (C7-T1) by using immunohistochemistry. Immunoreactivity of c-Fos, PKCγ, and GFAP was increased in ipsilateral side compared to those in contralateral side. However, immunoreactivity of CGRP was decreased in the ipsilateral side. These results suggested that the casting model showed the feature of neuropathic pain in the spinal dorsal horn.

## P2-j24 Studies on pain control system (Rept. 66): Involvement of endogenous opioidergic system in the suppression of the morphine-induced rewarding effect under the neuropathic pain-like state

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The present study was undertaken to investigate the mechanism of the suppression of the morphine-induced rewarding effect under a neuropathic pain-like state. Here we found that the suppression of the μ agonist-induced place preference with sciatic nerve ligation was reversed by pre-microinjection of a specific antibody to β-endorphin into the ventral tegmental area. Furthermore, the suppression of the morphine-induced rewarding effect under a neuropathic pain-like state was recovered by lacking the pro-opiomelanocortin gene. These findings suggest that the continuous release of β-endorphin may implicate in the suppression of the psychological dependence on morphine under a neuropathic pain-like state.

## P2-j25 Modulation of trigeminal spinal subnucleus caudalis neuronal activity following regeneration of transected inferior alveolar nerve in rats

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In order to clarify the neuronal mechanisms of abnormal pain sensation in the face reinnervated by the regenerated inferior alveolar nerve (IAN), the nocifensive behavioral and single neuronal activity were precisely analyzed in rats with IAN transection. The escape thresholds to mechanical stimulation of the mental skin was significantly higher at 3 days and lower at 14 days after IAN transection, and returned to the preoperative level at 60 days after that. The population of WDR neurons with background activity was significantly higher in rats 14 and 60 days after IAN transection compared with that of naive. Mechanical evoked responses were significantly larger in WDR and LTM neurons 14 days after IAN transection compare with naive rats, but not in WDR neurons at 60 days after transection. The present findings suggest that the regenerated IAN may have an ability to generate abnormal firing reflecting in the abnormal neuronal discharge in Vc neurons.

## P2-j26 In vivo patch-clamp analysis of plastic changes in descending serotonergic inhibition of noxious mechanical transmission to substantia gelatinosa neurons through 5-HT1A receptors in ovariectomized rats

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Presynaptic effects of 5-HT on EPSCs evoked in substantia gelatinosa (SG) neurons were compared between normal and ovariectomized (OVX) rat in vivo and in vitro. In normal, 5-HT inhibited the amplitude of EPSCs through the activation of C and Aδ fibers evoked by cutaneous pinch and brush stimuli. These inhibitory effects on C and Aδ fiber-evoked EPSCs were mimicked by $5HT_{1A}$ and $5\text{-}HT_{1D}$ agonists, respectively. In OVX, the 5-HT actions on pinch- and C fiber-evoked EPSCs were impaired without affecting on brush- and Aδ fiber-evoked EPSCs. The present results suggest that selective loss of presynaptic inhibition of 5-HT on noxious mechanical inputs to SG neurons through $5\text{-}HT_{1A}$ receptors in the presynaptic terminals of C fibers has an important role in the induction of chronic mechanical pain sensation in OVX rats.

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## P2-j27 Presynaptic TRPA1 activation enhances glutamate release onto substantia gelatinosa neurons of the adult rat spinal cord

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The role of TRPA1 in the CNS has never been documented yet, although it is proposed to be activated by cold stimuli or pungent ingredients in the periphery. In the present study, we examined whether TRPA1 activation affects synaptic transmission in substantia gelatinosa (SG) neurons of adult rat spinal cord slices. A chief ingredient of mustard oil, allyl isothiocyanate (AITC), superfused for 2 min markedly increased sEPSC frequency and amplitude, which was accompanied by an inward current. AITC also increased sIPSC frequency and amplitude; this AITC action was abolished in the presence of tetrodotoxin or glutamate-receptor antagonists. These results indicate that TRPA1 appears to be localized not only at presynaptic terminals on SG neurons to enhance glutamate release, but also in terminals of primary afferents innervating onto spinal inhibitory interneurons which make synapses with SG neurons. This central modulation of sensory signals may be associated with physiological and pathological pain sensations.

## P2-j28 Changes in new generation of glial cells under the neuropathic pain like state

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Pain pathways terminate in discrete brain areas that monitor the sensory and affective qualities of the initiating stimulus and show remarkable plasticity. Here we found that chronic pain caused a dramatic increase in glial fibrillary acidic protein (GFAP)-like immunoreactivity (IR) with expanding distribuation in the cingulate cortex (CG) of mice with sciatic nerve ligation. The branched GFAP-like IR in the CG of nerve ligated mice was associated with astrocyte activation. In contrast, OX42-positive microglial cells were dramatically increased in the hippocampus of nerve-ligated mice. The number of BrdU-positive cells in either the CG, subventricular zone (SVZ) or hippocampus was not changed by sciatic nerve ligation. These results suggest that chronic painful stimuli induce glial activation in both the CG and hippocampus with no changes in the cell proliferation/differentiation from neural stem cells.

## P2-j29 Pathological analysis of *XPG*-deficient mice

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The xeroderma pigmentosum group G (XP-G) gene (*XPG* ) encodes a structure-specific DNA endonuclease that functions in nucleotide excision repair (NER). Some XP-G patients exhibit skin abnormalities,growth failure,life-shortening and neurological dysfunctions, which are also characteristic manifestations of Cockayne syndrome (CS). We have generated mice lacking *XPG* alleles (*XPG* null mice). The *XPG* null mice exhibited postnatal growth failure, ataxia and premature death. These are similar to clinical symptoms of the XP-G, XP-G/CS, CS patients. We examined pathological changes corresponding to clinical manifestations of XP-G/CS in the *XPG* null mice, such as emaciation, hearing loss, pigmentary retinopathy, hypogonadism. In the cerebella of the *XPG* null mice at postnatal day 21, IHC for calbindin D revealed no staining of Purkinje cell dendrites, suggesting dysgenesis or degeneration of Purkinje cells.

## P2-j30 Dysregulation of Trk receptor signaling in β-galactosidase-deficient mouse brain

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$G_{M1}$-gangliosidosis is a fatal neurodegenerative disorder caused by a deficiency of lysosomal β-galactosidase (β-gal), resulting in accumulation of ganglioside $G_{M1}$ in the lysosome. In this study, we found that phosphorylation of Trk receptor was markedly up-regulated in the β-gal-deficient mouse brain. Treatment with a galactose derivative chemical chaperone, N-octyl-4-epi-β-valienamine to the β-gal-deficient mouse expressing an R201C mutant human β-gal enzyme protein resulted in a significant reduction of $G_{M1}$ accumulation and phosphorylated-Trk expression in the brain. These results suggest that abnormal regulation of Trk signaling may participate in the pathogenesis of $G_{M1}$-gangliosidosis.

## P2-j31 Trivial change of iron metabolism can cause or deteriorate neurodegenerative symptoms

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The relationship between oxidative stress caused by abnormal iron metabolism and neurodegenerative diseases were well described. To probe the mechanism underling iron-toxcity, we generated neuron specific IRP2, a regulator of iron uptake and storage, transgenic mice that expressed very small amount of IRP2 as compared with endogenous level. Although there was no substantial change of total expression level of IRP2, not massive but critical reactive-iron accumulation were observed in the transgenic brain. With aging, accumulation of oxidative stress marker and nuclear condensed apoptotic cells were observed in many parts of transgenic brain, accompanied low activity and low ability of coordinated movement compared with wild type mice. Those results suggested that the subtle imbalance of iron uptake and storage can cause oxidative stress, which may result in neuronal cell death and neurodegenerative symptoms and it can be one of the deteriorating factor for neurodegenerative diseases.

## P2-j32 Aberrant neurological development caused by genetic incompatibility

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Most of neurological diseases are complex and multifactorial, thus the underlying genetic mechanism mostly remains unknown. We have been analyzing a mouse model that exhibits neurological defects caused by an aberrant genetic interaction, so called as genetic incompatibility. We recently found neurologically defective mice, FURUE, indicating defective growth, degenerative eye development, tremor and ataxia. Furthermore, FURUE showed aberrant myelination, such as reduced number of myelin layers and occasionally incomplete compactions of the most inner layer of myelin sheath in spinal cord and optic nerve. FURUE emerged in a F2 (KLF2) population made between two wild-derived mouse strains, BLG2 and KJR. Because both BLG2 and KJR are never indicate such neurological and developmental defects, this aberration seemed to be caused by genetic incompatibility. Through a genetic analysis, we identified on of the causative loci, *Genetic incompatibility1* (*Genic1*).Here, we report results of detailed genetic and phenotypic analysis of FURUE.

## P2-j33 Development of database for experimental parameters of behavior analyses in mice

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Behavior analyses using rodents depends on various experimental or rearing parameters such as illuminance, space and handling procedures. Recently, collaborative saturation mutagenesis program using gene targeting techniques, such as KOMP and EuCOMM, have just started in USA and Europe. These programs required standardized large-scale phenotyping platforms for direct comparison of phenotype data. It is well recognized that comparison in detail of the procedures used among different institutions is needed. We are now developing a database for experimental parameters of behavior analyses based on the concept of " Standardized Description of Operating Procedures (SDOP) ". It enables easy identification of differences in parameters of the phentoyping platforms in different institutions and may useful for management of multiple versions of protocol in small labs.

## P2-j34 Analysis of α-synuclein and the associated protein in MSA mouse model

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Multiple System Atrophy (MSA) is a non-hereditary neurodegenerative disease. Glial cytoplasmic inclusions (GCIs) and neuronal inclusions, both composed by α-synuclein (α-syn), are characteristic markers in MSA pathology. Formation of α-syn inclusions might be the primary lesions that will eventually compromise nerve cell function and viability in MSA. There has not been studied to demonstrate α-syn accumulation in oligodendrocytes leads to neuronal degeneration until establishing a mouse model of MSA. We previously generated the transgenic (Tg) mouse model, in which human wild-type α-syn was overexpressed in oligodendrocytes. Tg mouse showed that the accumulation of α-syn as GCIs leaded to neuronal degeneration in the mouse CNS, suggestive of similar disease process in MSA. To elucidate the early process to cause the secondary neuronal degeneration in the mouse model of MSA, we prepared primary cultures of neurons and glial cells obtained from Tg and non-Tg mice. Insoluble endogenous mouse α-syn was accumulated in the Tg cultured neurons. Here we have identified a protein, which interacts with insoluble α-syn.

## P2-j35 Evaluation of pathophysiological features of migraine by micro PET study using rats

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Cortical spreading depression (SD) is characterized by the propagation of neuronal/glial membrane depolarization accompanied by temporal elevation of the cerebral blood flow throughout the cerebral cortical hemisphere, and has been implicated in pathophysiological states of migraine. We investigated here pathophysiological features of migraine by PET study with [18F]FDG using SD-induced rats. We demonstrated that uptake of FDG was suppressed in the cortical hemisphere which underwent SD. Moreover, the increase in FDG uptake was observed in several brainstem regions, including the trigeminal nucleus caudalis (TNC) which has been reported to be involved in a nociceptive pathway in migraine. These results indicate that the micro PET study is a useful tool for evaluation of pathophysiological features of migraine in such an animal model.

## P2-j36 Elevated 5-HT expression of spinal motoneurons of ataxic mutant mice, Pogo

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A novel neurological mutant mouse, pogo is characterized by involuntary movements of the extremities, difficulty in maintaining normal posture and side to side stability, faulty coordination between the movements of limb and the trunk, and displays abnormal hindlimb extension. In previous study, we reported increased serotonin expression in raphe nucleus of pogo mice. This study aimed to explain whether 5-HT expression of the spinal motoneurons was altered in pogo in relation with the abnormal hindlimb extension. In lumbar region of pogo mice, 5-HT immunoreactivity of motoneurons was about 2 fold higher than controls. However, in cervical and thoracic region of pogo mice, we observed no significant differences in 5-HT immunoreactivity of motoneurons, when compared to the control mice.

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## P2-j37 Deletion of Schnurri-2 causes multiple behavioral abnormalities related to psychiatric disorders in mice.

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Schnurri-2 (Shn2) is a zinc finger transcription factor, a mouse homologue of human HIVEP2, that binds to nuclear factor kB-binding site in the HIV long terminal repeat. Shn2 is known to play important roles in mammalian immune systems. However, the role of Shn2 in the central nervous system (CNS) is still unknown. To investigate the functional significance of Shn2 in mammalian brain, we analyzed Shn2 knockout (KO) mice using a behavioral test battery. Shn2 KO mice showed hyperactivity, impaired prepulse inhibition and impaired working memory. Gene expression analysis and morphological analysis of the hippocampus of the KO mice were also conducted. These results demonstrate a critical role of Shn2 in CNS and suggest that Shn2 KO mice may serve as an animal model of human psychiatric disorders. The potential mechanisms underlying the behavioral abnormalities of Shn2 KO mice will be discussed.

KAKENHI (16680015, 18016012, 18023022, 18053015, 18700315), JST BIRD

## P2-j38 Alpha-CaMKII deficiency causes dysregulated behaviours and immature dentate gyrus

Nobuyuki Yamasaki[1], Motoko Maekawa[2], Katsunori Kobayashi[3], Yasushi Kajii[4], Jun Maeda[5], Miho Soma[2], Keizo Takao[1], Kouji Kanzaki[4], Hidenori Suzuki[3], Makoto Higuchi[5], Tetsuya Suhara[5], Shigeki Yuasa[2], Tsuyoshi Miyakawa[1]

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The identification of endophenotypes is an essential step for investigating psychiatric illness, but represents a considerable challenge in human patients. Here we show that alpha-CaMKII heterozygous knockout mice have dysregulated behaviors, including a severe working memory deficit and an exaggerated infradian rhythm. The neurons of the dentate gyrus (DG) in the mutants resembled immature DG neurons in normal rodents. Biomarkers derived from the mutants classified individuals into two clusters, one of which had higher susceptibility to schizophrenia and differential expression of genes related to neural development. Based on these results, we propose the immature DG as a candidate endophenotype of psychiatric disorders.

KAKENHI (16680015, 18016012, 18023022, 18053015, 18700315), JST BIRD

## P2-j39 Alterations in cognitive behaviors of neuregulin-1 transgenic mice: An influence of genetic backgrounds

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Neuregulin-1 (NRG1) is one of the candidate risk genes for schizophrenia. Not only environmental factors but also the genetic backgrounds are involved in the pathophysiology of schizophrenia, though their interactions remain largely unknown. In various schizophrenia models, animal genetic backgrounds influence on levels of prepulse-inhibition (PPI). For instance, genetic background-dependent disruption of PPI was reported in NRG1 transmembrane region deleted mice. We found that a transgenic (Tg) mouse over-expressing NRG1 (TypeI $\beta$) gene also showed impaired PPI in hybrid genetic background of C57/BL6 : DBA/2 (F1, F2). To analyze the interactions between the over-expression of the NRG1 and the genetic background, we backcrossed it with C57/BL6 and DBA/2 parental strains more than five generations. We will show behavioral traits of the NRG1 Tg mice, and discuss an influence of genetic backgrounds on their behaviors.

## P2-j40 Establishment and behavioral analysis of conditional transgenic mouse lines carrying mutant ErbB1 gene.

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Epidermal growth factor (EGF) binds to ErbB1 receptors and induces proliferation and migration of various types of cells. Previously, we have reported that neonatal administration of ErbB1 ligands induces several abnormal behaviors in mice and rats. As ErbB1 are expressed in various types of neurons in the brain, the cellular targets for EGF that are responsible for the behavioral deficits remain to be unknown. Here we established transgenic mouse lines carrying a LoxP-silenced mutant ErbB1 gene and activated ErbB1 signaling in a neuron-specific manner. We constructed the vector carrying a constitutive active (CA) form of ErbB1. To activate the LoxP-silenced ErbB1 gene in pyramidal or GABAergic neurons, these mice were mated with the mice carrying Cre-recombinase gene driven by either Emx1 or glutamic acid decarboxylase (GAD)67 promoter. Currently we are testing these parental mice and offspring for several behavioral tasks.

## P2-j41 Antipsychotic effects of ErbB1 tyrosine kinase inhibitors on behavioral deficits in the neonatal hippocampal lesion

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We reported that there are EGF and EGF receptor (ErbB1) abnormalities in brain tissues and blood of schizophrenic patients. Here we examined ErbB1 signaling in the schizophrenia model using adult rats that received bilateral microinjections of saline or ibotenic acid in the ventral hippocampus as neonates. We also determined the antipsychotice effects of several ErbB1 tyrosine kinase receptor inhibitors in the above model rats. At 8 weeks of postnatal day, rats were tested for ELISA of ErbB1 ligands and behavioral tasks. In striatum of ibotenic acid treated rat, EGF protein levels were higher than those of control. Hippocampus-lesion significantly impaired the behavioral scores of PPI and latent learning. The abnormalities in PPI and latent inhibition were ameliorated by icv infusion of ErbB1 tyrosine kinase receptor inhibitors, ZD1835, OSI-774 and PD 153035. These results indicate that blockade of ErbB1 signals may be a novel antipsychotic drug.

## P2-j42 Central nervous system specific deficiency of lysosomal cathepsin D induces a phenotype resembling neuronal ceroid lipofuscinosis.

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*Department of Cell Biology and Neuroscience*

Neuronal ceroid lipofuscinosis (NCL) is one of lysosomal storage diseases with an autofluorescent lipopigment in neurons and a wide variety of extra neuronal cells. It has been shown that cathepsin D (CTSD)-deficient mice die at approximately postnatal day (P) 26 by massive intestinal necrosis, thromboembolia, and lymphopenia. We have previously demonstrated that CTSD deficiency induces accumulation of lysosomes with ceroid-lipofuscin in CNS neurons that resembles symptoms of NCL. We show here that CNS specific CTSD deficiency also induces a phenotype in mice (CTSDflox:NesCre) that resembles NCL. The mice were born at an expected mendelian frequency. Body weight of CTSDflox:NesCre mice increased normally over P20, whereas that of conventionally CTSD-deficient mice started to decrease around P15. The CTSDflox:NesCre mice died around P30, showing a phenotype that was quite similar to NCL. These lines of evidence suggest that CTSD plays an essential role in the degradation in lysosomes of CNS neurons.

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## P2-j43 Expression of a gene in response to traumatic experience in murine hippocampus

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Posttraumatic stress disorder (PTSD) is characterized by recurrent recalls of bad memories, insomnia with nightmares and emotional withdrawal, all of which are long lasting after initial traumatic experiences. Adult male mice were subjected to restraint stress in a metallic cage immersed in water up to the individual clavicle for 3 h as a traumatic experience, followed by various behavioral tests 14 days later. Stressed mice not only showed increased spontaneous locomotor activities, but also exhibited freezing behaviors in response to a recurrent recall signal on the tone fear-conditioning task and forced swimming test, respectively. Chronic treatment with imipramine significantly suppressed these stress-induced behavioral changes. Differential display analysis revealed that two different genes were expressed in response to traumatic stress in hippocampus 1 day later, while cloning studies identified these genes. These findings suggest that the aforementioned genes could play a pivotal role in mechanisms underlying the crisis associated with PTSD.

## P2-j44 Edaravone attenuates post-traumatic hyperactivity and impairment of LTP of excitatory synaptic transmission in rat hippocampal dentate gyrus

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We investigated the effect of edaravone, a free radical scavenger, on the post-traumatic neuronal function in dentate gyrus (DG) using conventional extracellular recording techniques. Horizontal brain slices were obtained from the control and the lateral fluid percussion injury (FPI) rats one week after the single moderate impact. The input-output relationship (I-O) of perforant path (PP) synapse was enhanced (hyperactivity), while induction of LTP of PP synapse was attenuated in FPI group. Edaravone (8mg/kg, IP) was administrated at 15 min after the impact. The I-O function and the induction of LTP of edaravone treated rats were similar to those of control rats. On the other hand, "in vitro" administration of edaravone blocked the induction of LTP. These data suggest that edaravone attenuates the post-traumatic malfunction of PP-granular cell synapse in rat DG.

## P2-j45 Transparent electrode for recording electrocorticogram from optical sampling area of the multiple-site optical detection of neural activity in the rat cerebral cortex

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Simultaneous use of multiple-site optical recording and conventional electrophysiological recording to monitor spontaneous neural activity provides a powerful tool for the cerebral cortical neurophysiology. However, the electrode and its lead wire are usually opaque and interrupting the light path when placed on the optical sampling area. Therefore, we have developed a transparent electrode. It was formed on the surface of a piece of glass that contacted and covered the entire region of the exposed cortex. Ga doped ZnO film, a transparent conductor, was deposited on the glass by radio-frequency magnetron sputtering and insulated with $SiO_2$ except for a hall ca 300 μm in diameter, which hall eventually served as an electrode. Using this transparent electrode, we succeeded in recording electrocorticogram at a site just within the optical sampling area of the rat somatosensory cortex.

## P2-j46 Real-time imaging of glucose uptake in the substantia nigra pars reticulata using a fluorescent glucose tracer

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In mammalian cells, measuring glucose uptake into cells of a heterogeneous population and/or differing activity status is of much interest. We have established a real-time imaging method for glucose uptake into single, living mammalian cells (Yamada et al, J Biol Chem, 2000; Yamada et al, Nature Protocol, 2007) by using a fluorescent D-glucose derivative, 2-[N-(7-nitrobenz-2-oxa-1,3-diazol-4-yl)amino]-2-deoxy-D-glucose (2-NBDG) (Yoshioka et al, Biochim Biophys Acta, 1996) as a tracer. For the last several years, 2-NBDG has been shown to be useful for monitoring glucose uptake into a variety of cells, especially activity-dependent uptake of glucose into neurons and astrocytes as well as intercellular transport of glucose through gap junctions. Here we visualize glucose uptake in substantia nigra pars reticulata, which we have proposed as a metabolic sensor in the brain (Yamada et al, Science, 2001; NIPS, 2002).

KAKENHI17590182

## P2-j47 Edema and cyclooxygenase-2 protein expression in the brain after local irradiation with carbon ion beams

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Formation of cerebral edema is acute response of brain tissue to a large radiation and may contribute to delayed pathology (vascular collapse and tissue necrosis) that often lead to permanent loss of function. Therefore, it is very important to reduce formation of cerebral edema for protect potential damage of normal brain tissue. Cyclooxygenase-2 (COX-2) is the inducible form of the 2 prostaglandin-synthesizing enzymes and is believed to be an important mediator of cell injury in inflammation. However, it is unclear what the role of COX2 activity in the brain irradiated with carbon ion beams. In the present study, the left cerebral hemispheres of adult rats were irradiated at dose 50 Gy with carbon ion beams, expression of COX2 protein and the effect of treatment with a COX2 inhibitor on cerebral edema were determined and compared at 2-24 hrs after irradiation. The results indicated that radiation-induced cerebral edema was dependent on COX2 activity.

## P2-j48 Single cell-based monitoring of dopamine release from a nerve model cell using a microelectrode for drug assessment

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We report single cell-based monitoring of dopamine release from a dopamine-producing nerve model cell using a microelectrode system for the purpose of its application to drug assessment for nervous system. PC12 cell was used as a dopamine-producing nerve model cell with acetylcholine receptor. A Pt microelectrode was settled on a single cell at 8 micrometer distance and was controlled at 0.55V vs Ag/AgCl to oxidize dopamine released from the cell. Dopamine release from a single PC12 cell was successfully monitored by real-time increase of oxidation current upon injection of DMPP(1,1-dimethyl-4-phenyl-piperazinium) as an acetylcholine-receptor agonist with dose-dependence. Furthermore, inhibition of dopamine release from a PC12 cell by CPZ(chlorpromazine) as an acetylcholine-receptor antagonist was also measured by decrease of dopamine release current upon DPMM stimulation. IC50 of CPZ using this method was almost same as reference value. Evaluation of another drug effect with this method will be presented in this paper.

## P2-k01 Glyoxylic acid induced fluorescence imaging of the larval nervous system of deuterostomia

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Deuterosome involves animals from echinoderms to vertebrates. Therefore, the making of the echinoderm's nervous system is an attractive issue for scientists of the brain. However, the neuroanatomy of a plankton-sized larva is less investigated due to its small size. We visualized the fine nerve net of the larva by the glyoxylic acid imaging technique. Major neurotransmitter species, monoamines and glutamate, can be sensitive by this method. Indeed, the fluorescent microscopic images of the entire nervous system exhibited the bilateral network, instead of 5-radial organizations in adult phase. Monoaminergic axons beneath the epithelia and around the esophagus were imaged. Yellowish 5-HT fluorescence, greenish dopamine fluorescence, and yellowish-green glutamate fluorescence were detected. The largest nerve cell clusters were located dorsally on the digestive tract. Intimacy of nerve fiber and cilia structure, and the location of the brain at the dorsal side indicate that the nervous structure of this animal resembles fundamentally the architecture of those of the deutrostome.

## P2-k02 Generation of novel fluorescent glutamate indicators by high-throughput screening system

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Visualization of glutamate dynamics in living neural cells is a powerful method to analyze neural functions in central nervous system. For glutamate imaging, we previously developed a fluorescent glutamate indicator (EOS) by conjugation of a glutamate binding protein and a thiol-reactive fluorescent dye which reacts with a cysteine residue in glutamate binding protein. In this process, the prediction of the adequate site for dye conjugation is difficult. Thus a process of search for optimal probe requires many trials and errors. To facilitate this process, we developed a high-throughput screening system based on 96-well plate format protocol including mutagenesis, protein expression, purification, dye-labeling, and functional assay. After screening, we obtained glutamate indicators showing larger fluorescence change upon glutamate binding than that of EOS (40% increase). This result suggests that our screening system should be applicable to develop the fluorescent indicator for other signaling molecules as well as for glutamate in short periods.

## P2-k03 Development of the high performance glutamate indicator for visualization of neurotransmission

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Analyzing spatiotemporal glutamate dynamics is expected to uncover the presynaptic mechanisms at a single synapse. Aiming at imaging glutamate, we developed a fluorescent glutamate indicator by conjugating a glutamate binding protein and a fluorescent dye. To obtain high performance glutamate indicators, we examined 540 candidates by high-throughput screening system carried out in 96-well plate format. After screening, we found a glutamate indicator consisting of G448C mutant and Alexa Fluor 488, which showed extremely large fluorescence changes upon glutamate binding (3-fold increase) and a low-affinity binding for glutamate (28 μM). Then aiming at glutamate imaging with high sensitivity, we generated improved probes by introduction of additional mutations on G448C, which are expected to use for glutamate imaging in neural cells with high spatial resolution. Also, a similar strategy should be applicable to development of high performance glutamate indicators consisting of other combinations of a cysteine mutant and a fluorescent dye.

## P2-k04 neuronal activity imaging in the rat cortico-striatum pathway using voltage-sensitive dye

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We examined the feasibility of the voltage-sensitive dye for imaging neuronal activity in the rat cortico-striatum pathway. The cortico-striatum slice preparations were dissected from postnatal rats (P3-18) and stained with the voltage-sensitive dye, NK2761. Transmembrane voltage-related optical changes were recorded simultaneously using the 464ch hexagonal diode array system, NeuroPlex. In the striatum, the optical signal was composed of a fast spike-like signal followed by a slow signal. The directions and sizes of the signals' were wavelength-dependent. The slow signal was blocked by APV and CNQX suggesting that the slow signal corresponds to the glutamatergic EPSP. On the other hand, the slow signal was enhanced by bicuculline. This further indicates that GABAA receptor function is expressed in the cortico-striatum pathway, which is detectable with our optical technique. Furthermore, we have been able to detect LTPs in this pathway.

## P2-k05 In vivo imaging indicates activation and differentiation of brain-migrated immature bone marrow cells in an artificial brain injury

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Some immature bone marrow cells (BMCs) migrate into the brain parenchyma at early stage of bone marrow transplantation and inhabit it for several months. It is unknown, however, what roles BMCs play in the brain. In this study, we demonstrated the dynamic response of BMCs to a local brain injury in GFP-expressing (GFP) bone marrow chimera mice using a magnetic resonance imaging (MRI) and an optical in vivo imaging system with immunohistochemical analysys. The position of local injury in the brain was detected by MRI, and an accumulation of $GFP^+$ cells around the injury was clearly indicated by an optical imaging. Some $GFP^+$ cells around the injury were proliferative and indicated mature phenotypes in brain sections by immunohistochemical analysis, but those in control and contralateral brain did not. Furthermore, about 70 % of them expressed iNOS. These suggested that exogenous BMCs which migrated into the brain as an immature cells might be activated and differentiate into phenotipically mature cells in response to a brain injury.

## P2-k06 Real time in vivo imaging of serine protease activity after theta-burst stimulation in the CA1 area of the mouse hippocampus

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We have developed an implantable biofluorescence imaging device by packaging the CMOS imaging sensor which enable on-chip imaging configuration and high spatial and temporal resolution optical imaging. By using the sensor, we have successfully imaged and measured activity-dependent serine protease activity in the hippocampus in vivo and in real-time. The protease activity was captured by imaging the fluorescence, which was excited by LED integrated in the sensor, from a fluorogenic substrate cleaved by the protease. Several minutes after theta-burst stimulation, serine protease activity was increased in the CA1 region. The upregulation of fluorescence associated with activation of the serine protease was dependent on the NMDA receptor activation, suggesting the fluorescence was fully linked with the synaptic plasticity. This technique could provide a methodology for imaging of synaptic plasticity of freely moving animals.

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## P2-k07 Engineering and characterization of an enhanced fluorescent protein voltage sensor

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Over the last decade, methods for optical imaging have been enriched by sensors based on fluorescent protein (FP) reporters that transduce biological events into optically detectable signals. Among this class of biosensors, the generation of FP voltage sensors is both highly desirable and challenging. A suitable such sensor would be an invaluable tool for monitoring activity of neurons or other electrically active cells. The most serious limitation of previous prototypes is their poor plasma membrane targeting, and thus a very small signal-to-noise ratio. Recently, we developed new FP voltage sensors (that we termed VSFPs, Voltage Sensitive Fluorescent Proteins) with efficient targeting to the plasma membrane. We demonstrated that our best variant can monitor membrane potential changes that occur during synaptic activation of neurons. This biosensor, which we termed VSFP2.1, is likely to lead to new methods of monitoring electrically active cells with cell type specificity, non-invasively, and in large numbers simultaneously.

## P2-k08 Analysis for multiple cellular signals by fiber-coupled realtime confocal microscopy

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To study cellular functions in situ in realtime, we developed a fiber-coupled confocal microscope, (FCM) and observed fluorescently-labeled cells in the organs of animal. The microscope system consisted of microlens-attached Nipkow disk (Yokogawa), and an imaging fiber (IF, FiberTech). The IF had 10,000 of single mode flexible fiber of 3.75 μm in diameter, which was scannned with multiple laser beams to form a confocal image. The tip of IF was 0.75 mm thick, for easier approach to the target inside the organ without a large physical damage. When the incidental face of IF was placed below the surface of organs, many spots (50 cells) were clearly visible in the field of 300 $\mu m^2$, showing fluctuation in fluorescence intensity (breathing and some cellular activities). Such an in situ imaging by FCM is very promising for detailed studies on the relationship between cell structure and functions.

Optronics Cluster Hamamatsu from ministry of academy of science

## P2-k09 Real-time monitoring of l-glutamate release from C6 glioma cells using a modified enzyme-luminescence method and its application to drug assessment

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Previously we reported real-time monitoring of dopamine release from nerve model cells by enzyme- luminescence method.Glutamate is also a major excitatory neurotransmitter in the brain. In this study, we modified the enzyme-luminescence method to detect L-glutamate sensitively and rapidly with high temporal resolution (<1 s). We applied the modified method successfully to perform real-time monitoring of L-glutamate release from brain model cell (C6 cell) upon stimulation with high concentration of KCl (> 10 mM) or 5-hydroxytryptamine (>1 μM). The measurement solution was not toxic and the continual L-glutamate release from the cell was measured after exchanging solution. Several drugs ($Ca^{2+}$ -channel inhibitors) were also administered to the C6 cells for evaluating the drug effects on voltage-dependent $Ca^{2+}$ channels and resulting L-glutamate release inhibition. we therefore conclude that the developed monitoring system is available for real-time detection of L-glutamate release in brain cell and can be applied in drug assessment for nervous system.

## P2-k10 3-dimensional reconstruction of neurons in the brain by using X-ray micro-CT technology

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It is thought that neuronal networks build our various higher brain functions. However, little is known about mechanisms underlying neuronal information processing in the brain. Anatomical structure of neuronal networks will provide us fundamental view to elucidate them. We aim at developing a three-dimensional atlas of neuronal circuits using X-ray micro-CT technology by synchrotron radiation. This experiment was performed at the beamline BL20XU of the large synchrotron radiation facility, SPring-8. We stained neurons of whole mouse brain with Golgi-Cox method. Heavy metals including mercury used in the method enhance X-ray absorption and phase contrast. X-ray micro-CT displayed 3D images of fibriform axons and dendrites of various neurons. X-ray microscopy with Talbot interferometry revealed stereographic structure of hippocampal pyramidal neurons in the CA1 region. This observation probably serves as a foundation for achieving the 3D mammalian brain atlas of neuronal networks.

## P2-k11 Roles of Myosin and myosin-ATPase for exocytosis in chromaffin cells

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Reorganization of actin network and actin-myosin interaction seem to have important roles in the vesicle recruitment for exocytosis. To clarify the roles of myosin-ATPase and of two myosin subclasses, we examined effects of 2,3-butanedione monoxime (BDM), an inhibitor of myosin-ATPase, and of myosin-antibodies on the secretory events and the vesicle movement in chromaffin cells. The secretory events were detected by amperometry, and the vesicle movement was studied by imaging of vesicles labeled with fluorescent dye, analyzing the vesicle movement during each 2 min of resting, stimulation and post-stimulation period.

BDM decreased the frequency of exocytotic events and vesicular movement throughout all the periods. Myosin-II-antibody showed similar effects as BDM, while myosin-V-antibody decreased the vesicular movement in the post-stimulation period. These results suggested that myosin-ATPase is required for vesicle recruitment, where myosin-II and -V have distinct roles.

## P2-k12 Complex topology underlies pairwise spike synchronization

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Synchronization in cortical networks is a prevalent feature that reflects dynamic processing of sensory input and internal information. Using high-speed functional multineuron calcium imaging (fMCI), we reconstructed the spatiotemporal structure of hippocampal CA3 network activity with millisecond precision. Spontaneous activity displayed a power-law distribution of synchrony size. To elucidate the profile of synchrony, we estimated the significance of synchronicity between pairs of neurons. Topography in which neurons are functionally connected based on strength of pairwise correlation was sparse, complex and locally clustered, exhibiting a small-world architecture. Simple Markov simulation on the graph replicated the firing rates of individual neurons and network synchronicity. These findings provide a simplified strategy for evaluating network operations.

## P2-k13 Metrics for multineuronal spike trains

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Functional multineuron calcium imaging (fMCI) is a large-scale optical technique to observe activity of neuron populations with single-cell resolution. But, there have been no simple measures that can effectively capture the entire properties of multineuronal activity. Here we developed entropy-based metrics that extract some specific aspects of activity patterns, which makes it possible to assess the whole activity with scalar quantities. We confirmed that these new parameters actually work on data obtained from about 200 hippocampal CA3 neurons in slice cultures at 100 Hz at various temperatures (24, 28, 32, 36 and 40°C). They may be useful for functional and pharmacological screening of the central nervous system.

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## P2-k14 Remodeling of information computation by hippocampal polysynaptic circuits

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The cortical memory system embodies the association, separation, and completion of neural information. Cortical networks respond dynamically to varying inputs through nonlinear computation. This complex processing allows functionally relating an input to another and retrieving the entire information from a partial input. In this research, we regarded a hippocampal polysynaptic network as an arithmetic logic unit-like operator and examined how network inputs are converted to an integrated output. Functional multineuron calcium imaging (fMCI) from hippocampal slice cultures revealed the spatial pattern of individual CA1 neurons activated by stimulation of the dentate gyrus. Patterned stimulation of two independent pathways induced a persistent change in neural representation, so that stimulation of one pathway recruited neurons that had previously been responsive only to the other stimulation. These phenomena may represent information completion and association at the network level.

KAKENHI (18021008, 17689004)

## P2-k15 Phase locking among multiple oscillators as a mechanism for generating the auditory evoked peak of N100m

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In contrast with the traditional "components" theory, the most prominent peak in the auditory evoked field N100m is mostly due to phase-locking of ongoing oscillations at around 6 Hz in the auditory cortex (T.Hamada, Int. J.Psychophysiol., 2005). Power of the oscillations is also increased, however, at the timing of the peak. For explaining these facts, a model is proposed where the auditory cortex was assumed to have several oscillators, whose phases were random each other when silent, but locked at around the timing of the peak (T.Hamada, Biol.Cyb., 2006). Oscillations in the model are different from those in the Singer's "binding theory" in two respects: Firstly, the frequency is at around 6 Hz (not in the gamma range), and secondly, they are ongoing (not evoked).

## P2-k16 A spike sorting method with optimal feature extraction and clustering

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Multiunit recording techniques to acquire spiking activities of many neurons simultaneously provide useful information to understand neuronal coding. The accuracy and efficiency of spike sorting in multiunit recordings largely depend on both feature extraction from spike wave forms and clustering based on the features, since the raw data generally contain complex temporal structure with unavoidable noise. However, conventional spike sorting methods, e.g. based on principle component analysis (PCA) and maximum likelihood (ML), have some theoretical or practical limitations, and a large amount of spikes often can not be available for analysis of spike ensembles. To perform spike sorting more effectively, we propose a novel method based on matching pursuit (MP) and variational Bayesian (VB). MP, a type of wavelet transform, can detect accurate timing of signals and filter out background noise from wave patterns, and VB provides much better generalization capability then ML. We compared the accuracy and efficiency of our novel method with those of conventional methods by using artificial and experimental data.

KAKENHI 17022036, 18109041

## P2-k17 Comparison of cell-specific spike patterns among different cortical areas

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The firing rates of cortical neurons change in time, but some aspects of their *in vivo* firing characteristics remain unchanged and are specific to individual neurons. Our recent study has demonstrated that cortical neurons can be grouped into several firing types according to a measure of local variation of inter-spike intervals, which we termed $L_V$. It was also revealed that the neocortex consists of heterogeneous neurons that differ not only from one cortical area to another, but also from one layer to another in their spiking patterns. We explored more cortical areas and found a wider variety of firing type distributions. In those cases, the temporal variation of $L_V$ value for each neuron is small compared to the variation among neurons. But there is a certain tendency that the $L_V$ value is slightly modulated according to the change of firing rate. In this contribution, we search for a better coefficient representing a firing characteristic stably intrinsic to each neuron.

KAKENHI 16300068,18020015

## P2-k18 Perturbation response measurements in hippocampal CA1 pyramidal neuron based on Bayesian statistics

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Recently, phase response curves (PRCs) of single neurons have been estimated by perturbation-response experiments. PRCs are the minimum representation for describing oscillatory dynamics in neural network. Therefore, the PRC measurement is one of effective methods to bridge the gap between single neuron dynamics and network dynamics. We propose an estimation method of PRCs based on the Bayesian statistics. It is possible to theoretically design the optimum perturbation-response experiments, because observation processes for RRCs have been rigorously modeled. Results of numerical simulation showed the effectiveness of the method. We will apply the method to In-Vitro perturbation-response experiments and report results of estimation for PRCs in hippocampus CA1 pyramidal neuron.

KAKENHI18700299

## P2-k19 A prediction of synaptic polarities of neural network regulating thermotaxis and chemotaxis in C.elegans

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We constructed a neural network that was thought to regulate thermotaxis and chemotaxis in C.elegans. Polarity of each synapse (excitatory or inhibitory) was assigned at random at the beginning of simulation. Responses of all neurons in the network to an input stimulus to a thermosensory neuron or chemosensory neuron was obtained. The polarity of a synapse was selected if the potential changes of several interneurons, that are considered to determine a forward / backward movement, coincided with the polarity. The extracted polarities were fixed and others were given at random in the next simulation. This process was repeated, and we obtained the polarities of synapses that are necessary to the forward and backward movement by a temperature or chemosensory stimulation. The fixed polarities of synapses were compared to those predicted in several papers.

## P2-k20 An analysis of multi-neuronal activities and local field potentials in the subthalamic nucleus in Parkinson's disease

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High-frequency deep brain stimulation (DBS) of the subthalamic nucleus (STN) has shown the clinical efficacy on the Parkinson's disease (PD). To maximize the efficacy, the DBS target, sensorimotor area in STN, should be localized exactly. In this study, to improve the precision of physiological targeting during surgery, we analyzed the data of multi-neuronal spike trains and local field potentials (LFPs) in STN. Spike trains were obtained by the waveforms of neuronal activities recorded from PD patients with four single microelectrodes; LFPs were acquired with DBS electrode. Here, the neuronal firing rate and the amplitude of $\alpha$ and $\beta$ oscillation of LFPs are analyzed to determine the boundary between white matter and STN. In addition, the statistical analysis of interspike intervals in sensorimotor area in STN was examined.

Aihara Complexity Modelling Project, ERATO, JST

## P2-k21 Determination of parameters of voltage-gated channel of *Caenorhabditis elegans*

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*Caenorhabditis elegans* (*C. elegans*)is one of the most suitable model animal for investigation of the relationship between the connection and the function of the neural network because the information of the connection was revealed with the electronmicroscopy. On the other hand, it has been difficult to build a precise model neuron because the neuronal electrophysiological data of *C. elegans* has not been sufficient. We have been developing a precise neural model with parameters required for model of voltage-gated channels from the electrophysiological data. The parameters were obtained by scanning in the parameter space by the genetic algorithm. More precise neuronal model was obtained by improvement of the fitting to the channel of which conductance is temporally decaying. We report validity of obtained neuronal model and the possibility of the existence of unknown channel.

KAKENHI 16500185

## P2-k22 Optimization of a histogram of spike data

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In Neuroscience research, a histogram is frequently used to estimate a time-varying rate of neuronal spikes. However, the bin size that critically determines the shape of the histogram has been subjectively selected by individual researchers. We provide an objective method for selecting the bin size from spike data: the optimized histogram best represents an unknown time-varyingrate. We demonstrate the effectiveness of the method by applying it to spike data of a neuron in area MT. It is notable that the optimal bin size may become as large as the entire observation period if only a few experimental trials are available. Even in this case, our method can suggest how many more experimental trials must be performed in order to construct a histogram. We also provide a method to construct a histogram with variable-bin-sizes, which can effectively estimate a non-stationary rate.

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## P2-k23 Fine spatio-temporal interactions in multielectrode LFP signals

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Recent studies have demonstrated the occurrence of "neuronal avalanches"(Beggs & Plenz 2003) in spontaneous local field potentials (LFP) recorded from cultured and acute slices of the rat cortex, revealing a putative global property of the neural network. We now complement these results by performing a temporally finer analysis of the same data to discover local properties of the network. Our technique consists of obtaining discrete, non-linear components from the LFP signal, on which Bayesian analysis is applied. The results suggest the possibility of recovering interactions both within and across electrodes in the 1-10 ms range. Interesting examples of potential interactions are discussed, along with their validation and possible interpretations.

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## P2-k24 On a novel measure for synchrony and its application to EEG

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We propose a novel measure to quantify neural dynamics, i.e., stochastic event synchrony(SES). It is a measure for the synchrony between two event strings (a.k.a. "point processes"). Intuitively speaking, two event strings can be considered as synchronous if they are identical apart from: (i) a time shift; (ii) small deviations in the event occurrence times ("event timing jitter"); (iii) a few event insertions and deletions; (iv) small dissimilarities in the events. SES captures this intuitive concept in a quantitative way: it is a four-tuple consisting of the average time shift, the standard deviation of the timing jitter, the fraction of inserted/deleted events, and the average similarity of the events.We applied SES to the early detection of Alzheimer's disease based on EEG signals. We approximate the EEG signals as a sum of basis functions ("bump trains"), which are a specific type of event strings. We found that SES significantly improves the sensitivity of EEG for the detection of Alzheimer's disease.

## P2-k25 A fast in-silico protocol for identification of anti epileptic drug leads

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A ligand based virtual screening procedure in tandem with Molecular Modeling and Neural Network was implemented to mine chemical databases for the identification of selective ligands for GABA A alpha 2 and 3 subtypes. A target specific Pharamacophore was developed using non congeneric molecules known to exhibit functional selectivity. The robustness of the Pharmacophore was assessed statistically. The pharmacophore was queried against databases. In order to create an focused library of putative Anti epileptic molecules Kohonen SOM was used to filter the hits. Those hits, which were not distributed among the known reference compounds, were regarded as outliers because a ligand based design capitalize on the fact that ligands similar to an active ligand are more likely to be active than random ligands. This strategy might be foreseen as powerful tool for identification of anti epileptic drug leads and, particularly, for projects where receptor based design is not feasible.

CSIR, India

## P3-a01 Association of gamma-5 and gamma-7 proteins with AMPA receptors

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Gamma-1 (γ-1) is a protein identified as a small subunit of voltage-dependent calcium channel of skeletal muscles. At least seven other homologous proteins are expressed in mammalian brain. Gamma-2 (stargazin), γ-3, γ-4 and γ-8 were shown to associate with AMPA receptors and designated as transmembrane AMPA receptor regulatory proteins, however, it has not been identified which proteins are associated with γ-5 and γ-7. We generated specific antibodies to γ-5 and γ-7 and studied their biochemical properties and expression in rodent brain. We found that both γ-5 and γ-7 proteins were N-glycosylated and were highly phosphorylated in brain. AMPA receptors were coimmunoprecipitated both with γ-5 and with γ-7 from synaptosomal proteins solubilized with either CHAPS or digitonin. No specific association was observed in Triton X-100. These results suggested that γ-5 and γ-7 may also play some regulatory roles in expression or function of AMPA receptors.

## P3-a02 Frequency-dependent response property of the dendrites in the hippocampal CA1 pyramidal cells

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Depending on the behavioral state, hippocampal CA1 pyramidal cells receive very distinct patterns of synaptic input. To investigate the relationship between temporal input pattern and the dendritic responsiveness, we used frozen-noise current stimuli with different distributions of power over frequencies to the dendrites and recorded voltage response with patch clamp method. FFT analysis of the responses allowed calculation of cell membrane impedances. Cell membrane sensitivity to the stimulation with particular frequency band was estimated by the impedance value. We find frequency-selectivity characteristics in the dendrite and the soma of the rat hippocampal CA1 pyramidal cells. Our results suggested that the apical dendrites operated in a frequency-dependent manner. Differences of the operation within the dendrite could explain distance-dependent processing in the dendrites of the cell.

## P3-a03 Specialized late-spiking pyramidal neurons in layer 2 of the rat retrosplenial cortex

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Callosally projecting neurons in layer 2 of granular retrosplenial (GRS) cortex of the rat are specialized to form distinct apical dendritic bundles. We report here that they show distinct electrophysiological firing properties. Standard whole-cell patch clamp recordings using IR-DIC video microscopy were performed in coronal and horizontal brain slices of rat GRS. Neurons just below the layer 1/2 border were targeted for recording. About 90% of neurons (n=81) demonstrated a "late-spiking" (LS) firing pattern in which the initiation of the first spike was delayed 500-1000 ms from the onset of supra-threshold step current injection. The delay was eliminated by bath application of 4AP (at 30 μm, a concentration specific for Kv1.4 channels). Biocytin-filled neurons typically had a thick proximal apical dendrite with extensive, spinous branches selectively in layer 1a, a thalamic input layer. Our results establish layer 2 pyramidal neurons in GRS as a distinct subtype, presumably related to the modular and functional specializations of the GRS.

## P3-a04 Influence of conductance-input signal on spike generation and stability of rhythmic activity in rat somatosensory fast-spiking neurons

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In the cortex, a profusion of electrophysiological cell types, which form specific synaptic connections, is becoming apparent. For understanding cortical network, a quantitative understanding of the dynamics of different cell types when responding to complex, natural inputs, is an important prerequisite. In this study, we focused on one type of interneurons, fast-spiking (FS) neurons and examined their properties of synaptic conductance inputs which are most effective in evoking spikes, by injecting broad-band excitatory and inhibitory conductance inputs, and using spike-triggered reverse correlation. Our analysis showed that average conductance injection trajectories reflect both previous stimulus history and previous discharge history. We also examined the effects of noisy synaptic inputs on stability of rhythmic activity of FS neurons. The results indicated that expression of Kv3 channels could contribute the dynamical stability/instability of FS neurons.

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## P3-a05 Effects of propofol on tonic GABAergic inhibition in rat cerebral cortex

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$GABA_A$ receptor mediated two inhibition models, phasic and tonic. Propofol, widely used anesthetic, potentiate $GABA_A$ receptor function. We studied the effects of propofol on the tonic current in neocortex layer V pyramidal neurons using whole-cell patch clam techniques from 2- to 3- week old rat brain slice. Tonic current recorded in the presence CNQX, APV, CGP 55845, and TTX. Propofol cause a concentration-dependent increase in the amplitude of the tonic current. Picrotoxin (50 micM) completely blocked propofol effect on the tonic current. In contrast, bicuculline (10 micM) completely blocked tonic current induced by propofol low dose (3 micM) but not high dose (100 micM). Tonic current mediated by high dose propofol did respond $GABA_C$ receptor blocker TPMPA (30 micM) but not blocked by strychnine (10 micM). These suggest that low dose propofol mainly activate $GABA_A$ receptor and high dose mediate tonic current via both $GABA_A$ and $GABA_C$ receptors.

## P3-a06 Estrogen modulates voltage dependent potassium currents in the EGFP-tagged neurons in the hippocampus of estrogen receptor gene promoter transgenic rats

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Estrogen exerts excitatory effects on neuronal functions in several brain regions, but underlying mechanisms for the actions remain uncertain. Because voltage dependent K channels play a fundamental role in controlling neuronal excitability, we hypothesized that estrogen may modulate K conductance. In the present study we examined the effects of estrogen on voltage dependent K currents recorded from primary cultured EGFP-tagged hippocampal neurons collected from newborn estrogen receptor gene promoter transgenic rats. Acute application of higher concentrations of estrogen decreased voltage dependent K currents. Chronic application of physiological concentration of estrogen for 48 hours up-regulated K currents in cells maintained for 8 to 10 days in vitro, on the other hand, in cells maintained in vitro for 12 to 13 days estrogen down-regulated the currents. The data indicate that estrogen regulates voltage dependent K currents and exerts different roles in developing neurons.

KAKENHI(17500215)

## P3-a07 PSD-95 modulates the sensitivity of NR1/NR2A subtype NMDA receptor channels for a NMDA receptor antagonist, MK801.

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PSD-95 functionally modulates the NR1/NR2A and NR1/NR2B-subtype NMDA receptor channels. It inhibits the PKC- and Src-mediated potentiation and decreases the sensitivity for glutamine. In this study, we examined the effects of a high-affinity open channel blocker, MK-801 on the complex of PSD-95 with NR1/NR2A or NR1/NR2B NMDA channels using *Xenopus* oocyte expression system. PSD-95 increased the sensitivity of the NR1/NR2A for MK-801 but not of NR1/NR2B channels. Activation of PKC increased the sensitivity of NR1/NR2A channels for MK-801 but not that of the complex with PSD-95. Deletion mutant of PDZ1 or PDZ2 of PSD-95 did not have such an effect on the NR1/NR2A channels, whereas substitution of N-terminal two cysteines to serines showed the same effect on the channels as wild type PSD-95. These results indicate that the PSD-95 modulates the sensitivity of NR1/NR2A channels for MK-801 through the interaction between the C-terminal of the receptor and PDZ1or PDZ2 of PSD-95.

## P3-a08 Functional roles of voltage-gated $Na^+$ channels for pain sensation in different subpopulations of small dorsal root ganglion neurons

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Small neurons of the dorsal root ganglion (DRG) that are associated with unmyelinated C fibers or thinly myelinated Aδ fibers mediate nociceptive information. Small DRG neurons can be divided neurochemically into two subpopulations of isolectin $B_4$ ($IB_4$)-positive and $IB_4$-negative neurons. It has been shown that these two subpopulations possess unique neurotransmitters and receptors, and may play distinct roles in pain sensation. Pain sensation is mediated by voltage-gated $Na^+$ channels expressed in small DRG neurons, which are responsible for the upstroke of action potentials. However, it is not fully understood how these subtypes of $Na^+$ channels are distributed in distinct subpopulations of small DRG neurons and are involved in propagation of pain sensation. In this study, therefore, we investigated functional roles of $Na^+$ channel subtypes in action potential electrogenesis in different subpopulations of small DRG neurons.

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## P3-a09 Superoxide retards the inactivation of voltage-gated $Na^+$ currents

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Our previous study showed that Lucifer Yellow CH, a fluorescent cell marker dye, retarded the inactivation of voltage-gated $Na^+$ currents irreversibly, and suggested that an unidentified radical species produced by the illumination for microscopy was responsible for the retardation (J. Physiol., 2003). We investigated the properties of this radical species with mouse taste bud cells under patch clamp conditions. Here, we show that the radical species is superoxide, as follows. The mean lifetime of the radical species calculated from the retardation effect was ~6 min in the absence of cell constituents, but less than ~60 s in the presence of them. The addition of cell constituents, typically superoxide dismutase (SOD), an enzyme specifically decomposes superoxide, inside the cell inhibited the retardation effect, but that of heat-inactivated SOD did not. The roles of superoxide in the retardation effect on $Na^+$ channels are discussed.

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## P3-a10 SCN1A missense mutation associated with infantile partial epilepsy

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Mutations in SCN1A, encoding the human neuronal voltage-gated sodium channel alpha1 subunit (Nav1.1) were linked to severe myoclonic epilepsy in infancy and related epileptic syndromes. Here we report the SCN1A missense mutation associated with infantile partial epilepsy in two unrelated patients. The SCN1A mutant was constructed in a recombinant human SCN1A and then heterologously expressed in HEK293 cells along with the human beta1 and beta2 sodium channel accessory subunits. Whole-cell patch-clamp recording was used to define biophysical properties of mutant and for comparison with the wild-type channel. SCN1A missense mutation exhibited incresed channel availability, faster recovery from inactivation and increased persistent sodium current. Infantile partial epilepsy can be caused by gain-of-function of Nav1.1.

KAKENHI (18591154)

## P3-a11 Analysis of a voltage-gated proton channel, VSOP in microglia

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In phagocytosis, voltage-gated proton channel has been believed to regulate reactive oxygen species generation and phagosomal/cytoplasmic pH that are critical for pathogen killing and elimination of apoptotic cells. But, its molecular basis has long been elusive. We identified the voltage-gated proton channel, called VSOP (voltage-sensor only protein). To understand the function of VSOP in phagocytosis, we examined the expression of VSOP in microglia of mouse. VSOP was expressed in microglia. We next introduced cryoinjury to brain to test the stimulus-dependent expression of VSOP. VSOP was expressed in macrophage/microglia in injured brain. We are now studying subcellular localization of VSOP in microglia.

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## P3-a12 Inhibition by opioids of compound action potentials in frog sciatic nerves

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Tramadol and its metabolite mono-*O*-demethyl-tramadol exhibit a high affinity to μ-opioid receptors while the former is much more effective in inhibiting compound action potentials (CAPs) than the latter, suggesting no involvement of opioid receptors in the CAP inhibition. To address further this issue, we examined the effects of opioids (morphine, codeine, dihydrocodeine and ethylmorphine) and a well-known local anesthetic cocaine on CAPs by applying the air-gap method to frog sciatic nerves. Opioids dose-dependently reduced CAP amplitudes with a potency sequence of ethylmorphine ($IC_{50}$ = 4.6 mM) > codeine > dihydrocodeine > morphine in a manner resistant to opioid-receptor antagonist naloxone. This action of opioids was much less than that of cocaine ($IC_{50}$ = 0.80 mM). Considering the CAP inhibition and opioid's chemical structure, it is suggested that the substituted groups of -OH bound to the benzene ring of morphine, codeine and ethylmorphine may play an important role in producing nerve conduction block.

## P3-a13 Interaction between h-channels and glutamate receptor channels in the mesencephalic trigeminal neurons

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To investigate the physiological roles of glutamate receptors (GluRs) in the mesencephalic trigeminal neurons, the current responses to puff application of glutamate (Glu) were recorded under the whole-cell voltage-clamp condition. When the peak amplitudes were plotted against the holding potentials, an N-shaped *I-V* relationship was obtained. At < -40 mV, the biphasic responses consisting of inward and outward components were observed. Glu-induced outward component was sensitive to ZD7288 and $Cs^+$, indicating an involvement of h-current ($I_h$) in generating the outward current. Provided that GluR channels shared the microdomain with h-channels, it is possible that a transient increase in $[Na^+]_i$ in the microdomain by activation of GluR channels would cause a transient reduction of $I_h$ due to the negative shift of its reversal potential, leading to a generation of the outward component of the biphasic response where the N-shaped *I-V* relationship can be seen. Thus, the biphasic response was seen at < -40 mV.

## P3-a14 Left-right asymmetry of mouse hippocampal pyramidal neurons

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The Number of synaptic receptors is an essential factor that influences on synaptic transmission. We previously reported left and right difference of NR2B contents in the mice CA1 hippocampal area. After synaptic inputs from contralateral hippocampus were eliminated by transaction of the ventral hippocampal commissure, antagonizing effects of NR2B blocker were larger in the left than right hippocampus stratum radiatum. Purification of postsynaptic density protein from those animals, followed by immunoblot also showed 1.5 times higher NR2B immunoreactivity in the left. However, postembedding method failed to detect this difference in wildtype mice probably because of steric hindrance hampering detection of two NR2B subunits in single channels. Here, we have tried SDS freeze-fracture immunolabeling method to overcome this problem. The density of immunogold particles for NR2B in synaptic areas was 1.5 times higher in the left than right CA1 stratum radiatum, consistent with the results from previous analysis. This study is the first demonstration of left-right asymmetrical contents in individual synapses.

18700344

## P3-a15 Active zone protein RIM1 functionally associates with presynaptic VDCCs

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The spacing between voltage-dependent calcium channels (VDCCs) and vesicles at active zones is thought to influence the dynamic properties of synaptic transmission. Here, we demonstrate a novel molecular interaction between active zone protein RIM1 and VDCC $\beta$-subunits, that controls neurotransmitter release. Biochemical experiments showed that RIM1 associated with VDCC $\beta$-subunits via its C-terminus. In neuroendocrine PC12 cells, acetylcholine release was significantly potentiated by the full-length, N-terminus, and C-terminus constructs, but the proportion of membrane-docked vesicles was enhanced only by the full-length. Thus, RIM1 associated with VDCC $\beta$-subunits near the active zone supports neurotransmitter release via anchoring of neurotransmitter-containing vesicles in the vicinity of VDCCs.

## P3-a16 The RIM1 mutation associated with an autosomal dominant cone-rod dystrophy, CORD7, alters effects of RIM1 on VDCC currents

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Recent genetic analyses have revealed an important association of the gene encoding the Rab3A-interacting molecule (RIM1) with CORD7, an autosomal dominant cone-rod dystrophy. However, the effects of RIM1 mutation which associated with CORD7 have remained unclear. We appeared effects of RIM1 on VDCC currents, anchoring of neurotransmitter-containing vesicles and neurotransmitter release. In this study, we demonstrate that the mouse RIM1 arginine-to-histidine substitution R655H, which corresponds to human CORD7 mutation, impaires RIM1 function in ACh release and regulation of VDCC currents. Thus, we suggest that phenotypes of familial cone-rod dystrophy with RIM1 mutation can be at least partly attributable to defects in regulation of presynaptic VDCC currents, presumably resulting in retinal deficits.

## P3-b01 Functional impact of RIM family sharing $C_2B$ domain on gating of voltage-dependent $Ca^{2+}$ channels

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RIM1 is multidomain adaptor proteins that was discovered as putative effectors for Rab3. RIM1 and RIM2 consist of an N-terminal $Zn^{2+}$-finger, PDZ, $C_2A$, and C-terminal $C_2B$ domain. On the other hand, RIM3 and RIM4 consist of only a $C_2B$ domain. While $C_2B$ domain is common to all RIM family, the function remains unknown. Moreover, the physiological roles played by RIM3 and RIM4 remain unclear. Here we show, $C_2B$ domains of all RIM family associate with voltage dependent $Ca^{2+}$ channel (VDCC) $\beta$ subunits to modulate currents of the VDCCs. The most prominent RIMs effect on VDCC currents was observed on inactivation parameters. Inactivation kinetics were markedly decelerated resulting in the predominance of high voltage inactivation and an inactivation-resistant current component in the 2-s prepulse protocol. This is the first report of common function for the RIM family.

## P3-b02 Mechanisms underlying subunit recognition and channel assembly in KCNQ channels

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KCNQ2 and KCNQ3 are voltage-gated $K^+$ channel $\alpha$ subunits expressed in the central nervous system. KCNQ2 can form homomeric channels, but co-expression with KCNQ3 robustly increases the current expression level. Recent reports from several groups have suggested that two separated cytoplasmic coiled-coil domains (CCD) are important for the subunit recognition and channel assembly. We reported previously that the deletion of the second CCD did not affect the properties of homo KCNQ2 channels but abolished the robust current increase by co-expression of KCNQ3. To understand the mechanism for it, we substituted CCD of KCNQ2 and KCNQ3 with CCD from KCNQ1 or GCN4-pLI, a known peptide of four-stranded CCD, and observed that the robust current expression seemed to be induced if only four-stranded coiled-coil structure was formed. We also identified several inter subunit salt bridges on CCD required for the subunit recognition. Recent structural data of CCD from KCNQ4 have also supported our findings.

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## P3-b03 Increased $Ca^{2+}$ channel currents in cerebellar Purkinje cells of the ataxic Groggy rat

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The groggy rat (strain name; GRY) is an autosomal recessive neurological mutant found in a closed colony of Slc:Wistar rats, whose phenotype is characterized by movement disorder, ataxia and unstable gait first observed at around postnatal day 15. Recently, genetic analysis has identified the missense (M251K) mutation in the coding region of Ca,2.1 $\alpha_1$ subunit in GRY rat. Here, we have evaluated the functional properties of $Ca^{2+}$ channel currents in the cerebellar Purkinje cells acutely isolated from GRY rats. Our results demonstrate that high-voltage-activated (HVA) currents in Purkinje cells of GRY rats are distinctly upregulated with respect to the current density, voltage-dependence of activation and inactivation, whereas those of low-voltage-activated (LVA) currents remain unchanged. These results suggest that increased HVA $Ca^{2+}$ channel function underlies the cerebellar dysfunction and ataxic phenotype of GRY rats.

## P3-b04 Effect of RIM 1 on inactivation of neuronal voltage-dependent calcium channels

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Active zones (AZs) are highly specialized sites for release of neurotransmitter in presynaptic nerve terminals. The spacing between voltage-dependent calcium channels (VDCCs) and synaptic vesicles at AZs is thought to influence the dynamic properties of synaptic transmission. Recently we have demonstrated a novel molecular interaction between an AZ scaffolding protein, RIM1, and VDCCs. To elucidate the functional significance of this direct coupling, we characterized whole-cell $Ba^{2+}$ currents through recombinant VDCCs. The RIM1 induced a pronounced deceleration of inactivation rate and a depolarizing shift of the inactivation curve. Currents evoked by trains of action potential waveforms, a more physiological voltage-clamp protocol used particularly to reveal closed-state inactivation, further support profound suppression of voltage-dependent inactivation by the RIM1. Thus, the RIM1 coordinates calcium signaling and spatial organization of molecular constituents in presynaptic AZ.

## P3-b05 Activity-dependent relief of G-protein-dependent inhibition of presynaptic P/Q-type $Ca^{2+}$ channels

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Activation of GPCRs reduces $Ca^{2+}$ influx via G $\beta\gamma$-subunits binding with voltage-gated $Ca^{2+}$ channels, thereby decreasing neuro transmitter release. Though the depolarization-induced relief of G-protein-dependent inhibition is well-known phenomenon, it is not known whether action potential can relief the inhibition. To address this issue, we recorded N- and P/Q-type $Ca^{2+}$ currents directly from calyces of Held nerve terminals. The G-protein-dependent inhibition of $Ca^{2+}$ channels was elicited by intracellular application of GTP$\gamma$-S or extracellular application of the $GABA_B$ receptor agonist baclofen. The duration and potential of depolarizing pulse required for disinhibition was shorter and lower in P/Q- than N-type channels, suggesting the higher affinity of G $\beta\gamma$-subunit for N-type channels. The high frequency trains of action potential waveform can relieve the inhibition in P/Q-type channels. We conclude that the developmental switch from N- to P/Q-type channels endows presynaptic terminals with the property of the activity dependent relief of G-protein-dependent inhibition.

JSPS Research Fellowships

## P3-b06 Effect of ciprofloxacin and biphenylacetic acid on voltage-gated ion channels in cultured rat hippocampal neurons

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Binding and electrophysiological studies have shown a novel antagonist interaction between ciprofloxacin and biphenylacetic acid (BPAA) at $GABA_A$ receptors. In the present study, the effect of these two drugs on voltage-gated $K^+$, $Na^+$ and $Ca^{2+}$ channels in the cultured rat hippocampal neurons was investigated using the whole-cell patch clamp techniques. BPAA (100 μM), ciprofloxacin (100 μM) and the combination of BPAA and ciprofloxacin (100 + 10 μM) had little or no effect on the $K^+$ or $Na^+$ currents. While ciprofloxacin had no effect, BPAA and the combination of BPAA plus ciprofloxacin did increase the amplitude of $Ca^{2+}$ currents. Voltage-dependentnce of activation was not however changed by BPAA remaining at approximately $V_{1/2}$ = -15 ± 1.4 mV in control and in the presence of the drug. These data show that ciprofloxacin and BPAA have little or no effects at voltage-gated $K^+$ and $Na^+$ channels but BPAA can enhance $Ca^{2+}$ currents in hippocampal neurons.

## P3-b07 Identification of the Ginsenoside Interaction Site in Human Kv1.4 Channels

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Voltage-gated K+ channels play critical roles in a variety of physiological processes in nervous systems and heart. We found that Rg3-induced inhibition of hKv1.4 channel currents was concentration-dependent and reversible. Raising extracellular [K+]o induced K+ activation by enhancing outward K+ currents and abolished the effect of Rg3 on hKv1.4 channels. We examined whether the K531 residue (K+ activation and external TEA binding site) is also involved in Rg3 interactions. Mutation of K531 to K531Y abolished the effects of Rg3 and rendered channels sensitive to TEA. Examinations of the effect of TEA on Rg3-induced regulation of wild-type channels and K531Y mutant channels revealed that the K531 is an overlapping interaction site for Rg3 and TEA. These results indicate that Rg3 is a novel hKv1.4 channel regulator that functions by interacting with the K531 residue.

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## P3-b08 Expression and localization of TREK-1 channel in Schwann cells

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The Schwann cell has widely studied for repair lesioned peripheral nerve and spinal cord. In the peripheral nervous system, the neuron-glial signaling is mediated by axon electrical activity through ion channels. TWIK-related $K^+$ channels (TREKs; TREK-1 and TREK-2) are functionally expressed in dorsal root ganglion (DRG) neurons. Whether TREKs are expressed in Schwann cells has not yet been reported. Here we report the expression and localization of TREK-1 and TRPV1 in Schwann cell-like glial (SCLG) cells obtained from rat DRG. Glial cells obtained from DRG demonstrated positive staining with antibodies of Schwann cell markers (S-100 and GFAP). Immunostaining data showed that TREK-1 was highly expressed in SCLG cells, but was localized only in nucleus. However, TREK-1-like channel was functionally expressed and activated by arachidonic acid, low pH, and stretch as judged by single channel recording. These results suggest that TREK-1 may serve for background $K^+$ conductance in SCLG cells.

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## P3-b09 Activation of presynaptically silent synapses - one of underlying mechanisms enhancing mossy fiber transmission in the hippocampus

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The activation of presynaptically silent synapses is hypothesized to be one of mechanisms of the synaptic enhancement. This hypothesis was tested for the PKC-dependent enhancement of mossy fiber (MF)-CA3 synaptic transmission in the hippocampus. Individual large MF boutons were identified in the acute slice of hippocampus and the activity-dependent changes of synaptopHluorin were measured. We found that the individual MF boutons are variably responsive to PKC. Among them some presynaptically silent boutons became active. The individual MF bouton consists of dozens of active zones. It is possible that each active zone has at least two states, releasable and non-releasable. These MF boutons may have mostly non-releasable active zones which become releasable by a PKC-dependent mechanism. All animal procedures were conducted in accordance with the guiding principles of NIH.

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## P3-b10 cAMP signaling cascade is reduced in hippocampal LTP of HPC-1/syntaxin 1A knock-out mice

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The membrane protein HPC-1/syntaxin 1A believed to plays a key role in synaptic vesicle exocytosis. Previously we reported that HPC-1/syntaxin 1A knock-out mice had impaired learning and behavioral abnormality which was recovered by administration of serotonin selective reuptake inhibitor. Moreover, although the properties of basic synaptic transmission of the matured hippocampal neurons in vitro was normal, the knock-out mice exhibit impairment in induction of long-lasting potentiation in the CA1 of hippocampal slice. In this study, we examined the effect of several agents which affect signal transudation pathways mediating LTP in the CA1. Among them the adenylyl cyclase activator forskolin rescued this impairment of LTP in knock-out mice. This result suggests that cAMP/protein kinase A signaling cascade may be disturbed in LTP of knock-out mice.

## P3-b11 RGMa inhibition enhances synapse formation of the cortico-spinal axons after spinal cord injury

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Several proteins have been identified as inhibitors of axonal regeneration followinginjury of the adult vertebrate central nervous system. The repulsive guidance molecule(RGMa) is considered a potent myelin-derived neurite outgrowth inhibitor. In rats,RGMa inhibition enhances the growth of injured axons and promotes functionalrecovery after spinal cord injury (SCI). Here, we demonstrate that RGMa inhibitioninduces synaptic rearrangements of spared axonal projections after SCI. Intrathecaladministration of a function-blocking antibody to RGMa enhances anatomical synapseformation of the corticospinal tract in the cervical region of rats with thoracic spinalcord hemisection. These findings suggest that the suppression of synapticrearrangements as well as axon growth inhibition in the adult spinal cord maycontribute to the limitation of functional recovery after SCI.

## P3-b12 Trapping of the Vesl-1S protein into dendritic spines is input-specific

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Input-specificity of late-phase plasticity involves yet-unknown mechanisms that permit somatically synthesized plasticity-related proteins (PRPs) to function locally in the activated synapses. Although synaptic tagging hypothesis has been proposed to solve this central problem, molecular identity of the tag, signals that tag a synapse and PRPs that substantiate the hypothesis are not known. We hypothesized that the regulated entry of PRPs into dendritic spines works as a synaptic tag. We previously reported that entry of Vesl-1S (Homer 1a)-EGFP (VE) proteins into spines of rat hippocampal neurons in culture was regulated by pre- and postsynaptic activities, and fulfilled some standards of synaptic tag. Here we applied microperfusion techniques to test whether VE trapping is input-specific. NMDA receptors in limited numbers of spines within a diameter of 10 micrometers were stimulated by microperfusion, which activated VE trapping only in the stimulated area. These results further supported that VE trapping works as a synaptic tag.

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## P3-b13 Proteolytic cleavage of telencephalin by plasmin in mouse visual cortex

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Ocular dominance (OD) plasticity reflects a transient loss of dendritic spines in the binocular zone of mouse visual cortex (VC) mediated by tissue-type plasminogen activator (tPA) (Mataga et al, 2004), whose molecular substrates remain unknown. The cell adhesion molecule, telencephalin (TLCN), is a negative regulator of spine maturation (Matsuno et al., 2006) and contributes to hippocampal plasticity (Nakamura et al., 2001). Here, we examined tPA-mediated proteolysis of TLCN as a potential substrate in the VC. TLCN expression emerged before eye-opening and independent of sensory experience. Partial proteolytic cleavage of TLCN was observed in vitro in a dose- and time-dependent manner by plasmin, but not by tPA alone or in the presence of serine protease inhibitor, aprotinin. TLCN was more sensitive to plasmin-dependent proteolysis than another candidate substrate, NCAM. Abnormal accumulation of TLCN was seen in tPA KO beyond the critical period (>P28). Thus, TLCN may be a site of tPA action during OD plasticity.

RIKEN BSI, KAKENHI

## P3-b14 Rapid morphological changes of dendritic spines induced by corticosteroids in rat hippocampus

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Modulation of hippocampal synaptic plasticity by corticosteroids has been attracting much attention, due to its importance in stress responses. Dendritic spines are essential for memory storage processes. We investigated the effect of dexamethasone (DEX), a specific agonist of glucocorticoid receptor, on density and morphology of dendritic spines in adult male rat hippocampus by imaging of Lucifer Yellow-injected spines in slices. The application of 100 nM DEX induced rapid modulation of the density and morphology of dendritic spines in CA1 pyramidal neurons within 1 h. Because the presence of cycloheximide, an inhibitor of protein synthesis, did not suppress the DEX effect, these responses are probably non-genomic and not mediated via slow genetic processes. We further examined the effects of corticosterone, on dendritic spine in hippocampal neurons. We found that corticosterone also significantly increased the density of CA1 dendritic spines and changed the morphology of dendritic spines.

## P3-b15 Time course of the neuropathic pain-evoked synaptic potentiation in the central amygdala of the rat

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The spino-parabrachio-amygdaloid (SPA) pathway plays the principal role in the expression of pain-related emotion. We have demonstrated that the synaptic transmission in the laterocapsular part of the central amygdala (CeLc) is potentiated in the chronic neuropathic pain model (Ikeda et al., Pain, 2007). To analyze the time course of this potentiation, we recorded EPSCs in the CeLc neurons evoked by parabrachial afferent stimulation in the brain slice of the rats that underwent left L5 spinal nerve ligation. At 24 hours after ligation, side-specific potentiation of EPSC was not apparent despite manifestation of unilateral allodynic responses. On the second day, the EPSC amplitude of the neurons in right CeLc was significantly larger than that in the left CeLc, which was however not correlated with the allodynic responses in each animal. It is suggested that the early phase of the synaptic potentiation in CeLc is triggered by persistent activation of SPA pathway for 1-2 days by neuropathic pain.

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## P3-b16 Isoflurane bidirectionally modulates the hippocampal paired-pulse responses *in vivo*.

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We studied the effects of isoflurane, a volatile anesthetic, on hippocampal synaptic properties in vivo, by using chronically electrode-implanted rats, measured in the hippocampal CA1 field by stimulating Schaffer collaterals. Low concentration of Isoflurane increased paired-pulse facilitation, accompanied with the depressed excitatory postsynaptic potentials. These changes indicate the inhibition of glutamate release by isoflurane, as are evident in previous in vitro studies. Isoflurane-anesthetized rats exhibited higher PSA values along with EPSP slope, indicating the greater synaptic efficacy, which may reflect the suppression of hippocampal inhibitory circuitry. High concentration of isoflurane inhibited the increase of PPF. The enhancement of synaptic efficacy and the bidirectional paired-pulse responses in this in vivo study are not shown previous in vitro studies, suggesting the crucial role of afferent intrinsic pathway on isoflurane-mediated synaptic properties in the hippocampus.

## P3-b17 Experience-dependent spine plasticity in the hippocampus

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The morphological plasticity of dendritic spines suggests fine-tuning of network connectivity. However, little is known about the relationship between spine plasticity and experience-dependent neuronal activities *in vivo*. Here we simultaneously imaged spines and Arc (activity-regulated cytoskeleton-associated protein, as a putative marker of neuronal activities) expression using Thy1-mGFP TG mice after spatial exploration in novel environment. After 15 min and 60 min of exploration, Arc was expressed in one third of hippocampal CA1 pyramidal cells, probably reflecting the neuronal activities during the exploration. We found that after 60 min of exploration, the spine density is lower and the mean spine-head size is larger in Arc-positive cells compared with Arc-negative cells, although these parameters showed no difference after 15 min of exploration. These results are the first evidence that spine plasticity *in vivo* can be induced as short as 60 min selectively in the subpopulations of neurons.

## P3-b18 Acute administration of the neurosteroid DHEAS primes the induction of NMDA-receptor independent LTP in rat hippocampal slices

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We have reported that chronic administration of dehydroepiandrosterone-sulfate (DHEAS, 20 mg/kg for 7 days) facilitates LTP induction in the hippocampal dentate gyrus via a σ1 receptor/src dependent phosphorylation of NMDA receptor (Chen et al, 2006a, 2006b). To know detailed mechanisms of the DHEAS effects on synaptic transmission and plasticity, the present study examined the effects of acute administration of DHEAS to rat hippocampal slices. A low dose of DHEAS (0.1μM for 10 min) caused a transient increase in the EPSP amplitude without altering PPF, which terminated within an hour. After this response, robust LTP could be induced by a subthreshold tetanus of 20 pulses at 100 Hz that cannot induce LTP in control slices. Surprisingly, this LTP was NMDA receptor-independent and was blocked by ryanodine and the mGluR5 receptor inhibitor MPEP (10μM).

## P3-b19 Neurosteroids rapidly modulate synaptic plasticity of hippocampus at CA1 region.

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It is well known that stress reduces several activity of brain. Especially, hippocampus is the largest target of stress. These phenomena are caused by glucocorticoids which are synthesized at adrenal when suffering stress. On the other hand, 17 $\beta$ estradiol is one of the neuro protective factors and rescues neural death caused by several neurotoxins,such as $\beta$-amyloid,glutamate,glucocorticoids. In this study, we focused attention on the acute effects of steroid hormones and researched the effects of glucocorticoids and estradiol on rat hippocampal long term potentiation(LTP), which is the index of learning and memory. The results was that corticosterone(glucocorticoid of rat) acutely reduced LTP via glucocorticoid receptor. 17 $\beta$ estradiol rescued this reduction via estrogen receptor $\alpha$ and $\beta$. So we found that 17 $\beta$ estradiol affected not only neuro protection but synaptic protection from stress-induced suppression of synaptic transmission.

## P3-b20 Inverted-U dopamine D1 receptor actions in NMDA receptor-dependent plasticity at hippocampal-prefrontal synapses in vivo

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The rat hippocampal-medial prefrontal cortex (mPFC) synapses develop NMDA receptor-dependent long-term potentiation (LTP), which dopamine D1 receptor action upregulates. However behavioral implementation of working memory expresses the synaptic depression. In vivo microdialysis in the mPFC demonstrated that either NMDA or D1 antagonist (D-APV or SCH23390) completely blocked induction of long-term depression (LTD), whereas a D2 receptor antagonist (sulpride) did not. Two kinds of D1 agonist paradoxically attenuated the early stage of partially-induced LTD. SKF81297 ceased to attenuate at the late stage and further reinforced the saturated LTD 1 hour after washout, but SKF38393 did not. On the other hand, induction of LTD did not vary mPFC extracellular dopamine, measured by HPLC at a time resolution of 6 min, suggesting that the spontaneous or active level of dopamine release regulates D1 receptors in the mPFC, preserving LTD or LTD in this pathway, respectively. The bidirectional function between-level crossover appears to underlie the inverted-U D1 receptor actions.

LRI (JCIA), KAKENHI (16530484)

## P3-b21 Spatiotemporal asymmetry of associative synaptic plasticity in fear conditioning pathways

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Input-specific long-term potentiation (LTP) in afferent inputs to the amygdale serves as essential function in the acquisition of fear memory. Factors underlying input specificity of synaptic modification implicated in information transfer in fear conditioning pathways remain unclear. Here we show that the strength of naïve synapses in two auditory inputs converging on a single neuron in the lateral nucleus of the amygdale (LA) is only modified when a postsynaptic action potential closely follows a synaptic response. The stronger inhibitory drive in thalamic pathway, as compared with cortical input, hampers the induction of LTP at thalamo-amygdala synapses, contributing the spatial specificity of LTP in convergent inputs. These results indicate that spike timing-dependent synaptic plasticity in afferent projections to the LA in both temporarily and spatially asymmetric, thus providing a mechanism for the conditioned stimulus discrimination during fear behavior.

## P3-b22 Functional significance of perineuronal nets; neuroprotection and plasticity

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Perineuronal nets (PNNs) consisting of chondroitin sulfate proteoglycans (CSPGs) and hyaluronic acids are known to be associated with distinct neuronal population in mammalian brain. In present study, we examined the function of neuroprotective action of PNNs against amyloid $\beta$ protein (A$\beta$) in cultured cortical neurons, and the effect of chondroitinase ABC (ChABC) on Fos expression in GABAergic interneuron in vivo. In vitro study, the application of A$\beta$ to cultured neurons induced apoptosis on PNNs-negative neurons, however, PNNs-positive neurons survived. In vivo study, immobilization stress caused Fos expression in the primary somatosensory cortex (S1). There was no significant difference of total number of Fos-positive neurons in the S1 after ChABC injection, however, double labeling study of Fos and GABAergic interneuron revealed that the percentage of Fos-positive neurons in GABAergic interneurons was fewer than control. These results indicate that PNNs are concerned with neuroprotection against A$\beta$ and neural activity of GABAergic interneuron.

## P3-b23 Effects of chronic blockade of NMDA receptors during synaptogenesis on glutamate receptor-mediated currents in cultured hippocampal neurons

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Chronic blockade of neuronal activity induces scaling up of AMPA receptor (AMPAR) -mediated miniature currents (mEPSCs). In the present study, we showed scaling up of AMPAR-mediated mEPSCs induced by chronic (48 h) treatment with AP5 in cultured hippocampal neurons without affecting inter-event-interval of mEPSCs, using whole-cell patch-clamp technique. The amplitude of mEPSCs in AP5-treated neurons was significantly larger than that in control neurons, whereas no difference in their frequencies. From a pharmacological study, GluR2-lacking AMPARs were unlikely involved in the increment of mEPSC amplitude. Since we have previously shown immunocytochemically that NR1 subunit of NMDA receptor is accumulated at dendritic spines by the same chronic AP5 treatement, we are now analyzing the change in NMDA-induced whole-cell currents and in the NMDAR-mediated mEPSCs.

## P3-b24 Alteration of lipid and protein composition in raft under continuous KCl stimulation

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The existence of microdomains (rafts) composed of a special lipid structure may play important roles in the formation of a place for transmitting functional signals. We separated and refined raft fractions, and examined the lipid and protein composition focusing on ErbB tyrosine kinase and related molecules under the condition of low and high KCl concentration in cerebellar granule cells.The conditions of a cholesterol/sphingolipid depletion at high K and low K led to loss of synapse-like structure, and the remaining punctuated signals became enlarged, compared with high K. In addition, our results indicate the possibility that at low K, membrane cholesterol and sphingolipid concentrations are low, while at high K, membranes can be formed that are rich in cholesterol and sphingolipids.

## P3-b25 Developmental change of heterogeneous distribution of the functional synaptic connections in a rat hippocampal dissociated neurons

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To elucidate brain function, it is important to analyze dynamics and interactions between neurons in network. For the purpose, we developed a reconstructed network of dissociated rat hippocampal neurons on multielectrode array dish. Using the system, we investigated the spatio-temporal pattern of spontaneous action potentials. Interestingly, each culture contained hub-like neurons with many functional connections. The neuronal networks cultured on the dish were not random networks but heterogenerously reconstructed networks. In addition, after the developmental stage with transient high-frequency-bursting activity of action potential (HFB), it increased gradually that variance in frequency of spontaneous action potential between each electrode. These results suggest that dissociated rat hippocampal neurons formed a particular self-assembled neuronal network, and synaptic plasticity induced by HFB was critical for the formation of the network structure.This work was partially supported by KAKENHI 18048043.

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## P3-b26 Effects of ZSET1446 on hippocampal spine density in female mice

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In previous studies, a newly synthesized drug ZSET1446 improved memory performance in castrated male and female mice. Estrogen also improved memory performance in castrated female mice, which is well supported by a theory that high spine density (SD) of hippocampal CA1 dendrites contributes acceleration of the memory performance. However, no report of SD in ZSET1446 administrated mice is available yet. In this study, we examined the effects of ZSET1446 on SD with Golgi stain in MK-801 (N-methyl-D-aspartate receptor (NMDA-R) antagonist) injected or medial septum lesioned, castrated female mice. ZSET1446 increased SD in saline control group. Furthermore, ZSET1446 increased SD in the medial septum lesioned group but not in the MK-801 injected animals. These results indicate that memory system is morphologically changed by administration of ZSET1446 not through cholinergic fibers originated from the medial septum but through NMDA-R.

## P3-b27 Developmental changes of presynaptic glycine receptor expression on hippocampal mossy fiber boutons

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Using direct recordings from mossy fiber boutons (MFBs) in rat hippocampal slices (34°C, $V_H$=-80mV), we demonstrate strychnine sensitive glycine-receptor-mediated presynaptic currents. In symmetrical chloride conditions, puff-application of glycine (1 mM) to MFBs evoked inward currents which decreased significantly in amplitude during postnatal development (P12: 51.7±4.4 pA, n=9; P23-26: 17.1±4.6 pA, n=9; P90-100: 10.8±3.4 pA, n=3, ANOVA $p<0.001$). Single channel recordings revealed a major single channel conductance of the presynaptic glycine receptor of about 53 pS (P=12, symmetric chloride). Thus, we estimate a glycine receptor density between 8 and 15 receptors per MFB in immature rats (P12) when presynaptic glycine receptor density was highest. The marked developmental decline of presynaptic glycine receptor expression suggests a specific role of presynaptic glycine receptors in the maturation of the hippocampal mossy fiber system.

## P3-b28 Characteristics of $Ca^{2+}$ responses induced by serotonin in differentiated NG108-15 cells

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We have reported that the response of $[Ca^{2+}]_i$ evoked by serotonin (5HT) and bradykinin was significantly augmented in differentiated NG108-15 cells. In this work, we investigated the characteristics for the enhanced response of $[Ca^{2+}]_i$ induced by 5HT in differentiated cells. The results were as follows. 1) 5HT-induced $Ca^{2+}$ response was inhibited by tropisetron (5-$HT_3$ receptor blocker), but not by other types of 5-HT receptor blocker, 2) 5HT-induced $Ca^{2+}$ response was mainly inhibited by calciseptine (a L-type $Ca^{2+}$ channel blocker), but not by other types of $Ca^{2+}$ channel blockers and TTX (a voltage-sensitive $Na^+$ channel blocker), 3) when the extracellular sodium ion was deprived by exchange with choline chloride or N-methyl D-glucamine, 5HT-induced $Ca^{2+}$ response was extremely inhibited. These results suggested that 5HT-induced $Ca^{2+}$ response in differentiated NG 108-15 cells was mainly due to the activation of L-type $Ca^{2+}$ channels followed by the influx of extracellular $Na^+$ via 5-$HT_3$ receptors, but not via voltage sensitive $Na^+$ channels.

## P3-b29 AFM structural observation of an ionotropic glutamate receptor reconstituted into artificial lipid bilayer

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Ionotropic glutamate receptors (iGluR) play an important role in signaling in the central nervous system, but their structure and function remain unclear. Atomic force microscopy (AFM) is a unique measurement technique which enables molecular imaging with nanometer resolution in a physiological environment, and it is able to observe functional biological molecules such as receptors and ion channels. In this study, we succeeded in observing the external structure of iGluR by AFM. iGluR was purified from over-expressed insect cells and reconstituted into a lipid bilayer by dialysis. We confirmed the protein as iGluR and determined its orientation after reconstitution using two different types of antibody. We also examined its conformational change by agonist. We believe that this is the first study to observe an iGluR protein in liquid, and it would be an useful tool for biological molecules.

## P3-b30 Intramolecular hydrocarbon chain of odorant may control switching between off and on signaling states of the odorant receptor S6

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As a piece of jigsaw puzzle of the odor encoding at odorant receptors (ORs), we need to know a ligand profile of a mirine olfactory receptor, OR-S6. Saito et al. (2004) had reported that the RTPs promote functional expression of ORs in HEK293T cells. Here, we constructed the heterologous functional expression system for the OR-S6 in HEK293 cells. Based on structure similarities to two agonists, octanedioic acid (cc8) and nonanedioic acid (cc9), we extended the odorant panel to include possible agonists and antagonists. The observed ligand profile was consistent with the reported one, and showed that cc9 was the most potent agonist among seven series of compounds. Furthermore, we found several antagonists of OR-S6. Comparison of the agonists with the antagonists indicated that the intrahydrocarbon chain of the odorant may control switching between off and on signaling states of OR-S6. These results also suggested that potencies of ligands are dependent on molecular length, functional groups, and interactions between receptor and medial part of odorant.

## P3-b31 Optical analysis of AMPA receptor motility in cultured hippocampal neurons.

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Recent studies revealed that AMPA receptors at postsynaptic membrane are constitutively recycled to keep an amount of receptors to mediate a constant synaptic response. Further an insertion and retrieval of receptors are dynamically controlled during expression of synaptic plasticity. Although there are many researches, the ratio of receptor turn-over at synapse is still controversial and the underlying machineries remain unknown. To explore the motility of AMAP receptors in living neurons, we generated photo-activatable GFP (PA-GFP) tagged AMPA receptors. PA-GFP is powerful tool for labeling with light at any time and any point in living cell. We used two-photon excitation to activate PA-GFP-AMPA receptors and monitored their GFP fluorescent by single photon excitation. Activation of dendritic shaft showed that two-components, rapid and slow decay of fluorescent intensity change. On the other hand, photo activation on spines showed relative slow fluorescent changes. This result showed that motility of AMPA receptors is dependent on their own location. This method can provide a useful tool for investigation of receptor trafficking.

## P3-b32 Lactic acid increases expression of aquaporin 4 in rat astrocytes

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AQP4 is highly concentrated in astrocytes. AQP4 is involved in the formation of brain edema, but little is known about the molecular mechanisms. On the other hand, it is well known that lactic acidosis plays an important role in the formation of brain edema. In this study, we investigated whether lactic acid induces expression of AQP4 in cultured rat astrocytes.Lactic acid increased expression of AQP4 protein in a concentration- and time-dependent manner without up-regulation of AQP4 mRNA levels. After blockade of protein synthesis by cycloheximide, lactic acid retained AQP4 protein. Additionally, lactic acid increased cell surface expression of AQP4.It may indicate that "lactic acidosis-induced AQP4 expression" is mainly regulated by attenuation in the degradation of AQP4 protein. This would be a new finding in regulatory mechanisms of AQP4 expression in the brain.

## P3-b33 Developmental change of the distribution of P2X purinoceptors in the mouse retina

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We have reported that P2X2-purinoceptors selectively expressed in the OFF-pathway but not in the ON-pathway. In the present study, we immunohistochemically examined whether such a pathway specific expression of P2X2-purinoceptors is generated by the intrinsic mechanisms. In the mouse kept in normal light cycle (12 h light, 12 h dark), immunoreactivity for P2X2 became detectable at 2weeks after birth. At 2-3 week, there were no significant differences of immunoreactivity between ON- and OFF-pathways. After 3 weeks, immunoreactivity in the ON-pathway became weak and tended to show pathway specific distribution. In the mouse retina kept in dark until 8 weeks after birth, we also observed the similar developmental change of the expression pattern of P2X2-purinoceptors. Our data indicate that the OFF-pathway specific expression of P2X2-purinoceptors is a result of extinction of the immunoreactivity of ON pathway during the development. The extinction process in the ON pathway might be determined by the intrinsic mechanisms and not by the environmental condition.

## P3-b34 Cloning and characterization of an ascidian AMPA receptor

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We cloned and characterized the ascidian AMPAR subunits. We show three findings here; 1, the ascidian has only one type of subunit, 2, the subunit does not have the pore RNA editing and 3, it has three types of splicing variants. Next, we used *in situ* hybridization technique for verifying the receptor's distribution in the nervous system of the ascidian. The staining pattern was unique compared with other higher organisms. Moreover, to study the ion channel properties, we recorded glutamate-gated currents from *Xenopus* oocytes, in which either ascidian or chimeric rat/ascidian receptors were expressed. In spite of trying any combination of splicing variants, all receptors did not show any currents.

## P3-b35 The role of Notch signaling in synaptogenesis

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Notch plays essential roles in the development of the nervous system. The canonical Notch signaling pathway requires an intramembrane proteolysis by gamma-secretase. However, the role of Notch signaling in mature nervous system is unclear, while Notch and its ligands are expressed in the adult mammalian brains. Here we examined the role of Notch signaling in the synaptogenesis. We found that the expression levels of synaptophysin 1 in primary cortical neurons were significantly increased by the coculture with nonneuronal NIH3T3 cells overexpressing mammalian Notch ligands. Moreover, synaptophysin 1 was accumulated as punctate structures on the ligand-expressing cells along the contacting neurites. Intriguingly, gamma-secretase inhibitor DAPT failed to abolish the augmentation of the synaptophysin levels. These data suggest that non-canonical Notch signaling is involved in the presynapse formation in synaptogenesis.

## P3-b36 The intracellular signal of RGM

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Repulsive guidance molecule (RGMa) is a potent inhibitor of axon regeneration in the adult central nervous system. RGMa inhibits mammalian CNS neurite outgrowth by a mechanism dependent on activation of the Rho/Rho-kinase pathway. Intrathecal administration of the RGMa-neutralizing antibody to rats with spinal cord injury results in significant axon growth of the corticospinal tract, and improves functional recovery. Here we present the ongoing study on the intracellular signal of RGMa. Understanding of the molecular mechanism of RGMa may be useful for developing methods to overcome the inhibitory signals.

## P3-b37 The existence of two distinct types of serotonergic fibers revealed by VGLUT3 expression in the rat brain

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Although vesicular glutamate transporter 3 (VGLUT3) is a glutamatergic neuron marker, midbrain raphe serotonergic neurons also express this transporter. This suggests that the serotonergic neurons may co-release serotonin and glutamate in transmission over their terminal fields. Immunohistochemical evidence suggests the presence of VGLUT3 in neocortical and hippocampal serotonergic fibers. However, whether other forebrain serotonergic fibers contain VGLUT3 is still unclear. Therefore, we here analyzed the serotonergic terminal fields in the rat forebrain with double-immunofluorescence staining. In general, double-labeled fibers were more frequently detectable in the telencephalic regions, among which a most prominent fiber plexus was found in the lateral septal nucleus. In contrast, few double-labeled fibers were seen throughout diencephalic regions, including the suprachiasmatic nucleus. These findings indicate that in the forebrain there are two functionally distinct types (glutamatergic and non-glutamatergic) of serotonergic fibers.

## P3-b38 Interaction between the metabotropic glutamate receptor 1 $\alpha$ and the $GABA_B$ receptor studied in the heterologous expression system

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The metabotropic glutamate receptor 1 $\alpha$ (mGluR1 $\alpha$) in the cerebellar Purkinje cell has recently been suggested to undergo enhanced sensitization through an involvement of the $GABA_B$ receptor (GBR). The results of IP and other biochemical characterizations suggest that there is a direct physical interaction between the two receptors. In the present study, we confirmed whether the above results can be reproduced in the HEK293T cell or not. When GB1/GB2 were coexpressed, the sensitivity of mGluR1 $\alpha$ to DHPG monitored as $[Ca^{2+}]_i$ response was increased by applying GABA. As a next step, combinations of FLAG-mGluR1 $\alpha$ and tagged GBR subunits were expressed in the HEK293T cells and assessed for IP. We observed that besides the native combinations of the GBR subunits (GB1a-Myc or GB1b-Myc/GB2, GB1a or GB1b/GB2-HA), each tagged GBR subunit alone can also be coimmunoprecipitated with FLAG-mGluR1 $\alpha$.

## P3-b39 Molecular sequence and distribution of vesicular glutamate transporter mRNA in the pigeon central nervous system

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Glutamate is a major excitatory neurotransmitter in the central nervous system and is stored in synaptic vesicles by the vesicular glutamate transporters (VGLUTs). Three isoforms of VGLUTs have been identified in mammals; VGLUT1, VGLUT2, and VGLUT3. The VGLUT1 and VGLUT2 are present in the glutamatergic neurons, whereas the VGLUT3 is found in the cholinergic and serotoninergic neurons. In the present study, we examined a partial sequence of pigeon VGLUT cDNA and its distribution in the central nervous system. The sequence of cDNA is 1,330 bp and the predicted amino acid sequence has 98 and 74% similarity with chicken VGLUT2 and VGLUT3 respectively, and has 70-73% with mammalian VGLUT1, 92% with VGLUT2, and 66-74% with VGLUT3. In situ hybridization revealed that pigeon VGLUT mRNA signals were expressed in the hyperpallium, mesopallium, nidopallium, entopallium, arcopallium, hippocampal formation, dorsal thalamic nuclei, and cerebellar cortex. In conclusion, the sequenced mRNA is suggested to be that of pigeon VGLUT by homology search.

KAKENHI(18590165)

## P3-b40 Altered motor function in vesicular GABA transporter knockout mice

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Vesicular GABA transporter (VGAT) accumulates inhibitory neurotransmitters, GABA and glycine into synaptic vesicles. We generated the VGAT knockout (KO) mice, which showed anteflexion and body wall defect called omphalocele. VGAT KO fetuses on E18 showed drastic reduction in spontaneous IPSCs by performing patch-clamp recordings from their motoneurons, indicating that VGAT is essential for inhibitory neurotransmission. Histopathological analysis revealed an increase in fibers of pectoral muscle and rectus abdominis muscle in VGAT KO fetuses. These observations suggest that the loss of inhibitory neurotransmission in VGAT KO mice causes their muscle hyperplasia via hyperexcitability in their muscle and further results in anteflexion and omphalocele.

## P3-b41 Expression analysis of VGLUT during the postnatal maturation of rat pineal gland

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In the rat pineal gland, pinealocytes contain synaptic-like microvesicles which have vesicular glutamate transporter (VGLUT) 1 and 2. Recently, we reported early postnatal VGLUT1 expression in the gland whose maturation completes in the first three weeks after birth. Despite inhibition of melatonin synthesis by exogenous glutamate in adult pinealocytes in vitro, the role of glutamate in the developing gland is still unclear. To address this issue, we studied ontogeny of rat pineal VGLUT with in situ hybridization and immunohistochemistry. VGLUT1-staining was always seen only in pinealocytes stained for synaptopysin (a pinealocyte marker) at all ages examined. In contrast, VGLUT2-staining was seen in both pinealocytes and synaptophysin-unstained cells until the second postnatal week and later confined to pinealocytes, with the dominant type of pineal VGLUT postnatally shifting from VGLUT2 to VGLUT1. Our data suggest that pineal maturation processes are regulated under the age-dependent and VGLUT type-dependent exocytotic release of glutamate.

## P3-b42 Warm temperature-activated $Ca^{2+}$ response in cultured fibroblasts, neuronal cells and keratinocytes

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Among six TRP channels, TRPV3 and TRPV4 are identified as "warmth" receptors, as their thresholds are around 33 to 35 °C. We investigated the effects of warm temperature on the intracellular $Ca^{2+}$ in cultured fibroblasts (NIH3T3), neuronal cells (NG108-15) and keratinocytes (M308). The "warmth" stimulus was applied by increasing the temperature of the bath solution from 33 to 38 °C. M308 cells expressing TRPV3 and TRPV4 showed rapid increase of cellular $Ca^{2+}$ in normal bath solution containing $Ca^{2+}$. Without the extra-cellular $Ca^{2+}$, slow and reduced response was observed, suggesting contribution of $Ca^{2+}$ mobilization from intracellular stores. NIH3T3 cells also responded to "warmth" in the normal solution, but not in $Ca^{2+}$-free solution. This suggests that they have only influx of $Ca^{2+}$. On the other hand, NG108-15 cells showed both $Ca^{2+}$ influx and mobilization. The variety of the mechanisms of "warmth" sensing will be discussed.

KAKENHI(17657052)

## P3-b43 Effect of oxidized galectin-1 on mRNA expression in macrophage

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Oxidized galectin-1 (GAL-1/Ox) stimulates macrophages to promote axonal regeneration in peripheral nerves after nerve injury. But, the mechanism of GAL-1/Ox, how to regulate macrophage, remains to be clarified. Here we demonstrated effects of GAL-/Ox on mRNA expression of four injury related molecules, IL-1 $\beta$, IL-6, LIF, and iNOS in cultured rat peritoneal macrophages under 10 ng/ml LPS treated condition. RT-PCR analysis revealed that IL-6 as well as iNOS mRNA expressions were significantly up-regulated by GAL-1/Ox, but the other mRNA did not show significant differences by the application of GAL-1/Ox. Increased IL-6 may promotes axonal regeneration in peripheral nerves, but it is not certain whether increased iNOS contributes to the regeneration.

KAKENHI 17500265

## P3-b44 Histone modifications involved in kainate-induced status epilepticus

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Systemic administration of kainate induces status epilepticus, but the molecular mechanisms of epileptogenesis are largely unknown. We found that kainate stimulation induced histones H3 phosphorylation and H4 acetylation in the hippocampus coupled with status epilepticus. Kainate stimulation also induced CBP, a transcription coactivator possessing histone acetyltransferase activity (HAT), and immunohistochemical analysis showed that the spatio-temporal distribution of histone H4 acetylation was well correlated with that of CBP in the hippocampus. Pretreatment of curcumin, a HAT inhibitor of CBP, attenuated histone modifications and also the severity of status epilepticus after kainate stimulation. On the other hand, pretreatment of TSA, a histone deacetylase (HDAC) inhibitor, increased the basal level of histone H4 acetylation and augmented the seizure severity at 6 h after kainate stimulation. Our finding suggests the involvement of histone modifications in the development of epilepsy.

## P3-b45 Essential role of activation of PKA and ERK by BDNF for AMPA receptor translocation to postsynaptic sites

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We have recently shown that brain-derived neurotrophic factor (BDNF) induced an AMPA-type glutamatergic receptor (AMPAR) subunit, GluR1 translocation to the postsynaptic density (PSD) of cultured cortical pyramidal neurons. The $Ca^{2+}$ signaling through IP3R as well as store-operated cation channel TRPC was critical in this translocation. The present study investigates the signaling mechanism of GluR1 translocation after the calcium raise. Acute BDNF application to the cultured neurons increased protein phosphorylation of protein kinase A (PKA) substrates and extracellular signal-regulated kinase (ERK) within 5~10 min at most of the dendritic spines. A PLCγ inhibitor blocked this effect. Half the PSD-translocation of GluR1 was prevented by each inhibitor of adenylyl cyclase (AC), PKA, or MEK. An AC (or PKA) inhibitor together with MEK one completely blocked the translocation which may partially involve exocytic process. These findings suggest that AC/PKA and ERK work downstream of PLCγ activated by BDNF.

Health sciences research grant of nano-1

## P3-b46 Amplification of depolarization-induced and ryanodine-sensitive cytosolic $Ca^{2+}$ elevation by synthetic carbocyclic analogues of cyclic ADP-ribose in NG108-15 neuronal cells

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Cyclic ADP-ribose (cADPR) is a second messenger of $Ca^{2+}$-induced $Ca^{2+}$ release (CICR). Analogues modified in the ribose unit (ribose linked to N1 of adenine) of cADPR was synthesized, and their biological activities were determined in NG108-15 cells that were pre-loaded with fura-2 AM and subjected to whole-cell patch-clamp. Application of the hydrolysis-resistant cyclic ADP-carbocyclic-ribose (cADPcR) through patch pipettes potentiated elevation of cytoplasmic free $Ca^{2+}$ concentration ($[Ca^{2+}]_i$) at the depolarized membrane potential. The increase in $[Ca^{2+}]_i$ evoked upon sustained membrane depolarization was significantly larger in cADPcR-infused cells than in non-infused cells. These results demonstrated that cADPcR have agonistic effects on the depolarization-induced $[Ca^{2+}]_i$ increase, and suggested the presence of functional reciprocal coupling between RyRs and VACCs via cADPR in neuronal cells.

KAKENHI 16650068

## P3-b47 Activation of CREB by ethanol and acetaldehyde in NG108-15 cells.

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It has been shown that ethanol promotes cAMP signaling, leading to phosphorylation of cAMP response element binding protein (CREB) and induction of CRE-mediated gene transcription. Acetaldehyde has long been suggested to be involved in a number of ethanol's pharmacological and behavioral effects, but the role of acetaldehyde in ethanol's effects has not been cleared. In the present study, we investigated the effects of ethanol and acetaldehyde on the activation of CREB. When NG108-15 cells were incubated with ethanol or acetaldehyde, activation of CREB was observed. The effect of ethanol was attenuated in the presence of catalase inhibitor, and was enhanced in the presence of aldehyde dehydrogenase inhibitor. Therefore, acetaldehyde appeared to be partly responsible for ethanol-induced CREB activation in NG108-15 cells.

## P3-b48 Regulation of the membrane association and kinase activity of Cdk5-p35 by phosphorylation of p35

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Cdk5 is a protein kinase predominantly expressed in postmitotic neurons by binding to an activation subunit called p35 or p39. Cdk5-p35 plays a variety of roles in neuronal functions, including neuronal migration, synaptic plasticity and neuronal cell death. Cdk5-p35 in adult rat brains, which is bound to membranes, is low kinase activity. However, the kinase activity of Cdk5-p35 in fetal/neonatal brains remains to be examined. We found that part of fetal/neonatal Cdk5-p35was a soluble complex dissociated from membranes with high kinase activity and that the soluble complex became bound to membranes after dephosphorylation of p35. These results suggest that the kinase activity of Cdk5-p35 is regulated through the association of Cdk5-p35 with membranes that is also regulated by dephosphorylation of p35 with protein phospatase-1.

## P3-c01 Characterization of novel CK1-mediated phosphorylation of SCS-binding Rho GTPases in vitro

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[Purpose] Recently, we found that casein kinase 1 (CK1) preferentially phosphorylates sulfatide and cholesterol-sulfate (SCS)-binding proteins (SCS-BPs), such as myelin basic protein and tau protein, containing novel phosphorylation motif [K/R-X-K/R-X-X-S/T] for CK1 *in vitro*. These findings suggest that CK1 may be responsible for the regulation of various SCS-BPs through their preferential phosphorylation at high level of SCS in brain. Therefore, the present study was carried out to identify novel SCS-BPs involved in the novel CK1-mediated signal transduction in brain.[Results and Discussion] It was found that (i) RhoA (a small GTPase) was a SCS-binding protein; and (ii) RhoA was highly phosphorylated by CK1 (δ and γ) in the presence of SCS *in vitro*. No effect of GTPγS and GDP on the CK1-mediated phosphorylation of RhoA GTPase was observed *in vitro*. These results suggest that the CK1-mediated phosphorylation of Rho family GTPases at the high level of SCS may be involved in the preferential modulation of the Rho-mediated signaling neuronal system in brain.

## P3-c02 Characterrization of SCS as a potent activator for the CK1-mediated phosphorylation of tau protein and its associated proteins from rat brain

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[Purpose] Recently, we reported that casein kinase 1 (CK1) phophorylates two functional basic proteins [myelin basic protein (MBP) and tau protein (TP)] in the presence of two sulfated lipids [sulfatide and cholesterol-3-sulfate (CH-3S), SCS] in vitro. The present in vitro study has been carried out to characterize the SCS-dependent phosphorylation of TP and its associated proteins by CK1 in the TP fraction from rat brain.[Results and Discussion] It was found that (i), in the presence of SCS, CK1 phosphorylated TP and its associated proteins (p82 and p55) in the partially purified TP fraction; and (ii) p82 and p55 was identified as eIF-4B and syndapin 1, respectively. These results suggest that the accumulated high level of SCS preferentially induces the CK1-mediated phosphorylation of TP, eIF-4B and syndapin 1, which are involved in the mechanisms of various neuronal diseases, including Alzheimer's disease.

## P3-c03 Computer simulation of target molecule of genipin with neurotrofic factor-like activity: Interaction of genipin-related compounds and tetrahydrobiopterin binding domain in neuronal NO synthase

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We have previously demonstrated that genipin, a natural iridoid compound, induces neuronal differentiation through NO-cGMP-PKG pathway in several types of neuronal cell lines which are expressed mainly neuronal NO synthase(nNOS). Therefore, in this study we have analyzed the possibility of the interaction of genipin- related compounds, mainly 1-O-alkyl derivatives, and nNOS by computer simulation. The results suggested that genipin has very similar configuration to that of tetrahydrobiopterin(H4B) and binds H4B-binding domain in nNOS, and the neuritogenic activities of genipin-related compounds correlate to the parameters (HOMO-LUMO energy, docking score, LogP) calculated. Thus, it is strongly suggested that the target molecule of genipin is nNOS. This work was supported by MEXT. FRONTIER(2005-2009).

## P3-c04 Activation of the *c-fos* promoter is modulated by PKA in PC12 cells.

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The *c-fos* protooncogene is thought to contribute to the regulation of normal cellular proliferation and differentiation. It has been reported that application of NGF, which leads to cellular differentiation, induced a transient *c-fos* gene expression in PC12 cells. NGF activates the *c-fos* promoter through two well characterized regulatory elements, the serum response element (SRE) and the cAMP response element (CRE). Many kinases are involved in the transcription through these elements, a contribution of PKA, however, has not been investigated in detail. Using a luminescence imaging system and a human *c-fos* promoter-driven luciferase expression vector, we analyzed physiological roles of PKA in NGF-induced *c-fos* promoter activation in a single cell level. Inhibitors for PKA enhanced the increase of the *c-fos* promoter activity without any effects to MEK/MAPK cascade. Mutational analysis of the CRE and/or the NFκB consensus sequence in the regulatory region of the *c-fos* gene revealed that *c-fos* promoter activity might be modulated by both CREB and NFκB. Finally, we propose a molecular mechanism under the modulation of the *c-fos* promoter by PKA.

## P3-c05 Tyrosin kinase is involved in the mGluR1-mediated inward current in the cerebellar molecular layer interneurons

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Activation of mGluR1 induces an excitatory slow inward current in the cerebellar molecular layer interneurons (MLIs), such as basket and stellate cells. We addressed the underlying mechanism with whole-cell patch-clamp recordings. Pressure application of the group I mGluR agonist DHPG produced a slow inward current in MLIs, which was blocked by an mGluR1 antagonist, CPCCOEt. Bath-applied protein tyrosin kinase inhibitors, genistein and AG490, considerably reduced the DHPG-induced inward current, although infusion of a PLC inhibitor U73122 into MLIs only partially inhibited the current amplitude. Furthermore, perfusion of a selective Src kinase inhibitor PP2 significantly suppressed the inward current. These results suggest that mGluR1 activation in MLIs induces the inward current through a Src-dependent pathway, which is different from the mGluR1-TRPC1 signaling cascade in cerebellar Purkinje cells.

KAKENHI (16700344)

## P3-c06 Distinct cellular expression of PKC α and PKCγ in particular interneurons of the mouse telencephalon.

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Protein kinase C (PKC) is classifed as a family of closely related serine/threonine protein kinases that regulate various biological events within the cell. PKC α and PKCγ, members of classical PKC, are $Ca^{2+}$-dependent and activated by diacylglycerol (DAG) in the process of a series of phosphorylation events. In this study, we studied cellular expression of PKC α and PKCγ in the mouse striatum, cortex, and hippocampus. PKC α and PKCγ were more or less coexpressed in principal neurons, including medium spiny neurons in the striatum, cortical neurons in the layer II/III, and pyramidal cells and granule cells in the hippocampus. However, PKC α was expressed predominantly in cholinergic interneurons and parvalbumin (PV)-positive interneurons in the striatum, layer V non-pyramidal neurons and PV-positive interneurons in the cortex, and nNOS-positive interneurons in the hippocampus. In contrast, pyramidal cells in the layer V selectively expressed PKCγ. Therefore, particular interneurons preferentially express PKC α, whereas most principal neurons express both PKC α and PKCγ.

## P3-c07 Algebraic method for the analysis of signaling crosstalk

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A unified mathematical description that expresses the characteristics of whole systems is necessary for an understanding of signal transduction cascades. In this study we explore an algebraic method named extreme signaling flow (ESF), enhanced from the concept of extreme pathway (EP), to analyze signal transduction systems. This method enables us to represent the long-term potentiation (LTP) and the long-term depression (LTD) of hippocampal neuronal plasticity in an integrated simulation model. The model is validated by comparing in silico knockout analyses with biological knowledge available from literature. Several properties, such as redundancy of kinase, are introduced by these computational analysis.

COE

## P3-c08 Localization of diacylglycerol kinase γ and -ε in neurons

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Diacylglycerol kinase (DGK) is involved in intracellular signal transduction as a regulator of a second messenger, diacylglycerol. To address the functional implications of DGK isozymes in the brain, we have raised specific antibodies against DGKγ and -ε. By immunoblot analysis, we found that immunoreactivities against DGKγ and -ε were detected in the light membrane/microsome enriched fraction of rat brains, suggesting that these isozymes localize to the internal membrane system. Immunohistochemical examination on the cerebellum revealed that immunoreactivity for DGKγ was detected as punctate or reticular structures in the cytoplasm and dendrites, but not in the axon, of Purkinje cells. The immunoreactivity was also detected diffusely in the cytoplasm of cerebellar granule cells. On the other hand, DGKε-immunoreactivity was observed as granular structures in the perinuclear region, dendrites and axon of Purkinje cells, but not in cerebellar granule cells. These data suggest that DGKγ and -ε play different roles in distinct internal membrane system in neurons.

KAKENHI 18700337

## P3-c09 Lipid components in the detergent-resisitant membrane microdomain from rat synaptic plasma membrane.

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Membrane microdomains are recovered as a detergent-resistant low-density membrane fraction(DRM) after Triton extraction and centrifugation. Since specific recovery of phosphatidylinositol 4,5-bisphosphate(PIP2) in DRM is known in cultured cells, the DRM is considered as a signal processing platform. Little information is however available on the PIP2 content in the DRM prepared from brain, for the RI-labeling of live animals are not practical. In this study, we assayed the amounts of PIP2 in SPM and SPM-derived DRM using the PH domain of phospholipase C-delta1 fused to GST. Extracted lipids were separated on HPTLC and heat-transfered to PVDF membrane. After blocking, anti-GST and ECL system was used to measure the bound GST. About 10% of PIP2 in SPM was recovered in DRM and no specific localization was detected. In contrast,nearly half amounts of sphingomyelin and cholesterol, and about 30% of phosphatidylcholine or phosphatidylethanolamine were recovered in DRM.

KAKENHI(16370062)

## P3-c10 Localization of phospholipase C $\beta$1 on detergent-resistant membrane microdomains prepared from synaptic plasma membrane of rat brain

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Membrane microdomains (MDs) such as a detergent-resistant membrane microdomain fraction (DRM) have been paid much attention, because many signal-transducing molecules are recovered in the fraction. In order to identify neuronal MD-proteins, monoclonal antibodies against the DRM-components prepared from synaptic plasma membrane of rat brain were produced. One of the antigens reacted with two closely positioned bands of about 140 kDa in SDS-PAGE and the antigen showed age-dependent localization on DRM. The antigen was immunoprecipitated and identified as phospholipase C $\beta$1 through mass analysis and western blotting. Lipid composition of $PIP_2$-containing liposomes affected the enzymatic activity and addition of NAP-22, a neuronal MD-protein, inhibited the activity. These results suggest the presence of MDs that perform important roles in neuronal functions through the lipid metabolism.

KAKENHI (16370062)

## P3-c11 DARPP-32-dependent regulation of spinophilin Ser94 phosphorylation in neostriatal neurons of direct and indirect pathways

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Spinophilin is a protein phosphatase-1 (PP-1)- and actin-binding protein that is enriched in dendritic spines. Spinophilin, by binding to actin filaments, anchors PP-1 in the vicinity of AMPA and NMDA receptors, and thereby modulates excitatory synaptic transmission. Binding of spinophilin to actin is regulated by phosphorylation of the actin-binding domain of spinophilin by cAMP-dependent protein kinase (PKA). Activation of PKA by forskolin stimulated the phosphorylation of spinophilin at Ser94. In this study, we investigated the regulation of spinophilin phosphorylation at Ser94 using mouse neostriatal slices. Activation of dopamine D1 and adenosine A2A receptors increased, whereas activation of dopamine D2 and NMDA receptors decreased, spinophilin Ser94 phosphorylation. D1, D2 and A2A receptors, not NMDA receptors, required DARPP-32 to elicit their effects on spinophilin Ser94 phosphorylation.

## P3-c12 Neurofibrillary tangles in Lafora disease: Functional link between laforin and tau proteins

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Mutations in *EPM2A* in human and mice leads to a neurodegenerative condition called as Lafora disease (LD). *EPM2A* encodes a protein phosphatase known as laforin. Our analyses on the *Epm2a* gene knockout mice reveal the presence of hyper-phosphorylated tau as neurofibrillary tangles (NFT's), a hallmark of Alzheimers disease. One of the kinases that phosphorylate tau is GSK3 $\beta$. Intriguingly, the phosphorylation levels of Ser-9 residue of GSK3 $\beta$ and the Ser-473 of AKT are lower in the Epm2a knockout mice. AKT is a serine-threonine kinase that phosphorylates and regulates the activity of GSK3 $\beta$. Dephosphorylation of residues Ser-9 of GSK3 $\beta$ and Ser-473 of AKT is known to activate and inactivate these two enzymes, respectively. In this presentation we would discuss the molecular links between laforin and tau proteins and propose that NFT's might underlie some of the symptoms associated with LD.

LSRB

## P3-c13 Modulation by brain natriuretic peptide of rectify outward K+ current on mouse Schwann cells

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Here we found that mouse Schwann cells expressed all the natriuretic peptide receptors (NPRA, NPRB and NPRC) by molecular techniques and immunohistochemistry. Moreover, extracellular application of BNP, one type of natriuretic peptides, could increase rectify outward $K^+$ current ($I_k$) on mouse Schwann cell, which was not concentration-dependent(5nM~200nM). BNP also had a significant effect on the kinetic parameters of the steady-state activation. Extracellular application of c-ANF, the NPR-C selective agonist, did not mimic the effect of BNP, while anatin, the blocker of NPR-A, could inhibit the effect. All this indicated that only the receptor NPR-A was involved. With the application of HS-142-1 or 8Br-cGMP, we further confirmed that only NPR-A and its intracellular cGMP pathway took part in the current-increased effect. By means of proliferation assay, BNP seemed to have facilitated effect on Schwann cell proliferation. All the results help us to investigate in terms of therapeutic applications of BNP and basic research of Schwann cell.

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## P3-c14 Lysophosphatidic acid stimulates astrocyte proliferation through $LPA_1$

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Lysophosphatidic acid (LPA) is a signaling phospholipid that regulates the nervous system functions through LPA receptors. Here we explored the role of LPA receptor subtypes in cortical astrocyte functions. Astrocytes cultured under serum-free conditions were found to express the genes of five LPA receptor subtypes, $lpa_1$ to $lpa_5$. When astrocytes were treated with dibutyryl cyclic adenosine monophosphate, a reagent inducing astrocyte differentiation, $lpa_1$ expression levels remained unchanged, but those of other LPA receptor subtypes were relatively reduced. LPA stimulated DNA synthesis in both undifferentiated and differentiated astrocytes, but failed to do so in astrocytes prepared from $lpa_1$-null mice. By contrast, LPA-induced inhibition of [$^3$H]-glutamate uptake was observed in not only both undifferentiated and differentiated astrocytes, but also in $lpa_1$-deficient astrocytes. These indicate that $LPA_1$ mediates LPA-induced stimulation of DNA synthesis but not inhibition of glutamate uptake in astrocytes.

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## P3-c15 Signaling mechanism inducing deleterious factors in microglia

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To clarify the molecular mechanism by which harmful molecules are induced in infected microglia, we investigated the effects of mitogen-activated protein kinase (MAPK) inhibitors and antioxidant reagent on the production of superoxide anion (SA), nitric oxide (NO) and tumor necrosis factor alpha (TNF $\alpha$) in lipopolysaccharide (LPS)-activated microglia. The inhibitor experiments indicated that two MAPKs, c-Jun N-terminal kinase (JNK) and p38MAPK are strongly associated with the induction of NO and TNF $\alpha$ in LPS-activated microglia. On the other hand, antioxidant N-acetyl cystein (NAC) was found to suppress the production of not only SA but also NO and TNF $\alpha$ in LPS-activated microglia. The antioxidant reagent also significantly reduced LPS-dependent activation of JNK and p38MAPK. Therefore, these results suggested that SA produced in infected microglia induces NO and TNF $\alpha$ by activating JNK and p38MAPK.

## P3-c16 The response of microglia and astrocytes to the infected cortical neurons induced by a coronavirus

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Following inoculation of a coronavirus (strain 67N) into the rat hind footpad, the time course of the neuronal infection and glial activation were immunohistochemically examined in the primary motor cortex (M1). The activated microglia and astrocytes associated with infected neurons were first detected in the layer 5 of M1 contralaterally to the injection side on day 4 post-inoculation (p.i.), and showed with enlarged cell bodies and thickened processes. Furthermore, activated microglia migrated to and gathered around the infected neurons. On day 5 p.i., activated microglia changed into amoeboid form and tightly ensheathed the infected neurons. Additionally, numerous OX42-immunoreactive cells in round shape appeared on the infected neural tissue. In contrast, astrocytes became more hypertrophic. On day 6 p.i., the OX42- and GFAP-immunoreactive cells dramatically decreased. These results suggest that microglia and astrocytes play a pivotal role in the innate immune response to the CNS viral infection.

## P3-c17 Nitric oxide mediates cytokine-induced enhancement of $Ca^{2+}$-dependent Glu release from astrocytes

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Aim; Cytokines that are released under several pathologic conditions enhance inducible nitric oxide synthesis (iNOS) expression. NO produced by iNOS is involved in several neural functions. To date, there are no studies on the effects of cytokines on $Ca^{2+}$-dependent Glu release from astrocytes. Therefore, we studied the effects of cytokines and the contribution of NO on Glu release. Methods; Glu release by the $Ca^{2+}$ influx was determined by HPLC.Results; Glu release was enhanced by the treatment with cytokines. Inhibition of iNOS canceled the cytokine-induced enhancement of Glu release, and the treatment with a NO donor, even in the absence of cytokines, increased the Glu release.Conclusion and Discussion; Cytokines enhance Glu release, and this enhancement is mediated by NO. Cytokine-activated astrocytes are known to protect the CNS from injuries. Our results suggest that the increased Glu release through NO by cytokines may participate in the protection from injuries.

## P3-c18 Spontaneous calcium activities of astrocytes *in vivo* differ between cortical layer 1 and layer 2/3

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Growing evidence obtained with *in vitro* experiments suggests involvement of astrocytes in neuronal signal processing. For example, increase of intracellular calcium ($Ca^{2+}$) concentration in an astrocyte propagated to neighboring astrocytes, and induced $Ca^{2+}$ increase in their adjacent neurons; astrocytes release neurotransmitters such as glutamate and ATP upon intracellular $Ca^{2+}$ increases. While detailed research on $Ca^{2+}$ dynamics of astrocytes *in vitro* have been performed, *in vivo* investigation is sparse. We examined $Ca^{2+}$ dynamics of rat cortical astrocytes in layer 1 and layer 2/3 *in vivo* using two photon laser microscopy. Following parameters of astrocyte $Ca^{2+}$ surge were investigated: number, duration, peak, and spatio-temporal correlation. We found that astrocytes in layer 1 had 2-3 times more spontaneous calcium surges than that of layer 2/3, which may reflect the difference in the cytoarchitecture between these layers. Relation between glial $Ca^{2+}$ increase and electrical neuronal activities are under analysis.

KAKENHI(18053026)

## P3-c19 Astrocyte-mediated regulation of synaptic glutamate release in the NTS

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In the nucleus of the solitary tract (NTS), spatially delimited activation of presynaptic P2X receptors with laser-based photoactivation of caged ATP in the brain slices facilitates glutamate release, affecting the excitability of the postsynaptic neurons (Shigetomi & Kato, 2004; Imura et al., 2006). We examined the hypothesis that ATP of astroglial origin could activate these presynaptic P2X receptors and affect synaptic transmission in the NTS. Electron-microscopic analyses revealed that 99% of presynaptic terminals showed partial or full contact with astrocyte-like processes. Two lines of evidence, as confirmed in the rat brainstem slice using patch-clamp recording of synaptic currents, that (1) activation of P2Y1 receptors, that are mostly expressed in astrocytes, increased mEPSC frequency and (2) a treatment with fluoroacetate decreased mEPSC frequency support the notion that these presynaptic P2X receptors might function as an interface between astrocytic ATP signal and synaptic transmission.

KAKENHI (17300123,18053022)

## P3-c20 Functional cold/menthol receptor expressed in cultured astrocytes

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It is established at present that astrocytes express a variety of neurotransmitter receptors and control the brain functions including synaptic plasticity directly and indirectly. However, little is known to date whether astrocytes express sensory receptors to regulate the brain functions as well. Here we found that a population of cultured astrocytes isolated from the rat brain (hippocampus) possessed functional cold/menthol receptor, which raised the concentration of cytoplasmic $Ca^{2+}$ when activated by menthol. The population of responsive cells took bipolar configuration and became less in number when the days of culture proceeded. It is possible that reactive astrocytes express transiently the cold/menthol receptor.

## P3-c21 Neuronal stimulation leads microglia to more neurotrophic cells

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The regulation of microglial properties by neurons was characterized in vitro from a neurotrophic perspective. To evaluate the neurotrophic effects of microglia stimulated with neurons, the effects of conditioned medium (CM) of microglia stimulated with neuronal CM (NCM) were assessed in neuronal cultures. The experiments revealed that the CM of microglia stimulated with NCM enhances the survival/maturation of GABAergic, catecholaminergic, and cholinergic neurons. As the effective factors increased in the CM of microglia stimulated with NCM, NGF, NT-4/5, TGF $\beta$1, GDNF, FGF2, IL-3, and IL-10 were identified. The promoted neurotrophic effects of CM of microglia stimulated with NCM were abrogated by deprivation of neurotrophic factors by means of an immunoprecipitation. Thus, neuronal stimulus was found to activate microglia to produce more neurotrophic factors, thereby changing microglia into more neurotrophic cells.

## P3-c22 The localization and non-genomic function of the membrane-associated estrogen receptor in oligodendrocytes

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The physiological functions of estrogen involve activation of cytoplasmic signaling mediated by a membrane-associated estrogen receptor (mER). We recently showed that a mER is present within myelin plasma membrane. To understand the physiological function of mER in oligodendrocyte (OL), its cellular localization and involvement in rapid signaling were investigated in OLs. An ER $\alpha$ was expressed along the lacy network of veins in the membrane and in the perikaryon in OLs but not in the nucleus. ER $\beta$ was located in the nucleus, and to a lesser extent along the veins. OLs were pulsed with 17 $\alpha$ and 17 $\beta$-estradiol for various times and the total lysates were analysed for phosphorylated kinases. Both estradiols elicited rapid phosphorylation of p42/44MAPK, Akt, and GSK-3 $\beta$. Here, we show that mER is involved in rapid signaling pathways in OLs, which are activated via both estradiols.

Multiple Sclerosis Society of Canada

## P3-c23 Hydrogen peroxide at a low concentration protects BV-2 cells against hydrogen peroxide-induced apoptosis through the production of catalase.

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In this study, we investigated the effect of pretreatment with $H_2O_2$ at a low concentration on $H_2O_2$-induced death in mouse microglial cell line BV-2 and human astrocytoma cell line U-251 MG. Cell viability was determined by a colorimetric MTT assay. Pretreatment with $H_2O_2$ at a low concentration (50-100 μM) for 24 h inhibited $H_2O_2$ (0.5 mM)-induced cell death in BV-2 cells. On the other hand, the cytoprotection by pretreatment with $H_2O_2$ at a low concentration was not observed in U-251 MG cells. In BV-2 cells, the cytoprotective effect of pretreatment with $H_2O_2$ at a low concentration was reduced by a catalase inhibitor, 3-amino-1,2,4-triazole. Treatment with $H_2O_2$ at a low concentration increased the level of catalase and its activity in BV-2 cells, but not in U-251 MG cells. These results indicate that pretreatment with $H_2O_2$ at a low concentration protects BV-2 cells against $H_2O_2$-induced apoptosis through the production of catalase.

## P3-c24 Negative feedback regulation of NO production in LPS-activated microglia.

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The overproduction of nitric oxide (NO) from activated microglia has been proposed to play a pathogenetic role in neurodegenerative diseases, such as Alzheimer's disease and Parkinson's disease. In this study, we investigated the effect of NO preconditioning on the production of NO by microglial cell line BV-2 stimulated with lipopolysaccharide (LPS). The production of NO was assessed as the accumulation of nitrite in the culture supernatants, using a colorimetric reaction with the Griess reagent. Treatment of BV-2 cells with LPS for 24 h increased the levels of NO and inducible NO synthase (iNOS) protein in a concentration-dependent manner. Pretreatment with 1-hydroxy-2-oxo-3,3-bis-(2-aminoethyl)-1-triazene, an NO donor, for 24 h concentration dependently attenuated the LPS (100 ng/mL)-induced increase in the levels of NO and iNOS protein. These results suggest that the production of NO by activated microglia is regulated by negative feedback mechanisms.

## P3-c25 Distribution of oxytocin receptors in the neonatal brain stem

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It has been proposed that oxytocin is implicated in development and plasticity, and maternal oxytocin may increase the resistance to the stressful delivery associated with high risks to the fetal brain. In order to understand the role of oxytocin at the birth and neonatal period, we examined the distribution of oxytocin receptors (OTRs) in neonatal brain stem of rat by immunohistochemistry. Dense OTR-immunoreactive fibrous structures were observed uniformly in the area postrema, reticular formation and most nuclei in medulla oblongata and pons, however not observed in motor nuclei such as ambiguus, hypoglossal, facial and trigeminal motor nuclei, which were intensely immunoreactive for p75 neurotrophin receptor at postnatal day 1 (P1). These OTR immunoreactive structures were morphologically considered glial fibers. They decreased at P5 and progressively disappeared by P14 and then OTR immunoreaction became to be observed sporadically in small blood vessel of the brain. These results support that oxytocin may play important roles in the delivery and/or neonatal period of brain development.

## P3-c26 Local GABAergic system in trigeminal ganglion

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Trigeminal ganglion (TG) neuronal cell bodies are generally devoid of synaptic contacts and are thought to play only supportive roles in the maintenance of neurite. However, our preliminary study in vivo autoradiographic study with $^{14}$C-GABA showed a radioactivity in TG, which suggested presence of GABAergic system. The aim of this study is to investigate a local GABAergic system in rat TG. Immunohistochemical analysis showed that GAD65, GABA and $GABA_A$ and $GABA_B$ receptor subunits were expressed in neuronal cell bodies. Satellite cells were strongly immunopositive for GABA. In patch clamp experiments, GABA and $GABA_A$ agonist induced $Cl^-$ currents and also $GABA_B$ agonist increased voltage-depend $K^+$ currents in neuronal cell bodies. In addition, we found that GABA was released from TG cells by increasing extracellular $K^+$ concentration. These data indicate that neuronal cell bodies in TG express functional $GABA_A$ and $GABA_B$ receptor and satellite cells may have function to store and release GABA. We conclude that GABA may have important physiologic roles in TG.

KAKENHI16790898

Poster Session

## P3-c27 The mode of AMPA-type glutamate receptor trafficking in Bergmann glia

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AMPA-type glutamate receptors (AMPARs) in Bergmann glia accumulate on the plasma membrane facing glutamatergic synaptic clefts and mediate glial enwrapping of glutamatergic synapses. To elucidate the trafficking mechanism of AMPARs in Bergmann glia, we performed immunofluorescence and immunoelectron microscopies using antibodies for GluR1 and GluR4, two subunits expressed in Bergmann glia. By immunofluorescence, intense signals for GluR1 and GluR4 subunits were found at particular cytoplasmic regions of the cell body, but showed no apparent overlap with rER and Golgi apparatus markers. By pre-embedding immunoelectron microscopy, the immunolabeling in the cell body was selectively detected on the membrane of vesicular elements of trans-Golgi network. Moreover, such immunolabeled vesicles were occasionally observed inside Bergmann fibers and lamellate processes adjacent to synaptic clefts. These results suggest that AMPARs in Bergmann glia are transported intracellulary as transport vesicles, and the exit from the trans-Golgi network appears to be the regulatory point.

KAKENHI (18053001)

## P3-c28 Analysis of Alexander disease model mice

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Alexander disease is a specific astrocytic disease caused by a dominant heterozygous mutation in the glial fibrillary acidic protein (GFAP) gene. Tanaka et al. found that mutant mice which have three copies of transgene showed Rosenthal fiber (GFAP aggregates) formation in hippocampus and olfactory bulb. The intermediate filament structure was entirely disrupted under these conditions, however, over all morphology of astrocytes, including their fine processes, were unaffected. In the Kainate challenge, the mortality of transgenic mice which have aggregates after kainate challenge was dramatically altered. But, they did not show megalencephaly, leukodystrophy or seizure, characteristic of Alexander disease. In electrophysiological analysis LTP was easily induced in mutant mice and mutant mice showed good memories in short term memory test. We also generated homozygous mutant mice which showed much higher expression of human mutant GFAP. These mice showed the expression of human mutant GFAP even in the cortex, and in many other regions which were devoid of Rosenthal fiber in the heterozygotes. The homozygotes had reduced body weight and seem to have severer phenotype than heterozygotes.

## P3-c29 Analysis of myelin proteins using CTAB/SDS-PAGE

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The analysis of proteins in damaged myelin is crucial to clarify the mechanism of dys and demyelination. Recently, we performed two-dimensional electrophoresis (2-DE) using the cationic detergent benzyldimetyl-n-hexadecylammonium chloride for the analysis of myelin proteins. While we obtained improved separation and identified major myelin proteins, highly hydrophobic proteins were partially retained in the first dimension gel. In order to increase the solubility and resolution of myelin hydrophobic proteins, we tested four cationic detergents for 2-DE. We found that hexadecyltrimethylammonium bromide (cetyltrimethylammonium bromide; CTAB) was the most effective agent for the solubilization and separation of myelin proteins. We applied CTAB/SDS-PAGE 2-DE for developmental analysis of myelin proteins and demonstrated that fourteen protein spots were enriched in 2- and 3-week-old rat brain myelin samples. We conclude that CTAB/SDS-PAGE 2-DE provides a powerful technique facilitating the proteomic analysis of myelin proteins in developmental and possibly pathological changes.

## P3-c30 The functional regulatory mechanism in reactive astrocytes via VIP/VPAC2 system

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Vasoactive intestinal peptide (VIP) is a neuropeptide that functions as a neuroprotective agent for brain injury. To elucidate the regulatory mechanism of VIP, we examined the regulation of VIP/VPAC2 system in reactive astrocytes. The expression of VPAC2, which is one of the VIP receptors, increased in dibutyryl cAMP-stimulated reactive astrocytes in vitro. Immunohistochemistry revealed that the VPAC2 expression up-regulated in GFAP-immunoreactive reactive astrocytes in the cerebral lesion. VPAC2 activation by a selective VPAC2-agonist induced morphological changes of cultured astrocytes associated with increased expression of glial glutamate transporters, suggesting that VPAC2 signal induces functional alterations in reactive astrocytes. In conclusion, VIP/VPAC2 system in reactive astrocytes may have an important role for tissue repair processes of the pathological conditions with the prevention from glutamate excitotoxicity in the CNS.

## P3-c31 Dysfunction of MBNL1 involved in aberrant splicing of SERCA1 in myotonic dystrophy type 1

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Myotonic dystrophy type 1 (DM1) is caused by unstable expansion of CTG trinucleotide repeats in the 3'-UTR of the myotonic dystrophy protein kinase (DMPK) gene. Previously, aberrant splicing of SERCA1, that showed exclusion of the exon 22, was found in skeletal muscle of DM patients. There are five 'YGCU(U/G)Y motif' that could potentially serve as splicing regulator, MBNL1, binding motifs downstream from the SERCA1 exon 22. Exon trapping experiments showed that MBNL1 acts the 'YGCU(U/G)Y motif' in its intron 22, and positively regulates the exon 22 splicing. Overexpression of the CUG repeat expansion of DMPK mRNA resulted in exclusion of the exon 22 of SERCA1. These results indicate that sequestration of MBNL into the CUG repeat expansion of DMPK mRNA could cause the exclusion of SERCA1 exon 22, and expression of this aberrant splicing form of SERCA1 could affect the regulation of $Ca^{2+}$ concentration of sarcoplasmic reticulum in the DM patients.

## P3-c32 Analysis of a brain specific protein arginine N-methyltransferase 8 (PRMT8) in the mouse brain

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Arginine methylation is a common post-translational modification that is mediated by the protein arginine methyltransferases (PRMTs). PRMT8 is a member of the PRMTs, which is specifically expressed in the brain, suggesting that is specifically involved in some brain functions. However, the biological function of PRMT8 has not been known.In this study, to elucidate the function of PRMT8, we developed polyclonal antibody for PRMT8 and examined the distribution of the neurons expressing PRMT8 in the mouse brain. Though almost neurons in brain were stained, cortex, hippocampus, thalamic nuclei and Purkinji cells expressed PRMT8 strongly. Next, we performed immunofluorescence analysis in primary cultured neuron from hippocampus to investigate the subcellular localization of PRMT8. PRMT8 was localized in the nuclei. The ontogenetic analysis of PRMT8 using Western blot methodology clearly revealed that PRMT8 develops during the perinatal stage.

## P3-c33 Dynamic localization of eIF4E mRNA in dendrites of cultured rat hippocampal neurons

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It is known that availability of eukaryotic translation initiation factor 4E (eIF4E) is the major rate-limiting factor, thus subserving as a major control site for protein synthesis. In this study we carried out in situ hybridization and showed that the mRNA for eIF4E is present in the dendrites of cultured rat hippocampal neurons. Fluorescence in situ hybridization (FISH) revealed that treatment of the neurons with KCl increases very significantly in dendritically localized eIF4E mRNAs by 50.1 and 74.5% at 2 and 6 hr after treatment, respectively. By combining FISH with immunocytochemistry, we further showed that KCl treatment increases very significantly in the PSD95-associated eIF4E mRNA punctae by 21.6 and 32.5% at 2 and 6 hr after treatment, respectively. These results demonstrate an activity-dependent increase of eIF4E mRNA at synaptic sites and provide a potential mechanism for enhanced synaptic translation upon synaptic stimulation.

This work was supported by the Korea Research Foundation Grant funded by the Korean Government (Basic Research Promotion Fund)

## P3-c34 Cystatin C regulates glial differentiation

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During development, proliferation and differentiation of neural stem cells are regulated by series of factors. We identified cystatin C, a cystein protease inhibitor, as a factor promoting astrocyte development. Given that cystatin C induces the differentiation towards astrocytes, it may also affect the proliferation and/or the maturation of oligodendrocytes. We hypothesized that cystatin C regulates oligodendrocytic differentiation in embryonic stage, when the glial induction and fate determination between astrocytes and oligodendrocytes occur. To test this hypothesis, we used the dissociation cell culture of E14.5 mouse brain and neurosphere culture of E14.5 ganglionic eminence, in which neural stem/progenitor cells are enriched. Treatment with exogenous cystatin C suppressed oligodendrocytes differentiation from neurospheres, and in contrast, addition of anti-cystatin C neutralizing antibodies into the dissociation cell culture increased the number of oligodendrocytes. These results suggest that cystatin C suppresses the differentiation of oligodendrocyte, in addition to the effects on astrocytes.

## P3-c35 Regulation of proliferation by sustained magnetism in cultured neural progenitor cells

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In the central nerves system, static magnetic fields could affect a variety of cellular functions in neurons as well as glia through modulation of electrical currents and mechanical mobility. In this study, we have attempted to elucidate the possible modulation by static magnetism of the proliferation in neural progenitor cells prepared from 18-day-old embryonic rat neocortex. Cells were clustered to form neurospheres during culturing in DMEM/F12 medium containing epidermal growth factor for a period up to 12 days under floating conditions. Sustained exposure to magnetism at 100 mT induced a marked decrease in the MTT reduction activity and the total area of neurospheres on Day 12, without significantly affecting the cell viability assessed by propidium iodide staining. Sustained magnetism failed to markedly alter the expression of immunoreactive nestin in neurospheres cultured for 12 days. These results suggest that static magnetism may negatively modulate the cellular proliferation activity in undifferentiated neural progenitor cells.

## P3-c36 Sox6 is expressed in mouse embryonic neural stem/progenitor cells (NSPCs) and contributes to their maintenance and/or survival

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The detailed function of Sox6 in the CNS including NSPCs has been unclear. In this study, we analyzed the expression of Sox6 in the mouse developing brain and the function in NSPCs. By immunohistochemical analysis, we found that Sox6 was expressed in the ventricular zone of mouse telencephalon at 14.5 dpc, co-localizing with NSPCs's markers, nestin and Sox2. The expression of Sox6 in undifferentiated neurospheres also showed the co-localization with nestin, suggesting that Sox6 may play some roles in NSPCs. To evaluate the function of Sox6 in NSPCs, we performed a neurosphere forming assay in vitro. Over-expression of Sox6 using retrovirus vector in neurosphers caused an increase in the number of secondary neurospheres without changing the proliferation ability as determined by BrdU incorporation assay. These results suggest that Sox6 is expressed in NSPCs in the developing CNS and involved in the maintenance and/or survival of NSPCs.

KAKENHI18500299

## P3-c37 Roles of the LGN gene in the mouse neural development

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Asymmetric cell division has an important role to give rise to various types of cells from progenitor cells in *Drosophila* and *C.elegans*. On the other hand, the role and mechanisms of asymmetric division remains unclear in mammalian development although many similarities can be found in neural progenitor types and the molecular toolbox between the mouse brain and the *Drosophila* nervous tissue. To address these issues, we have investigated the mouse brain as a model system. We have knocked out the LGN gene encoding an orthologue of *Drosophila* PINS that regulates spindle orientation during neuroblast divisions. In the LGN-KO mouse, neuroepithelial cells acting as apical progenitors that divide at the ventricular surface, randomize their mitotic spindle orientation. In these mice, the basal progenitors, which divide once into neurons in the subventricular zone, are increased at the expense of the apical progenitors. We will discuss how neural progenitors generate various types of daughter cells based on those phenotypes observed in the LGN-KO mice.

## P3-c38 Regulation by cAMP signals of proliferative activity in murine neural progenitor cells

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Neural progenitor cells were prepared from neocortex of 15.5-day-old embryonic ddY mice, followed by culturing in DMEM/F12 containing epidermal growth factor (EGF) for 10 days. Under these in vitro conditions, round spheres were formed with clustered cells within 2 to 4 days. These cells were immunoreactive for nestin but not for microtubule-associated protein 2 (MAP2) or glial fibrillary acidic protein (GFAP) when determined before and after the cultivation for 10 days. These round spheres were further cultured in the absence of EGF added for spontaneous differentiation for an additional 4 days, when cells were immunoreactive for MAP2 and GFAP but not for nestin. A significant increase was induced in the mitochondrial activity and the total area of neurosphere in cells exposed to forskolin, dibutyryl cAMP and pituitary adenylyl cyclase activating polypeptide for 10 days, respectively. These results suggest that cAMP signals would modulate the proliferative activity for self-replication in murine neocortical neural progenitor cells.

## P3-c39 Unc5h4 and netrin-4 regulate survival and neurite outgrowth of mouse neocortical layer IV neurons in culture

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We have previously found that unc5h4, a member of netrin receptors, is expressed specifically in layer IV of the developing rodent neocortex. Here, we studied the effect of unc5h4 and its possible ligands on layer IV neurons, by overexpressing unc5h4 or netrin-4 with enhanced green fluorescent protein (EGFP), by in utero elecroporation into the cells that are destined for layer IV. After 72 hr, the cortical region containing the transfected cells was dissected and subjected to dissociated cell culture. Then, the number and morphology of EGFP-labeled and unlabeled neurons were examined after several days in culture. The result demonstrated that unc5h4-overexpressing labeled neurons tended to die compared with unlabeled neurons. Conversely, netrin-4 overexpression suppressed cell death and promoted neurite outgrowth. These results suggest that unc5h4 and netrins may regulate survival and differentiation of layer IV cells during cortical development.

KAKENHI(18021021)

## P3-c40 Premature neurogenesis in the *Pax6* heterozygous mutant cerebral cortex

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Pax6 is a transcription factor that plays essential roles in the developing cerebral cortex. *Pax6* homozygous mutant mice/rats exhibit severe abnormalities in cortical development and die soon after birth. Therefore, the role of Pax6 in neuronal specification and laminar formation in the developing cerebral cortex is not fully unclear. To address this issue we examined cortical development of *Pax6* heterozygous mutant rats (*rSey²/+*) that are viable and fertile. Histological analysis indicated a reduction of cortical thickness in the *rSey²/+* compared to that in wild type rats (WT). BrdU-pulse labeling and immunostaining with an anti-neuronal antibody revealed enhanced neuronal differentiation in the *rSey²/+* cortex. Furthermore, layer II/III neurons were precociously generated in the *rSey²/+* cortex compared to those in WT. These results suggest that premature neurogenesis occurs in the developing *rSey²/+* cerebral cortex, and that Pax6 regulates timing of neuronal differentiation during cortical development.

CREST

## P3-c41 RhoG regulates neural progenitor cell proliferation in mouse cerebral cortex

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In early cortical development, neural progenitor cells (NPCs) expand their population in the ventricular zone (VZ), and postmitotic neurons, which are sequentially generated in the cortical VZ, then migrate radially toward the pial side and form six-layer structure. To date, a series of studies have revealed the mechanisms of cortical development. However, the molecular control underlying it is still largely unknown. Here we investigated that the role of RhoG, a member of Rho-family GTPases, in the developing cerebral cortex. RhoG was specifically expressed in the VZ at E12 and E14. Expression of constitutively active RhoG promoted NPC proliferation and it was suppressed by RNAi-mediated knockdown of endogenous RhoG. However, there was no significant difference in the percentages of apoptotic cells between them. On the other hand, overexpression but not knockdown of RhoG led to abnormal cell morphology and migration defect. These results indicate that RhoG regulates NPC proliferation in cortical development.

KAKENHI(17079003)

## P3-c42 Origin of climbing fiber neurons and their developmental dependence on *Ptf1a*

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Climbing fiber (CF) neurons in the inferior olivary nucleus (ION) extend their axons to Purkinje cells. However, little is known about their birthplace and developmental molecular machinery. We found that *pancreas transcription factor 1a* (*Ptf1a*) is expressed in a discrete dorso-lateral region of the embryonic caudal hindbrain neuroepithelium. Analysis of mice which express β-galactosidase from the *Ptf1a* locus revealed that the majority of CF neurons are derived from this neuroepithelial region. In *Ptf1a* null mutants, putative immature CF neurons produced from this region were unable to migrate or differentiate appropriately, resulting in a failure of ION formation. These findings clarify the precise origin of CF neurons and the pivotal role of *Ptf1a* in CF neuron development.

KAKENHI, UEHARA MEMORIAL FOUNDATION

## P3-c43 Modulation of proliferation and differentiation by GABAergic signals in murine neural progenitor cells

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We have evaluated the possible modulation by signals mediated by GABA receptors ($GABA_AR$) of proliferation and differentiation activities in neural progenitor cells prepared from neocortex of 15.5-day-old embryonic ddY mice. The preparations before cultivation were immunoreactive to nestin but not to either microtubule-associated protein-2 or glial fibrillary acidic protein, while *in vitro* culture of these cells resulted in the formation of round spheres with similar immunoreactivities within 10 days. Constitutive expression was seen with mRNA for particular subunits of ionotropic GABAR ($GABA_AR$) and metabotropic $GABA_BR$ in cells before and after the culturing. A significant increase was induced in the mitochondrial activity in cells exposed to the $GABA_AR$ agonist muscimol and the $GABA_BR$ agonist baclofen at 100 μM for consecutive 10 days, respectively. These results suggest that both $GABA_AR$ and $GABA_BR$ may be functionally expressed by undifferentiated neural progenitors to modulate their proliferation activity.

## P3-c44 Transient overexpression of Ntan1 gene relevant to N-end rule in murine neuroblastoma Neuro2a cell line

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Ntan1 (amidohydrolase for N-terminal asparagine) is an essential component of the ubiquitin proteasome pathway in the N-end rule, whereas we have previously shown marked upregulation of Ntan1 in response to the static magnetism in primary cultured neurons. This study was aimed to investigate possible functional alterations by overexpression of Ntan1 in murine neuroblastoma Neuro2a cell line. Neuro2a cells were transiently transfected with Ntan1 expression vector, followed by the cultivation in either the presence or absence of all-*trans* retinoic acid (ATRA) for 2 to 5 days *in vitro*. Transient overexpression of Ntan1 not only significantly decreased mRNA expression of the synapse marker proteins synapsin-I and synaptotagmin-I in cells cultured with ATRA, but also markedly inhibited neurite outgrowth with a concomitant increase in the number of undifferentiated cells. These results suggest that Ntan1 may play a pivotal role in mechanisms underlying the neurite outgrowth required for the synapse formation in neuronal cells.

## P3-c45 Runx1 is required for the proper cell fate specification of dorsal root ganglion neuron

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The runt-related transcription factor Runx1 is expressed in cutaneous dorsal root ganglion (DRG) neurons. However, the function of Runx1 in the development of DRG neurons is not well known, because *Runx1*-deficient (*Runx1*$^{-/-}$) mice die around embryonic day 11.5 due to impaired hematopoiesis. To overcome the early embryonic lethality, we adopted a new approach to utilize transgenic *Runx1*$^{-/-}$ mice (*Runx1*$^{-/-}$::Tg) in which hematopoietic cells are selectively rescued by Runx1 expression. In *Runx1*$^{-/-}$::Tg, the total number of DRG neurons was increased, probably because of an increased proliferative activity of DRG progenitor cells and decreased apoptosis. In the mutant DRG, TrkA$^{+}$ neurons and peptidergic neurons were increased, while c-ret$^{+}$ neurons were decreased. In the dorsal horn of the spinal cord, projections of CGRP$^{+}$ axons expanded to the deeper layer. Our results suggest that Runx1 is involved in the cell fate specification of cutaneous neurons, as well as their projections.

the 21st Century COE Program

## P3-c46 RNA interference for p27kip1 induce supporting cell proliferation in organotypic cultures of postnatal mammalian cochlea

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In mammals, cochlear hair cell loss causes permanent hearing loss. Studies of avian indicated that hair cell regeneration occurs through supporting cell (SC) division. In this study, we examined whether RNA interference (RNAi) technique for p27Kip1,a cyclin-dependent kinase inhibitor, induces cell proliferation of SCs in the mammalian cochlea. A short hairpin RNA-expressing vector targeting to *p27Kip1* coding sequence (kindly provided by Drs. Kawauchi and Hoshino, Kyoto University) and an EGFP expression vector were co-electroporated into cochlear SCs of organotypic cultures obtained from postnatal 3 day ICR mice. After the electroporation, the organotypic cultures were incubated for 3, 5 and 7 days *in vitro*. Immunohistological studies showed that incorporation of BrdU was detected in RNAi-induced p27kip1 negative SCs and BrdU positive ratio was promoted as time advances. Our results suggested that RNAi for p27Kip1 induces cell proliferation of the postmitotic SCs.

KAKENHI

## P3-c47 Decoupling of gap junctions between neuroepithelial cells for neuronal differentiation

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Neuroepithelial cells are interconnected by gap junctions. However, the functional role of gap junction in neurogenesis remains unknown. In the present study, electrophysiological properties of neuroepithelial cells and newborn neurons were investigated by intracellular recordings in embryonic retinae of E3-6 chick. The neuroepithelial cell showed resting membrane potential of -72.0 ± 2.1 mV ($n$ = 12, mean ± S.E.) and input resistance of 12.5 to 80 MΩ (28.6 ± 5.2 MΩ, $n$ = 12). The input resistance was dramatically increased by application of carbenoxolone (CBX), a gap junction channel blocker; 10 μM of CBX increased the input resistance by 2-4 times and 100 μM CBX increased it by 3-8.5 times. The resting membrane potential became unstable and shifted in depolarizing direction with CBX. Newborn retinal ganglion cells showed higher input resistance and immature $Ca^{2+}$-spikes after losing gap junctional coupling. These results suggest that the gap junctional coupling stabilizes the membrane potential of neuroepithelial cells by lowering the input resistance and the decoupling of gap junctions is essential for the generation of spikes.

## P3-c48 A drastic change in gene expression profile of human neural progenitor cells under serum-free and serum-containing culture conditions

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Neural progenitor cells (NPCs) show a self-renewing and proliferating capacity. NPCs expanding in culture provide an unlimited source for transplantation therapy of neurodegenerative diseases. To obtain a desirable source, the interaction of environmental and genetic factors that regulates stem cell fate should be elucidated in the human NPCs. We cultured human NPCs in the DMEM/F-12 medium containing EGF, bFGF and LIF. After several months, nestin$^{+}$ GFAP$^{+}$ floating spheres were incubated for 72 hours in the medium with or without 10% FBS. They were processed for analyzing on a microarray (Agilent, 41,000 genes), validated by real-time PCR. The 46 genes (ID1, ID2, ID3, CTGF, TGFA, METRN and GFAP) were upregulated, while 23 genes (NPTX1, DLL1, DLL3, SOX4 and HES5) were downregulated in the culture with the serum. Thus, the serum upregulated the genes that enhance astrocytic differentiation but repressed those involved in maintenance of the stem cell capacity.

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## P3-d01 Roles of p38 MAP kinase in mouse neural progenitor cells survival and proliferation

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p38 Mitogen-activated protein kinase (p38 MAPK) is one of the MAP kinase families, and supposed to play significant roles in survival of mature neuronal cells. In developing mouse brain, however, the localizations and roles of p38 remain unclear. In the present study, p38 MAPK was preferentially expressed in the brain from embryonic 10th day (E10) to E14 rather than that from postnatal 8 weeks. In the E10-E14 brains, most of the p38 MAPK-positive cells were located in the subventricular zone, and were positive for nestin. The early embryonic brain cells and cultured neural progenitor cells exposed to the selective chemical inhibitors of p38 MAPK for 7 days showed increase in the number of neurosphere, intracellular amount of ATP, and number of Ki-67-positive cells. These results suggest that p38 MAPK could involve in the survival and/or proliferation of neural progenitor cells in developing mouse brain.

## P3-d02 Systematic immunostaining and RNAi experiments for finding the novel molecular marker candidates of the growth cone.

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The growth cone (GC) is the tip of the extending neuronal processes for the accurate synaptogenesis. There were no methods discovering the molecules involved in the GC functions systematically, thus, the molecular basis of the GC behavior is poorly understood and its molecular markers were not known except GAP-43. Here we take the strategy to identify the GC proteins that are important for axonal growth for combining purification of the GCs from rat brain, proteomic analysis by multidimensional protein identification technology of the GC; immunostaining using antibodies against the proteins identified by proteomics; and RNAi that inhibits the axonal growth. Using this approach, we found 90 proteins which are more concentrated in the GC area than GAP-43. Among more than siRNAs against 50 proteins, those against 15 proteins were shown to inhibit the axonal growth significantly. In conclusion, our approach succeeded in effectively finding the protiens that opereates the GC behavior.

KAKENHI(#17023019)

## P3-d03 Cxcl12/Cxcr4 chemokine signaling is required for placode assembly and sensory axon pathfinding in the zebrafish olfactory system

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In the zebrafish olfactory system, precursors of olfactory sensory neurons (OSNs) assemble into a compact cluster to form the olfactory placode. Subsequently, OSNs differentiate and extend their axons to the presumptive olfactory bulb with high precision. The chemokine receptor *cxcr4b* is expressed in the migrating olfactory neuron precursors, and its ligand *cxcl12a* is expressed in the abutting anterior neural plate. The expression of *cxcr4b* persists in the olfactory placode at the initial phase of OSN axon pathfinding. Interfering with Cxcl12a/Cxcr4b signaling perturbs the assembly of the olfactory placode, resulting in the appearance of ventrally displaced olfactory neurons. Moreover, OSN axons frequently fail to exit the olfactory placode and accumulate near the placode-telencephalon border in the absence of Cxcr4b signaling. These data indicate that chemokine signaling contributes to both the olfactory placode assembly and the OSN axon pathfinding in zebrafish.

KAKENHI 16300105, CREST

## P3-d04 Key role of Rho/Rho-kinase signaling in hindbrain motor axon patterning

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In this study, we performed conditional expression of a dominant-negative form for Rho-kinase in transgenic mice by use of Cre-loxP system to suppress the activity of Rho/Rho-kinase signaling in developing hindbrain motor neurons. The patterning of somatic motor (SM) axons, but not of visceral motor axons, was specifically impaired in transgenic embryos. In addition, chemoattractivity for SM axons in response to hepatocyte growth factor (HGF) was inhibited in the explant culture derived from transgenics. These findings suggest that Rho/Rho-kinase pathway dependent on HGF signaling plays a critical role in the patterning of SM axons during hindbrain development.

## P3-d05 Localization of functional reelin receptors as revealed by alkaline phosphatase-reelin fusion protein

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Reelin is essential for proper layer formation of mammalian brain. Reelin is received by cells that express Reelin receptors (ApoER2 and VLDLR), but when and where such interaction occurs remains obscure. Here we tried to know the localization of functional Reelin receptors. In situ staining of developing mouse forebrain, both from wild-type and reeler mice, with alkaline phosphatase fusion protein of the receptor-binding domain of Reelin revealed that functional Reelin receptors were mainly localized in the subventricular-intermediate zones, suggesting that Reelin is received by migrating neurons before they path through the subplate and that the receptors are down-regulated thereafter. In developing cerebellum Reelin receptors are expressed in not only purkinje cells but also various kinds of neurons. These observations will help understand how Reelin and its receptors organize neuronal layer formation.

KAKENHI17082007

## P3-d06 Roles of 5-$HT_{1A}$ and 5-$HT_{2A}$ receptors in the dendrite formation and synaptogenesis of cerebral cortex pyramidal neurons

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Recent studies have suggested 5-hydroxytryptoamine (5-HT) affects neural development, but roles of receptor subtypes in neuronal development are largely unknown. We have previously examined roles of 5-$HT_{1A}$ and 5-$HT_{2A}$ receptors in the formation of dendrites and synapses by dissociation culture (Shiga et al., 2006). Embryonic day 16 or 18 rat cerebral cortex was cultured in the presence of 5-$HT_{1A}$ or 5-$HT_{2A}$ receptor agonists. In the chronic presence of the agonists, dendritic outgrowth was promoted by 5-$HT_{1A}$ receptor, while inhibited by 5-$HT_{2A}$ receptor. In addition, synaptogenesis was promoted by 5-$HT_{2A}$ receptor, whereas inhibited by 5-$HT_{1A}$ receptor. In order to clarify the mechanisms of 5-$HT_{1A}$ and 5-$HT_{2A}$ receptors underlying the dendrite formation and synaptogenesis, we examined acute effects of 5-$HT_{1A}$ and 5-$HT_{2A}$ receptor agonists. The agonists were added at 7-14 days of culture. We fixed the culture within 24 hours, and analyzed synaptogenesis and spine formation.

The Long-range Research Initiative by Japan Chemical Industry Association

## P3-d07 R-Ras controls axon specification upstream of GSK-3 $\beta$ through integrin-linked kinase

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Spatially regulated glycogen synthase kinase 3 $\beta$ (GSK-3 $\beta$) activity is critical for specifying axon-dendrite fate; however, the upstream signaling of GSK-3 $\beta$ in determination of neuronal polarity still remains obscure. Here we found that in cultured hippocampal neurons, the small GTPase R-Ras selectively localized in a single neurite of stage 2 neurons, and that its activity increased after plating and peaked between stages 2 and 3. Ectopic expression of R-Ras induced global inactivation of GSK-3 $\beta$ and formed multiple axons, whereas knockdown of endogenous R-Ras by RNA interference blocked inactivation of GSK-3 $\beta$ and axon formation. The inactivation of GSK-3 $\beta$ and axon formation by R-Ras required integrin-linked kinase (ILK), and subcellular localization of ILK is strictly regulated by R-Ras-mediated phosphatidylinositol 3-kinase (PI3K) activity. In addition, membrane targeting of ILK was sufficient to inactivate GSK-3 $\beta$ and form multiple axons. Our study demonstrates a novel role of R-Ras and ILK upstream of GSK-3 $\beta$ in regulation of neuronal polarity.

JSPS, KAKENHI

## P3-d08 The Cdc42 activator Zizimin1/Dock9 regulates dendritic development in hippocampal neurons

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Rho family small GTPases are key regulators for morphological changes of neurons. Cdc42 is involved in actin cytoskeletal reorganization in this process. Recent studies have identified Zizimin1, a member of Dock180-related protein family, as a Cdc42-spicific GEF. However, the physiological function of Zizimin1 is totally unknown. Here we studied expression pattern and neuronal function of Zizimin1 in developing rodent brains. Zizimin1 is strongly expressed in developing brain including hippocampus and cerebral cortex in late developmental stages. Expression of a Zizimin1 mutant lacking GEF activity in primary cultured rat hippocampal neurons reduced the total dendrite length and the number of branch points. In addition, knockdown of Zizimin1 by siRNA also suppressed dendritic development. These results suggest that Zizimin1 plays an important role in dendrite development through the regulation of Cdc42 activity. We are now further dissecting the detailed function of Zizimin1 in neuronal development.

KAKENHI(17079003)

## P3-d09 The exremly conserved C-terminal region of Reelin is not nsecessary for secretion but is required for efficient activation of downstream signaling

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Reelin is a secreted glycoprotein essential for correct development of the mammalian brain. Reelin has the short and highly positively-charged C-terminal region (CTR), and its primary sequence of CTR is almost completely conserved among all vertebrates except fishes, indicating its importance. It is widely accepted that CTR is necessary for secretion of Reelin. Here, we found that CTR is not necessary for secretion of Reelin. We then found that Reelin mutants without CTR were less potent in activating the downstream signaling in cortical neurons. Interestingly, these mutants did not bind well to the isolated membrane bearing Reelin receptors or receptor-expressing cells. Therefore, it is concluded that CTR is required for efficient activation of downstream singaling, presumably via binding to unidentified "co-receptor" molecules on the cell membrane.

KAKENHI(17082007)

## P3-d10 Different functions of two RPTP β isoforms in neurons

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The receptor-type protein tyrosine phosphatase β (RPTP β) is a member of transmembrane chondroitin sulfate proteoglycans. There exist two variants: the full-length form of RPTP β (fRPTP β); the shorter receptor form of RPTP β (sRPTP β). Although they have two intracellular catalytic phosphatse domainsin, sRPTP β lacks greater part of glycosminoglycan attachment region. Until now, little is known about the functional difference between fRPTP β and sRPTP β in neurons. In the present study, the transfection of sRPTP β vector made cultured hippocampal neurons to promote dendritic branching, however, that of fRPTP β vector causesd neurons to attenuate synaptogenesis. Both sRPTP β and fRPTP β were not extracted by detergent treatment. These results suggest that fRPTP β and sRPTP β have different functions concerning synaptogenesis and dendritogenesis possibly via cytoskeletal interations.

## P3-d11 tPA-plasmin-dependent degradation of extracellular matrix causes structural plasticity in vasopressin neurons

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Osmotic homeostasis is controlled mainly by arginine-vasopressin (AVP) in mammalians. Here, we show tissue plasminogen activator (tPA) and plasminogen mediate AVP elevation of blood circulation in animals. Tissue plasminogen activator (tPA)-deficient mice showed more hyperosmolar and became higher mortality than wild-type littermates against chronic osmotic challenge. Plasma levels of AVP was significantly lower in tPA-deficient mice than wild-type littermates after acute and chronic osmotic challenges. Similar deficiency of AVP increase into blood in response to osmotic challenges was observed in plasminogen-deficient mice. The abnormality of AVP release in tPA- and plasminogen-deficient mice was not due to the reduction of neuronal activity in brain regions related with osmotic control. Chronic osmotic challenge significantly reduced the expression of laminin. Thus,tPA-plasmin axis is responsible for the degradation of extracellular matrix and AVP release.

## P3-d12 Crk is required for Reelin-mediated hippocampal dendrite outgrowth

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The Reelin signaling pathway has been implicated in both neuronal positioning and process extension. Here we have examined a role for Dab1, a docking protein on the pathway, in the formation of hippocampal processes during postnatal development by Cre- mediated excision of a conditional *dab1* allele. We found that excision of the conditional *dab1* allele by the activation of a ubiquitously expressed Cre-estrogen receptor fusion at postnatal day 3 leads to a simplification of the hippocampal processes compared to control animals.
Recently, it has been demonstrated that tyrosine phosphorylated Dab1 binds Crk and CrkL, SH2-SH3 containing adaptors, but their function in the Reelin signaling pathway remains unknown. We have examined a role for Crk and CrkL in postnatal Reelin-induced hippocampal dendrite outgrowth using cultured neurons. We found these molecules are required for Reelin-induced hippocampal dendritogenesis, by reducing Crk and/or CrkL with a Lentiviral gene knockdown approach, providing evidence that these Dab1 binding proteins are required for downstream events in Reelin signaling.

NINDS intramural funding

## P3-d13 Screening and functional analyses of the molecules that are preferentially expressed in the upper cortical plate of the developing cerebral cortex

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During the cerebral cortical development, major population of excitatory neurons originate in the ventricular zone and migrate to beneath the marginal zone (MZ) to form the cortical plate. To clarify the events that occur beneath the MZ, we screened for the molecules that were preferentially expressed beneath the MZ using GeneChip analysis and in situ hybridization. Consequently, we identified 40 molecules that were preferentially expressed in the upper cortical plate. Especially, Ndrg1 mRNA was strongly expressed in the neurons that were located just beneath the MZ. Interestingly, the Ndrg1 protein was detected as punctates mainly in the MZ, suggesting that it was located on the primitive dendrites of neurons that had just reached beneath the MZ. Thus, to examine the functions of Ndrg1 in cortical development, we applied in utero electroporation system. Here we report the results of loss-of-function experiments of Ndrg1 and discuss its functions during cortical development.

## P3-d14 Observation on developing cerebellar cortex using acoustic impedance microscope and characterization of L-type Calcium channel distribution

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Two-dimensional acoustic impedance imaging is useful for observation on living organs with no invasion. We have reported that three-layer structure in cerebellar cortex was observed as the contrast in acoustic impedance. In order to observe more clearly or specifically, we examined substrate conditions and specimen treatments. Either $SiO_2$ or $TiO_2$ coated substrate, which had a contact angle between 25 to 55 degree, gave us good discrimination. In addition, the application of 100 μM $CdCl_2$ induced increment of molecular layer impedance at P21. Neither 50 μM $NiCl_2$ nor 2 mM $CaCl_2$ treatment showed that increment. $Cd^{2+}$ has high affinity for L-type Ca channel, so it suggested that selective binding of $Cd^{2+}$ to L-type Ca channel could show the distribution of functional L-type Ca channel in living cerebellar cortex.

## P3-d15 Neural zinc finger (NZF) transcription factors regulate development and survival of dorsal root ganglia neurons

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NZF-2b and NZF-3 transcription factors, which has C2HC-type zinc finger motifs, are expressed in subsets of DRG sensory neuron, suggesting that they may regulate development of specific sensory neurons. To elucidate in vivo functions of NZF family, we generated knockout mice of NZF-2 and NZF-3 gene, and double knockout mice both of NZF-2 and NZF-3. Double knockout mice exhibited a forelimb posture abnormality. To find the cause of this defect, we examined the neuronal differentiation and the trajectories of spinal nerves. Double mutant mice embryos showed spinal nerve hypoplasia especially projecting into limb buds. In the cervical DRG of the double mutants, the number of sensory neurons was fewer than that of wild type embryos. Furthermore, TrkC- and parvalbumin-positive proprioceptive neurons were markedly decreased in the mutant DRG. Our data suggest that NZF-2 and NZF-3 are important for regulating the development of proprioceptive DRG sensory neurons and may help to elucidate the molecular mechanisms underlying somatosensory- related ataxia.

## P3-d16 Phenotype analysis of *crmp5*-deficient mice

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Collapsin response mediator proteins (CRMPs) are involved in neuronal cell migration and axonal guidance. The expression of CRMP5, one of the CRMP members, is abundantly expressed in fetal brain. CRMP5 is also thought to play a critical role in neuronal degeneration associated with paraneoplastic syndrome. To elucidate the function of CRMP5 *in vivo*, we examined phenotype of *crmp5* $^{-/-}$ mice. CRMP5 protein was detected at P21 in all layers of cerebellum by anti CRMP5 antibody staining. In *crmp5* $^{-/-}$ mice, decreased staining of MAP2 and aberrant dendritic morphogenesis were observed in the cerebellar Purkinje cells. Furthermore, tail hanging test revealed that *crmp5* $^{-/-}$ mice, but not *crmp5* $^{+/-}$ mice and *crmp5* $^{+/+}$ mice, exhibited abnormal limb-clasping reflexes. These findings suggest that CRMP5 plays an important role in cerebellar development and funciton.

## P3-d17 Phosphorylation of CRMP2 at S522 is required for proper dendritic patterning of layer V cortical neurons in vivo

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We report that CRMP2 is phosphorylated by Cdk5 at S522. This phosphorylation is essential for the sequential phosphorylation by GSK3 $\beta$ at T509, and caused reduction in its affinity to tubulin. To determine the role of the phosphorylation *in vivo*, we generated CRMP2 knock-in (KI) mice in which S522 was replaced with A. Since the phosphorylated CRMP2 at S522 was abundant at dendrites in cultured cortical neurons, we examined dendritic morphology using YFP-H transgenic mice. CRMP2 KI mice did not show any defects in dendritic patterning. In CRMP2S522A KI;*crmp1* double mutant mice, however, we observed disorientation of basal dendrites of Layer V cortical neurons. These findings suggest that phosphorylation of CRMP2 at S522 is essential for proper development of basal dendritic patterning *in vivo*.

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## P3-d18 The role of *calmodulin 1* in the migration of precerebellar neurons

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Migration of neurons from their birthplace to the appropriate positions is a fundamental event, and calcium signaling is implicated in regulating this process. Calmodulin (CaM) is an upstream regulator of calcium signaling cascade, and is encoded by three non-allelic genes (*calm-1,2,3*) in vertebrates. Since all three *calm* genes encode an identical protein with no amino acid substitutions, they are thought to play differential roles by providing distinct subcellular CaM pools through the regulation of mRNA sorting and translation. Here we investigated the role of CaM signaling in migration of precerebellar neurons (PCNs). We examined the expression profiles of three *calm* genes in PCNs by *in situ* hybridization with gene-specific cRNA probes. We found that *calm1* is expressed during migration, followed by the expression of all three genes around the period of nucleus formation. RNA-interference-mediated knockdown of *calm1* resulted in delayed migration of PCNs. These results suggest that the *calm1* signaling is required for the proper tangential migration of PCNs.

## P3-d19 Inhibition of axonal outgrowth in the tumor environment: Involvement of class 3 semaphorins

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That tumors lack innervation is dogma in the field of pathology, but the molecular determinants of this phenomenon remain elusive. We studied the effects of conditioned media from Colon 26 and B16 mouse tumor cell lines on the axonal outgrowth and cellular differentiation of embryonic ICR mouse dorsal root ganglion cells. Tumor-conditioned media suppressed dorsal root ganglion axonal extension but had no effect on its neuronal or glial differentiation. We found that the tumor cells expressed most of class 3 semaphorins - axon guidance molecules. Blocking activity of class 3 semaphorins with the soluble receptor neuropilin-1 significantly counteracted the tumor-induced inhibition of axonal extension. Together, these results suggest a role for tumorsecreted class 3 semaphorins in selectively inhibiting axonal outgrowth of dorsal root ganglion neurons.

## P3-d20 Measurements of changes in $[Ca^{2+}]_i$ and $[Cl^-]_i$ during glioblastoma cell movement

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Malignant glioblastoma cells invasively migrate into widespread regions of brain with changing cellular morphology. Alterations in intracellular ion levels such as $[Ca^{2+}]_i$ or $[Cl^-]_i$ are involved in the regulation of cell motility or cellular volume. However, it has well not known about the spatial-temporal changes in these ion levels, especially $[Cl^-]_i$ during invasive migration. To assess it, we have attempted to visualize the cell movement with dynamic morphological alterations and changes in $[Ca^{2+}]_i$ or $[Cl^-]_i$ simultaneously. Combination of time-lapse observation and $Ca^{2+}$ imaging demonstrated that $Ca^{2+}$ oscillations were observed in the glioblastoma cells which have the structures like leading process during cell movement. Furthermore, FRET imaging with Yellow-camleon and Clomeleon to calculate $[Ca^{2+}]_i$ and $[Cl^-]_i$ levels, respectively, allowed us to detect the changes in FRET efficiencies by pharmacological applications. These results raise the possibility of analysis of the spatio-temporal characteristic of $[Ca^{2+}]_i$ and $[Cl^-]_i$ by FRET imaging during long-time migration.

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## P3-d21 Induction of cell migration of neural progenitor cells *in vitro* by α-1 adrenergic receptor stimulation

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Previous studies have reported that classical neurotransmitters such as *l*-dopamine and *l*-adrenaline regulate the proliferation of neural progenitor cells (NPCs). However, the effects of these neurotransmitters on the cell migration are unknown. We therefore attempted to determine whether *l*-dopamine and *l*-adrenaline regulate cell migration *in vitro*, using embryonic NPCs. Neurospheres on the cover glass coated with fibronectin began to migrate out of the sphere with or without various drugs. Migration was assessed by measurement of the number of migrated cells from the edge of the sphere. We found that the treatment with *l*-adrenaline significantly increased the number of migrated cells and these effects are inhibited by α-1 adrenergic receptor antagonists. In contrast, treatment with apomorphine, dopamine receptor agonist, had no effects on cell migration. These results suggest that the activation of α-1 adrenergic receptors enhances the cell migration of embryonic NPCs, but not adult NPCs *in vitro*, and may be an important process in embryonic brain development.

## P3-d22 Calcium dependent-regulation of neurite outgrowth mediated by a protein translational factor eEF2 within nerve growth cones

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Local protein synthesis within growth cones is known to regulate growth cone behavior. However, the mechanism of the regulation remains unclear. We found that eukaryotic elongation factor-2 (eEF2) in growth cones regulates neurite outgrowth in calcium-dependent manner. Acute localized loss of eEF2 in growth cones by chromophore-assisted laser inactivation (CALI) resulted in growth arrest of neurite. Extracellular ATP-evoked calcium transient activated EF2 kinase (EF2K) and increased eEF2 phosphorylation in growth cones, and also inhibited neurite outgrowth. This ATP-induced growth arrest of neurite was abolished by CALI of EF2K. The data suggest that local mRNA translation regulated by eEF2 in the growth cone may play an important event in the neurite outgrowth.

CREST, KAKENHI(13680851)

## P3-d23 Neuronal calcium sensor-1 in nerve growth cones independently regulates both the neurite outgrowth and growth cone morphology

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Neuronal calcium sensor-1 (NCS-1) is a calcium binding protein, which is specifically expressed in the nervous system. NCS-1 was distributed in a cluster and co-localized with the type 1 IP3 receptor in the central domain of the growth cone. The acute localized loss of NCS-1 in the growth cone induced by chromophore-assisted laser inactivation (CALI) resulted in the growth arrest of neurites and lamellipodial and filopodial retractions. The CALI-induced growth arrest of neurites was abolished by the pharmacological inhibition of IP3 receptor, while CALI-induced lamellipodial and filopodial retractions were unaffected. These findings suggest that NCS-1 in growth cones may independently regulate both the neurite outgrowth and growth cone morphology.

CREST,JST

## P3-d24 How does EphrinB3/EphA4 signaling regulate motor-circuit formation?

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The ephrin/Eph system plays a central role in neural-circuit formation; however, its downstream effectors are poorly understood. Here we show that a-chimerin Rac-GAP mediates ephrinB3/EphA4 forward signaling. We discovered a spontaneous mouse mutation, *miffy (mfy)*, which results in a rabbit-like hopping gait, along with impaired corticospinal axon guidance and spinal central pattern generators. Using positional cloning, transgene rescue and gene targeting, we demonstrated that dysfunction of a-chimerin leads to *mfy* phenotypes similar to those of *EphA4*$^{-/-}$ and *ephrinB3*$^{-/-}$ mice. a-chimerin interacts with EphA4, and, in response to ephrinB3/EphA4 signaling, inactivates Rac, which is a positive regulator of process outgrowth. Moreover, down-regulation of a-chimerin suppresses ephrinB3-induced growth-cone collapse in cultured neurons. Our findings indicate that ephrinB3/EphA4 signaling prevents growth-cone extension in motor-circuit formation via a-chimerin-induced inactivation of Rac.

PRESTO, KAKENHI (18021041)

## P3-d25 CRMP1 regulates spine maturation through mediating Semaphorin3A signaling *in vivo*

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CRMP1 is one of the CRMP family members that mediate signal transduction of axon guidance molecules. We here show evidence that CRMP1 is involved in Sema3A-Cdk5 signaling to regulate spine maturation in the cerebral cortex. In the cultured cortical neurons from *crmp1*$^{+/-}$ mice, Sema3A increased the number of clusters of synapsin I and PSD-95, but this increase was attenuated in *crmp1*$^{-/-}$ mice. This phenotype seen in *crmp1*$^{-/-}$ mice was rescued by introduction of wild-type CRMP1 but not CRMP1-T509A/S522A, a mutant unphosphorylatable by Cdk5. Golgi-impregnation method showed that the *crmp1*$^{-/-}$ layer V cortical neurons showed lowered density of synaptic bouton-like structures and this phenotype had genetic interaction with *sema3A*. These findings suggest that CRMP1 regulates spine maturation through phosphorylation of CRMP1 by Sema3A-Cdk5 cascade.

JST CREST

## P3-d26 IgLON adhesion molecules participate in synapse formation of hippocampal neurons

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IgLON cell adhesion molecules are known to belong to a subfamily of immunoglobulin superfamily and comprise four members: LAMP, OBCAM, neurotrimin, and kilon. Several in vitro studies showed that members of IgLON subgroup play a role in neurite outgrowth. However, although ultrastructural analysis demonstrated the localization of IgLON at synaptic region in the adult brain of rat, it has not been studied whether IgLONs are involved in the formation of synapses. In this study, we examined the overexpression of IgLONs on synapse formation of hippocampal neurons. Hippocampal neurons were transfected with a vector containing the GFP and each IgLON coding sequence. The quantitative analysis showed that overexpression of LAMP or OBCAM increased the number of synapses. In contrast, overexpression of kilon or neurotrimin surpressed the synapse formation. These results indicate that IgLON are the synaptic cell adhesion molecules concerning the regulation of synapse formation.

## P3-d27 Regulatory mechanisms of Cdh6 gene expression in the postnatal mouse brain

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Cdh6 is a synaptic adhesion molecule whose expression demarcates neural circuits such as auditory, somatosensory and other systems in the postnatal mouse brain. While crucial roles of cadherins in formation and/or modulation of synaptic junction have been implicated, what brings cadherin expressions in restricted sets of neurons along functional circuitries remains largely unknown. Here we investigate genetic control elements that could confine Cdh6 expression within subsets of neurons by using an innovative method in which systematic integration of a reporter cassette into bacterial artificial chromosomes with extensive coverage of huge Cdh6 gene locus was followed by fine visualization of reporter activities in the postnatal mouse brain after the transgenesis. Our screening revealed that divisible genomic segments controlled each nucleus specific expression of Cdh6 at right timing for circuit formation, suggesting developmental and/or evolutional value of cadherin regulatory machineries to shape elaborated neuronal circuitries in the nervous system.

## P3-d28 Localization of Protocadherin-α protein in the mouse hippocampal neuron.

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The Protocadherin-α (Pcdh-α) proteins, members of clustered protocadherin family, are putative cell recognition/adhesion molecules in the brain. Here, we examined localization of Pcdh-α proteins in hippocampal culture by using monoclonal antibody against Pcdh-α cytoplasmic region, and by time-lapse imaging of GFP fused Pcdh-α proteins. The double-label immunostaining using antibodies against Pcdh-α, Pcdh-γ, synapsin-1 (pre-synapse), PSD95 (post-synapse), and γ-adaptin (golgi apparatus) showed that the dotted signals of Pcdh-α were observed on growth cone, neurite and soma. Although no Pcdh-α was double-labelled with synapsin-1, PSD95 nor γ-adaptin, most of Pcdh-α were colocalized with Pcdh-γ. Next, GFP fused Pcdh-α protein was transfected to mouse hippocampal culture. The fused proteins were detected on soma, neurite, and growth cone, similar to endogenous proteins. The time-lapse imaging revealed that the part of fused proteins moved along with neurites. Understanding of Pcdh-α protein localization will contribute to uncover molecular mechanisms underlying neural circuit formation.

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## P3-d29 Lamina-specific expression and subcellular localization of protocadherin9 in the developing cortex

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During development, sensory thalamic axons form branches in layer4 and 6. To explore the layer-specific targeting mechanism, we searched for the genes that are expressed in the target layers, by constructing a subtraction cDNA library. We found that protocadherin9 (pcdh9) was expressed specifically in layers 4 and 6 of P7 rat cortex. In terms of area specificity, pcdh9 was expressed strongly in the occipital region. In the developmental time course, pcdh9 was already expressed in the cortical plate at E18. The expression was gradually increased by P7 and thereafter decreased. Pcdh9 expressed in the thalamus as well, particularly in the lateral geniculate nucleus and ventral posteromedial nucleus. Furthermore, the expression analysis of pcdh9-EGFP in dissociated cell culture demonstrated that the protein was more localized in cell bodies, dendrites, and synaptic buttons of cortical neurons. These results suggest that pcdh9 may be involved in the interaction between thalamocortical axons and cortical neurons in the target layers.Research funds: KAKENHI(18021021)

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## P3-d30 Expression of NEPH family of immunoglobulin superfamily in the hindbrain

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NEPH family molecules including NEPH1,2,3 and nephrin belong to the transmembrane immunoglobulin superfamily and are involved in cell adhesion in the kidney. Although their expression has been reported in the nervous system, their function remains unclear. In the present study, we analyzed the expression of NEPH family molecules in the hindbrain. Among NEPH family members, mRNA of NEPH3 and nephrin are strongly expressed in the ventricular zones of the dorsolateral pons and the cerebellum at E11.5, and their expression gradually decreases at later stages. NEPH3 and nephrin-positive cells are not Tuj1 positive, suggesting that they are not mature neurons. Within the ventricular zones, NEPH3 and nephrin cells are not localized in Math1 or Neurogenin1-positive area. We are currently analyzing the identity of NEPH3 and nephrin-positive cells, and the function of these molecules in the hindbrain.

KAKENHI

## P3-d31 Protocadherin-α family is required for sensorimotor gating and spatial learning

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Protocadherin(Pcdh)-a family is a subfamily of the clustered protocadherin family that occurs as diverse splicing variants. Pcdha proteins are located in axons during the neurodevelopment and partially at the synapses. However the roles of the Pcdha function in the brain are not well understood. To address *in vivo* function of the Pcdha family, we generated several lines of Pcdha gene-targeted mice and subjected them to anatomical, biochemical and behavioral approaches. Among these mutant, Pcdha$^{\Delta Bneo/\Delta Bneo}$ which lacked a splicing variant of Pcdha-B and downregulated Pcdha-A proteins to approximately 20% showed no obvious morphological abnormalities in the brain. They, however, displayed enhancement of contextual fear conditioning and also deficit in prepulse inhibition (PPI) of the acoustic startle response in behaviors. Moreover, the abnormality of PPI was often rescued by administration of haloperidol, which is dopamine D2 receptor antagonist. These results suggest that Pcdha family proteins involve in behavioral regulation for fear learning and sensorimotor processing.

KAKENHI 17024034

## P3-d32 Protocadherin-α family is required for early establishment and maintenance of the olfactory axonal projection

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Olfactory sensory neurons (OSNs) expressing the same odorant receptor project precisely their axons to specific glomeruli in the olfactory bulb. This process is directed by axonal identity of odorant receptor and neural activity. However, other molecules must also contribute to the mechanisms underlying the appropriate olfactory sensory map formation. Here we demonstrate that protocadherin-a (Pcdha) proteins, diverse cadherin-related molecules that are encoded as a gene cluster, are highly concentrated in the axons of OSNs and olfactory glomeruli. Pcdha gene-targeted mice, in which a conserved region of the Pcdha gene cluster is deleted, show disturbed OSN axonal sorting to the glomeruli, and small, ectopic glomeruli. Interestingly, the abnormal projections were observable in neonates and these ectopic glomeruli do not disappear with age. Thus, Pcdha molecules are involved in the early establishment and maintenance of OSN axonal sorting into specific glomeruli in the olfactory bulb.

KAKENHI (17024034)

## P3-d33 Alteration of the cell adhesion molecule CHL1 expression in a specific subset of primary afferent neurons and dorsal horn following nerve injury

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We investigated expression of cell adhesion molecule close homologue of L1 (CHL1) in the dorsal root ganglia (DRG) and spinal cord in the rat neuropathic pain model. Expression of CHL-1 was examined in the rats (Sprague-Dawley, weighing 200-250 g) received tibial and common peroneal nerve ligation (spared nerve injury model; SNT). Constitutive expression of CHL-1 mRNA was detected in the DRG. Following SNI, expression of CHL-1 mRNA showed up-regulation in L4/5 DRG. Induction of CHL1 mRAN was mainly observed in small size neurons and in the satellite cells around large size neurons. CHL1 immunoreactivity (ir) was increased in cell membrane of small size neurons and formed ring structures. In the spinal cord, CHL1 ir was increased in the ipsilateral laminae I-II of dorsal horn and showed a button like staining pattern following SNI. The alterations of CHL1 may be involved in the structural plasticity that associates with neuropathic pain.

## P3-d34 The fifth splice variant of neuroglycan C, a CNS-specific transmembrane chondroitin sulfate proteoglycan

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Neuroglycan C (NGC) is a membrane-spanning chondroitin sulfate proteoglycan that is predominantly expressed in the brain. We have reported that four transcripts for NGC splice variants (NGC-I to -IV) exist in the human brain as well as the rat brain. In the present work, we examined the expression of NGC-V that was reported as a new splice variant of NGC in databases (accession number AK053891.1). The NGC-V cDNA comprises five exons (2, 3, 4, 4', and 4") of the NGC gene, and the deduced NGC-V protein, which consists of 344 amino acids, has the longest cytoplasmic domain among five splice variants. Northern blot and RT-PCR experiments showed that the transcript for NGC-V existed in the mouse brain. Now, we are focusing on the function of NGC-V in the brain.

KAKENHI (17500246)

## P3-d35 Molecular dynamics of cell adhesion molecule L1 induced by theta burst stimulation in rat hippocampus

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One prominent form of synaptic change in the hippocampus is long-term potentiation (LTP) that occurs following specific patterns of synaptic activity. Changes in synaptic activity can lead to changes in neuronal function and morphology. Cell adhesion molecules (CAMs), such as L1, are known to influence cell-cell interactions and can regulate the kinds of cell motility that could underlie neuronal plasticity. However, the mechanism by which neuronal activity influences the expression and distribution of L1, as well as the signal transduction machinery affecting subsequent changes in cell-cell interactions remain poorly understood. Here we present that Y1176 of the YRSL motif with in L1 cytoplasmic domain is dephosphorylated in LTP-induced hippocampus, leading to the association of L1 with AP-2 that are required for clathrin-mediated endocytosis. These data suggest that clathrin-mediated recycling of L1 at presynaptic sites is enhanced by certain kinds of neural activity, and that maintenance of LTP-induced synaptic changes is regulated by L1 recycling.

## P3-d36 Postsynaptic accumulation of cell adhesion molecules imaged in vivo

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Innervating axon can induce postsynaptic assembly through transsynaptic cell adhesion. However, there is little evidence directly showing such mechanisms do work in vivo. Here we examined the dynamics of the cell adhesion molecule Fasciclin2 (Fas2) in Drsophila neuromuscular synaptogenesis. We previously showed YFP-tagged Fas2 strongly accumulated at the nascent postsynaptic site depending on axonal Fas2 and found Fas2 was involved in postsynaptic assembling of scaffold protein Dlg/PSD-95. Here, to further study the dynamics of postsynaptic Fas2, we performed photobleaching analysis. Turnover of Fas2 was faster before innervation than after innervation which suggests axon entraps mobile Fas2. Genetic and deletion analyses indicate Fas2 on motoneuron is crucial for holding Fas2 at postsynaptic site. These results support the model that axonal Fas2 entraps postsynaptic mobile Fas2 to represent newly innervated region, which in turn induce postsynaptic assembly of Dlg and other molecules in vivo.

## P3-d37 Expression of neurocan in the rat nucleus of tractus solitarius during postnatal development

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Neurocan is thought to be associated with barrier functions and provide nonpermissive environment for neural plasticity. Our previous studies have reveled that local neural network in the rat nucleus of tractus solitarius (NTS) are reorganized around at postnatal day (P) 10. To investigate whether neurocan might be involved in reorganization of local neural network in NTS, we examined expression of neurocan mRNA and protein during postnatal development. In situ hybridization for neurocan mRNA showed that it expressed in the caudal NTS and the intensity of expression decreased between P2 and P10. Quantification of anti-neurocan immunoreactivity showed that the amount of expression of neurocan protein decreased significantly between P6 and P10. The timing of neurocan expression decrease appears to be consistent with that of beginning of local neural network reorganization in NTS. Thus down-regulation of neurocan might provide permissive environments for neural plasticity and facilitate reorganization of local neural network in NTS during postnatal development.

## P3-d38 Protocadherin-alpha family in the olivo-cerebellar system; their isoform expression and loss of function analysis

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Protocadherin alpha (Pcdha) family, one of the clustered Protocadherins, expressed in the nervous system. In particular, Cerebellar Purkinje cells highly expressed the Pcdha transcripts and have their differential and allelic expression patterns at single cell level. In present study we focus to role of the Pcdha family in the olivo-cerebellar nervous system . Using in situ hybridization analysis for each Pcdha isoforms, we found that the Pcdha isoforms have specially and temporally diagnostic expression pattern in the olivo-cerebellar system. Next we analyzed abnormalities of neuronal networks in the olivo-cerebellar system of the Pcdha gene-targeted mice which deleted the common conserved exons in the Pcdha gene cluster. We found a few unusual synapse formation in the climbing fiber of the Pcdha mutants. These results indicated that the diverse Pcdha family involves in acquiring the neuronal identity and in building the neuronal circuit formation in the olivo-cerebellar system.

## P3-d39 Expression of gicerin in glial cells of the rat sensory nervous pathway

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Gicerin is a cell adhesion molecule belonging to the immunoglobulin superfamily isolated from a chicken. In the chick nervous system, gicerin appears transiently in fetal life and promotes neurite extension and migration of embryonic neurons. In adult rat nervous system, gicerin expression is in the hippocampal cells, Purkinje cells, and spinal cord. We reported that gicerin expression is observed in sensory nervous pathway, espesially spinal cord dorsal horn, column and dorsal roots, which the sensory pathway of pressure and deep sensation pass through. But the cell type of gicerin expression is unknown. In this study, we visualize gicerin by immunological electric microscopy to define the cell type expressing gicerin in spinal cord. Gicerin expressed in the membrane of the oligodendrocytes constituting myelin seath and a part of the process of astrocytes as well as neurons.

## P3-d40 AMPA protects cultured neurons against glutamate cytotoxicity via activation of phosphatidylinositol-3 kinase /Akt and inactivation of GSK3 $\beta$ and caspase-3

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In this study, we revealed that the mechanism of neuroprotective effect of AMPA against glutamate-induced cytotoxicity in cultured rat cortical neurons. Pretreatment with AMPA (10 μM, 24hrs) decreased glutamate (100 μM, 24 hrs)-induced caspase-3 activation. Simultaneous administration of LY294002 (10 μM), a blocker of phosphatidylinositol 3-kinase (PI3K), attenuated neuroprotective effect of AMPA. Phosphorylation at Ser473 residue of Akt was enhanced by the exposure to AMPA (10 μM, 10 min). Phosphorylated glycogen synthase kinase 3 $\beta$ (GSK3 $\beta$) at Ser9, an inactivated state, was also augmented by the exposure to AMPA (10 μM, 10 min). From these data, we concluded that AMPA protects neurons through the activation of PI3K/Akt pathway and, as a consequence, the inactivation of GSK3 $\beta$ and caspase-3. These results suggest that AMPA protects neurons via activation of survival signals and, as a result, inhibits signals which relate to neuronal death.

## P3-d41 Effects of tributyltin on rat neural stem cells

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We recently demonstrated that exposure of neonatal rats to environmental chemicals caused the degeneration of mesencephalic dopaminergic neurons, resulting in hyperactivity. Our concerns were then moved to examine whether these chemicals would affect the neural stem cells during rat development. To this end, we tried to isolate neural stem cells from the mesencephalon of E16 rat. The tissue was digested and cultured in serum-free media containing bFGF and EGF. A few weeks later, proliferative spheres were formed. The sphere was identified to be nestin positive, indicating that the mesencephalon contained neural stem cells. The neurosphere was seeded in poly-L-ornithine/ laminin-coated dishes and cultured 4 days, followed by a subject to examine the neurotoxicity of tributyltin (TBT). It was found that a wide range of concentration of TBT induced TUNEL-positive cells, suggesting that the chemical caused neural cell apoptosis. Thus, the neurosphere in vitro system would facilitate the evaluation of developmental neurotoxicity of environmental chemicals.

## P3-d42 Prothymosin-alpha1 switches the neuronal necrosis to neurotrophin-reversible apoptosis

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A stroke delivers a double whammy. First, cells near the clot begin to perish from necrosis, which is triggered by ATP scarcity. Later, more distant cells start dying through apoptosis. We initially identified, prothymosin-alpha1 (ProT $\alpha$), which inhibits the neuronal necrosis in cultures that lack serum or have been oxygen deprived. However, it also boosts their apoptotic death rates. ProT $\alpha$ activated caspase-3 and caspase-9, but not for caspase-8 or caspase-12 in in vitro ischemia model. Moreover, ProT $\alpha$ -treatment up-regulates pro-apoptotic $Bcl_2$-family proteins (Bax and Bim) and down-regulates anti-apoptotic protein (Bcl-xL). Adding growth factors (BDNF, NGF, bFGF or IL-6) that inhibit apoptosis completely protects neurons. Knockdown of PKC $\beta_I$ and $\beta_{II}$ expression prevented ProT $\alpha$ -induced Bax expression. Taken together, these results suggest that ProT $\alpha$ causes apoptosis through Bax-cytpchrome c release-caspase-9/-3 activation pathway. Co-treatment of ProT $\alpha$ with neurotrophins might spare neurons after a stroke.

KAKENHI 13470490, 15390028, 17025031

## P3-d43 Prothymosin-alpha1 prevents neuronal necrosis by reversing the decreased membrane localization of GLUT

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A stroke delivers necrosis near the clot (ischemic core), which is triggered by ATP scarcity. We initially identified a nuclear protein, prothymosin-alpha1 (ProT $\alpha$), as a key protein inhibiting necrosis by subjecting conditioned media from serum-free cultures of cortical neurons. We found that the cellular ATP levels of cortical neurons rapidly decreased, possibly through an inhibition of membrane translocation of glucose transporters (GLUT1/4) under the serum free condition. ProT $\alpha$ inhibited rapid loss of cellular ATP levels by reversing the decreased membrane localization of GLUT1/4 through activating $G\alpha_{i/o}$-PLC-PKC $\beta$ II pathway. Taken together, these results suggest that ProT $\alpha$ is an extracellular signal protein that prevents neuronal necrosis by reversing the decreased membrane localization of GLUT.

KAKENHI 13470490, 15390028, 17025031

## P3-d44 Staurosporine-induced apoptosis in NG108-15 cells.

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Staurosporine (STS), a bacterial alkaloid, is a broad-spectrum protein kinase inhibitor, and is frequently used as an inducer of apoptosis in many cell types including neurons. Since the intracellular cascades that can be modulated by STS are not completely identified in NG108-15 cells, we examined the effects of STS on proteins that are related to the mitocnondrial apoptotic pathway. When NG108-15 cells were incubated with 0.2 μM STS, cell death became evident after treatment for 18 h. The pro-apoptotic protein Bax had shifted from the cytosol to the mitocdhondrial fraction, which was accompanied by a release of cytochrome c from the mitocnondria into the cytosol fraction.

## P3-d45 Neurotrophic effect of genipin: Neuroprotective action on tunicamycin-induced cytotoxicity in Neuro2a cells

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We have demonstrated previously that genipin, a natural iridoid compound, shows not only induction of neuronal differentiation in Neuro2a cells and PC12 cells, but also protective effect on 6-hydoroxydopamine-induced cell death in Neuro2a cells. To investigate whether genipin exerts protective effect on endoplasmic reticulum (ER) stress-induced cell death in Neuro2a cells, we demonstrated effect of genipin against tunicamycin (TM), the specific ER-stress-inducer. Treatment with TM for 24 h significantly decreased MTT reduction and increased LDH exclusion in a dose dependent manner, and markedly increased the protein level of GRP78, PERK and IRE1 $\alpha$ using western blotting. Genipin significantly prevented cell death in a dose dependent manner and suppressed the expression of GRP78 protein in the Neuro2a cells. We are now in progress to do western blotting and RT-PCR for analysis of PERK and IRE1 $\alpha$ expression. This work was supported by MEXT. FRONTIER (2005-2009).

## P3-d46 Possible involvement of RNA degradation in glutamate-induced neuronal death

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Recent reports have shown the possibility that an activation of ribonuclease (RNase) is involved in cell death. In this study, we evaluated whether RNase is involved in neuronal death induced by glutamate and trimethyltin (TMT) in cultured cortical neurons. Cells were exposed to either glutamate for 15 min or TMT for 2-24 h. The glutamate and TMT exposure led to a significant reduction in the cell viability 12 h post the exposure. An increase was seen in the level of RNase-L mRNA at least 2 h after glutamate exposure, with being prevented by NMDA receptor antagonist MK-801. However, TMT was ineffective in changing the expression of RNase-L. We next determined the degradation of total RNA after glutamate exposure. Marked RNA degradation was determined at least 4 h post exposure, with being prevented by MK-801 and NR2B antagonist ifenprodil. These results suggest that RNA degradation induced by the activation of NMDA receptor is at least in part involved in Glutamate-induced neuronal death in the cortical neurons.

## P3-d47 Rap1 is involved in the signal transduction of myelin-associated glycoprotein

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Myelin-associated glycoprotein (MAG) is a well-characterized axon growth inhibitor in the adult vertebrate nervous system. Several signals that play roles in inhibiting axon growth have been identified. Here, we report that soluble MAG induces activation of Rap1 in postnatal cerebellar granule neurons (CGNs) and dorsal root ganglion (DRG) neurons. The p75 receptor associates with activated Rap1 and is internalized in response to MAG. After MAG is applied to the distal axons of the sciatic nerves, the activated Rap1, internalized p75, and MAG are retrogradely trafficked via axons to the cell bodies of the DRG neurons. Rap1 activity is required for survival of the DRG neurons as well as CGNs when treated with MAG. The transport of the signaling complex containing the p75 receptor and Rap1 may play a role in the effect of MAG.

## P3-d48 TRPV1 mediates vanilloids and low pH-induced neurotoxicity in rat cortical cultures

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Transient receptor potential vanilloid 1 (TRPV1) is a non-selective cation channel, sensitive to several vanilloids including capsaicin, protons and heat. In this study, we investigated the roles of TRPV1 in pathophysiological processes using cultured rat cortical neurons. Capsaicin (10 μM) induced severe neuronal death with apoptotic features, which is completely inhibited by capsazepine and nifedipine, even though the addition of blockers was delayed for 2-4 h. Vanilloids including capsaicin and *N*-arachidonoyl dopamine and $G_q$-coupled receptor agonists, such as histamine and bradykinin could robustly induce TRPV1-mediated neurotoxicity under slightly low pH conditions (pH 6.5). Pharmacological interventions of arachidonic acid metabolism by lipoxygenase pathway or $H_1$ receptor-PKC pathway resulted in full inhibition of neuronal death induced by histamine at pH 6.5. We conclude that brain TRPV1 channels could be regarded as an important target in pathogenesis of neurodegenerative disorders, especially mild acidosis.

KAKENHI (18390166,18890087)

## P3-e01 Neuronal glutamate transporter EAAT3 functions as cysteine transporter in neurons

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Glutathione, one of the most important anitoxidant, plays an essential role in cellular antioxidative defense. The biosynthesis of glutathione is late-limitted by L-cysteine uptake. In the pure neuronal cultures neurons expressed EAAT3 and not EAAT1 or EAAT2. Pure astrocyte cultures expressed EAAT1 and EAAT2 and not EAAT3. In mixed cell cultures, glutamate transporter inhibitors inhibited not only glutamate uptake but also L-cysteine uptake, though these inhibitors did not influence L-cysteine uptake in the pure astrocyte cultures. Increased level of extracellular cysteine enhanced the L-cysteine uptake and intracellular glutathione level, and inhibitors of glutamate transporters inhibited this effect of extracellular cysteine. Antisense oligonucleotide for EAAT3 decreased cysteine uptake and glutatione level in the neurons, and increased neuronal vulnerability against oxidative stress. These facts indicate that neuronal glutamate transporter EAAT3 plays an important role in neuronal cysteine uptake and glutathione synthesis.

HIGHT TECH RESEARCH FOUNDATION

## P3-e02 A new type of ER stress transducer, BBF2H7 expresses in injured neurons

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Endoplasmic reticulum (ER) stress transducers are well known to transduce signals from the ER to the cytoplasm and nucleus when unfolded proteins accumulate in the ER. Recently, we identified OASIS as a novel ER stress transducer expressed in astrocytes. More recently, we found that BBF2H7, structurally homologous to OASIS, is markedly induced at the translational level during ER stress. In a mouse model of focal brain ischemia, BBF2H7 protein is prominently induced in neurons in the peri-infarction region. Furthermore, in a neuroblastoma cell line, siRNA knockdown of BBF2H7 promotes ER stress-induced cell death. Taken together, our results suggest that BBF2H7 is a novel ER stress transducer and could play important roles in preventing accumulation of unfolded proteins in damaged neurons.

KAKENHI(17200026)

## P3-e03 Intracellular TAGE (toxic AGEs) trigger neuronal degeneration

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To examine the effect of intracellularly generated glyceraldehyde-derived advanced glycation end-products (AGE-2), one of the main components of TAGE, on neuronal cells, SH-SY5Y neuroblastoma cells were exposed to various concentrations of glyceraldehyde. Immunofluorescence microscopy and immunoblotting analysis indicated that the intracellular AGE-2 were increased by adding glyceraldehyde in a dose dependent manner. Reduced proliferation and apoptosis of the neuroblastoma cells were observed preceded by the intracellular formation and accumulation of AGE-2. Furthermore, the expression of glyceraldehyde-3-phosphate dehydogenase (GAPDH) was significantly increased and the distribution of the enzyme was changed from the cytosol to nucleus. These results suggest that intracellular production of AGE-2 can trigger apoptosis through GAPDH overexpression and apoptotic signaling pathway, implicating the onset of Alzheimer's disease.

## P3-e04 Carnosic acid and carnosol as nuroprotective electrophilic compounds

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In a previous study we found that carnosic acid (CA) protected cortical neurons by activating the Keap1/Nrf2 pathway, which activation was initiated by S-alkylation of the critical cystein thiol of the Keap1 protein by the "electrophilic" *quinine*-type of CA.In the present study, we used HT22 cells, a neuronal cell line, to test CA derivatives that might be more suitable for *in vivo* use, as an electrophile like CA might react with other molecules prior to reaching its intended target.CA and carnosol protected the HT22 cells against oxidative glutamate toxicity.CA activated the transcriptional anti-oxidant-responsive element of phase-2 genes including hemeoxygenase-1, NADPH-dependent quinine oxidoreductase,and γ-glutamyl cysteine ligase,all of which provide neuroprotection by regulating cellular redox.

JSPS gant (2004-2006) JST grant(2007)

## P3-e05 A new aspect of carnosic acid as a neuroprotective electrophilic compound: activation of the Keap1/Nrf2 pathway

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Electrophilic compounds are a newly-recognized class of neuprotective compounds through S-alkylation of specific cysteines on targeted proteins. Catechol ring-containing electrophilic compounds have attracted attention, since they become an electrophilic quinone by autooxidation. Here, we focused on the neuroprotective effects of carnosic acid (CA), a catechol-type electrophilic compound from Rosmarinus officinalis. We discovered that CA activated the Keap1/Nrf2 pathway by binding to the cysteines of Keap1 and activating specific transcriptional elements, thus protecting neurons from oxidative stress and excitotoxicity. Further, CA was translocated into the brain and regulated the redox (increasing the levels of glutathione) capacity in vivo, suggesting that CA could be a new type of neuroprotective electrophilic compound.

## P3-e06 NADPH oxidase is not directly involved in neuronal death induced by hypoglycemia-glucose reperfusion

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The aim of this study was to clarify whether activation of NADPH oxidase was involved in neuronal death induced by glucose deprivation treatment followed by glucose reperfusion (GD/R) of primary cultured rat cortical neurons. The GD/R treatment decreased the neuron viability, which was accompanied with the decreased $NAD^+$ content and accumulation of poly(ADP-ribose) (PAR) and nitrotyrosine, and these alteration was reversed on coadministration of N-acetyl-L-cysteine and 3-aminobenzamide. The neurons expressed functional NADPH oxidase, which was activated by the GD/R treatment, but it did not generate detectable ROS and its pharmacological inhibition decreased the GD/R-induced decrease in neuron viability. A zinc chelator, CaEDTA, abolished the GD/R-induced ROS generation and PAR accumulation, and improved the neuron viability. These findings suggested that NADPH oxidase activation is not directly involved in GD/R-induced neuronal death, and furthermore demonstrated that chelatable zinc plays a central role in GD/R-induced neuronal events.

## P3-e07 Analysis of neuronal differentiation of GFAP-expressing neural precursors in the adult mice hippocampus

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In the adult hippocampal neurogenesis, primary neural precursors that express GFAP proliferate and finally differentiate into neurons. Here we studied how the GFAP-expressing neural precursors differentiate into neurons in transgenic mice with mouse GFAP promoter-controlled enhanced green fluorescent protein (mGFAP-EGFP Tg mice). To label proliferating neural precursors, mice were injected with BrdU. Thirty minutes after BrdU injection, only 36% of EGFP+/BrdU+ cells express GFAP+ and 39 % was positive for a neuronal marker, Hu. The number of Hu+/EGFP+/BrdU+ cells was increased during 3 days after BrdU injection, but was reduced to zero level 2 weeks later, suggesting that EGFP remains in the proliferating neuronal precursor cells for a short time after GFAP disappears. These results indicate that mGFAP-EGFP Tg mice can be used to examine the daughter cells directly derived from GFAP+ primary neural precursors in the adult hippocampal neurogenesis.

## P3-e08 Injury-related molecular profiling of the subgranular zone in young and aged brain, relative to neurogenesis

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The hippocampal dentate gyrus contains neural progenitors that continue to differentiate into glias and neurons. Active maintenance declines with aging. The environment modulates neurogenesis with the critical factors. The mouse SGZ was isolated by laser capture microdissection, applied to microarray analysis. The molecular profile differed as a function of age with 398 genes. When the SGZ was examined following injury induced by the hippocampal toxicant, TMT, levels for 1083 genes were altered. Differential responses with injury occurred as a function of age. Pathway mapping in the neurogenic environment included the IL-1 $\alpha$ pathway in immature brain and the IL-6 pathway in aged. The level of neurogenesis suggests a greater response in young compared to the response in aged. Characterization in the neurogenic environment will identify novel signaling factors to promote repair in the adult brain.

Intramural Research Program, NIEHS/NIH

## P3-e09 Rho-associated coiled-coil kinase (ROCK) regulates cell cycle of SH-SY5Y cell through β-catenin/TCF pathway

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Up-regulation of adult neurogenesis including an acceleration of cell cycle might be beneficial to the treatment of psychiatric disease. One of the cascade has been shown that both glycogen synthase kinase-3 β (GSK-3 β) and histone deacetylase (HDAC) regulate β-catenin/TCF pathway, and theactivation of it increases Cyclin D1. Here we investigated the role of ROCK, originally identified as an effector of Rho, on cell cycle. Using SH-SY5Y cell derived from human neuroblastoma, Y72632, the specific inhibitor of ROCK, decreases Cyclin D1 and inhibits the proliferation of cells. The decrease are recovered by both Li, inhibitor of GSK-3 β, and VPA, inhibitor of HDAC. Moreover, the recovery was inhibited by Quecertin, which decreases β-catenin and TCF in cell nucleus. Taken together, these observations may suggest that ROCK regulates cell cycle of SH-SY5Y cell through β-catenin/TCF pathway.

## P3-e10 Social isolation rearing impairs neurogenesis and behavioral development

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Experiences during a critical period of the brain development may influence the pathogenesis of developmental disorders. Thus, isolation rearing immediately after weaning is thought to be useful for studying the pathogenesis of such psychiatric diseases. In the present study, we examined the effect of social isolation on neurogenesis in the hippocampus (HIP) of juvenile mice by BrdU-staining, relating to aggressive behaviors and spatial memory impairment. Isolation for 4 weeks decreased the neurogenesis in the HIP and caused aggressive behaviors and spatial memory impairment, compared with group-housed control. Chronic treatment of fluoxetine, a selective serotonin reuptake inhibitor, reversed the reduction in BrdU-positive cell numbers in the HIP and alleviated memory impairment as well as aggressive behaviors induced by the isolation rearing. These findings suggest that communication with others in juvenile is important in neurogenesis in the HIP, which is associated with the development of psychiatric function including memory and emotion.

## P3-e11 Intracranial self-stimulation enhances neurogenesis in the rodent brain

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Van Praag et al.(1999) reported that a wheel running promoted neurogenesis in the mice hippocampus, suggesting that activated brain reward system by exercise might benefit to the adult cytogenesis. The present study was conducted to examine whether activation of brain reward system by intracranial self-stimulation (ICSS) could also enhance the cytogenesis in adult brain. Immediately after ICSS, 5-bromodeoxyuridine (BrdU) was injected intraperitoneally (rat, 150 mg/kg; mice, 50 mg/kg), and the number of BrdU-posi cells were counted at 1 day. 1 week, and 4 weeks after the injection. We found that ICSS group showed a significantly higher number of BrdU-posi cells in hippocampus than control at all time points. At 4 weeks,surviving cells of ICSS animals were found to be co-localized with a mature neuron marker NeuN. These results suggest that activated reward system by ICSS enhances neurogenesis in the rodent brain.

## P3-e12 Increased incorporation of ablated subtype of newborn granule cells in the mouse olfactory bulb

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In the olfactory bulb (OB), granule cells (GCs) are incorporated into preexisting circuitry throughout life. GCs are differentiated into distinct functional subtypes. However, little is known about how the incorporation of subtypes of newborn GCs is regulated. We hypothesized that local environment of the OB might influence the incorporation of subtypes of newborn GCs. To address this point, we selectively ablated mGluR2-expressing subtype of GCs in a restricted area of the OB with immunotoxin, and found that the incorporation of mGluR2-expressing newborn GCs was significantly increased in the immunotoxin-damaged area. The result suggests the compensatory incorporation of ablated subtype of newly-generated GCs in the neuronal circuit of the adult OB.

## P3-e13 Immunohistochemical analysis of nuclear lamins in the glial cells of adult rat brain

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Lamins are intermediate-filament proteins that form a nuclear lamina, a filamentous meshwork underlying the nuclear envelope. Three lamin subtypes, A/C, B1 and B2, predominate in mammalian somatic cells. We previously demonstrated that the neuronal progenitor cells, which are resided in the two neurogenic regions of adult brain and immunoreactive for PSA-NCAM, are lamin A/C-negative, but highly positive for lamin B1 and weakly positive for lamin B2 compared with mature neurons.To clarify the relationship between nuclear lamina composition and cell differentiation process, we performed immunohistochemical studies on three lamin subtypes in the glial cells of adult rat brain using confocal microscopy.Lamin B2 was weakly positive in astrocytes, oligodendrocytes and microglia compared with neurons. This data indicates that the composition of lamin subtypes is different in glial cells and neurons. We also found that lamin A/C-negative cells exist in the cerebral cortex, and these cells were immunoreactive for nestin, which is a marker for immature cells.

## P3-e14 Long-term effects of voluntary running on neurogenesis of adult mice

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It is shown that exercise brings about a positive regulation of adult hippocampal neurogenesis. However the majority of neurogenesis research on voluntary running is short-term (i.e. a followup trial is during 7 to 30 days), furthermore, it is reported that extended voluntary running inhibits exercise-induced neurogenesis. To clarify whether long-term voluntary running is beneficial to adult hippocampal neurogenesis, we allowed C57BL/6 mice to access to running wheel during 6 months after weaning. Although there is no difference in body weight between experimental groups, we found that both the rate of cell proliferation and survival of the hippocampal progenitor cells in exercised group was significantly increased up to 1.6 and 2.5-fold respectively in comparison to that of control group. Moreover the expression of brain-derived neurotrophic factor in the hippocampus was increased in exercised group. These results suggest that the modest exercise is beneficial to production of new neurons throughout a life.

## P3-e15 Direct evidence for neuronal differentiation of astrocyte-like progenitors in the postnatal hippocampus

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New neurons continue to be generated from progenitor cells in the postnatal hippocampus. The primary neurogenic progenitors have been characterized as astrocyte-like cells. To directly observe the neuronal differentiation of astrocyte-like progenitors and understand how neuronal differentiation occurs in postnatal dentate gyrus, we observed progenitor cell divisions and traced their daughter cell fates under the time-lapse imaging of mGFAP-EGFP transgenic mouse. First we characterized EGFP+ dividing cells at P5. Most of the EGFP+ dividing cells were GFAP+, thus defined as astrocyte-like progenitors. Next we followed these EGFP+ daughter cell fates for up to 30 hr using time-lapse imaging system. Approximately half of these EGFP+ daughter cells became expressing a neuronal marker Hu at the end of the time-lapse imaging. The neuronal differentiation process mostly occured in a symmetric manner. This study is the first direct observation and elucidation of neuronal differentiation modes of the astrocyte-like progenitors.

## P3-e16 Cognitive impairments in diabetic rats in relation with IGF-1 and hippocampal neurogenesis

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Recently,it has been pointed out diabetic patients have impaired learning/memory,and two-fold increased risk of dementia.Such cognitive dysfunction is more associated with $HbA_{1c}$ and duration of diabetes mellitus rather than MRI imaging such as periventricular hyperintensities,deep white matter lesions,medial temporal lobe atrophy,cerebral atrophy and lacunar infarction. In this study,working memory studies with use of 8 radial maze were performed on diabetic rats.After sacrificing,IGF-1 mRNA expression in the hippocampus was examined by real time PCR assay.In addition, the hippocampal formation was processed for immunohistochemical staining of PSA-NCAM,Ki67,NeuN and GFAP.Effects of estrogen and isoflavone on intracerebral IGF-1 expression in diabetic rats were also examined.

Conclusions were as follows.1)In diabetic rats, working memory was impaired.2)In the hippocampus of diabetic rats, there were increased expressions of IGF-1 mRNA which were inhibited by chronic estradiol administration.3)In the hippocampus of diabetic rats,neurogenesis was inactivated when compared to non-diabetic rats.

## P3-e17 The function of Ephrin-Eph signaling in postnatal neurogenesis

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Neurogenesis occurs in the subgranular zone (SGZ) of the hippocampal dentate gyrus (DG) in mammalian adult brain, where astrocytes, blood vessels constitute a "neurogenic niche". Recently, Eph-Ephrin signaling is reported to act as one of the factors establishing a niche in the intestinal epithelium. In this study, we focused on EphrinA5 (EfnA5) function for postnatal neurogenesis. *EfnA5* mRNA was expressed in $GFAP^+$ cells, but not in neurons. BrdU labeling showed decreased proliferation in the SGZ of $EfnA5^{-/-}$ mice at 4 weeks. Immnohistochemical analysis showed that EfnA5 protein was localized at the endfeet of astrocytes surrounding capillaries. Contrary, EphA4 was expressed in capillary endothelial cells (EC). Moreover, the diameter of capillaries in the DG was decreased in $EfnA5^{-/-}$ mice. These data suggest a possibility that EfnA5-expressing astrocytes may regulate the diameter of capillaries through interacting with EphA4 receptor of ECs controlling the blood flow, thereby contributing to establish the neurogenic niche around the capillary in the DG.

## P3-e18 Morphological analysis of axons of newly generated granule cells in adult mouse hippocampus

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It is known that adult neurogenesis occurs in the subgranular zone of the dentate gyrus (DG) in the mammalian hippocampus. In the present study, we labeled the nestin-expressing cells in the DG of the Nestin-Cre transgenic mouse with EGFP by adenovirus-mediated Cre-loxP system. We have revealed that nestin-positive cells differentiate to granule cells and project their mossy fibers to CA3. In this region, EGFP-labeled mossy fibers formed large boutons and some of them expressed vesicular glutamate transporter 1, indicating that these boutons are glutamatergic. To compare the maturation of the newly generated granule cells with that of granule cells in the developmental stage, we analyzed the morphology of the newborn large boutons with electron microscopy and confocal immunofluorescence microscopy.

## P3-e19 Cell proliferation in the rat hippocampal formation under the conditions of dysfunctional mastication

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Recently, the hippocampal neurogenesis has been shown to be changed in various environment conditions, e.g. exposure to enriched environments or stress. We examined in Wistar rats whether cell proliferation in the hippocampal formation varies by either chewing behavior with differential food texture or loss of the molar teeth. The experiments were done in the three groups: 1) molarless group whose molar teeth were extracted, 2) soft-diet group which had fed with a powder diet, and 3) control group without any treatments. The animals were applied with a thymidine analog, 5-bromo-2'deoxuridine(BrdU). When BrdU positive cells were counted using BrdU immunohistochemistry, significant decreases in the number of BrdU positive cells in the dentate gyrus was seen in the molarless and soft diet groups. The data suggest the link between abnormalities in mastication and hippocampal cell proliferation.

## P3-e20 Cessation of neuronal migration in adult rodent olfactory bulb is paralleled with the disappearance of drebrin E from the cell body

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We have previously shown that migrating neuroblasts in the rostral migratory stream of the adult rat forebrain express drebrin E but not drebrin A (E+A-). To clarify how it participates in neuronal migration in the adult brain, the image subtraction method using anti-pan-drebrin and anti-drebrin-A antibodies was applied to the olfactory bulb. The migrating neuroblasts at the core region showed E+A-signals in their cell bodies, but postmigratory young neurons at the outer layers showed only dot-like E+A- signals. On the other hand, doublecortin, a microtubule-associated protein, was expressed in the cell body through migratory and postmigratory stages. Our results highlight the unique characteristics of actin cytoskeleton within the migrating neuroblasts and open the way to identify the migrating neuroblasts in the adult brain.

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## P3-e21 ΔFosB promotes neurogenesis in the dentate gyrus of mouse hippocampus after brain damage caused by excitotoxicity

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We have shown that expression of *fosB* gene, which produces FosB and ΔFosB, the latter lacking the C-terminal transactivating domain (101 aa) of FosB, by alternative splicing of its transcript, is induced in hippocampus after systemic administration of kainate, an agonist for subsets of glutamate receptors. In order to clarify the significance of increased expression of FosB and ΔFosB in the hippocampus under the excitotoxicity, we compared the extent of excitotoxicity-induced neurogenesis in the dentate gyrus among wild-type, *fosB*-null and *fosB*$^{d/d}$ mice which express only ΔFosB, using unbiased stereology. The number of BrdU-positive cells in the dentate gyrus was significantly decreased in *fosB*-null mice, but slightly increased in *fosB*$^{d/d}$ mice, in comparison to wild-type mice after kainate administration. These results suggest that ΔFosB may promote neurogenesis in the dentate gyrus of mouse hippocampus after brain damage caused by excitotoxicity.

## P3-e22 A novel vanadyl compound enhances neurogenesis via Akt and ERK activation in the hippocampal subgranular zone after mouse focal cerebral ischemia

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Neurogenesis is well documented in the subgranular zone (SGZ) of hippocampus and subventricular zone (SVZ). However, the mechanism underlying neurogenesis in the adult brain remains to be unclear. We here assessed whether Akt and extracellular signal-regulated kinase (ERK) pathways mediates vanadyl compound-induced neurogenesis in ischemic mouse brain. Intraperitoneal administration of a vanadyl compound, which is a potent inhibitor for protein tyrosine phosphatases, stimulated neurogenesis in the adult SGZ after brain ischemia. The compound led to activation of Akt and ERK in the new born cells after brain ischemia. Furthermore, behavioral studies also showed that the vanadyl compound-induced neurogenesis accounts for improvement of memory deficits following brain ischemia. These results suggested that brain ischemia-induced neurogenesis is mediated by Akt and ERK activities and is essential for improvement of memory deficits following ischemia.

## P3-e23 Mitotic hippocampal cells expressing β III-tubulin in secondary cultures from organotypic slices

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Neurons are believed not to be proliferative or mitotic. However we observed mitotic hippocampal cells expressing a neuron specific protein, β III-tubulin, in seconday cultures produced by re-cultivation of cells dissociated from neonatal rat hippocampal slices cultured for more than 2 weeks. Immunocytochemical analysis revealed that β III-tubulin exprssing cells (BT cells) increased during 2 weeks in secondary cultures. BT cells incorporated bromodeoxyuridine and expressed mitotic morphology. Furthermore, binucleate BT cells were observed suggesting impaired cytokinesis after mitosis. These phenomena were observed also in cells secondarily cultured in presence of brain-derived neurotrophic factor, basic fibroblast growth factor or 17 β -estoradiol. Mitosis was suggested to be a neuronal reaction to injury of matured nervous tissue.

## P3-e24 Proliferation of neural stem/progenitor cells in adult rats after facial nerve avulsion

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We found proliferation of nestin-immunoreactive (IR) neural stem/progenitor cells (NSPCs) associated with motoneuron death in lesioned facial nuclei after facial nerve avulsion, but not axotomy, in adult rats. When intact facial nuclei were dissociated and cultured in the presence of FGF2 and EGF, neurospheres formed and NSPCs could be expanded and passaged over 6 months. In vitro proliferation of NSPCs derived from avulsed facial nuclei was much more vigorous than that from intact nuclei. These NSPCs differentiated to neurons, astrocytes and oligodendrocytes in the presence of retinoic acid. In vivo local treatment of an adenoviral vector encoding FGF2 after avulsion significantly increased nestin-IR cells in lesioned facial nuclei. These results indicate that adult rat facial nerve avulsion model is a novel tool to investigate endogenous NSPCs in adult rat brain, and can be used to screen candidate therapeutics to promote in vitro and in vivo neurogenesis against neurodegenerative disorders.

KAKENHI (17590908)

## P3-e25 Galectin-1 promotes neurogenesis in the dentate gyrus of mouse hippocampus after brain damage caused by excitotoxicity

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Galectin-1, a major beta-galactoside-binding lectins, has been implicated in regulation of various biological functions. In the present study, we examined the expression level of galectin-1 in mouse hippocampus after excitotoxic damage. Expression of galectin-1 was remarkably increased in activated astrocytes in CA3 subregion and dentate gyrus a week after kainate administration. In order to clarify the significance of an increased expression level of galectin-1 after excitotoxiciy, we compared the extent of neurogenesis between galectin-1-null and wild-type mice after kainate administration. In the galectin-1-null mice, number of BrdU positive cells in dentate gyrus decreased to 75% level seen in the wild type, indicating that galectin-1 secreted from astrocytes activated after excitotoxic damage, promotes neurogenesis in the hippocampus.

## P3-e26 Inhibition of Renshaw cells during spinal reflexes in the neonatal mouse spinal cord

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Renshaw cells (RCs) are known to provide recurrent inhibition to motoneurons in the mammalian spinal cord. However, their modulation by motor circuits remain unclear. We examined the synaptic inputs during spinal reflexes to RCs. We performed whole cell recordings from identified RCs in the lumbar ventral horn using isolated spinal cord preparations taken from GAD67-GFP (Δneo) mouse neonates (Postnatal day 0-4). Electrical stimulation (intensity 1.5-3 times of threshold) of dorsal roots evoked a short latency EPSP followed by a long lasting IPSP. Both PSPs persisted in the presence of mecamylamine, a nicotinic receptor antagonist, indicating that they are unlikely to have derived from motor axon collaterals. They were both blocked by CNQX and AP5 indicating that EPSPs are mediated by glutamate receptors and IPSPs are polysynaptically elicited. These results indicate that RC activity is strongly modulated by spinal reflex pathways. Moreover, 5-HT enhanced the EPSPs suggesting that descending pathways modulate synaptic inputs to RCs during spinal reflexes.

KAKENHI(18700388)

## P3-e27 Postural muscle tone synchronizes with automatic nervous activity in very low frequency domain

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In this present study we measured postural sway, muscle tone and heart rate variability simultaneously to elucidate the relation between spontaneously postural sway, muscle tone and automatic nervous activity in very-low-frequency (VLF: 0.0033-0.04Hz) domain of the heart rate variability. Ten healthy young subjects take part in this study. Subjects were asked to stand on a force-plate to keep a static upright stance in five minutes holding a force-transducer consistently by using his/her thumbs and indices. Center-of-Pressure (COP), heart-rate-interval and clip forces were recorded and analyzed. The results shown that postural sways are correlated to heart-rate-variability with a correlation coefficient of 0.47+/-0.12 ($p<0.01$), and also coherent with muscle tone significantly in frequency less than 0.03Hz.The results suggest a common oscillator be situated on the controller center which have an effect on balance-keeping and automatic-nervous-system control simultaneously.

17560220

## P3-e28 Presynaptic AMPA receptors on the corticostriatal terminals enhance the release probability of the synaptic vesicles

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Presynaptic AMPA receptors (preAMPARs) have recently been reported on corticostriatal terminals (CsTs). Constructing a simulation model of a CsT with preAMPAR and a medium-sized spiny neuron (MSN), we tried to reveal the role played by the preAMPARs in the cortico-basal-thalamic loop. High-frequency stimulation of the model CsT enhanced the release probability of synaptic vesicles, and depolarized the membrane potential of the model MSN supralinearly. Injecting a sequence of high- and low-frequency stimulations to the model CsTs, we observed membrane potential fluctuations closely similar to up- and down-state transition *in vivo*.

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## P3-e29 Patch-clamp analysis of membrane properties of bursting neurons in the young adult rat ventral horn in vitro

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Electrophysiological and morphological properties of ventral horn neurons were investigated using blind patch-clamp recording from spinal cord slices. In lamina IX of the spinal ventral horn, we classified recorded neurons into two types, bursting and silent types (BT and ST), based on their firing properties. BT neurons had rhythmic firing pattern at resting membrane potential. On the other hand, ST neurons did not have any rhythmic firing patterns. Under the voltage-clamp condition, any rhythmic activity of spontaneous EPSCs was not observed in both BT and ST neurons. The results suggest that rhythmic firing in BT neurons is mainly caused by intrinsic membrane properties such as persistent Na+ channel or voltage-dependent Ca2+ channel activities. Such BT neurons may contribute to the central pattern generation in the ventral horn.

KAKENHI

## P3-e30 Asymmetric adjustments of human target-reaching by target switch

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We examined adjustments of ongoing human target-reaching evoked by unexpected switch of targets. Right-handed subjects were required to reach to a lighted center LED (target, 40 cm ahead) by either arms, as rapidly and accurately as possible. The target sometimes switched to either a left or right LED (10 cm from the center) randomly, 25 or 100 ms after the movement initiation. 3D positions of an index finger and EOG were recorded. As reported, subjects could adjust movements at short latencies (>120 ms) after the target switch. But the adjustment was faster and more accurate, when the new target was in the horizontal adduction than abduction direction. In the former, the adjustment was better coordinated with the saccade (onset latency, >120 ms). The tendency was clearer in the dominant arm. The results suggest that normal humans tend to attend around the target asymmetrically, already during reaching. The coordination indicates that the adjustment of the arm movement could use the same drive as the regular saccade.

## P3-e31 Neural correlates of estimating graspability

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Cognition of one's own motor capacity in the visual space is essential to select an appropriate motor behavior. In the present study, we utilized functional magnetic resonance imaging to investigate the neuronal correlates of judging one's own motor capacity.

We measured brain activity of 17 right-handed participants while they judged whether visually presented objects are graspable (with their right hand) or not, without making any actual movement. As a control condition, they compared the size of the object with that of their fist. We found that the left dorsal premotor cortex (PMd) was specifically activated under the condition where the participants judged their own graspability, and its activity was correlated with the time required for the judgement.

The left PMd is involved in the neuronal computation of one's own motor capacity, that may be critical for planning an adaptive motor behavior.

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## P3-e32 GABA-containing γ-motor neurons

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GABA-containing fibers, identified by antibodies for GABA and GAD67, were found in the ventral roots of C5-T7 spinal segments. They were immunopositive for ChAT, a marker of cholinergic neuron, and were thinner than GABA-negative fibers. GABA-containing fibers were also found in the brachial plexus. Furthermore, GABA-containing terminals made contact with the capsular sleeve of the intrafusal muscle fibers of the upper limb, and were considered as terminals of γ-motor fibers. After injection of fluorogold (FG), a retrograde tracer, in the forelimb muscles, we processed spinal cord sections for a combined method of immunohistochemistry for FG, and in situ hybridization for GAD67 mRNA, and found that some retrogradely labeled medium-sized neurons in the ventral horn, presumably γ-motor neurons, express GAD67 mRNA. These observations indicate that some γ-motor neurons contain GABA.

KAKENHI 18700339

## P3-e33 Fiber connections of the intermediate mesopallium in the pigeon

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The intermediate part of the caudal nidopallium (NCI) in the pigeon has a strong, reciprocal fiber connection with the intermediate mesopallium and is suggested to be a subdivision in the nidopallium (Neurosci.Res.55 (suppl.1), S150, 2006).The present study was examined afferent and efferent fiber connections of the inermediate mesopallium of the pigeon by a tract-tracing method. CTB and BDA were injected approximately at levels of A 8. Following a CTB injection into the intermediate mesopallium, a large number of neurons was labeled in NCI and a small number of labeled neurons were found in the nucleus basalis magnocellularis (NBM), arcopallium, thalamic posterior dorsolateral nucleus (DLP), subrotundal nucleus, pretectal area, and locus coeruleus. Following a BDA injection into the intermediate mesopallium, a large number of labeled fibers extended in NCI. A small number of labeled fibers were seen in the arcopallium. Few labeled fibers were located in NBB. In summary, the intermediate mesopallium has a strong reciprocal connection with NCI but no connections with the primary sensory areas or limbic system in the telencephalon.

KAKENHI(18590165)

## P3-e34 Single cell tracing of striatofugal neurons in neonatal rat brain

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Mammalian striatum consist of two compartments, i.e., patch and matrix, which differ in neurochemical properties and afferent/efferent connections in adulthood as well as in development. In rat embryos, neurons of patches (also known as dopamine islands) are produced ~5 days earlier than those of matrix. In rat neonates, both glutamatergic and dopaminergic afferents are scarce in matrix and abundant in patches. Previous investigations with bulk injection of retrograde tracers into rat neonates indicated that the patch neurons, but not matrix neurons, project to the substantia nigra as early as at birth. In addition, an anterograde labeling study confirmed the striatofugal projection to the substantia nigra and external and internal globi pallidi in newborn rats. To examine how the striatofugal projection systems from patch and matrix are organized in neonates, we injected Sindbis virus as an anterograde tracer expressing membrane-targeted GFP into the striatum of newborn rats to reconstruct entire axonal arborization of single striatofugal neurons.

## P3-e35 A new choice reaction-time task in the rat using air-puff stimuli and lever-release responses

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We have developed a choice reaction time (RT) task in the rat to investigate the neural mechanisms of stimulus-response (SR) compatibility. In this task, the rat pressed two levers with its forepaws during the preparation period of each trial, and then quickly responded to an air-puff stimulus on its left or right forepaw by releasing the lever on the same side (compatible) or the opposite side (incompatible) of the stimulus. Twenty rats successfully learned the task in both the compatible and incompatible conditions. RTs were shorter and error rates were lower in the compatible condition than in the incompatible condition. A lesion in the forepaw sensorimotor cortex increased RTs in trials that required stimulus detection and/or response movement with the contralesional forepaw. However, the lesion did not disrupt re-learning of the reversed SR conditions. We conclude that this instrumental task for rats can be regarded as a model for human choice RT tasks and be used to investigate the neural basis of the SR compatibility.

## P3-e36 Functional brain areas involved in the use of chopsticks: a PET study

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Using PET, we examined the brain areas of tool-use by comparing the use of the chopsticks with that of the fingers in well-trained human subjects (N = 7). Chopstick prehension was associated with higher activities in the ventral premotor (PMv) area, superior parietal area, intraparietal lobe, middle temporal gyrus, primary motor-sensory areas, occipital area, and cerebellum (lateral and anterior hemispheres including the dentate nucleus) as compared to finger prehension. These cortical and subcortical areas are suggested to form a tool-use-associated neural network in humans. Activation of the left PMv and superior parietal cortex supported the hypothesis that the parieto-frontal circuit developed for manipulation control using a hand-held tool.

KAKENHI(18500450)

## P3-e37 Functional connectivity of sensorimotor oscillatory activities associated with electromyogram in the monkey

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To study the functional coupling between the cortical oscillatory activities and electromyogram (EMG), we examined the directed transfer function (DTF) between EMG and local field potential (LFP) oscillations at the monkey S1 and M1. Compared to the ordinary coherence, the DTF computation based on the multivariate model is suitable for analyzing the directional structure of the reciprocally interconnected system. We found that DTF between the LFP oscillations and EMG at 14 - 30 Hz is predominantly centrifugal flow and strongest at M1. As regard to the cortico-cortical coupling between M1 and S1, the DTF in the beta band was reciprocal. The information flow from S1 to M1 was larger than that from M1 to S1. These findings suggest the importance of LFP oscillations in M1 as well as S1 for generating the EMG oscillations.

KAKENHI(18019020,18020014,18500239)

## P3-e38 Effect of visuomotor adaptation for arm reaching on the manual following response induced by visual motion

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We investigated an effect of visuomotor adaptation for visually guided reaching on short latency manual following response (MFR) induced by a sudden visual motion applied during arm movement. Subjects practiced fast center-out reaching in six directions with visual feedback of hand position cursor rotated around the start position. One group of subjects was exposed to 60 deg cursor rotation throughout the training. After training, the reaching start direction was shifted almost 60 deg from the target direction, indicating that the motor planning was well adapted. However, the shift of MFR direction, measured after training, was extremely less than the cursor rotation angle. The other groups were exposed to the incremental cursor rotations up to 24 or 48 deg. Although the final cursor rotation angles were smaller than that in the first group, the shifts in the MFR directions were the same as or larger than that in the first group. These results suggest that the visuomotor adaptation for the MFR is partially independent of that in the motor planning.

## P3-e39 Gain modulation of manual following response associated with galvanic vestibular stimulation

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In order to make stable interactions with various environments, our motor system is required to provide quick processing for sensory inputs. Recently, we found that a sudden motion of the visual background during target reaching induces change of hand trajectory with an ultra short latency (manual following response: MFR). This is considered as a compensatory response for online control of hand trajectory when having a postural imbalance. However, this idea has yet to be directly verified. If the MFR plays an important role for maintaining reaching performance while less postural stability, we would expect an increase in the MFR gain against the visual motion in order to achieve the reaching task. To test this idea, we examined whether or not perception of unstable postural condition motivated by frequently applied galvanic vestibular stimulation (GVS) during experiment would cause a gain modulation of the MFR. In the results, we found evidence that a perception of instability can affect the MFR gain.

## P3-e40 Do the similar quick visuomotor controls of arm and eye share a gain modulation caused by the spatial dissociation of gaze and pointing?

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Background visual motion induces reflexive manual (MFR) and ocular (OFR) responses. We recently revealed that the MFR is modulated by the spatial relationship between reaching target and gaze point. To explore its mechanism, we here examined whether or not the OFR share this spatial dependent modulation. In the experiment, participants were asked to hit the NEAR (placed at same horizontal direction to the gaze point) or FAR (different direction) target by index finger while looking at the center of the screen placed in front of the face. In the FAR condition, participants rotated their body without change of the face-screen configuration in order to keep reaching kinematics. Upward or downward visual motion was given on the screen during arm movement to induce MFR and OFR. The results show that the OFR didn't change for the NEAR and FAR, whereas the MFR was greater for the NEAR than for the FAR. This suggests that the MFR gain was modulated after the common visual motion processing for MFR and OFR.

## P3-e41 Spatiotemporal tuning of short-latency manual and ocular following responses associated with sudden visual motion in monkeys

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A sudden drifting movement of a visual stimulus evokes short latency manual following response (MFR) in both humans and monkeys. It helps to adjust an arm movement during body movement accompanied by visual motion. The same visual motion also evokes ocular-following response (OFR), which helps to stabilize gaze. To clarify whether a common visual processing pathway provides inputs to both manual and oculo- motor systems, the manual and ocular responses were simultaneously recorded in arm reaching tasks in monkeys. The spatiotemporal tuning characteristics of the OFR were similar to those of the MFR: the amplitude of both MFR and OFR increased markedly with temporal frequency, generally reaching a peak at 10 - 20 Hz. This result suggests that the early visual processing pathway in monkey may be shared in generating the MFR and OFR.

## P3-e42 Localization of V-ATPase in the retina: evidence for proton feedback hypothesis

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In order to interpret the formation of receptive field surround in retinae, Hirasawa and Kaneko (2003) proposed a pH-mediated mechanism to substitute for GABA-mediated feedback hypothesis from horizontal cells (HCs) to cones. To verify the idea that the depolarized HCs release protons, we measured, by a fluorescence ratio imaging method, pH of the immediate external surface ($pH_o$) of isolated HCs of goldfish. Bafilomycin A1, a specific inhibitor of V-ATPase, suppressed depolarization-induced acidification of HC $pH_o$ , suggesting the existence of V-ATPase on HC membranes. Anti-V-ATPase immunoreactivity (IR) was found at the cone terminals and on the surface of GAD-positive external HCs in retinal slices. In ribbon synapses, anti-V-ATPase IR was found in lateral elements of dendrites (post-synapse) wrapped in anti-V-ATPase IR cones. These results suggest that $pH_o$ reduction at the narrow invaginating synaptic cleft would suppress $Ca^{2+}$ channels at the cone terminals, resulting in suppression of release of glutamatergic synaptic vesicles from cones to the second neurons.

18500313, 17657051

## P3-e43 A retinal circuit model accounting for functions of amacrine cells

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Recent studies have demonstrated the higher-level functions of visual processing, such as object motion sensitivity and spatio-temporal pattern adaptation, in vertebrate retinas. In those visual processing, diverse types of retinal third-order interneurons, amacrine cells are believed to play essential roles by integrating excitatory and inhibitory signals on their dendrites to shape the ganglion cell responses. Previously, a simple " linear-nonlinear" model was utilized to explain a specific function of the retina, and could capture the spiking behavior of retinal output, although each class of the retinal neurons were largely omitted in it. In the present study, to reveal the cellular mechanisms underlying the retinal functions, we developed a circuit model based on the response function for each class of the retinal neurons and the anatomical wirings between them. By this model, not only the spatio-temporal filtering properties of outer retina but also the higher-order nonlinear computations in the inner retinal circuitry involving amacrine cells could be realized.

KAKENHI 17700398

## P3-e44 Motion picture effects to eye movement and cerebral blood flow

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We presented that motion pictures representing rider's view of motocross bike elicited two types of horizontal eye movements; coherent or noncoherent to the motion vectors in the movie, while those representing passenger's view of roller coaster evoked similar eye movements in all subjects (29th JNS, 2006). We investigated the relationship between their eye movements and cerebral blood flow (CBF) while watching these vehicle type movies on ten subjects. Eye movements were measured by a binocular video oculography (Newopto), and head movements were measured by a magnetic motion sensor (Polhemus). CBF was simultaneously monitored with a near infrared spectroscopy (Shimadzu). Both the eye movements and the CBF variation had relevance to the motion components. It might depend on the differences in the strategies for the recognition of these movies.

Tsukada Medical Foundation, N S Promotion Foundation for Science of Perception

## P3-e45 Determining factors for endogenous saccadic eye movement during visual search in complex scene

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It is known that when we search for a target object in a complex scene, we tend to make saccades to the visual features that stand out from the background (exogenous saccades). However, it seems less likely that we find the target object solely based on exogenous saccades during visual search. To investigate additional factors (endogenous components) involved in the saccades, we asked human subjects to perform visual search tasks in which a target object was chosen from one of three object categories that were familiarized to the subject beforehand. We then calculated the category specific information content of each local feature, centered on the saccade end-points. We found that the end points of saccades tend to provide more information about the category of the target object than randomly selected features. Thus, a determining factor for saccades during visual search could be visual features specific to the target object category.

## P3-e46 Perceptional orientation-dependent interaction between color and luminance

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Isoluminant color modulation such as a red-green color grating perceptually fades by gaze fixation when presented on uniform background (Troxler fading) or superimposed on a orthogonal luminance grating (monocular rivalry). In this study, we found that a moving luminance grating enhances the perceptual fading of the color grating when superimposed orthogonally, and abolishes the fading when superimposed parallel. Perception of a static color grating was suppressed by superimposing a moving orthogonal luminance grating. When the color grating was moving, Troxler fading and monocular rivalry did not occur. The moving luminance grating, however, suppressed the perception of the moving color grating. When the orientation of the luminance grating was the same as that of the color grating, the perceptual suppression never occurred even when the luminance grating was smaller than the color grating. These results suggest that there are enhancing and suppressive perceptual interactions between color and luminance when their orientations are the same and orthogonal, respectively.

## P3-e47 Functional maps of visual responses with a multiple-electrode array in macaque inferotemporal cortex

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Previous studies with intrinsic signal imaging revealed that an object image is represented by a combination of columns each representing visual features. To address a question how object recognition relates to object representation with multiple columns, it is essential to investigate spatiotemporal patterns of activity maps with monkeys working on visual recognition tasks. Intrinsic signal imaging, however, is not appropriate for this purpose because the signals are indirect measures of neural activities and because of their low S/N ratio. In this study, we developed a densely arranged multiple electrode array that is available in behaving monkeys and have spatial resolution roughly comparable to intrinsic signal imaging. The array consisted of 16(4x4) or 49 (7x7) electrodes (spacing, 350 um) which were arranged to cover 1.05 x 1.05 mm or 2.1 x 2.1 mm area along the cortical surface. With this array, we successfully reconstructed a two-dimensional functional map elicited by object stimuli.

## P3-e48 A mathematical model of retinal rod photoreceptor network

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It is known that the network structure of the rod photoreceptor improve signal to noise ratio and signal transmission to the second order neuron. However, it is difficult to understand the function of the rod network due to the complexity of the subcellular, cellular and intercellular systems.
In this study, to analyze the underlying mechanism of rod photoresponses, we developed a mathematical model of the rod network based on anatomical and neurophysiological findings. We modeled single rod photoreceptor with five ionic currents ($I_{Ca}$, $I_{K,v}$, $I_{Cl(Ca)}$, $I_{K(Ca)}$, $I_l$), intracellular calcium system and phototransduction mechanism. Each rod compartment was connected by gap conductance according to the finding on rod electrical coupling (Zhang and Wu, 2005). In simulation, the responses of the model produced by small and large spots were in good agreement with the experimental results. The time course of the photoresponse produced in the rod model depended upon the area of illumination. Our results suggest that the ionic mechanisms of single rod itself can account for these photoresponses.

KAKENHI(17500195)

## P3-f01 Different roles of the parietal and lateral occipito-temporal cortex for 3D perception from motion

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We can perceive a 3D object when a special figure rotates in the frontal plane about the visual axis, which is termed the stereokinetic effect (SKE). To reveal the roles of visual areas along the dorsal pathway for SKE, we compared fMRI activity in the lateral occipito-temporal (LOT) region with that in the dorsal intraparietal (DIP) region, while subjects viewed a variety of SKE stimuli or performed a spatial attention task. DIP was activated only for the class of SKE stimuli that evoked a spatial attention shift, whereas LOT did not show such preference. This suggests that LOT plays a central role in constructing 3D structures from motion, while DIP is involved in spatial attention to constructed objects.

JSPS 17-2093

## P3-f02 A model of spatiotemporal processing in X retinal ganglion cell

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Several computational models of X retinal ganglion cell have been proposed to understand the behavior of the X ganglion cells. However, there are three major problems in the previous models: (1)the model developed as one dimensional structure; (2)the spike firing mechanism was not included; and (3)the contrast sensitivity was not satisfactory reproduced. We propose an improved model of the X retinal ganglion cell.
We extend the previous model (Gaudiano, P., 1994) by introducing two dimensional receptive field structure and stochastic spike generation mechanism. Our model consists of photoreceptors, bipolar cells, and X ganglion cells. The spike generation is described by Poisson spike generator.
In simulation, our model showed responses similar to the experimental data such as the null response to contrast-reversed gratings. The model also showed the spatial bandpass characteristics and reproduced the contrast sensitivity quantitatively.

KAKENHI(17500195)

## P3-f03 Receptive field structures of transcallosally connected neurons in the cat's visual cortex

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How are receptive fields organized for transcallosally connected visual neurons? Although previous studies showed that hand-plotted receptive field of interhemispheric synchronized neurons are typically overlapped and their orientation preferences are similar, structures of the receptive fields were not clarified. To investigate the spatial organizations of receptive-fields these neurons, a reverse correlation analysis is used. Simultaneous recordings were made in left and right hemispheres from the border areas between area 17 and 18 (transitional zone). Interhemispheric connectivity was examined by cross-correlation analysis. Contrary to previous studies, however, we found that some pairs of connected neurons have different orientation preference. In addition, subregions of the paired neurons show various response polarities, where the overlaps were observed not only for ON-ON or OFF-OFF, but also for ON-OFF combinations. These results suggest greater complexity in the pattern of callosal connectivity than that previously thought.

MEXT(18020017), JSPS(13308048), COE21

## P3-f04 Characteristics of surround modulation and stimulus shape for consistent determination of border ownership

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Border Ownership (BO) that indicates the side of figure along the border is an essence for figure/ground assignment. We propose a computational model for the BO determination based on the surround modulation (SM) reported in early visual areas. To examine the characteristics of the SM necessary for consistent BO determination among stimuli, we tested hundreds of the model cells, whose regions were given randomly, with a set of pseudo-random stimuli. Our analysis showed that the significant factor for the consistency is dominance of suppression that is a major physiological characteristic of SM. To analyze overall consistency of the model cells as a population, we compared the consistency of the model with the reaction time of human subjects, and found that a stimulus with the longer reaction time yields the lower consistency. The result provides phenomenological evidence for supporting SM in BO determination.

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## P3-f05 Voluntary activity of neurons projecting centrifugally to the retina

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In birds, neurons in the isthmo-optic (IO) nucleus project to the contralateral retina. The centrifugal pathway is thought to be involved in local and transient enhancement of retinal output. We recorded activities of the IO neurons in awake, behaving Japanese quails. The IO neurons showed brisk responses to light stimuli applied to their receptive fields. Besides the passive response, the IO neurons also showed voluntary activities without any visual stimulation. Voluntary activity was observed not only in the middle of eye-head fixation but also during about 200 ms before the onset of head saccades. The IO neurons showed a large phasic elevation of activity before the onset of head saccades toward their receptive field, but substantially less response before the onset of head saccades in other directions. Furthermore, neighboring IO neurons showed high correlation of activity before the onset of head saccades toward their receptive fields, but not in the middle of head fixation. These results suggest that population of active IO neurons varies in dynamically and topographically in the course of various phases of visually-guided behavior.

## P3-f06 Perceptual color spreading induced by visual motion

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The neon color effect is known as one of the visual illusions that the color of a figure spreads in the surrounding achromatic area. It occurs when the figure is static. We found out a new color spreading illusion that occurs only when a figure moves. We placed four chromatic squares on a circular orbit at even intervals against white background and rotated them along the circular orbit. When we continue to watch the center of the rotation, we perceive the same color as the square color on the white area inside the circular orbit. When the motion speed is too fast or too slow, the color spreading illusion does not occur. We consider that color induction occurs at the border area between the motion stimulus and the white background, then the induced color spreads and fills in the circular area surrounded by subjective contours made by the circular motion. By using a linear motion stimulus, we found that strong color induction occurs near the motion stimulus. When the color spreading area was limited by contours, the color filled in the area. The strong color spreading illusion we found may resolve the mechanism determining the attribute of surfaces.

## P3-f07 Burst ISI coding is conserved across species

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Under visual stimulation, the output neurons of the vertebrate retina generate spike bursts. We have reported that the interspike intervals (ISIs) of these bursts have distinct combinations depending on the stimulus and thus covey information about the stimulus. In salamanders, the ISIs of two-spike bursts generated by Off ganglion cells code the rate of light intensity modulation. In the present study we asked if the ISIs code similar stimulus features across species. We recorded from the retinas of mice and asked what the ISIs of two-spike bursts code. The results showed that similar to the ISIs in salamanders, the ISIs of two-spike bursts code the rate of light intensity modulation. In particular, the stimulus feature coded by the ISIs of two-spike burst generated by On cells are almost identical to that coded by the ISIs generated by salamander Off cells, except the sign of light intensity modulation. Thus burst ISI coding is conserved across species.

## P3-f08 Analysis of neuronal activation in the telencephalon in the visually imprinted chicks

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During a few days after hatching, chicks recognize and memorize a moving object which they see frequently, distinguish it from others and follow it. In this visual imprinting, it is known that the visual Wulst (VW) and intermediate medial mesopallium (IMM) play critical roles. Using optical imaging techniques with a voltage-sensitive dye, we found that a neural circuit that extends from the VW to the IMM was established by visual imprinting, and good learners which showed imprinting behavior until P7 retained high neural activities along this circuit. The expression of an immediate-early gene Arc revealed that the synaptic activity of the surrounding region was decreased while the activity within this circuit was maintained. Furthermore, we also observed the increased expressions of NMDA receptors and increased number of parvalbumin-positive neurons in the VW. These results indicate that the visual imprinting specifically enhanced neuronal activity within the circuit extending from the VW to IMM.

## P3-f09 Transcranial electrical stimulation for activating cortico-cortical connections in anesthetized mice

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Neural activities elicited by sensory stimuli in the primary auditory, somatosensory and visual cortices can be easily visualized using transcranial flavoprotein fluorescence imaging in anesthetized mice. However, activities in higher cortical areas are hardly elicited by natural sensory stimuli. Therefore, we developed a new method of transcranial electrical stimulation for activating cortico-cortical connections *in vivo*. The skull of a mouse anesthetized with urethane was thinned with a dental bar. A tip of a bare metal needle was gently pushed on the thinned skull, so that the thinned skull was deformed and the space between the skull and the cortex was minimized around the tip. Stimulus currents applied to the needle directly flew into the cortex just under the tip via the thinned skull, since current escape through the minimized subdural space was small enough. Using this method, we have succeeded to visualize the functional cortico-cortical connections between the primary and higher cortices and between motor and somatosensory cortices *in vivo*.

## P3-f10 Multiple generators model based on neural mass model for MEG/EEG

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Models for generating Magnetoencephalogram (MEG) and Electroencephalogram (EEG) have been previously developed on the basis of the neural mass model. These models regarded the cortical column as the generator of the alpha rhythm. Although several experimental studies indicated the existence of multiple generators of the alpha rhythm, these previous models had a single generator for each cortical area. Then, we developed a multiple generators model for V1, based on the neural mass model. Our model regarded the column as the hyper-column in V1, and therefore multiple generators are horizontally linked. The multiple generators model could reproduce not only the waveforms of the alpha rhythm and event-related potential but also the event-related synchronization/desynchronization of the alpha rhythm. Moreover, the multiple generators model could reproduce the dynamics that the alpha rhythm is reset by an external stimulus. These results suggest that the multiple generators model is useful for understand the dynamics of MEG/EEG.

## P3-f11 Relation between depth and flicker perception in motion transparency

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Motion transparency is perceived when two groups of dots moving in different directions are presented in the same region of the visual field. In this case, one group is perceived as a near surface, the other as a far surface. In the present study, we investigated the relation between depth and flicker perception in motion transparency. Firstly, effects of depth perception on flicker perception were examined. When two groups of dots moving in different directions are presented alternately at high frequency, transparent motion and flicker are expected to be perceived. However, subjects did not perceive the flicker if the moving dots were perceived as the far surface. This phenomenon suggests that the depth perception affects the flicker perception in motion transparency. Secondly, effects of flicker on depth perception were investigated. When one of two groups of moving dots was flickering and the other not, the subjects perceived the flickering dots as the far surface. This means that the flicker becomes a depth cue in motion transparency.

## P3-f12 Effect of the stimulus size on the human brain response to visual motion

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Visual detection of motion is one of cardinal property of human brain. In this study, we investigated how spatial extent of motion stimulus affects human brain response using 306 ch whole head magnetoencephalograph (MEG). We recorded MEG response to motion onset of sinusoidal grating with different size (2.2 × 2.9 deg to 44.8 × 57.4 deg) and speed (2.9 deg/s to 23.5 deg/s). The first component of MEG response (peaked at 114 ms to 216 ms) was observed around occipito-temporal area. The latency and amplitude was affected by the motion speed as previous study for the 2nd and 3 rd stimulus sizes. The latency was also affected by the stimulus size and was the shortest at the 2 nd largest stimulus size. In contrast, the amplitude was not affected by the size. The results show that the motion size affects human brain response in a manner different from motion speed, suggesting that the neural process of motion detection interacts with neural information about spatial extent of motion image.

## P3-f13 Perceptual cost for motion transparency: psychophysics and model analysis

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Transparency perception is recognized as an important phenomenon for the theory of the human visual system. This is because transparency perception indicates a simple theory reconstructing a single-valued field of a visual attribute cannot model the neural mechanism for early vision and raises the fundamental issue of how the visual attributes are represented and computed in the brain. Here we present a systematic study that examined the perceptual "cost" for motion transparency psychophysically. The perceptual performance for motion transparency is worse than that predicted by assuming overlapping motions are detected individually, and the cost would reflect the neural encoding mechanism for transparent motions. The results showed that the detection threshold became smaller as the direction difference between overlapping motions decreased, whereas the precision of directional judgement became worse. A computational analysis suggests that the cost for motion transparency results from the discriminability of a whole population profile of direction selective neurons as well as the inhibitory interactions between them.

KAKENHI (18700252)

## P3-f14 Real-time vergence control of binocular robot vision employing disparity energy computation in visual cortex

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Vergence is a binocular movement to keep or change fixation by rotating both eyes inward or outward. It is an important function for humanoid robots to control the vergence eye movements robustly in real-time. In order to implement the vergence in robot vision, however, costly computation is required in conventional machine visions in which CCD cameras are directly connected to the digital computer operating with a serial algorithm. As a consequence, they are often confronted with excessive power consumption and large-scale hardware. In the present study, we developed a binocular robot vision inspired by functional architecture of the brain. The developed system consists of two silicon retinas and FPGA circuits. The outputs of silicon retinas are integrated in FPGA and the disparity maps were computed based on the disparity energy model. The vergence was controlled with spatially pooled responses of disparity neurons. The robot vision developed here can emulate the vergence eye movements for random-dot patters with compact hardware and low power dissipation.

## P3-f15 Odor representation in the anterior piriform cortex of an *in vitro* isolated whole brain with the olfactory epithelium

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In order to examine how the differences of odorants are represented in the anterior piriform cortex (APC), we recorded odor-evoked local field potentials (LFPs) in multiple sites of the APC during olfactory stimulation, using an isolated guinea-pig brain with the olfactory epithelium. Several discrete concentrations of amyl acetate and ethyl butyrate were used as stimulants. We found that the steepness of the initial negative-going potential change after olfactory stimulation, which was indicated to reflect inputs from the olfactory bulb in our previous study, varied between odor stimulants and recording sites. The difference of spatial distribution of the dose-dependent potential change by odorants may suggest the differential coding of odorants within the APC.

## P3-f16 Olfactory neuromagnetic responses measured by passive and active olfaction

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This study is to analyze olfactory cognition in human by non-invasive measurements using a whole cortex bio-magnetometer. In this magnetoen cephalography(MEG), we have used two methods for measuring olfactory cognitive responses. The first is the experiment of "passive olfaction" on odd-ball paradigm using two kind odorants. From this experiment a later response was obtained at superior-temporal area. The second olfactory experiment was tested by "active olfaction" using a sniffing method. Subjects were instructed for sniffing odor actively by themselves. From the second test a sniffing response was only estimated at right orbital frontal area. Above two olfactory experiments gave us the different results and the different active areas on olfactory cognitive responses. This difference may be considered as the results of different olfactory cognitive mechanisms between a passive olfaction and an active olfaction.

research funds in Japan

## P3-f17 Neurons responsible for *C. elegans* chemosensation to pH

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Neuronal networks involved in pH responses have still remained to be explored in any animals. *C. elegans* senses acid pH and alkaline pH as nociceptive and attractive stimuli, respectively. We have developed an assay system for the study of *C. elegans* chemotaxis to alkaline pH. On an assay plate with a pH gradient from pH 7.0 to pH 9.5, wild-type worms were attracted toward alkaline pH along the gradient. In contrast, *che-1* mutants were found to avoid higher pH regions. Because *che-1* is deficient in differentiation of ASE chemosensory neurons, this result suggests that ASE neurons are required for chemotaxis toward alkaline pH, and other neurons than ASE are responsible for the higher alkaline pH avoidance. Furthermore, ASE-specific *dyf-3*(+) expression in *dyf-3* mutants defective in all gustatory sensory neurons rescued the defect in their chemoattraction toward alkaline pH. These results indicate that ASE neurons are necessary and sufficient for the chemotaxis toward alkaline pH, and that two neuronal networks may regulate the behaviors. We will also discuss about neurons responsible for the higher alkaline pH avoidance.

## P3-f18 The neprilysin gene *nep-2* is involved in olfactory adaptation in *C. elegans*

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With prolonged exposure to an odorant, *C. elegans* exhibit a diminished response to the odorant. In the early adaptation assay which we have previously developed, the response to an odorant diminishes with only 5 minutes of exposure to the odorant. To elucidate the mechanisms of early adaptation, we screened for mutants that show defects in this assay. We identified one of the isolated mutants as an allele of *nep-2*. *nep-2* encodes one of the neprilysin homologs in *C. elegans*. Neprilysin is a membrane protein with a large extracellular domain having endopeptidase activity. We found *nep-2* is expresssed in muscles and several neurons, and NEP-2 seems to act cell nonautonomously. We also show that the early adaptation defect of *nep-2* mutants is not due to developmental abnormality. Because a conserved HExxH motif is necessary for the activity of NEP-2, NEP-2 appears to act for degradation of extracellular peptides. This speculation also suggests that peptide signaling is important for early adaptation.

## P3-f19 Behavioral strategy of salt chemotaxis in *C. elegans*

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*C. elegans* is an ideal model organism for understanding the neural basis of behavior, because the whole neural circuit is known for this animal. *C. elegans* shows chemotaxis to various chemoattractants, but how it performs this task has not been fully investigated. In this study, video tracking analysis of chemotaxis was conducted, and two mechanisms for chemotaxis were identified. Freely moving worms occasionally change the direction of locomotion by a stereotyped behavior called pirouette. In the previously reported mechanism, the frequency of pirouette is modulated by temporal change of chemoattractant concentration, so that worms switch the direction of locomotion more frequently when the chemoattractant concentration decreases. In the other, newly identified, mechanism, worms sense the spatial gradient of chemoattractant concentration, and slowly turn towards the direction of higher chemoattractant concentration. Computer simulation suggests that neither of the two mechanisms is by itself sufficient for good chemotaxis, but real worms utilize combination of the two strategies to achieve efficient chemotaxis.

## P3-f20 Glutamate responses of mouse fungiform taste cells with action potentials

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Glutamate is one of the transmitter candidates for cell communication in taste buds as well as a umami compound. To investigate the dual functions of glutamate in taste cell activity, we recorded action potentials from single taste cells when glutamate is apically or basolaterally applied to the taste buds isolated from mouse fungiform papillae. The apical application of 100-300 mM monosodium glutamate (MSG) induced an increase in firing frequency in a subset of the taste cells. The response profile of these cells was classified into four categories by best stimulus and synergistic response with inosine monophosphate. The basolateral application (<1 mM) changed the spontaneous spike frequency in some of the taste cells responding to apical MSG, suggesting that basolateral glutamate modulates the basal activity of MSG-sensitive taste cells. Our results support the hypothesis that glutamate functions both as an umami compound and as a modulator of taste cell activity.

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## P3-f21 Taste receptor cells generating action potentials within a single taste bud of mouse fungiform papillae

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We have recently shown that taste receptor cells generating action potentials are the cells that transmit major taste information to gustatory nerve fibers. However, it is unclear whether these cells in a single taste bud would possess different taste selectivity or they would share the same taste selectivity. Here, we recorded action potentials from more than two different taste cells within a single taste bud separately or simultaneously by using a loose patch recording technique, and compared their taste selectivity to 4 conventional taste stimuli. In some cases, two taste cells in a bud responded to the same class of taste stimuli, whereas in other cases they did to different classes of taste stimuli, such as saccharin vs. quinine, saccharin vs. NaCl, etc. These results suggest that a single taste bud may contain the plural numbers of taste receptor cells having similar and different taste responsiveness.

※KAKENHI 18077004, 18109013 (YN) and 17791325 (RY).

## P3-f22 Voltage-gated currents of taste bud cells in infant mice.

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In adult mouse taste bud cells, the magnitude of voltage-gated currents depended on their cell types. The magnitude of $Na^+$ and outward currents was much smaller in type I cells than in other cell types, and that of TEA-insensitive outward currents was much larger in type II cells than in the others. Here we show that these relations are inapplicable to infant counterparts; some infant type II cells fail to elicit either $Na^+$ or outward currents though adult type II cells elicited both currents, and their currents elicited are much smaller in magnitude than that of adult type II cells. We also show that the number of taste bud cells increases markedly during the infant period. These results suggest that newborn type II cells express molecules involved in a taste transduction cascade such as G-protein γ-13, PLC β 2, and $IP_3R3$, before increasing the number of functional voltage-gated channels.

※21th century COE program in K. I. T. and a Grants-in-Aid for Scientific Research (#16300094) from JSPS

## P3-f23 Polymorphisms of ENaC subunits relate to mouse strain differences in amiloride sensitive salt responses

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Amiloride, epithelial $Na^+$ channel bloker, is known to inhibit NaCl responses of the chorda tympani (CT) nerve innervating anterior tongue in mice. C57BL/6(B6) exhibited inhibition of NaCl responses by amiloride to -50% of control, whereas slightly inhibition (-20%) was observed in 129P3/J(129) mice. Amiloride-sensitive epithelial $Na^+$ channel (ENaC) is a potent candidate for salt taste receptor. In this study, using B6, 129 and their $F_2$ hybrids, we investigated possible relationships between the amiloride sensitivity and SNPs of ENaC subunits ( α , β ,γ). Sequencing analysis detected a SNP resulted in an amino acid substitution, R616W, in α subunit. Responses of the CT nerve to NaCl decreased after amiloride treatment in B6 and $F_2$( α R616W genotypes: B6/129 and B6/B6), whereas only weak inhibition was evident in 129 and $F_2$(129/129). These results suggest that α R616W may be one of factors responsible for mouse strain differences in amiloride sensitive salt responses.

## P3-f24 Modulations of sweet taste responses by salts, acids and bitter compounds in the mouse chorda tympani nerve

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To investigate gustatory mixture interactions, responses to sweet compounds with and without salts, acids and bitter compounds were examined in the chorda tympani nerve of wild type (WT) and TRPM5-KO mice. In WT mice, whole nerve responses to 0.1-0.5 M sucrose increased when they were mixed with 30 mM NaCl (more than the sum of response to each component), whereas no such increase of sucrose responses was observed when mixed with 1-10 mM HCl. Responses to 0.3 M sucrose were suppressed to ~ 60% of control by addition of 3-10 mM QHCl. Consistently, responses of sweet-sensitive fibers to sucrose were significantly suppressed by 1-10mM QHCl. In contrast, sucrose responses in TRPM5-KO mice were not affected by QHCl. These results suggest that sweet taste responses are modulated by other taste substances at peripheral taste cell level and that TRPM5 may be involved in the pathway responsible for inhibition of sucrose responses by QHCl.

## P3-f25 Quantitative differences between taste buds in mouse fungiform papillae and soft palates

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The different taste responsiveness of the chorda tympani nerve and the greater petrosal nerve suggested the dissimilar expression of taste transduction proteins such as SNAP-25, $IP_3R3$, Gγ13 and G α gustducin in the taste bud cells innervated by them. We investigated their expression immunohistochemically; the density (cells/μm²) of immunoreactive cells (IRCs) per single taste buds in both regions was calculated by slicing each taste bud optically and horizontally with a confocal microscope. The density of Gγ13- and G α gustducin-IRCs was higher in soft palate taste bud innervated by the greater petrosal nerve than that in fungiform taste bud innervated by the chorda tympani nerve. The density of SNAP-25- and $IP_3R3$-IRCs did not differ. The present results suggest that the dissimilar expression of G proteins contributes to the differences in the neural responsiveness.

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## P3-f26 Temperature modulation of gurmarin sensitive sweet responses in the mouse chorda tympani nerve

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It is known that sweet responses of the mouse chorda tympani (CT) nerve are classified into two components; one is inhibited by gurmarin [Gur-sensitive (GS)] and the other is not [Gur-insensitive (GI)]. The C57BL strain possesses both components, whereas BALB mice have only the GI component. Recently, TRPM5 is found to be a temperature-sensitive channel through which temperature-denpendent increase (TDI) in response to sweeteners may be generated. Here, we showed that by examining GS and GI components to sweeteners in the mouse chorda tympani nerve at 15, 25, 35°C of temperature, we found that both GS and GI components exhibited the TDI. The magnitude of TDI in GS component, however, was smaller than that of GI component at higher temperatures (25-35°C). These results suggest that the GS component may be occur through not only temperature-sensitive (TRPM5-dependent) pathway, but also other unknown pathway(s) which may be temperature-insensitive and/or temperature-sensitive (TRPM5-independent).

## P3-f27 Organization of the projection targets of the *Drosophila* gustatory receptor neurons visualized with GAL4 enhancer-trap strains

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In *Drosophila melanogaster*, gustatory receptor neurons (GRNs) are located mainly on the labellum of the mouth, sending their axons to the primary gustatory center in the suboesophageal ganglion (SOG). Projection patterns of various types of GRNs have been analyzed by expressing reporter genes driven by the regulatory sequences of gustatory receptor (*Gr*) genes. To elucidate a more comprehensive organization of the primary gustatory center, we screened 4,000 GAL4 enhancer-trap strains for those that can visualize specific subsets of the GRNs, and obtained 7 lines. The flanking genes of the insertion site of each strain did not match with any known *Gr* genes, indicating that the expression patterns of these strains would reflect those of novel types of taste-associated genes. For example, one of the strains visualized mechanosensory neurons that are specifically associated with taste sensilla. We will report detailed neural architecture in the SOG visualized by these strains.

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## P3-f28 Phylogenetic comparison of serotonin-immunoreactive neurons in the insect olfactory neural systems

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In a previous study we compared the morphology of the neural circuits formed by the serotonin immunoreactive neurons (SIRNs) in the olfactory neural systems of various insect species. We found that, whereas the SIRN circuits were modified drastically during evolution in the antennal lobe (AL) -- the primary olfactory center, they were essentially preserved in the lateral accessory lobe (LAL) -- a candidate control center of olfactory-induced turning behavior. To evaluate the effect of specific lifestyles on the SIRN morphology, we here compared the SIRNs between male and female brains of the same species, as well as between olfactory-dependent and visual-dependent / diurnal and nocturnal / aeronautical and ambulatorial species within the same order. No significant sexual differences were observed. Moreover, species that belong to the same order never showed morphological differences of SIRN circuits both in the AL and LAL. These suggest that evolution itself, rather than the lifestyle-dependent factors, should play major role in defining the architecture of the SIRN circuits.

## P3-f29 Transmembrane adenylyl cyclase, encoded by *AC78C*, is required for sugar perception in *Drosophila*

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At '05 Neuroscience meeting, we reported that DGs $\alpha$ is coupled to sugar receptor, namely Gr5a, and conveys the sugar signal in sugar-sensitive receptor neurons. DGs $\alpha$ is a *Dorosphila* homologue of stimulatory G-protein $\alpha$ subunit (Gs). Since it is well known that Gs activates adenylyl cyclase (AC), we now asked which AC is required for sugar signaling. By the RT-PCR analysis, we found two AC genes are expressed in the taste organ. We employed the RNAi and GAL4/UAS system to specifically depress the expression of two genes in sugar-sensitive receptor neurons and found that the electrical sugar responses in the transgenic flies carrying RNAi construct against *AC78C* were depressed. Finally, we generated mutant flies of *AC78C* and found that the sugar responses in the mutant flies were also depressed. Taken together, we conclude that Gr5a, DGs $\alpha$ and AC78C are a set of transduction molecules to convey the sugar signal in receptor neurons.

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## P3-f30 Connectivity in the larval mushroom body calyx, a secondary olfactory center of Drosophila

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The mushroom bodies of insect brains are essential for associative olfactory learning. Here we show that the relatively simple Drosophila larval mushroom body calyx, is organized in about 34 glomeruli, which we have mapped. Individual glomeruli receive specific innervation from second order olfactory neurons. By contrast, they contain dendrites from hundreds of mushroom body neurons (Kenyon cells), which show low specificity for individual glomeruli. Glomeruli therefore potentially transmit specific sensory inputs to a large fraction of Kenyon cells. Dendritic termini of single larval-born Kenyon cells arborize in about 6 glomeruli in an apparently random manner. This pattern of connectivity is consistent with a model in which Kenyon cell dendrites process olfactory input by a combinatorial mechanism that can discriminate a large number of odors. To test the functional models suggested by the neuroanatomy, we are developing tools to image neuronal activity in the larval calyx.

## P3-f31 Differential impact of Lhx2 deficiency on expression of class I and class II odorant receptor genes in mouse

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Odorant receptor (OR) genes can be classified into two types: fish-like class I OR genes and terrestrial-specific class II OR genes. We have previously shown that Lhx2, a LIM-homeodomain protein, binds to the homeodomain site in the promoter region of mouse *M71*, a class II OR, and that a knockout mutation in *Lhx2* precludes expression of all tested class II OR genes including *M71*. Here, we report that most class I OR genes, which are expressed in a dorsal region of the olfactory epithelium, are still expressed in Lhx2-deficient embryos. There are two exceptions: two class I OR genes, which are normally expressed in a more ventral region, are no longer expressed in Lhx2 mutant mice. Lhx2 is transcribed in olfactory sensory neurons irrespective of expression of class I or class II OR genes. Thus, a deficiency of Lhx2has a differential impact on class I and class II OR gene expression, suggesting that two distinct pathways underlie OR gene expression in mouse.

※KAKENHI (18570206)

## P3-f32 Expression of TRPM8 in rat dorsal root ganglion neurons innervating the urinary bladder

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The transient receptor potential (TRP) channel family contributes to transmit sensory information. TRPM8 is an nonselective outwardly rectifying channel and activated by cold temperature or cooling compounds such as menthol. Afferent pathways innervating the urinary bladder consist of Aδ- and C-fibers, the neuronal cell bodies of which correspond to medium and small-sized cell populations in DRG neurons, respectively. Afferent fibers passing in the pelvic nerve to the spinal cord are responsible for initiating the micturition reflex and pelvic nerve afferent fibers innervating the bladder enter the spinal cord mainly via L6 and S1 DRG. To elucidate the role of TRPM8 in DRG neurons innervating the urinary bladder, the expression of TRPM8 mRNA and protein in DRG L6 and S1 was examined. TRPM8 mRNA and protein were colocalized in single DRG neurons that were primarily small-sized. TRPM8 protein might play a role in the transmission of cold sensation from the bladder wall.

## P3-f33 Brain-derived neurotrophic factor-immunoreactive neurons in the rat vagal and glossopharyngeal sensory ganglia; co-expression with other neurochemical substances

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Immunohistochemistry for brain-derived neurotrophic factor (BDNF) was performed on the rat vagal and glossopharyngeal sensory ganglia. In the jugular, petrosal and nodose ganglia, 56.1 ± 5.5%, 52.4 ± 9.4% and 80.0 ± 3.0% of sensory neurons, respectively, were immunoreactive for BDNF. These neurons were small to medium-sized, and observed throughout the ganglia. A double immunofluorescence method demonstrated that BDNF-immunoreactive neurons were also immunoreactive for calcitonin gene-related peptide (CGRP),the capsaicin receptor (VR1) or vanilloid receptor 1-like receptor (VRL-1) in the jugular (CGRP, 43.5%; VR1, 71.7%; VRL-1, 0.5%), petrosal (CGRP, 33.2%; VR1, 54.2%; VRL-1, 23.3%) and nodose ganglia (CGRP, 1.8%; VR1, 70.7%; VRL-1, 11.5%). The present findings suggest that BDNF-containing vagal and glossopharyngeal sensory neurons have nociceptive functions.

## P3-f34 Role of histaminergic neurons in cognitive control of hypersensitive visceral perception

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Molecules that act during cognitive modulation of visceral pain are not known. We tested our hypotheses that the abnormality of central pain modulatory system is conditional to the patients with visceral hyperalgesia and that these conditions are in part dependent on histaminergic neurons. Twelve male subjects with irritable bowel syndrome were studied on two separate days: a day of treatment with histamine $H_1$ receptor antagonist and another day of that with placebo in a randomized order. We recorded cortical evoked potentials to 100 rectal electrical stimuli following hypnotic suggestions. Following analgesic suggestion, the amplitude of P1/N2 component was significantly increased, and the latency of P1 component was significantly shorter than neutral condition ($p<0.01$). Administration of histamine $H_1$ antagonist diminished this reversal of analgesic effect. These results suggest that the central pain modulatory system activated by hypnotic suggestion are abnormal in visceral hyperalgesia and that brain histamine is a mediator in this pathway.

## P3-f35 Immunohistochemical mapping of NPR-A in the brainstem of macaca fascicularis

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Natriuretic peptide receptor-A (NPR-A) mediates the biological effects of atrial and brain natriuretic peptides (ANP and BNP) in maintaining cardiovascular homeostasis. Here, we examined the distribution of NPR-A immunoreactivity in the monkey brainstem. NPR-A immunoreactivity was localized to perikarya and fibers in specific but widely distributed brainstem regions. Interestingly, NPR-A immunoreactivity was found in cholinergic neurons of the parabrachial nucleus, dorsal motor nucleus of the vagus, hypoglossal nucleus, and nucleus ambiguus. However, there was no colocalization of NPR-A and tyrosine hydroxylase. The wide distribution of NPR-A suggests that natriuretic peptides, besides having a role in the central regulation of endocrine and cardiovascular homeostasis, may also mediate diverse physiological functions.

## P3-f36 Osmosensitivity in the synaptic inputs to the magnocellular neurosecretory cells in the supraoptic nucleus of rat

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The magnocellular neurosecretory cells (MNCs) in the supraoptic nucleus (SON) project their axons to the posterior pituitary and secrete vasopressin into the systemic circulation. The neuronal activity of MNCs is modulated by synaptic inputs and humoral factors such as osmotic change. In the present study, we examined the effects of osmotic stimulation on excitatory synaptic inputs to the MNCs in the SON in in vitro brain slice preparation, using whole-cell patch-clamp recordings. Bath application of mannitol (60 mM) caused a significant increase of the frequency of the spontaneous ecitatory postsynaptic currents (EPSCs) without affecting the amplitude. The increased frequency of EPSCs persisted in the presence of tetrodotoxin. Osmotic stimulation-induced potentiation of EPSCs was suppressed by previous exposure to the transient receptor potential vanilloid (TRPV) blocker, ruthenium red (10μM). These results suggest that TRPV family may be involved in osmotic modulation of synaptic inputs to the MNCs in the SON.

## P3-f37 Injection of biocytin and golgi staining of medaka brain

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VT/IT-ir neurons are sexually dimorphic and reduce in number after spawning in the female medaka fish. Further, VT/IT-ir fibers form a plexus in the medulla. In the present study, by injecting biocytin into the spinal cord of the medaka fish *in vitro*, we observed fibers extending from the diencephalon, and fibers consisting the plexus in dorsal and ventral portion of the medulla. Furthermore large neurons were labeled in the nucleus of medial longitudinal fasciculus (nMLF). The present observation is in good agreement with a previous report in the dwarf gourami, that the neurons of medial longitudinal fasciculus (MLF) form several different clusters, since we detected at least three kinds of cells in the MLF by golgi staining in the medaka fish brain.In the present experiment we failed to obtain any direct evidence that shows direct projection of the NPO neurons to the spinal cord. Since, the plexus in the medulla, which is labeled by the biocytin applied to the spinal cord and VT/IT-ir fibers form, it is possible that VT and/or IT neurons detected in the NPO may play roles as interneurons.

## P3-f38 Immunohistochemical observation of the postnatal development of the orexin neuron in the rat brain.

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The orexin neurons localized in the lateral hypothalamic area are thought to participate in regulations of various physiological functions. In the present study, we immunohistochemically investigated the postnatal development and aging of orexin-A or -B neurons in the rat brain at 9 day-, 2 week-, 3 week-, 6 week-, 9 week-, 8 month-, and 24 month-old. No immunoreactivity for orexin-A or -B was found in 9 day-old rats while small number of orexin-A or -B neurons was observed at 2 week-old rats. After that, the number of orexin-A and -B immunoreactive neurons increased gradually to the adult rats. On the other hand, the number remarkably decreased at 8 months. Although it was observed that the number of immunoreactive neurons was almost same as the 8 month-old rats at 24 month-old, the immunoreactivity apparently decreased. These results indicate that both orexin-A and -B immunoreactive neurons were accompanied by the maturation and the aging of animals, suggesting that the orexin neurons were correlated with some physiological and energy homeostasis conditions in the age.

## P3-f39 Identification of thyroid hormone dependent genes in the developing mouse cerebellum: effect of thyroid hormone on cell proliferation and death

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Although the indispensable role thyroid hormone (TH) plays in brain development, its precise mechanism of action remains largely unknown. In the present study, we screened for TH-responsive genes in the cerebellum at PND 4 when morphological alterations are not apparent. Among the new candidate genes selected by comparing gene expression profiles of experimentally hypothyroid, hypothyroid with postnatal TH replacement, and control animals using microarrays, 7 genes were confirmed by real-time PCR to be positively (Orc1, Galr3, sort1, Nlgn3, cdk5r2, zfp367) or negatively (akap9) regulated by TH. Examination of TH-dependent differences in cell proliferation and apoptosis by BrdU incorporation and TUNEL assay, respectively, revealed that TH enhances cell proliferation through increase of Orc1 and, at the same time, enhances neuronal apoptosis possibly through increase of sort1 and cdk5r2 which is an activator of cdk 5 whose hyperactivation leads to cell death.

## P3-f40 Effects of prolactin-releasing peptide administration upon anxiety-related behavior in rats

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We have previously shown that emotional stimuli activate prolactin-releasing peptide (PrRP) -containing neurons in the brain. Here we examined effects of PrRP administration upon anxiety-related behavior in rats. Wistar male rats were used. Animals were implanted with a guide cannula into the brain under avertin anesthesia. PrRP was injected into the lateral cerebral ventricle, or microinjected into the bed nucleus of the stria terminalis, dorsomedial hypothalamus, or hypothalamic paraventricular nucleus via the guide cannula. Fifteen minutes after the injection, rats were tested with an elevated plus maze test. Rats injected icv with PrRP or microinjected into the dorsomedial hypothalamus showed significantly reduced anxiety-related behavior. These data suggest that PrRP within the dorsomedial hypothalamus has anxiolytic effects in rats.

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## P3-f41 Glucocorticoid receptor interaction with nuclear factor κB in the nervous system

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Stress has been known to induce increases of inflammatory activity, through the activation of nuclear factor (NF)- κB. In the immune system, GR has been found to associate with NF-κB (p65). However, it is not clear whether GR also interacts with p65 in the nervous system. In this study, we investigated the protein-protein interaction between GR and p65 in the mouse brain. Since immunohistochemistry showed abundant distribution of GR and p65 in the hippocampus, we investigated the interaction of GR with p65 in the whole cell extracts from the cultured hippocampal neurons by using immunoprecipitation. We further examined the interaction of GR with p65 using the cell extracts from the nervous tissues of the mice subjected to restraint stress by immunoprecipitation, and compared the results to that of the control mice. Fluorescence resonance energy transfer was performed on the interaction of CFP-GR with YFP-p65.

## P3-f42 Immunohistochemistry of CD38 in hypothalamus and pituitary

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Oxytocin (OT) and arginine vasopressin (AVP) are neurohypophysical hormones. CD38 with cyclic ADP-ribose (cADPR) formation is identified in the hypothalamus and is critical for OT secretion, but not AVP, with profound consequential changes in social behaviors. In the present study, we examined the immunolocalization of CD38, OT and AVP in the hypothalamus of mice. CD38 was co-localized with neither OT nor AVP completely. It means that CD38 is expressed in cells other than hormone-producing neurons. To identify the distribution of CD38, we are now investigating experiments whether neuron-glia interaction is involved in the cADPR-dependent OT release and subsequent social behaviors.

Kanazawa University 21st Century COE Program on Innovative Brain Science on Development, Learning and Memory

## P3-f43 Effect of estrogen on spine regulation in hippocampus

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Estrogens modulate transcription known as genomic effects in the brain as same as in peripheral organs. But estrogen also elicit rapid non-genomic effects on the synaptic transmission. Recently, we elucidated the localization of estrogen receptor alpha (ER $\alpha$) at synapse in principal neurons in the hippocampus by using purified antibody RC-19. Here, we demonstrated the rapid effect of estrogens on the density and morphology of spines in each layers by imaging Lucifer Yellow-injected CA1 neurons in adult male rat hippocampal slices. The application of 1 nM estradiol-induced a rapid increase in the density of spines of pyramidal neurons within 2 h. ER $\alpha$ agonist PPT but not ER $\beta$ agonist DPN induced the same enhancing effect of spinogenesis as that induced by estradiol. This increase was blocked by Erk MAP kinase inhibitor and estrogen receptor inhibitor in all regions.

## P3-f44 Effects of ghrelin on neuronal activity of anterior hypothalamic area in rats

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Ghrelin participates in the regulation of growth hormone (GH) release and sleep-wakefulness via GH secretagogue receptors (GHS-Rs). Since neurons in the anterior hypothalamic area (AHA) express abundant GHS-Rs, we examined effects of ghrelin on AHA neurons electrophysiologically using rat brain slice preparations. Application of ghrelin depolarized AHA neurons, and increased firing rate dose-dependently in about 60% of AHA neurons. During the depolarization, membrane resistance significantly decreased. I-V relationships obtained before and during application of ghrelin indicate that the reversal potential for depolarization is around -43 mV. The ghrelin-induced depolarization was further disappeared in the ACSF containing flufenamic acid (FFA). These results suggest that ghrelin depolarizes AHA neurons by opening of nonselective cation channels, and participates in the regulation of GH release and sleep-wakefulness.

## P3-f45 Cold-sensitive phenotype in mice lacking oxytocin receptor

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Oxytocin (Oxt) receptor deficient mice (Oxtr -/-) failed to maintain their body temperature after acute cold exposure. In Oxtr -/- mice, their brown adipose tissue showed typical features observed in mice with dysfunction of thermogenesis, lower levels of $\beta$3 adrenergic receptor (AR) mRNA at room temperature, and higher levels of $\alpha$2 AR mRNA at room and low temperature. Since these ARs have opposite effects on thermoregulation, the inbalance of ARs might cause this dysfunction in thermoregulation of Oxtr -/- mice.
Then we analyzed the thermoregulatory center of the mutant. In contrast to wildtype animals, Oxtr -/- mice exposed to cold condition showed attenuation of c-fos immunoreactivity in the ventromedial hypothalamus. These results suggested that Oxt/Oxtr systems controlled body temperature via central nervous system.

KAKENHI (18380063)

## P3-f46 Neurosteroids synthesis in the rat hippocampus

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It is widely accepted that dihydrotestosterone (DHT) and 17beta-estradiol (E2), the main androgen and estrogen in mammals, reaches the brain via blood circulation and modulate efficiency of learning and synaptic plasticity. However, recent study suggests the possibility that those steroids are synthesized in the rat brain. This indicate that brain-derived androgen and estrogen act as a modulator of synaptic plasticity. DHT, E2 and their precursors and metabolites are synthesized by enzyme cytochromeP450(17a), P450(arom),3b-HSD, and 17b-HSD, but presence of those enzymes in the rat brain are still unclear. To prove the presence of androgen and estrogen synthesis pathway, we examined the expression level of mRNA of those enzymes in adult male rat brain by means of the semi-quantitative RT-PCR. This experiment demonstrated the presence of the enzymes required E2 and DHT synthesis pathway in the brain. These results indicate that the pathway of synthesis of androgen and estrogen exist in rat hippocampal neurons. We are also investigating the pathway of stress steroid synthesis in hippocampus now.

## P3-f47 Morphological characteristics of gonadotropin-releasing hormone (GnRH) neurons in the preoptic area of GnRH-EGFP transgenic rat

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To understand better regulation mechanism of puberty onset, we examined morphology and synaptic input of GnRH neurons in the preoptic area (POA), using GnRH-EGFP transgenic rats. Observations with confocal laser scanning microscopy indicated that GnRH neurons were generally unipolar and bipolar. Both of them were subdivided into the smooth type cell with few small processes and the irregular type cell with numerous spine-like processes in the proximal dendrites. Prepubertal GnRH neurons mainly appeared unipolar, whereas adult GnRH neurons principally exhibited the typical bipolar feature, suggesting a morphological change of the GnRH neurons in the POA during the course of reproductive maturation. Synaptophysin immunofluorescence experiments revealed that presynaptic terminals mostly innervated the spine-like processes of GnRH neurons, suggesting excitatory inputs to GnRH neurons through synaptic contacts with these processes.

## P3-f48 Increase of heat shock proteins by treatment of cultured hypothalamic cells with 17beta-etstradiol

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To elucidate roles of 17beta-estradiol (E2) on sexial differentiation of neuronal cells, we comprehensively analyzed effects of E2 on cultured hypothalamic cells by proteomics. Hypothalamic and cortical cells, obtained from rat fetuses (E15), were cultured with or without E2 for 8 days. By quantitative 2-dimensional electrophoresis, differentially expressed proteion spots were detected, a part of which were further identified by mass spectrometry. We found that heat shock protein (HSP) 1 and HSP90 were increased by E2 in the hypothalamic cells, but not in the cortical cells, while HSP70 were increased in the both cell types. Together with known essential roles of molecular chaperones in signal transduction, the E2-induced different regulation of HSPs between the hypothalamic and cortical cells, presented here, implicates involvement of HSP1 and HSP90 in the E2 signaling in the hypothalamus.

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## P3-g01 Neurophysiological analysis of GABAergic synapses in the terminal nerve GnRH neurons

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GABA is the major inhibitory neurotransmitter in the brain. In immature neurons, however, the action of GABA is mostly excitatory. It has recently become clear that GABA induces excitation in some specific synapses in adult brain. Thus, the clear-cut dichotomy that glutamate is excitatory and GABA is inhibitory may not be always valid. It is therefore necessary to reexamine whether the nature of each GABAergic synapse is inhibitory or excitatory, which should facilitate our understanding of synaptic transmissions. Here we focused on the terminal nerve (TN)-GnRH neurons whose GABAergic afferents remain to be investigated. TN-GnRH neurons are peptidergic neurons and suggested to modulate neuronal excitability in wide areas of the brain simultaneously. Our neurophysiological analysis using *in vitro* brain preparation of adult teleosts indicated that (1)TN-GnRH neurons express functional $GABA_A$ receptors, and (2)activation of $GABA_A$ receptor induces excitation under the condition maintaining the intracellular milieu of the neurons. We also found the voltage-dependence of $GABA_A$ receptors.

KAKENHI 18370029 18021009 17657027

## P3-g02 Function analysis of Pituitary adenylate cyclase-activating polypeptide associated with schizophrenia in hippocampal dentate gyrus

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PACAP was first identified as a novel hypothalamic neuropeptide, based on its ability to stimulate adenylate cyclase in cultured rat anterior pituitary cells. Recently, it was reported that pituitary adenylate cyclase-activating polypeptide (PACAP) is a promising schizophrenia candidate gene. This time, to study a physiology of PACAP, we screened mobilizing factor in hippocampal dentate gyrus of PACAP knockout mice. As a result, we identified the gene associated with neurogenesis. And its positive cells exist in subgranular zone. These data suggest that alterations in PACAP signaling might affect a neurogenesis and contribute to the pathogenesis of schizophrenia.

## P3-g03 Dysfunctional mastication increases dynorphin A-like and β-endorphin-like immunoreactivities in the amygdala and hypothalamus

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We immunohistochemically investigated the effects on endogenous opioids (dynorphin A and β-endorphin) in the cerebral limbic system and hypothalamus of the bite-raising condition that was made by increasing the height of mouse teeth with a dental resin. The mice under the bite-raising condition revealed increases in their immunoreactive intensity and their immunopositive cell numbers in both the amygdala (especially in the central amygdala) and hypothalamus (especially in the paraventricular hypothalamic nucleus), in comparison with sham-operated mice. The data suggest the involvement of dysfunctional mastication in the emotional integration processes that may be coupled with endogenous opioids.

## P3-g04 Elimination of sex differences in the principal nucleus of the bed nucleus of the stria terminalis (BSTp) in estrogen receptor-α and -β and aromatase knockout mice

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The BSTp of males is larger than that of females in mice. Aromatized androgen, estradiol, which is locally synthesized by aromatase and binds to estrogen receptors (ERs) in the brain, is implicated in sexual differentiation of the rodent brain. In the present study, we examined morphometrically whether sex differences in the volume of the BSTp might be altered in ERα, ERβ, or aromatase knockout (KO) mice. In wild-type (WT) mice, the volume of the BSTp was significantly larger in males than in females, whereas these sex differences were completely abolished in all three KO mice. The abolition of sex difference was due to a reduction of the size of the BSTp in KO male mice, compared to that in WT male mice. The female BSTp was not affected by deletion of any gene. These results indicate that ERα, ERβ, and aromatase are involved in the sexual differentiation of the BSTp in mice.

## P3-g05 Sexually dimorphic metastin neurons in Medaka brain

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Novel peptidergic neurons, metastin neurons, are thought to facilitate sexual maturation and ovulation by stimulating gonadotropin releasing hormone (GnRH) neurons, but their physiological functions are still largely unknown. Here we focused on the relationship between the metastin and hypothalamic/extrahypothalamic GnRH neurons. Taking advantage of well-developed teleost GnRH systems, we used medaka, which is a useful teleost for the application of various molecular genetic tools. First, we cloned and sequenced the metastin gene (Kiss1) in medaka for the first time in non-mammalian species. Then, Kiss1 expression in the brain was analyzed by in situ hybridization. We found two hypothalamic nuclei with Kiss1 expression and discovered prominent sexual difference in cell number (male>>female) in one of the two nuclei. On the other hand, we found no circadian variation in expression of Kiss1 by real-time PCR, although medaka shows daily ovulatory cyclicity.

※KAKENHI 18370029 18021009

## P3-g06 Synaptic estrogen receptors in principal neurons of hippocampus

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Estrogens elicit rapid non-genomic effects on the synaptic transmission and spinogenesis in the hippocampus. However, the existence of estrogen receptor alpha (ERa) still remains elusive. With highly purified antibody RC-19, mass spectrometric analysis identified ERa in the hippocampus and immunohistochemistry showed ERa localization in principal neurons of CA1, CA3, and granule cells in dentate gyrus. Further we found the synaptic distribution of ERa by immunoelectron microscopy. We also examined the distribution of putative G-protein coupled estrogen receptor, GPR30. These results imply that synaptic estrogen receptors mediate the effects of estrogen in hippocampal neurons.

## P3-g07 Accumulated fatigue induces over-activation and subsequent degeneration of melanotrophs in rat pituitary gland

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Accumulated fatigue causes dysfunction of cellular mechanisms or homeostasis in various organs. In this study we have employed an animal fatigue model in which rat was kept in a cage filled with water height of 1.5 cm for up to 7 days. The melanotrophs in the intermediate lobe (IL) of the fatigued rat pituitary demonstrated marked morphological changes of rER and Golgi apparatus, and subsequent cell degeneration. The tyrosine hydroxylase positive nerve fiber in IL was decreased during the fatigue stimulation. The application of dopamine antagonist to normal rats mimicked the morphological changes of rER and Golgi apparatus, and dopamine agonist to fatigued rats inhibited the changes. These results suggested that prolonged fatigue suppressed dopamine release in IL, and thereby caused an over secretion of α-MSH from melanotrophs, leading to ER dysfunction and subsequent cell death.

## P3-g08 Isolated primary culture of the terminal nerve (TN)- GnRH neurons derived from the brain of a tropical teleost

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The TN-GnRH neurons release GnRH peptides from widely-branched neural processes, depending on their firing activity, and the released GnRH is suggested to exert their neuromodulatory action via GnRH receptors expressed in nearby as well as distant target neurons. To address questions on how electrical activities of the TN-GnRH neuronal cell bodies affect GnRH release from the widely-branched neural process, we tried to establish the primary culture of TN-GnRH neurons. The whole brain of the adult dwarf gourami (*Colisa lalia*, a tropical freshwater fish) was dissected out and was briefly treated by protease. The TN-GnRH neurons were sucked out from the cluster of TN-GnRH neurons with the aid of a suction pipette and IR-DIC optics and cultured on coverslips. The isolated TN-GnRH neurons were kept at room temperature in a darkened humidity chamber and could survive up to around two weeks. Both axon- and dendrite-like processes grew out from the cultured TN-GnRH neurons, and characteristic growth cone and varicosity-like structures were observed.

KAKENHI (17770059,18370029,18021009)

## P3-g09 Oxytocin receptor-Venus knock-in mice enable direct visualization of oxytocin receptor-expressing neurons

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The oxytocin receptor (OXTR) regulates reproductive physiology and sociosexual behaviors. Recently, we generated *Oxtr* gene-deficient mice (*Oxtr* -/-) and reported that Oxtr -/- exhibited pervasive social deficits (1). To further study the functions of OXTR, we newly generated knock-in mice that express Venus (variant of YFP), which was replaced with the *Oxtr* coding region. Distruption of *Oxtr* gene locus, absence of *Oxtr* transcripts and expression of *Venus* transcripts were comfirmed in OXTR-Venus knock-in mice. Interestingly, in these mice, the fluorescence was observed not only at regions previously identified to be expressing OXTR, but also at where its expression had not been described. The OXTR-Venus knock-in mice would be a powerful new tool to study the physiological role of OXTR-expressing neurons and the dynamics of OXTR signaling in the living neurons.(1) Takayanagi et al. PNAS. 2005

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## P3-g10 Taste may not affect salt appetite in zinc deficient rats

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Some reports demonstrated that zinc deficient (ZnX) brought about taste disturbance and that ZnX rats enhanced their preference to sodium solution. In the present study, we conducted some experiments to investigate whether or not taste affected this prefrence for sodium. Results were as follows; in the long-term (48 h) two-bottle preference test, the preference percents for 0.1 and 0.3M NaCl in the ZnX rats were higher than those in the control rats. But in the short-term (10 min) test, there was no significant difference in the preference percents between ZnX and control rats. There was no significant difference in the neural responses of chorda tympani nerves to some sodium solutions between ZnX and control rats. By the assay of aldosterone in serum of the ZnX rats, a disorder of sodium metabolism was found in these rats. These results suggest that the enhancement of preference for sodium is caused by the disorder of sodium metabolism was found in these rats. These results suggest that the enhancement of preference for sodium is caused by the disorder of sodium metabolism rather than taste disturbance.

※KAKENHI(15390629, 15591984), Miyata Fund.(A)

## P3-g11 Synaptic organization between cortical axon terminals and Melanocortin 4 receptor-expressing thalamic neurons of the transgenic mouse

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The melanocortin 4 receptor (MC4-R), distributed exclusively in the central nervous system, has been considered to play a critical role in the regulation of feeding behavior and autonomic homeostasis. Using a transgenic mouse model in which green fluorescent protein (GFP) is produced under the control of the MC4-R promotor, we examined the synaptic organization between cortical axon terminals and MC4-R expressing thalamic neurons. Axon terminals from the infralimbic cortex (ILC) and insular cortex (IC) formed asymmetrical synaptic contacts with dendrites and dendritic spines of the GFP-immunoreactive neurons in the reuniens thalamic nucleus and gustatory thalamic nucleus, respectively. Moreover, these two sets of cortical axon terminals were revealed to be immunoreactive for vesicular glutamate transporter 1. The present data suggest that excitatory inputs from the ILC and IC to the MC4-R expressing thalamic neurons may be involved in the control of food intake and autonomic function.

## P3-g12 Synaptic organization between amygdaloid axon terminals and feeding-related peptide containing or melanocortin 4 receptor-expressing neurons in the mouse hypothalamus.

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Orexin (ORX)- and melanin-concentrating hormone (MCH)-containing neurons as well as melanocortin 4 receptor (MC4-R)-expressing neurons in the lateral hypothalamus (LH) are involved in the regulation of feeding behavior. To know whether or not these neurons are under the influence of the amygdala, here we examine synaptic organization between these neurons and axon terminals originating from the central amygdaloid nucleus (CeA). MC4-R-expressing neurons were visualized as green fluorescent protein (GFP)-immunoreactive (ir) neurons using a transgenic mouse model in which GFP is produced under the control of the MC4-R promoter. After biotinylated dextran amine (BDA) injection into the CeA, overlapping distribution of BDA-labeled fibers and ORX-, MCH-, or GFP-ir neurons was seen in the LH. Under the electron microscope, the CeA axon terminals formed asymmetrical synapses predominantly with somata and dendrites of MCH- and GFP-ir neurons, and occasionally with those of ORX-ir neurons.

## P3-g13 Reduction of feeding behavior by *in vivo* knockdown of NPY mRNA using siRNA in hypothalamic arcuate nucleus

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NPY is a potent molecule for feeding in the CNS. NPY cooperated in feeding regulation with orexigenic peptides such as AgRP. RNAi has become an important tool to knockdown of gene expression. Here, we demonstrated that siRNA vector against NPY was stereotaxically injected into the hypothalamic arcuate nucleus, which is the center of feeding. siRNA vector, which lead to transcription of small RNA by U6RNA polIII promotor, were transfected with lipofection reagents. Knockdown of NPY gene expression significantly decreased feeding in nighttime for 3days, but not in daytime. Body weight decreased with NPY siRNA injection. These results suggested that NPY signaling in nocturnals contributes to feeding in nighttime, which is major feeding period. Moreover the change in gene expression of other feeding regulated neuropeptides was also studied by RT-PCR and ISH.

In summary, NPY is a key molecule in circadian rhythm of feeding. siRNA-induced knockdown of gene expression serves as a powerful method for assessing the function of appetite-related genes.

## P3-g14 Olanzapine increases body weight and cell mitotic activity in the hypothalamus of mice

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Olanzapine, an atypical antipsychotic drugs, has been used to treat schizophrenia. The effects of atypical antipsychotic drugs for positive symptoms are comparable to typical ones and also effective to negative symptoms. Given the less extrapyramidal side effects of the atypical antipsychotics, they are more frequently used, but at the same time the risk of weight gain, hyperglycemia and hyperlipidemia are emerging. Recently it was reported that neurogenesis also occurs in the hypothalamus of mice and the newborn neurons are involved in controlling body weight and energy metabolism. In the present study, we hypothesized that olanzapine treatment may influence neurogenesis in the hypothalamus, which may be related to weight gain. So far we revealed that food intake, body weight change of olanzapine-treated mice was increased and the number of BrdU positive cells in the hypothalamus was increased. We now study the characteristics of the newborn cells.

## P3-g15 Cannabinoid type 1 receptors in the nucleus accumbens shell are involved in the intake of palatable solutions

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Systemic injections of cannabinoid agonists have been shown to facilitate the intake of palatable food and fluids. However, it is unclear which brain regions are responsible for the cannabinoid-mediated feeding. In the present study, we investigated the role of cannabinoid type 1 receptor (CB1R) in the nucleus accumbens shell for the palatability-induced ingestion in rats. The intake of taste solutions or distilled water was measured for 3 hours after bilateral administrations of each drug. Microinjections of anandamide, an endogenous agonist of CB1R, significantly increased the intake of a palatable saccharin solution but not of water and an unpalatable quinine solution. In contrast, microinjections of an antagonist of CB1R AM251 significantly reduced the consumption of saccharin solution but not of water and quinine. These results suggest that the CB1 receptors in the nucleus accumbens shell are selectively involved in the intake of palatable solutions.

## P3-g16 WD-repeat protein 6 is involved in nutrient sensing in rat hypothalamus

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The hypothalamus is organized as a collection of distinct, autonomously active nuclei that regulate discrete functions, such as feeding activity and metabolism. We used suppression subtractive hybridization (SSH) to identify genes that are enriched in the hypothalamus of the rat brain. We found four genes that were predominantly expressed in the hypothalamus, compared to other brain regions. The mRNA for a member of the WD-repeat family of proteins, WDR6, was abundantly expressed in the hypothalamus, and we found that WDR6 interacts with insulin receptor substrate 4 (IRS-4) in the rat brain. WDR6 gene expression in the hypothalamic arcuate nucleus was decreased by caloric restriction, and in growth hormone (GH)-antisense transgenic rats, both of which are associated with an increased life-span. Our results might suggest that WDR6 participates in insulin/IGF-I signaling and the regulation of feeding behavior and longevity in the brain.

## P3-g17 A novel leptin-induced transcript in the expression of growth hormone secretagogue-receptor

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Ghrelin promotes food intake via the activation of growth hormone secretagogue-receptor (GHS-R), resulting in the increase of body weight. In this study, we identified a novel leptin-induced transcript, named Clone 1-42, which was expressed in the GHS-R-positive cells in the hypothalamus and pancreas. Luciferase reporter assay revealed that Clone 1-42 repressed the GHS-R promoter activity in a hypothalamic neuronal cell line, GT1-7 cells, and a mouse insulinoma cell line, MIN6 cells. Overexpression of Clone 1-42 reduced the expression of GHS-R protein in these cells. It has been reported that ghrelin inhibits glucose-induced insulin secretion in MIN6 cells. Clone 1-42 repressed the inhibitory effects of ghrelin on glucose-induced insulin secretion. Inhibition of ghrelin action in obese mice may lead to the reduction of food intake and body weight, suggesting the therapeutic potential of Clone 1-42 for the treatment of obesity.

※KAKENHI 17019047

## P3-g18 Fat binge eating alters dopamine response in rat nucleus accumbens

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Intermitted ingestion of fat induces binge eating, a type of addictive behaviors. To investigate dopamine (DA) involvement in this behavior, we measured DA level of the nucleus accumbens (NAc) with fat binge eating (FBE) rats using in vivo microdialysis. FBE was induced by intermitted access to fat for 1 h every 2nd day. In this condition, rats developed binge eating and consumed over twice amount in 5th fat access compared to 1st access. As control groups, long- (LI) and short- (SI) interval group were made. LI and SI group had 24h fat access only once 12 days or 2 days before microdialysis experiment, respectively. After these schedules, a microdialysis probe was implanted to the NAc and DA responses to 20min fat eating were measured. Fat ingestion increased DA level in NAc in each groups in schedule dependent manner (LI > FBE > SI). Repetitive consumption of fat reduced the DA response, but this reduction was attenuated in FBE group. The present results suggest that DA change in NAc may be involved in developmental process of FBE.

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## P3-g19 Palmitate-induced increase in long-chain fatty acid receptor GPR120 mRNA level in L$\beta$T2 pituitary gonadotrope cells

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Our previous study suggested that fasting-induced increase in G-protein-coupled receptor 120 (GPR120) mRNA level in the anterior pituitary gonadotrope. The aim of present study was to determine the GPR120 mRNA modulating factor in the gonadotrope. For this purpose, changes in GPR120 mRNA level in response to long-chain fatty acids and low glucose were examined in L$\beta$T2 pituitary gonadotrope cells. Long-term exposure of L$\beta$T2 cells to 0.1 mM palmitate induced increase in GPR120 mRNA level. The result suggests that long-chain saturated fatty acid, palmitate might be one of the factors that regulates GPR120 mRNA level in the gonadotrope at the time of fasting.

## P3-g20 Molecular basis of associative eyeblink conditioning: New player involved in motor memory formation

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To clarify the molecular basis of motor learning, we investigated classical eyeblink conditioning by the combination of KO mice and pharmacological studies. We elucidated that the delay conditioning paradigm, in which the CS overlapped the US in time, is critically dependent on mGluR1 to PLC$\beta$4 signaling cascade in cerebellar PCs. Furthermore, we found that delay conditioning is severely impaired in KO mice deficient in cannabinoid CB1 receptor. (CB1R). Intraperitoneal injection of the CB1 antagonist SR141716A (rimonabant) also caused severe impairment of delay conditioning to wild-type mice, indicating that endogenous cannabioid signaling through CB1 is essential for acquisition of delay conditioning. In this study, the relationship between the mGluR1 and cannabinoid signaling pathways in motor memory formation was also evaluated.

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## P3-g21 The role of amygdala as a neural substrate for conditioned taste preference learning in mice

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We examined the neural mechanism of discriminative taste preference learning using the modified procedure of flavor-postingestive consequence learning paradigm in C57BL/6 male mice. Under a food restriction schedule, mice given bottle exposures of 0.15 M sucrose (Suc) paired with intragastric (IG) 0.5 M Suc infusion and those of 5 mM saccharin (Sacch) paired with IG water infusion with an alternative sequence showed more preference to Suc than Sacch, but the simultaneous IG infusion of 2 mM phloridzin dehydrate and 0.5 M Suc suppressed the development of the preference to Suc. Lesions of the amygdala with ibotenic acid impaired development of the preference for. These results suggest that the amygdala plays a pivotal role in discriminative association of the sweet taste quality of Suc with hedonically positive visceral signal of glucose absorbed at the intestine. .

## P3-g22 Altered levels of BDNF protein following prenatal auditory stimulation

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Prenatal sound stimulation results in early maturation of the auditory pathway and stimulation by species-specific sounds or sitar music enhances postnatal responsiveness and preference of the chicks to species-specific maternal calls, indicating the presence of perceptual learning. The hippocampus, associated with learning and memory, shows an increase in volume, neuronal size and number in music stimulated group. The proportion and expression of calcium-binding proteins are increased in these stimulated chicks. BDNF, important for neuronal survival during development, regulates synaptic strength. We studied the levels of BDNF protein during the development of hippocampus. From embryonic day (E)10 till hatching, the embryos were exposed to patterned species-specific sounds or sitar music at 65dB for 15 min/h round the clock (frequency range:100-6300). Results show a significant increase in the levels of BDNF protein at E16, E20 and PH1 in both the auditory stimulated groups as compared to the age-matched control. The increased levels of BDNF may impact the strength of synaptic plasticity.

DBT, India

## P3-g23 Hypobaric hypoxia induces neuronal damage and impair spatial memory in rat brain

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Hypobaric hypoxia impairs several physiological processes including cognitive function.The aim of present study was to investigate the duration dependent neuronal changes in hippocampus, prefrontal cortex and striatum of rat brain on chronic exposure to hypobaric hypoxia. Rats were exposed for 3, 7 and 14 days to simulated high altitude equivalent to 6100 m in an animal decompression chamber. Morris maze was studied for spatial memory testing. The Golgi staining was done to study the dendritic morphology. The histology including morphometry, neurodegeneration,TEM, caspase-3IR, and TUNEL staining were studied.There was significant increase pathlength and latency in hypoxia-exposed animals to reach the target platform. The Dendritic branching and spine number also decrease in hypoxia-exposed animals. The shrunken, pyknotic neurons were observed in hippocampal CA1, CA3 region,prefrontal cortex and striatum of hypoxia-exposed animals along with significantly increased numbers of dead cells. Hypobaric hypoxia induces degeneration and apoptosis.

Defence R&D Organization, Min of Def, Govt. of India

## P3-g24 Facilitation of brain plasticity during food intake

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During food intake glucose concentration in the blood plasma and cerbrospinal fluide becomes twice. Responding to this increase, 1) acidic FGF is released from the ependymal cells in the III cerebral ventricl, and 2) leptin released from the adipose cells and enters into the brain. These two substances first reached to the hypothalamus and suppressed food intake. Then both reached into the hippocampus, and facilitated learning and memory. Namely neurophysiologically long-term potentiation in the CA1 neurons was facilitated, and neurochemically the phosphorylations of CaMKII, PKC, ERK,GLUR1 etc. were faciltated. These phosphorylations were necessary for the facilitation of learnig and memory. The increase of glucose in the brain itself also faciltated learning and memory through these two factors. These redundancies occurred in the brain during food intake are the fundamental characteristics in the living body machinery.

## P3-g25 Dopamine D1 receptors in neural responses to rewards of amygdala and accumbens neurons

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The amygdala (AM) plays an important role in stimulus-affect association, and the nucleus accumbens (NAc) has been postulated as the limbic-motor interface. These two structures receive dopaminergic innervation. To investigate the involvement of dopaminergic systems in neural responses to rewards in association with place, we recorded neural activity from the AM and NAc in mice lacking dopamine D1 receptor (D1R-KO) and their wild-type littermates in spatial tasks. A lack of D1R eliminated the prereward excitatory response, which is suppressed by D1 antagonist SCH23390, in both the AM and the NAc. These results suggest the role of the D1R in neural responses of the AM and NAc in incentive stimuli.

Kakenhi (18700312)

## P3-g26 Distinctive neuronal networks and biochemical pathways in appetitive and aversive memory in *Drosophila* larvae

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In classical conditioning, animals learn association of originally neutral stimuli (conditioned stimuli, CS) with unconditioned stimuli (US), which elicit reflexive responses. Whereas associative strength between CS and US is thought to determine learning efficacy, little is know on the neuronal processes that underlie differential CS-US association in the brain. In *Drosophila* larvae, appetitive but not aversive training induces medium-term memory, which is dependent on *amn* and CREB, whereas both conditionings produce short-term memory depending on cAMP signaling. Neurocircuitry analyses suggest that, in either training, memory is stored in mushroom bodies and/or antennal lobes despite that neural output of octopaminergic and dopaminergic pathways, which exhibit distinct innervation patterns, are required for appetitive and aversive memory formation, respectively. These results suggest that genetically programmed US pathways involved in memory acquisition may determine different memory spans activating additional cAMP-dependent molecular components in the brain.

## P3-g27 Involvement of a *Drosophila* transient receptor potential ion channel gene *painless* in long-term courtship memory

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The transient receptor potential (TRP) channels play various roles in sensory transduction, but little is known about their physiological function in the brain. The *Drosophila painless* gene (*pain*) encodes a TRP channel of the TRPA subfamily. Interestingly, strong *pain* expression is observed in the adult mushroom bodies (MBs) that are involved in various learning and memory paradigms. Hence, we tested *pain* mutants for associative memory using the courtship-conditioning assay, where experience with mated females causes males to reduce their courtship toward virgins. *pain* mutants were defective in long-term courtship memory, which are rescued by wild type *pain* preferentially expressed in the mushroom body neurons. Furthermore, Flies expressing *pain* RNAi in the MBs showed the defective long-term courtship memory. Our results indicate involvement of the brain TRP channel in the behavioral plasticity.

## P3-g28 Comprehensive behavioral characterization of mice lacking ICER, an endogenous antagonist of CREB

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Inducible cAMP early repressor (ICER) is an inducible repressor of CRE-mediated gene transcription. ICER knockout (KO) mice were assessed using test battery. KO mice had similar body weight and home cage activity to wild-type littermates. While demonstrating unchanged behavior in the open field and elevated plus maze under dim conditions, KO mice showed lower locomotor activity in the brightly lit open field and in the light/dark test, taking longer time to enter the light compartment. KO mice performed better than wild-type littermates in the water maze task: shorter escape latency and path length, less time spent near the wall and better performance in the probe test. At the same time, KO mice did not differ from controls in tests on motor performance. Finally, KO mice showed tendency to freeze more during contextual fear test 24 hr after conditioning. The results may suggest roles of ICER in regulation of emotional state in anxiogenic situations and certain types of learning.

## P3-g29 Effect of perirhinal cortex lesion on relearning of recognition memory tasks with large and small sets of stimuli in monkey

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Behavioral studies in monkeys have demonstrated that relearning of recognition memory with a large set of stimuli is impaired by perirhinal cortex (PRC) lesion. However, it has not been known whether experience of recognition memory with a small set of stimuli prior the PRC damage gives impairments inrelearning of it with large and small sets of stimuli after the lesion or not. We examined this issue using 4 Japanese monkeys (*Macaca fuscata*) and 2 rhesus monkeys (*Macaca mulatto*) in WGTA. Prior PRC lesion, each monkey was trained on delayed matching to sample with two sets of stimuli, first with a large set of stimuli (300 pictured stimuli: DMS-300) and next with a small set of ones (3 pictured stimuli: DMS-3). After PRC ablations in 3monkeys, all monkeys were re-trained first on DMS-3 and next on DMS-300.After relearning of each DMS task, delay test (30, 60 and 120 sec delays) was given.As a result, no impairment was shown on relearning of the 2 DMS tasks and on delay tests.

※KAKENHI (14510115)

## P3-g30 Overexpression of ICER, an endogenous antagonist of CREB, impairs fear memory

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Inducible cAMP early repressor (ICER) is an endogenous antagonist of CRE binding protein (CREB). Negative feedback regulation of CREB activity by ICER might play an important role in memory formation process. Three lines of transgenic mice showing overexpression (O/E) of ICER constitutively in forebrain were examined. ICER-I line 23 mice (~125-fold O/E) showed impaired context fear memory tested 24 hr after conditioning, despite unaltered short-term context memory and tone-induced fear memory. These mice also showed unchanged general behavioral profiles. ICER-I line 15 mice (~80-fold O/E) did not exhibit any major changes in behavioural profiles examined. In contrast, mice overexpressing ICER-II, another isoform of ICER (~585-fold O/E), showed not only impaired long-term fear memory, but also altered phenotypes in a number of other behavioral tests. The results show importance of the negative regulation exerted by ICER for long-term fear memory, the effect that seems to depend on the O/E level of transgene.

## P3-g31 Dependency on the ipsilateral cerebellum in rat eyeblink conditioning

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The ipsilateral cerebellum to the trained eye has been reported to be essential for classical conditioning of eyeblink responses in rabbits. However, recent reports suggest that the ipsilateral cerebellum might not be essential in mice and humans. In this work, we examined roles of the ipsilateral and contralateral cerebellum in rats. They were conditioned for 7 days with a 150-, 250- or 500-ms interstimulus interval (ISI) in delay paradigm using a tone and a periorbital electrical shock. Then, they received aspiration of the ipsilateral or contralateral cerebellum. After more than three weeks, they were conditioned again for 10 days. Irrespective of the ISI, the ipsilateral group showed a severe impairment in retaining their conditioned response (CR). However, many of them reacquired precisely timed CRs during the re-conditioning. Contralateral cerebellar ablation in 500-ms ISI caused a similar but less impairment. These results suggest an involvement of the ipsilateral cerebellum in rats, which, however, can learn without it.

## P3-g32 In the rat, auditory short-term memory is better than visual short-term memory

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The visual acuity of rats is poorer than their auditory acuity. This study examined whether visual or auditory memory is superior in rats. Either a light stimulus (L) or a tone stimulus (T) was presented for 3 seconds as a discriminative stimulus (SD) in an operant chamber. Then, there was a delay of 0.25, 3, 6, or 9 seconds. After the delay, the rat had to press one of two levers depending on the SD:When L was presented, pressing the right lever gave a food pellet reward; when T was presented, pressing the left lever gave a similar reward. In order to perform this task correctly, the rats had to remember the SD during the delay interval. Therefore, performance of this task reflects their short-term memory (STM). When L was presented as the SD, the percent of correct responses declined as the delay interval lengthened. When T was presented as the SD, the decline in the percent of correct responses was slower than in the trials in which L was presented. These results imply that in rats STM is better for auditory information than for visual information.

There is no conflict of interest in this study.

## P3-g33 Signs of generalization of tool-use in monkeys

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Our monkeys had already mastered forceps-use for food-taking (Hirai et al J Physiol Sci Sup 2007). This study examined whether and how they would apply this acquired tool-using behavior to a new tool, a tapered stick with grip, where we gave no guidance-by-hand. Monkey's first response to a new tool was that he reached for but did not grasp it, indicating monkey distinguished stick from his familiar forceps. On day 2, however, he began to take the stick without regarding the ends (point/grip) and tried to stick food in vain. Because of repeated failure, monkey refused to act and brushed it off from the table. Then, an experimenter stuck food using the same kind of stick before his eyes. Initially, monkey observed experimenter's action to steal the food on the stick. Gradually, he grasped the grip end of the stick correctly with readiness and joined in sticking food with the experimenter. The results suggest that monkey became to have representation of tool-use after forceps-use training, and that observation of others' acts promotes this into actions.

## P3-g34 Learning deficit of OLETF in visual discrimination task

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The Otsuka Long-Evans Tokushima Fatty (OLETF) rat, an outbred strain of Long Evans Tokushima Otsuka rat (LETO), lacks CCK-1 receptor and is an animal model of type-2 diabetes. It has been reported that the OLETF was selectively impaired learning ability, depending on types of learning. In this study, we examined OLETF on visual discrimination task and effect of pioglitazone, peroxisome proliferators activated receptor-γ agonist. OLETF and LETO (18 weeks age, male, n = 10 each) were used as subjects. Subject's body weight and blood glucose level were measured. Half of each group was administrated pioglitazone (20 mg/kg/day) orally. All subjects were trained to discriminate illuminated key from two keys. OLETF showed significantly lower correct reaction ratio and slower reaction time than LETO. Effect of the pioglitazone was clear in body weight and blood glucose level but not in the learning indexes.

## P3-g35 Study of brain activation related to memory organization by Near-infrared spectroscopy

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The purpose of this study was to examine the change of oxygenated hemoglobin concentrations during memory organization task using Near-infrared spectroscopy (NIRS). Participants were ten healthy volunteers aged 20-26. Hemoglobin concentration changes were measured with an 103-channel NIRS instrument during the Japanese Verbal Learning Test (JVLT). Behavioral indices were the number of total words recalled and stimulus category repetition (SCR) based on the JVLT analysis. We examined the correlation between SCR and change of [oxyHb] in each brain region. As a result, activation in the left lateral prefrontal region was significantly positively correlated with SCR. These results suggest the left lateral prefrontal function has an important role in memory organization.

Core Research for Evolutional Science and Technology, Japan Science and Technology Corporation

## P3-g36 Detection of phase difference in random delay recurrent networks

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The spike timing, or the phase of oscillatory neuronal firing, has been regarded to play an important role for signal processing in a brain. In the present study we demonstrate a model for detection of phase difference between oscillatory neurons by using Hebbian learning. We use Fitzhugh-Nagumo type neuron model that have the both stable limit cycle and fixed point solutions in the neighborhood of a subcritical Hopf bifurcation. The coupling strengths are modulated by the firing of each neuron activity based on the Hebbian learning rule. We take the following procedure in numerical simulation: In the learning mode, we set the both input and detection neurons in oscillatory state to maintain the phase relation equal to the the learning phase difference and renew the coupling strengths. After leaning we examine the amplitude of the detection neuron, that is set the steady state, against the various phase difference between the input neurons. We get the results that the detection neuron is firing with the maximum amplitude when the phase difference between input neurons come close to the learning phase difference.

## P3-g37 Studies on neuronal function of a novel and conserved protein TTX-8

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*C. elegans* can memorize the food condition in relation to the cultivation temperature and migrate to the cultivation temperature when looking for the food. This response to temperature is called thermotaxis. Several neurons and genes required for thermotaxis have been identified, but molecular mechanism of thermotaxis is poorly understood.

The *ttx-8* mutants are defective in several behavior including thermotaxis. We found that *ttx-8* is expressed in many neurons and functions in several neurons responsible for thermotaxis. Since the predicted protein structure of TTX-8 is similar to RIC-3, TTX-8 might be required for the maturation of some membrane protein like RIC-3. (Halevi et al., 2002; Halevi et al., 2003). In fact, TTX-8 seems to localize to peri-nuclei region of cytoplasm. The FLJ10747, a novel human protein homologous to the *ttx-8*, weakly but apparently rescued abnormal thermotaxis phenotype of *ttx-8*, suggesting that FLJ10747 plays a similar role to TTX-8. Now, we are looking for the protein that interacts with TTX-8 by Yeast two-hybrid screening.

## P3-g38 Neuronal activation related to auditory memory in parrot brain

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Many songbird species learn their song early in life, and do not learn new song when they are adult. In contrast, adult males of the budgerigar (*Melopsittacus undulatus*, a parrot) can learn the vocalizations of their female partners (open-ended learners). Very little is known about the neural mechanisms of vocal learning in open-ended learners such as budgerigars. We investigated the forebrain expression of the immediate early gene product Zenk related to auditory memory in budgerigars. Males were exposed to a recording of their mate call (MATE), or to a novel call of an adult female (NOVEL) or kept in silence (SILENCE). There was a significant difference in levels of Zenk expression for the caudomedial nidopallium (NCM) between MATE and SILENCE, but not between NOVEL and SILENCE. It seems likely, therefore, that NCM is (part of) the neural substrate for the representation of the learned mate call, which is activated when the animal is re-exposed to that call.

NWO grant

## P3-g39 Genetic analyses of the sensory integration process in the neuronal circuits in *C. elegans*

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Sensory integration is essential for informational processing in nervous systems, but little is known about the molecular and cellular basis of the sensory integration. In *C. elegans*, HEN-1, a protein secreted from neurons, regulates the sensory integration in the neural circuit. To further analyze the mechanisms at the molecular and cellular levels, we screened new mutants showing a defect similar to the *hen-1* mutants. By using Mos1 transposon insertional mutagenesis, we isolated 6 mutants defective in the sensory integration process. Among them, the *gcy-28* mutant shows a defect in the integration of two sensory stimuli, although each response to the sensory stimuli in the *gcy-28* mutants appears to be the same as that in wild type animals. *gcy-28* encodes the transmembrane-type guanylyl cyclase and GFP driven by the *gcy-28* promoter is located in many neurons. We are currently identifying neurons in which GCY-28 regulates the sensory integration process.

## P3-g40 A suppressor screen for genes that regulate salt chemotaxis learning in *C. elegans*

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*C. elegans* is attracted to NaCl, but starts to avoid it after a conditioning with NaCl and starvation. We have shown that mutants of DAF-2 (insulin receptor) and AGE-1 (PI 3 kinase) showed defects in this form of learning. On the other hand, mutants of DAF-18 (PTEN phosphatase) showed reduced chemotaxis to salt even without conditioning. To identify genes that regulate salt chemotaxis learning, we screened for suppressors of the reduced chemotaxis of *daf-18* mutants.One suppressor mutant was found to have a missense mutation in *egl-30*, which encodes the a subunit of $G_q$. Previously known gain-of-function mutation of *egl-30* also suppressed the chemotaxis defect of *daf-18* mutants, raising the possibility that insulin-like signaling antagonizes the $G_q$ signaling. Another suppressor mutant was found to have a nonsense mutation in *gcy-22* receptor-type guanylyl cyclase. We are currently trying to determine the function of GCY-22 in salt chemotaxis learning.

## P3-g41 Involvement of IL-1 in LPS-induced learning and memory deficits

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We have designed the animal model of learning and memory deficits with the activation of microglia. Lipopolysaccharide (20 μg/2 μL) injected into the mouse hippocampus CA1 region activated microglia resulting in an increased production of inflammatory cytokines such as IL-1B and TNF α. In this animal model, increased expression of cytokines might trigger activation of microglia resulting neurodegeneration. Therefore, we demonstrated whether IL-1 and TNF α participate in degeneration of neuron and loss of neuronal function, especially learning and memory, using IL-1 KO mice and TNF α KO mice. When IL-1 KO mice were injected with LPS for 5 consecutive days, learning and memory deficits were not observed. Activation of microglia was not observed in the IL-1 KO mice, whereas in the TNF α KO mice, slight activation of microglia was observed and then learning and memory deficits were observed. These results suggested that expression of IL-1 has a important role for activation of microglia caused learning and memory deficits.

## P3-g42 Study of nicotine dose effects on temporal discrimination in rats

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Rats were trained by using the peak interval (PI) 30-s procedure that included fixed interval (FI) trials and probe trials. Thw PI procedure is the best method to investigate for timing behavior. Ten Wistar rats were trained for PI 30-s over 30 sessions. The 3-s bin of lever press responses on probe trials showed a clear peak point. The evaluation indices on temporal discrimination were the peak time, fitting regression ratio and the variation of distribution of the lever responses. The peak time corresponded to near the 30-s with reinforcement durations. Then a dose of nicotine was administrated to the rat by I.P. before PI 30-s session. The dose of nicotine administration varied for 0.0625, 0.125, 0.25, 0.5 and 1.0mg/kg. The 0.25mg/kg dosage is especially stable effects for time perception as saline treatment. The temporal distribution of responses for the other doses of nicotine tended to come down the behavioral peak pattern. It suggests that the dose response curve was like a reversal U shape on the time perception in rats.

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## P3-g43 Orexin attenuates the rewarding effect of intracranial self-stimulation in rats

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Anatomical findings imply the involvement of hypothalamic orexin neurons in reward system. We used immunohistochemical staining of orexin and Fos, therefore, to examine whether the orexinergic cells are activated during the intracranial self-stimulation (ICSS) of the medial forebrain bundle. ICSS increased the double-stained cells in the PFA region of lateral hypothalamus, but without any difference between sides ipsilateral and contralateral to the stimulation. In addition, intra-VTA orexin A injection (0.1, 0.5 and 1.0nmol) caused a clear and dose-dependent increase in the reward threshold of ICSS, an effect which is reversed by an orexinergic antagonist, SB334867. Our unexpected results suggest that orexinergic neurons in the PFA, which projects to the VTA, attenuate the rewarding effect of ICSS.

## P3-g44 Activity of MPK-1 MAP kinase regulates gustatory adaptation in *C. elegans*

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In *C. elegans*, well-fed animals show chemotaxis to NaCl, whereas after exposure to NaCl without food, animals change their behavior not to show the chemotaxis to NaCl by the sensory adaptation and other forms of sensory plasticity. Interestingly, the adaptation to NaCl is suppressed by the presence of food. We revealed that *mpk-1* MAP kinase mutants (loss-of-function) adapt to NaCl even in the presence of food. Mutants of the Ras-MAPK pathway also showed the similar behavioral defect as *mpk-1* mutants, suggesting that the Ras-MAPK pathway regulates the suppression of NaCl adaptation by food. Conversely, constitutive activation of MPK-1 disrupted adaptation after exposure to NaCl without food. By neuron-type specific and developmental stage specific expression of MPK-1, we revealed that function of MPK-1 in some inter- or motor neurons in the mature nervous system is important for the regulation of adaptation by food. These results indicate that MPK-1 might act in an integrative process of multiple sensory inputs in the nervous system, such as NaCl and food signals.

## P3-g45 CNG channel, calmodulin and CaMKII underlie olfactory long-term memory formation in the honeybee

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Several signaling molecules underlie long-term memory (LTM) formation in both vertebrates and invertebrates. Here we investigated the effects of inhibition of cyclic nucleotide-gated channel (CNG channel), calmodulin or CaMKII on olfactory learning and memory in the honeybee *Apis mellifera*. We used the olfactory conditioning of proboscis extension reflex (PER), a protocol in which bees learn that a given odor (CS) is reinforced with sucrose solution (US). Bees were conditioned by three spaced CS-US pairing trials that establish LTM 3 days after conditioning. Bees were injected with L-*cis*-diltiazem, a CNG channel inhibitor, with KN-62, a CaMKII inhibitor, or with W-7, a calmodulin antagonist. Injection of each drugs before 3-trial conditioning significantly impaired 3-day retention. Acquisition and 3-hour retention (medium-term memory test) were normal. These results suggest that the CNG channel, calmodulin and CaMKII participate in olfactory LTM formation in the honeybee.

## P3-g46 Involvement of the dorsolateral prefrontal cortex in inductive reasoning

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Reasoning enables animals to respond correctly in unexperienced situations. The purpose of this study was to examine the involvement of the dorsolateral prefrontal cortex (DLPFC) in inductive reasoning, which is regarded as the most intuitive and simple type of reasoning. We trained a monkey to perform a repeated group-reversal task, which requires inductive reasoning at the stimulus-reward reversal, using 8 discrete visual stimuli. During the performance of this task, we recorded single-unit activity from DLPFC neurons. Of neurons responding selectively to the visual cue according to the predicted liquid (either juice or saline), the majority reversed the stimulus selectivity when it was presumed that the monkey realized the stimulus-liquid reversal by inductive reasoning. We also found that some neurons show elevated tonic activity during the presumed inductive reasoning. These results suggest an important role of DLPFC neurons in inductive reasoning.

## P3-g47 Oscillatory beta activity predicts successful feature binding in face processing

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Oscillatory activity plays an important role in successful feature binding. Some recent studies reported that behavioral performance was predicted by brain activity before an event. In the present study, we investigated whether brain activity before an event can predict the successful feature binding in the processing of ambiguous face (Mooney faces). Participants judged whether the presented stimuli looked like face. EEG data were recorded from 32 electrodes and analyzed by wavelet based time-frequency analysis. We found that the beta activity just before the presentation of Mooney face in the trials in which the participant successfully bound the features and perceived the stimuli as a face was stronger compared to that in the trials in which the participant did not successfully bound the features. These results indicate the beta activity before the stimulus presentation plays an important role in feature binding in face processing.

## P3-g48 Synchronized cortical network activity of perceptual ambiguity: a magnetoencephalographic study

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Perceptual ambiguity such as ambiguous figure perception and binocular rivalry reflects the flexibility of our brain, because it produces fluctuating perception though an unchanging stimulus. Recently neural processes underlying perceptual ambiguity are suggested to be concerned with neuronal interactions among multiple cortical areas. In this study, we analyzed the magnetoencephalographic signals during the percept of bistable apparent motion to study the dynamics of brain activity during perceptual ambiguity. We applied Hilbert transform and cortically constrained minimum-norm estimation to localize the frequency-specific active sources and calculate phase locking values among these sources. As results, we detected significant frontoparietal-posterior networks in gamma (30Hz-50Hz) range during perceptual alternation. These results suggest that synchronized activities among frontoparietal-posterior networks play important roles in perceptual ambiguity.

KAKENHI18700265

## P3-h01 Neural mechanisms for temporal integration of the fluctuating component of a balanced input

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Temporal integration of information plays a crucial role in a variety of cognitive processes, such as sensory discrimination, decision-making or interval timing. However, neural mechanisms of this computation remain elusive. Here we propose a neural-network model that performs temporal integration of the fluctuating component of a balanced external input. We consider a uniform recurrent network of excitatory leaky-integrate-fire neurons. All the neurons are initially in the resting state ('off' state); if a neuron fires, then it moves to an active state ('on' state) where after-depolarization produces regenerative spike discharges. Each neuron receives a common external input that consists of excitatory and inhibitory bombardments, which generates a rapidly varying postsynaptic current. It is analytically or numerically shown that, if these excitation and inhibition are balanced, the time course of $n$, the number of on-state neurons, represents accurate temporal integration of the variance but not the mean of this current.

KAKENHI16500190

## P3-h02 Predictive control of saccadic eye movements during errors

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Inhibiting inappropriate responses and correcting errors are critical in enabling goal directed behaviour.To probe the nature of its control in relation to eye movements, subjects performed a REDIRECT double-step task in which they were asked to cancel the initial saccade to the first target and look at the second target when it appeared.Failure to inhibit resulted in an erroneous saccade to the initial target followed by a corrective saccade to the final target.To study the time course of error correction, the final target was shifted to a new location during the execution of errors.Corrective saccades directed at the location of the target prior to the shift support the idea of predictive error correction.The distribution of corrective saccade end points was used to estimate their preparation time and its relation with inhibition explored.A significant negative correlation between the time it takes to inhibit a planned saccade and the rate at which the decision to correct evolves for a subject, implies that predictive error correction may occur as a consequence of the brain's capacity to predict the likelihood of an error as it is trying to inhibit an unwanted movement.

DST

## P3-h03 Does covert inhibition potentiate online inhibitory control?

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A planned action awaiting execution requires withholding a prepared response. We asked whether such a form of inhibitory control would interact with online decision processes that change a planned response. To study this issue with respect to oculomotor control, subjects performed visually (VG) guided, memory guided (MG) and delayed visually guided (DVG) saccades. In contrast to the VG task, subjects had to withhold a prepared response during the MG and the DVG task. In all three tasks, on a random fraction of trials a second target was presented after a variable delay, which instructed subjects to redirect their gaze to the newly specified target. The time taken to cancel a saccade, the target switch reaction time (TSRT) was calculated using a race model. We observed that although the reaction times were same for all the three tasks, the average TSRT was found to be significantly smaller for MG and the DVG compared to the VG task. Since, both MG and DVG require withholding a prepared saccade, a lower TSRT indicates that a tonic level of inhibition interacts with online decision processes to potentiate inhibitory control.

## P3-h04 Model-based reward prediction in the primate prefrontal cortex

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Several brain areas are involved in reward processing, including the middle brain, striatum and prefrontal cortex (PFC). It is not clear whether those areas have different functions for reward prediction. One hypothesis is that subcortical neurons apply model-free reinforce learning (eg: TD learning), while PFC uses model-based rule (eg: tree search method). In order to examine the PFC mechanism for predicting reward, we trained monkeys a sequential association task with asymmetric reward. They learned two sequences of stimuli: A1-B1-C1 and A2-B2-C2. An asymmetric reward rule was instructed by pairing C (C1 or C2) with a reward block by block. Out of 373 neurons from the lateral PFCs, 32% showed reward-related activity in the first cue period. These neurons showed reward-predicting activity for the stimulus A after paring C with reward. Furthermore, PFC neurons predicted A2 would be unrewarded if they experienced that A1 was rewarded, and vice verse. These results could not be explained by classical condition, but are consisted with predictions from the model-based reinforcement learning.

## P3-h05 Differences in representation of emotional information among nuclei of monkey amygdala

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The amygdala has been implicated in emotional processing. This brain area consists of several nuclei. To clarify functional differences of the nuclei of the amygdala in emotional processing, we compared the responsiveness of neurons among the lateral, basal, and central nuclei of the monkey amygdala. The activity of 227 neurons was recorded from several nuclei of the amygdala while presenting video clips of 13 stimuli belonging to 3 categories (monkey, human, and object). Of these, 37, 39, and 37 neurons in the lateral, basal, and central nuclei, respectively, responded to at least one of the stimuli. We applied a principal component analysis and a cluster analysis to the responses of population neurons in these nuclei. We found that the stimuli can be categorized by neuronal responsiveness according to 3 categories more properly in the central nucleus than in the lateral and basal nuclei. The results suggest that emotional information is highly processed in the central nucleus of the amygdala.

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## P3-h06 Cholinergic signal in the striatum during multi-step choice task for reward

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To study the role of cholinergic interneurons in the striatum in reward based decision making, we recorded activity of 149 tonically active neurons (TANs) of 2 Japanese monkeys performing choice 1 of 3 target buttons in a trial-and-error basis. Correct choices were followed by high-tone beep and reward water, while incorrect choices were followed by low-tone beep without reward. Correct choice rates at first (N1), second (N2), third (N3) choices were 33, 50, 85 %, respectively. Once correct button was hit, monkeys got one more reward by choosing the known button (R1, 95 %). TANs responded to start cue for each trial with similar magnitudes in every trial type. They responded to the beeps in N1, N2 and N3 trials with similar magnitudes, but showed almost no responses at R1 trials. TANs differentiated correct and incorrect beeps in terms of temporal pattern of responses. The selective activation of TANs by action outcome during trial-and-error search trials suggests involvement of cholinergic interneurons in learning through reinforcement process in the striatum.

KAKENHI17022032

## P3-h07 Performance of monkeys in a repeated group-reversal task requiring inductive reasoning.

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We trained 3 monkeys to perform a repeated group-reversal task with a stimulus set of 8 abstract figures. In every trial, a stimulus was presented to the subject, and then the associated liquid, either juice or saline, was delivered through a spout in front of the mouth. The correct response to the stimulus was to lick if it was associated with juice, and to withhold lick if it was associated with saline. The stimulus-liquid associations (4 of 8 stimuli associated with juice, and the rest associated with saline) were constant within a session of 48-96 trials, and between sessions, they were reversed without any explicit cue. After an extensive training of this group-reversal, the subjects learned to reverse their response to the stimuli after making a single error. This behavior, correctly responding to unexperienced reversals in 7 stimuli after experiencing a reversal in one stimulus, may suggest that the monkeys have ability to perform inductive reasoning, that is, to generalize an experience with a certain stimulus to the other stimuli of the same kind.

## P3-h08 Brain magnetic responses to biological motion

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With only about 10 dots representing joints movements, we can recognize specific human motion that is known as biological motion (BM). The objective of this study is to disclose a detailed spatio-temporal profile of the information processing related to BM. We measured visual event-related magnetic fields following BM and scrambled motion (SM) in 11 healthy volunteers using magnetoencephalography. Brain surface current density maps were calculated by minimum-norm estimates, and then time courses of the regional brain electrical activity around the bilateral posterior superior temporal sulci (pSTS) and middle temporal areas were visualized by ROI analysis. Results demonstrated that only the right pSTS showed significantly larger activation by BM than by SM (ANOVA, $p<0.01$) in the latencies around 150-200ms. However, there were considerably big inter-individual variations in the hemispheric predominance for BM processing. These results suggest that pSTS plays an important role in processing BM.

## P3-h09 Representation of reward probability in the rat striatum

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To investigate how the reward probability is coded in the striatum, we performed single-unit recordings from striatal neurons in head-restrained rats, which were conditioned in a Pavlovian procedure with distinct visual or auditory stimuli indicating the distinct reward probability. Of all striatal neurons recorded, 38% (72/189) responded to the conditioned stimuli (CSs), and 38% (27/72) of these showed probability-dependent responses. Of these neurons, 81% (22/27) showed positively correlated responses, and the rest showed negatively correlated responses. Most (89%, 24/27) of neurons showing probability-dependent responses to the CSs also responded at the timing of the reward delivery; and the majority (92%, 22/24) of these showed similar responses to reward regardless of the probability indicated by the CS. These results indicate that neurons in the striatum code reward probability similarly to the midbrain dopaminergic neurons, which code reward prediction error and project to the striatum. On the other hand, the manner of responding to the reward itself was different.

## P3-h10 Effect of catechol-*O*-methylferase (COMT) Val$^{158}$Met genotype on encoding, maintenance, and retrieval of visual working memory

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COMT is related to the efficiency of prefrontal cortex (PFC) activity. We identified whether polymorphism in the COMT (Val$^{158}$Met) gene affects brain activity during encoding, maintenance, or retrieval periods. We recruited 20 healthy participants from 70 adults: Ten were Met carriers and the other ten were Val/Val carriers. They were asked to memorize a visual pattern in the fMRI experiment. Memory accuracy was greater for the Met carriers than for the Val/Val carriers. We found activations in the PFC, anterior cingulate cortex, supramarginal gyrus, parietal areas, and visual areas during encoding period. Signal intensity of the PFC during encoding period was greater for the Val/Val carriers than for the Met carriers. These results suggest that the Met allele leads to more efficient activity in the PFC during encoding of memory than during maintenance and retrieval of it.

## P3-h11 Spatial activation patterns of spatial and non-spatial visual information in primate DLPFC

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We investigated how spatial and non-spatial information are represented within DLPFC and how these representations change along the progress of the trial by using three tasks. Each task required monkeys to make a saccade toward a target based on a spatial or non-spatial (color) visual cues. We made expanded flat cortical maps of DLPFC, and plotted the position of each neuron and the magnitude of each single-neuron activity on the expanded flat cortical maps. Although three tasks required monkeys to make a same oculomotor output, we found that spatial activation patterns by color cues were different from that by spatial cues. We also found that those by oculomotor outputs were different from those by visual cues. These result indicated that DLPFC may play a role in integrating various kinds of information to produce particular motor outputs.

17300103 to S. F.

## P3-h12 Prefrontal activities under a go/no-go conditional discrimination task

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There are two major functions related to attention: selective and divided attention. Although the neuronal basis of selective attention has been investigated systematically (Sakagami 1994a, b; Sakagami & Tsutsui, 1999), neural underpinning of the divided attention remains unclear. We examined neuronal activity in the lateral prefrontal cortex (LPFC) while the monkey performed a divided attention task -- a go/no-go conditional discrimination task that required the monkey to make a go or no-go response depending on the combination of the features present in a random-dot color kinematogram as a cue. The critical features of the kinematogram were color and motion direction and the monkey had to pay attention to the specific combination of these two features to make go/no-go decisions. We found the neurons that showed differential activity for go and no-go stimuli in the wide range of LPFC, but the differential activity in the dorsal part started earlier by 300-400 ms than in the ventral part. These results suggested that the dorsal part of LPFC has more critical role for the go/no-go decision in this task situation.

## P3-h13 The effect of random delay length on prefrontal delay-period activity during free-choice ODR tasks

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Our previous studies showed that prefrontal directional delay-period activity contributes to the selection of the forthcoming saccade direction during the 3-s delay period in the free-choice ODR task (S-ODR1). In the present study, we introduced a task with variable delay length (1.5, 3 or 4.5 sec: S-ODR2). In both tasks, 4 identical visual cues were presented in the cue period. Monkeys were required to make a saccade to any one of 4 directions after the delay. The mean saccade latency and error rates were not affected by the delay length in S-ODR2, suggesting that the monkeys were ready to make saccades before the end of the delay, even in the 1.5-s condition. In concert with the behavioral data, single-trial based analysis of neuron activity showed that the onset latency of delay-period activity in S-ODR2 tended to be shorter than that of S-ODR1. A comparison of delay-period activity between two tasks suggests that directional delay-period activity reflects the selection process of saccade direction in S-ODR tasks.

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## P3-h14 Internal model of the human hand affects object recognition

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In this study, we investigate a relation between an internal model of the human hand and object recognition of graspable tools. Object concepts acquired through motor experience make object recognition possible. Such a mechanism seems to relate to the internal model acquired through motor experience. Thus, we examined whether the novel internal model trained under the condition that changes a displayed hand shape on a monitor affects object concept and/or object recognition. In the first experiment, the internal models were trained under the three conditions: observation, passive movement and active movement. As a result, active movement plays an important role for the learning. For the second experiment, we found that object concept is influenced by the novel internal models, although visual image of a favorite glass is independent from the models. In the third experiment, we ascertained that the novel internal model affects object recognition such a glass. Finally, we conclude that an internal model of the human hand plays an important role for object recognition of graspable tools.

## P3-h15 Auditory evoked N1m latency reflects categorical perception of timbre

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To clarify the cortical representation of timbre perception, we recorded neuromagnetic responses to periodic complex sounds with equivalent acoustic parameters except for 2 different fundamental frequencies and 12 different spectral envelopes, which were categorized into vocal, instrumental and linear shapes. We analyzed N1m peak latency and N1m offset latency, which gives first minimal RMS values after the N1m peak. No significant difference was detected in the N1m peak latency across timbre categories, while the offset latency for the instrumental sound was significantly longer than that for the linear sound. These results suggest that the duration of auditory information processing may be affected by the shape of spectral envelopes and this process may be reflected in the duration from the N1m peak to the offset.

## P3-h16 Cue response of monkey striatal neurons during a duration discrimination task

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To clarify neural mechanisms for time perception, neuronal activity was recorded from the monkey striatum during a duration discrimination task. A monkey was presented two visual cues (the first cue, C1 and the second cue, C2), blue and red-colored squares, successively for different duration. Each cue was followed by a delay period. After the second delay period (D2) the subject was required to choose the longer-presented colored cue. Fifty-nine neurons responded to C2, and only 5 neurons responded to C1. Of the 59 C2 response neurons, 20 neurons responded to C2 with a constant latency after C2 onset in short-long trials. These neurons increased their firing rates during D2 with a short latency after C2 offset in long-short trials. The C2 and D2 activities were associated with trial types, but not necessarily correlated with C2 duration in each trial type. This result suggests that the striatum neurons are involved in temporal discrimination between two cue durations during the second cue presentation.

KAKENHI18500314

## P3-h17 Organization of multisynaptic inputs from parietal cortex to prefrontal cortex in macaque monkeys

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To investigate the organization of multisynaptic inputs from the parietal cortex to the prefrontal cortex, we injected rabies virus into the dorsal (d) and ventral (v) parts of area 46 in macaque monkeys. Three days after the viral injections, second-order neurons were retrogradely labeled in transynaptic fashion. Neuronal labeling occurred in the medial and lateral banks of the intraparietal sulcus (IPS). The neurons labeled from area 46d were distributed mainly in the caudal portion of the IPS, whereas those labeled from area 46v were in the rostral portion. These results, together with our previous data showing the differential disynaptic projections from the inferior temporal area to areas 46d vs 46v, indicate that there are two distinct pathways responsible for information outflow from the parietal and temporal association areas toward area 46.

## P3-h18 Independence of percepion and action by using a task change strategy

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In this study, we investigate the independence of perception and action for grasping movement. Several researchers reported that action is not affected by an illusion, although perception is affected by an illusion. However, the independence may not be investigated directly, because these studies use illusion tasks. Thus, we focused on grasping movement in daily life. In the first experiment, by using different objects, we measured grasping positions with three tasks: visual estimation task (VT), pinch task (PT) and lift-up task (LT). As a result, even after iterative trials of LT, grasping positions of VT are different from those of LT and moreover VT is the same as PT amazingly. For the second experiment, we ascertained that visual information during prehension movement doesn't affect the grasping positions. In the third experiment, We found that a task change from PT to LT can't be executed during movement and also grasping position and the task change are independently calculated. These findings are quite important for investigating a mechanism of perception and action in the brain.

## P3-h19 Laterality of human prefrontal cortex function during motor inhibition

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Abrupt inhibition of initiated movement is an important piece of human motor control. Typical experimental paradigm to quantify motor inhibition is Stop-signal task which may reflect the prefrontal function. Although the right side dominance of prefrontal cortex for motor inhibition has been suggested, the exact laterality is not known. We examined the brain activity during Stop-signal task by fMRI and analyzed the laterality of activation using SPM5 in ten healthy subjects. In Go trials (75%) one reacts to Go cue on the screen and presses the button on left or right hand which the cue arrow directs to. In Stop trials (25%), one reacts to Stop cue following Go cue and inhibits the movement. Event-related analysis of fMRI revealed that the right prefrontal cortex is more activated than left in successful inhibition even when the right and left hand Stop trials were analyzed separately. This indicates the functional relevance of the right prefrontal cortex in inhibition of initiated response.

## P3-h20 Effects of isolation rearing on the development of social behavior and central neurotrophin levels in male Mongolian gerbils

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We investigated influences of rearing conditions on development of social behaviors and central neurotrophin levels in male Mongolian gerbils. Subjects were divided at weaning into three groups; the isolation-reared (IR), the group-reared (GR), and the screen-divided-reared (SDR) males, which were housed in a pair but separated individually by a wire mesh screen. Social interaction tests revealed that IR and SDR males increased time spent in the investigation into unfamiliar males, but the increase of aggressive behaviors was observed only in IR males, not in SDR and GR males. This suggests that distant sensory interactions with another individual reduce isolation rearing-induced aggression. However, no significant difference among groups was found in NGF and BDNF levels in four brain areas at different developmental stages, which implies that other neurochemical factors would be involved in the mechanism underlying isolation-induced behavioral alternation.

## P3-h21 Genetic analysis of inter-male aggression using consomic mouse strains established from C57BL/6J and MSM

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Aggression is very important emotion for animals and evolutionarily conserved behavior. Recent studies with gene-altered mice have successfully elucidated several genes related to aggressive behavior. However, the attempts to identify naturally occurring genetic variation related to aggressive behavior have not been sufficiently done yet. We here report the forward genetics approach for inter-male aggression by using new genetic resource, consomic mouse strains. We found male of one consomic strain B6-15MSM, which have MSM chromosome 15, showed increased aggressive behavior in resident-intruder test. Behavioral analysis showed that the increased aggression of B6-15MSM was mainly caused by the effect of intruder. Several congenic strains of B6-15MSM were established to identify the genetic locus/loci related to the aggressive behavior, and revealed that there are several genetic loci that increased or decreased the aggressive behavior on the chromosome 15.

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## P3-h22 Soldier-specific neural modification in termites

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Social insects possess highly organized social system that is accomplished by elaborate division of labor. To increase the colony performance, colony members differentiate into various castes that show morphological and behavioral differences. For example, in termites, soldier castes show aggressive defense behavior with well-developed mandibles. The comparison of CNS between soldier and worker indicates that soldiers have larger suboesophageal ganglia than workers. Histological observations showed that the mandibular motor neurons were enlarged in subesophageal ganglion of soldiers. The enlargement of neurons should cause effective defense behavior by increasing nerve conduction velocity and amount of neurotransmitters. The soldier-specific giant motor neurons was shared by all examined termite species, suggesting that this characteristic was highly linked to soldier defensive behavior and acquired in the common ancestor of termites. We also analyze differential gene expression in the CNS to reveal molecular basis underlying the social behavior.

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## P3-h23 ADP-ribosyl cyclase activity and oxytocin levels in relation to mouse maternal behavior

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Oxytocin has been extensively implicated in the initiation of contact between mother and pups, resulting in the expression of maternal behavior. Maternal behavior of primiparous females and experienced mothers is not same. Therefore, we addressed the question whether or not good maternal behavior along experience is based upon ADP-ribosyl cyclase activity in the hypothalamus and plasma or tissue oxytocin levels.We prepared membrane fractions from the hypothalamus and fluorometrically measured ADP-ribosyl cyclase activity. It was observed that the mouse hypothalamus has the highest ADP-ribosyl cyclase activity in comparison with the pituitary, cerebrum or cerebellum tested. According with this result, the expression level of CD38 mRNA was also the highest in the hypothalamus, which is roughly identical level as that in the spleen. We show the close relationship between the cyclase activity and maternal behavior.

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## P3-h24 CD38-and cyclic ADP-ribose-independent vasopressin secretion in social behavior of mice

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Oxytocin(OT) and vasopressin(VP) are nonapeptides, synthesized in the hypothalamus, stored in the posterior pituitary and released into the blood stream. Apart from the physiological effects, recent studies have shown that OT and VP also play an important role in social behavior throughout a wide range of animal species. CD38, a glycoprotein with a ADP-ribosyl cyclase activity. CD38 catalyses NAD to produce cADPR, which is considered as a potent second messenger to regulate intracellular calcium concentration by CICR mechanism. Thus, we focused on whether CD38-cADPR-[Ca2+]i pathway participated in OT and VP release, and further affect social behavior. Here, we demonstrated that VP secretion was not enhanced by cADPR from isolated hypothalamic cells and their nerve endings, though OT release was increased by identical treatment in wild-type mice. We also showed that the correlation between plasma or CSF levels of VP and social behavior.

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## P3-h25 Decision-making of the common marmoset in gambling task

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To analyze decision-making under risk of monkeys, 17 common marmosets were trained to select and remove one of two colored caps on vertically-arranged wells in front of them. Each well contained constant reward (3 grains of sugar-coated rice) or risky reward (0 or 6 grains; possibility, 50%:50%). For each marmoset, white or black color were determined randomly as symbols of constant or risky choice. Arrangement of white and black caps were determined randomly in each trial. After 200 trials (5 trials per day), the marmosets were classified according to the pattern of their selection. Seven of 17 marmosets (41.2%) were risk-aversive, while 5 marmosets (29.4%) were risk-prone. Remaining 5 marmosets (29.4%) became to select one side constantly (left n=4, right n=1). These results showed individual difference in decision-making of the marmoset. Expected value of the number of reward grains did not depend on selection strategy of marmosets in the present experiment. Thus, constant selection of one side was interpreted as reasonable way to reduce brain energy consumption.

## P3-h26 Spontaneous categorization of social behaviors in primate prefrontal cortical neurons

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In group-living primates, visual information of social behaviors is important for making adequate social interactions. Since the lateral prefrontal cortex (LPFC) has been implicated in coding of faces/vocalizations that are critical for social behaviors, it is likely that visual information of social behaviors is processed by LPFC neurons. To address this problem, we examined neuronal activities in the LPFC of two monkeys that were fixating on various animation-stimuli (4-8 s in duration) of two types of social behaviors (grooming, mounting) or non-social behavior of plural monkeys. We found that a large proportion (about half) of LPFC neurons responded preferentially to a particular type of social behaviors, even though the subject was not required to recognize or memorize it. These findings suggest that many LPFC neurons categorize social behaviors spontaneously. Such a categorization by LPFC neurons should be a critical neuronal substrate of the social cognition, and the LPFC may be a "social cognition area".

## P3-h27 Premotor neurons related to pointing behavior

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Pointing the arm toward a desired object is an important behavior to request it. Here, we examined the relationship between macaque premotor (PM) neuronal activity and pointing behavior. Two types of tasks were developed. One was Food-pointing (FP) task in which the monkey pointed at the most desired one among three kinds of foods (sweet potato, apple, and carrot) to request it and then pointed at the second most. Another was LED-pointing (LP) task in which the monkey had to point at a certain LED to get reward. Of 112 PM neurons recorded, 60% (67/112) fired during either or both of the tasks. Of these, 34% (23/67) were more active during the perimovement period in the FP task than in the LP task. 13% (3/23) exhibited stronger responses when the monkey pointed at the most desired food than at the second most. The present results suggest that the PM is involved in pointing behavior.

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## P3-h28 The right temporal region contributes to the self-prospective direction of stress coping style

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In this fMRI study, we examined the brain mechanisms underlying stress coping styles, that is a direction of demand (Self vs. Other) and a direction of view (Prospective vs. Retrospective). We focused on the Self Prospective style as a common goal of psychotherapy. Thirty two healthy subjects performed a pretend task. They acted the protagonists of the stressful situations shown in pictures, and evaluated the degree of how spontaneously each response could be acted (Spontaneity). A significant negative correlation between the Spontaneity and neural responds for the Self Prospective condition was observed in the right temporal pole. The results indicated the advantage of the low response in this region to the stressful situation for adaptive social behavior.

## P3-h29 Prefrontal cortex activities in the successful inhibition of habitual responses: an fMRI study

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In our daily life on some occasions, habitual responses have to be well suppressed, and it is known that the patients with lesions in the frontal cortices often have difficulties in such suppression. The rock-paper-scissors game is known as an example of the familiar habitual behaviors, and in this study we measured fMRI signals to investigate how the frontal cortex activities change corresponding to the subjects' performance when they try to lose (successfully inhibit to win) against a presented gesture of a rock, paper, or scissors (n = 36). Their successful inhibition rate ranged from 50 to 100 % in a block of 4 experimental trials, and we found that the activations in the left superior frontal gyrus (Brodmann area 10) were parametrically increased corresponding to the subjects' successful inhibition ($P<0.005$, uncorrected). This result suggests that the left superior frontal gyrus plays a role in the successful inhibition of the habitual responses.

HLSRG(H16-014)

## P3-h30 Building a good reputation in others' eyes: the neural basis of the self-presentation motivation

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Everyday experience and theoretical research on the evolution of human cooperation suggest that establishing a good reputation is important. However, the neural basis of the motivation to achieve a good reputation remains unknown. By measuring brain activity using fMRI while subjects present themselves in front of other people, we found that dorsal striatum activity was specifically associated with the motivation to gain a good reputation and co-varied with mPFC activity. Moreover, an individual's self-report tendency to behave in a socially desirable manner predicted activities in both of these regions. Our findings suggest that dorsal striatum and mPFC constitute a *social reward system* that motivates individuals to seek a good reputation and play a key role in regulating our social behavior.

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## P3-h31 Neural mechanisms for preference-based decision making

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We speculate that neural mechanisms for positive emotions overlap with those for preference-based decision making. Using fMRI, we attempted to uncover how this preference for certain things over other things, focusing on the commodity preference, is represented. Forty-four healthy female college students answered questionnaires, in which 560 commodity brands from 100 categories were given with their logos. Based on the results of them, 80 pairs of preferred and neutral brands were determined. 80 pairs of positive and neutral pictures were also selected from the International Affective Picture System (IAPS). 18 subjects participated in the fMRI study. In test task, each subject chose her preferred brand. In control task, she chose the logo judged as visually more striking. In brand-logo sessions, parahippocamal gyrus, midbrain, dorsolateral prefrontal cortex and cerebellum were activated. In IAPS sessions, activated areas were similar to those in brand-logo sessions, but appeared more extensive and included amygdala.

## P3-h32 Ventrolateral prefrontal response to taste stimuli

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The role of the lateral prefrontal cortex (LPFC) in taste processing is gradually drawing attention. In our previous taste studies using multi-channel functional near-infrared spectroscopy (fNIRS), we have noticed prefrontal activity in response to taste-related stimuli even when no cognitive task is performed. To examine the observed activity further, we conducted a meta-analysis of our previous fNIRS data from 32 subjects (of which, 15 participated in two experiments). We found significant activation centering at inferior prefrontal gyrus (IFG) in both hemispheres in both taste and flavor tasting conditions. The cortical response did not show any significant correlation with subjective rating scores to the stimuli (intensity, familiarity, and hedonisity) at group level, but at the subject level, some subjects showed strong correlation to some of these rating scores. Our study suggests that, in addition to dorso-LPFC reported by Kringelbach, et al. (2004), IFG plays certain role in taste-related intra-oral stimulus processing. This work is supported by Grant-in-Aid for Young Scientists (B) 18700625 from MEXT of Japan.

## P3-h33 An fMRI study: Processing of item-situation relationship

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This study examines the cortical mechanism of monitoring item-situation relationship and detecting irrelevant items (Ir). Healthy subjects judged whether a presented item was relevant to a situation or not. As items, object or behavior pictures were presented in separate sessions (O or B sessions). Each control task (Co or Cb) was detection of inverted item pictures. Greater activation during O/B than Co/Cb was analyzed to identify the activation for monitoring item-situation relationship and neural responses to Ir were analyzed. Activation during O and B was observed in several frontal, temporal regions and the posterior cingulate cortex. The right lateral prefrontal cortex was activated during B only. Activation for Ir was observed in the left lateral orbitofrontal cortex during O, and in the medial prefrontal cortex, and the left prefrontal cortex during B. No common activation was identified between them.

## P3-h34 5-HT2A receptor gene polymorphism modulates activation for monetary reward in the human ventrolateral frontal lobe during Go/No-go task

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We examined whether the serotonergic system plays an important role in human impulsive behavior formonetary reward by measuring how 5-HT2A receptor gene polymorphism (A-1438G in the promoter region)affects inhibitory motor control in a Go/No-go task using fMRI. Twenty seven participants were instructed to learn to respond to Go stimuli and inhibit their response to No-go stimuli under boththe reward-only (R) condition and the punishment-only (P) condition. During the No-go trials underthe R condition, activation of the right ventrolateral frontal lobe was higher in participants for the AA allele than in participants for the GG allele. In contrast, under the P condition, the rightamygdala activation was higher in participants for the AA allele than in participants for the GGallele.

## P3-h35 Social transmission in avoidance learning is dependent on learning experience in rats

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Social transmission, transmitting one's individual experience to others, has been shown in certain learning paradigms such as bar-press learning or spatial learning in which naïve unlearned animals receive facilitative transmission from learned animals. Social transmission can be reciprocally elicited between learned and unlearned animals but detail is unknown. In the present study, we evaluated social transmission in learned animals affected by other unlearned or learned animals in the passive avoidance test. Male Wistar rats at age of 9-10 weeks were used. Retention time to stay in the light room to avoid entering the dark room was measured with or without coexistence of other individual. Avoidance responses of learned rats were sensitively suppressed by existence of naïve rats and facilitated by learned behavior of other individuals. The performances of unlearned rats were not affected by other learned or unlearned animals. The results suggest that social transmission depends on learning context and facilitated by learning experiences.

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## P3-h36 The Urbilaterian genes implicated to underlie the evolution of advanced brain functions

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Both honey bees and mammals develop many complex behaviors to maintain their social organizations. These behavioral traits are based on the brain functions, however, the brain structures of honey bee and mammal are dramatically different. This raises a question how the advanced brain functions have evolved without sharing the apparent structural similarity. To give insights into this question and to identify the genetic features underlying the advanced brain functions, we carry out comprehensive analysis on the genes shared between vertebrates and honey bees but not in other sequenced Ecdysozoan genomes (HVS genes). Our results suggest that the significant fraction of the HVS genes may underlie the advanced brain functions by increasing the network of gene interactions. The identification of HVS genes should provide a new venue to study the origin and evolution of brain functions in a variety of metazoans.

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## P3-h37 Enhanced susceptibility to MPTP neurotoxicity in magnesium-deficient C57BL/6 mice

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Magnesium (Mg) deficiency may be associated with stress-related disease. We hypothesize that Mg deficiency plays a critical role in the stress-induced exacerbation of the pathogenesis in Parkinson disease. To clarify this possibility, we investigated the effect of 1-methyl-4 phenyl-1,2,3,6-tetrahydropyridune (MPTP) in C57BL/6 mice fed a Mg-deficient diet. On the 3 weeks of Mg depletion, Mg-deficient mice displayed depression-like behavior in the forced swim test. MPTP (15 mg/kg) treatment decreased dopamine transporter protein levels in the striatum and increased glial fibrillary acidic protein in Mg-deficient mice but not in control mice. These results indicate that Mg deficiency increased vulnerability to MPTP neurotoxicity in C57BL/6 mice.

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## P3-h38 Preventive effects of phytoestrogens on the paraquat-induced toxicities in the cellular model of Parkison disease.

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Epidemiological studies reported that paraquat, a hervicide, is one of the environmental risk factors of Parkinson disease. Paraquat induced apoptosis in PC12 cells. The endoplasmic reticulum (ER) stress was found to be involved in this paraquat -induced apoptosis because upregulation of CHOP and activation of Jun-N-terminal kinase (JNK), ER stress activated substrates, were observed. Upregulation of mRNA of BiP, an ER-specific chaperone, and splicing of the mRNA of the X box binding protein were observed, indicating the occurrence of the unfolded protein response (UPR). Prominent production of reactive oxygen species (ROS) was observed after paraquat exposure. Pre-incubation of phytoestrogens such as resveratrol prevalent in the red wine and genistein derived from soybean reduced the paraquat -induced ROS. Resvertrol reduced the UPR and increased the viability of PC12 cells was increased. In contrast, genistein was less effective to prevent the ER stress and the paraquat-induced cell death.

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## P3-h39 Quantitative relationship of parkinsonian thalamic β-band activities with rigidity

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Excess β-band activities are observed in parkinsonian thalamic ventrolateral nucleus (VL). We examined how these activities were related with rigidity alleviated after the thalamotomy. Rigidity on the UPDRS and postoperative reduction (dR) were estimated in 100 patients with PD and 9 patients with essential tremor divided into six groups of rigidity preoperatively scored 0 (N), 0.5 (Ia), 1 (Ib), 2 (II), 3 (III), and 4 (IV). Filtered β-band local field potentials (β-waves) were quantified by the mean time integral (mβ) of 13-27 Hz wavelets in percent of 3-sec sample records. After the surgery, rigidity disappeared in groups Ia, Ib, and II, and remained partially in III or heavily in IV. The VL mβ was progressively increased through N, Ia, Ib to II, and decreased in III to IV. The mβ-to-dR relationship was positive, but non-linear of histeresis nature. The histeresis may arise by the time lag from the pathology represented by β-waves to the expression of rigidity. The VL mβ represented only a part of severe rigidity, indicating that the late pathology involved other sites beyond the VL.

## P3-h40 Neurosin cleaves the NAC region of α-synuclein

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Background: Neurosin is one of serine proteases predominantly expressed in a central nervous system. Neurosin is presumed to play an important role in metabolism of α-synuclein (α-syn), because the previous report showed that neurosin exists in Lewy bodies and degrades α-syn in vitro. However the details of α-syn degradation by neurosin are little known.
Method: Neurosin-mediated cleaving sites were investigated by LC/MS/MS. Degradations of wild and mutant α-syn by neurosin were evaluated by SDS-PAGE.
Findings: The dominant cleavage site was between Lys80 and Thr81. Neurosin digested A30P mutant α-syn less efficiently than the wild type one.
Interpretation: The dominant cleavage site we determined is the center of the NAC region, which is responsible for polymerization of the α-syn. This result suggests a hyothesis that neurosin inhibits the polymerization of α-syn by cleaving it in the NAC region.

## P3-h41 Nicotinic receptor stimulation is protective for dopaminergic neuron.

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Nicotine could play a protective role for dopaminergic neuron, but its mechanism is still unclear. We demonstrated that nicotine prevented rotenone-induced dopaminergic cell death in primary cultures of rat ventral mesencephalon. The neuroprotection was inhibited by nicotinic acetylcholine rececptor(nAChR) antagonists, PI3 kinase inhibitor, and we uncovered elebvated pAkt/PKB by stimulating nAChRs,thus nicotine showed antiapoptotic effect via PI3 kinase-Akt/PKB pathway.Also we showed nicotine improved behavior and nigral dopaminergic cell loss of chronic rotenone-treated parkinsonian model mice(n=8~12 per group). Stimulation of nAChRs may be useful as a treatment for Parkinson disease.

## P3-h42 Glutathione synthesis-related molecule in astrocytes in dopamine quinone neurotoxicity

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Recently, the neurotoxicity of dopamine (DA) quinone, which conjugates with functional proteins to form quinoproteins, has been focused as dopaminergic neuron-specific oxidative stress in Parkinson's disease (PD). Glutathione (GSH) is thought to diminish DA quinone-induced neurotoxicity through its conjugation with DA quinone. GSH synthesis in neurons is limited by availability of cysteine and GSH, which are synthesized from cystine uptaken through cystine/glutamate exchanger in the astrocytes. In the present study, we revealed that repeated L-DOPA treatment caused lesioned side-specific marked elevation of striatal quinoprotein levels in hemi-PD mice, coinciding with increase in expression of cystine/glutamate exchanger in the astrocytes. These results suggest that uptake of the substrate in the astrocytes is elevated against the long-term L-DOPA treatment-induced DA quinone neurotoxicity in PD models, to enhance the synthesis of GSH which quenches DA quinone.

KAKENHI (17590878, 18700364)

## P3-h43 Protective effects of metallothionein against dopamine quinone-induced neurotoxicity

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The neurotoxicity of dopamine (DA) quinone (DAQ) which conjugates with cysteine residues of functional proteins to form quinoproteins (QP) has been recently focused as DA neuron-specific oxidative stress in Parkinson's disease (PD). In the present study, we examined effects of a cysteine-rich metal binding protein metallothionein-I,II (MT) on DAQ-induced neurotoxicity. MT quenched DA semiquinones generated in vitro. In dopaminergic CATH.a cells, DA exposure increased QP and decreased cell viability. These DA-induced changes were ameliorated by pretreatment with MT inducer zinc. Repeated L-DOPA administration caused lesioned side-specific marked elevation of striatal QP levels and reduction of DA nerve terminal in MT knock out PD mice, but not in wild-type mice. These results suggest that intrinsic MT exerts protective effects against the long-term L-DOPA treatment-induced DAQ neurotoxicity in PD models by its quenching DAQ.

KAKENHI (17590878, 18700364)

## P3-h44 MPTP neurotoxicity is enhanced by a restraint stress in C57BL/6 mice

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Although the pathogenesis of idiopathic Parkinson's disease (PD) is not fully understood, a number of environmental factors as well as genetic ones are thought to contribute to the demise of dopaminergic neurons of nigrostriatal pathway. The neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) has been used to create an animal model of PD in nonhuman primates and mice. This study investigated the effect of a various kinds of stress, one of the environmental factors, on dopaminergic neurodegeneration induced by MPTP intoxication. C57BL/6 mice were treated with single injection of MPTP (20 mg/kg, i.p.) and immediately exposed to stress for 2 hours. We found that an exposure to restraint stress in MPTP-treated mice caused a severer loss of striatal nerve terminals as indicated by decreases in dopamine transporter protein levels as well as dopamine levels as compared with MPTP (alone)-treated mice. This result suggests that restraint stress might promote the dopaminergic neurodegeneration in MPTP-treated mice.

## P3-h45 Inhibition of catecholamine metabolism reduces α-synuclein related cytotoxicity in PC12 cells

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An intermediate polymer of quinone-binding α-synuclein (α-syn) has been reported to show cytotoxicity in the pathogenesis of Parkinson's disease. However, its detailed mechanism of action is still not well understood. We established PC12 cell lines that overexpress α-syn in a tetracycline-inducible manner and then examined their vulnerability against various stressors ($MPP^+$, Tunicamycin, Etoposide). An increased number of apoptotic cells (205%, 179%, 232%, respectively) were observed in the cells that overexpressed α-syn in comparison to cells not expressing α-syn. In addition, α-methyl-tyrosine, a specific inhibitor of tyrosine hydroxylase, reduced the number of apoptotic cells (26.3%, 49.6%, 62.5% reduction). Catecholamine is thus suggested to enhance cytotoxicity under various stressors such as ER stress, mitochondrial toxicity, genotoxic stress in cells demonstrating an overexpression of α-syn.

## P3-h46 Comparison between high and low MPTP sensitive mice after MPTP systematic treatment: alterations of behavior, glia and cytokines

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In order to reveal how glia contributes to the development of Parkinson's disease, differences in behavioral and glial alterations in the nigra-striatal region of high MPTP sensitive mice and low MPTP sensitive mice were studied. After MPTP treatment (4 ip injection at 2h intervals), transient movement dysfunction was observed in C57BL/6 (B6), but not in BALB/c (BALB). 1h after the second MPTP injection, microglia processes became shorter and thicker compared with resting microglia in both B6 and BALB. In B6, microglia processes became thicker and 24h after the fourth injection, hypertrophic microglia were observed but not ameboid. After 72h, processes and cell bodies became thinner and smaller compared with microglia after 24h. On the other hand, in BALB, recognizable progression of microglial activation was not observed. The expression of S100B in astrocytes of B6 was increased transiently after the fourth injection but not in BALB. Time dependent interesting changes of cytokines in CSF after MPTP injection were found.

## P3-h47 Advances in parkin gene therapy -a primate study-

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We investigated the delivery of serotype-1 rAAV (rAAV1) vector into the substantia nigra (SN) of monkeys, using a navigation system based on the magnetic resonance imaging (MRI) system. By viewing the MRI images of the monkey brain, we could precisely inject the rAAV1 vector into 12 different sites in the SN of monkeys. Immunohistochemical study for their brains found that the delivered gene was highly expressed in the dopaminergic neurons in the SN. This technique will be useful for the generation of primate models of neurological disorders and for the successful introduction of parkin gene into the SN dopaminergic neurons in primate models, and further, the patients with PD.

the Program for Promotion of Fundamental Studies in Health Sciences of the National Institute of Biomedical Innovation (NIBIO)

## P3-h48 Development of a Parkinson disease model by *parkin* gene knockout in medaka

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We utilized medaka (*Oryzias latipes*) as a new vertebrate gene engineered animal model of Parkinson disease other than mouse and rat. We choosed *parkin*, causal gene of autosomal recessive juvenile parkinsonism (AR-JP), as one of our target genes and identified medaka orthologue. A cryopreserved library of chemically mutagenized fish was screened by high-throughput resequencing for induced point mutations and one mutaion was found to be resulted in the introduction of a premature stop codon. Very similar nonsense mutation in human *parkin* gene was reported in some AR-JP patients and we thought that homozygote for this mutation could be a null mutant of medaka *parkin*. We are raising mutant fish and have found no apparent behavioral change till now. They need further observation, and should be analyzed biochemically and histologically.

Grants-in-aid from the Ministry of Education,Culture,Sports,Science and Technology and the Ministry of Health,Labor and Welfare of Japan

## P3-j01 Involvement of p35 Cdk5 activator metabolism in $MPP^+$ or 6-OHDA-induced neuronal cell death

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1-Methyl-4-phenylpyridinium ion ($MPP^+$) and 6-hydroxydopamine (6-OHDA) are widely used neurotoxins to induce neuronal cell death that is considered as a model for Parkinson's disease (PD). However, it is not known how $MPP^+$ and 6-OHDA induce cell death. In addition to their role as oxidative stressors, both are reported to affect proteasome activity. Cyclin-dependent Kinase 5 (Cdk5) is a neuron-specific proline-directed serine/threonine kinase activated by p35 or p39. Cdk5 is suggested to be involved in pathogenesis of PD. Further, p35 is a short life protein degraded by proteasome. We studied the effect of $MPP^+$ and 6-OHDA on p35 metabolism in primary cultured cortical and striatum neurons. $MPP^+$ induced the degradation of p35 by proteasome at low concentrations and cleavage of p35 to p25 by calpain at high concentrations. We are now investigating the relationship between p35 metabolism and neuronal cell death.

## P3-j02 Molecular basis of anti-apoptotic effect of D2 agonists against 6-OHDA-induced ER stress

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A dopaminergic neuronal toxin, 6-OHDA, is widely used to make a model as Parkinson's disease (PD) both in vivo and in vitro. It is known that 6-OHDA treatment induces a large number of genes involved in not only apoptosis-related molecules as caspase family but also ER stress-related molecules as ER chaperones. We examined that 6-OHDA-induced ER stress, which was characterized by an up-regulation in the expression of BiP, CHOP and caspase-12 in the mouse striatum and substantia nigra. In addition, we determined the effect of several dopamine $D_2$ agonists on 6-OHDA-lesioned mice, to clarify the beneficial effect of combined therapy with $D_2$ agonists and levodopa in PD. Daily treatments of bromocriptine, cabergoline and pergolide for 7 days beginning at first day after 6-OHDA i.c.v. injection decreased BiP, CHOP and caspase-12 expressions. In addition, we have a plan to examine the effects of $D_2$ agonists on 6-OHDA-induced apoptosis in mice and / or in cells, to clarify the molecular basis of inhibition of $D_2$ agonists on 6-OHDA-induced apoptosis.

## P3-j03 Phosphorylated alpha-synuclein contributes to accumulation of ubiquitinated proteins

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Lewy bodies (LBs) and Lewy neurites (LNs) are pathologic hallmarks of idiopathic Parkinson disease. Alpha-synuclein ( α )and ubiquitin (Ub) are major protein components deposited in LB and LN. Almost 90% of α syn in LBs is phosphorylated at serine 129 (S129). Here, we show that coexpression of phospho-mimic mutant α syn (S129D) and Ub results in an increase of Ub-conjugates and the formation of ubiquitinated inclusions. Furthermore, in the presence of a K63R mutant Ub, S129D α syn fails to increase the Ub-conjugates and form ubiquitinated inclusions. Our results suggest that the contribution of S129 phosphorylated α syn to the K63-linked Ub-conjugates and aggregation of itself may play a role in the biogenesis of LBs in PD and other related synucleinopathies.

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## P3-j04 Microglial migration is up-regulated by alpha-synuclein-mediated induction of CD44 and MT1-MMP in Parkinson's disease

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Alpha-Synuclein is involved in dopaminergic neuronal loss in the subtantia nigra (SN) in Parkinsons disease (PD). We hypothesized that microglia is critical for neuronal loss in SN and alpha-synuclein may drive microglial migration into the SN inducing neurodegeneration. Here we demonstrate that alpha-synuclein induces both CD44 and MT1-MMP expressions in microglia, followed by increased generation of soluble CD44 (sCD44) which facilitates microglial migration by interference with cell-to-ECM interactions. All these events are regulated through the ERK signaling pathway. Furthermore, we confirmed alpha-synuclein-induced microglial migration into the SN in both mouse PD model and the human PD patients with increased proinflammatory cytokines, IL-1beta, IL-6, and TNF-alpha. In this manner, alpha-synuclein displays a functional link with microglial migration to recruit them into the region of neurodegeneration as a possible key pathological process of Parkinsons disease.

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## P3-j05 Genetic Polymorphism in susceptibility genes for PD

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An attempt was made in a case control study to investigate the association between functionally important polymorphisms in the genes involved in xenobiotics metabolism and dopaminergic pathways and sporadic PD. PCR-RFLP based methods were used to detect polymorphisms in CYP2D6, 2E1, GSTT1, DRD2, DAT, MAOB genes.CYP2D6*3, CYP2D6*4, CYP2E1*5, CYP2E1*5B, GSTT1(null), DRD2(Taq1A&Taq1B), MAO-B(intron13A/G) were detected in 60 cases and equal numbers controls. The risk was found to be increased two fold in CYP2D6*4(heterozygous) genotype carriers in PD cases. CYP2E1*5 (heterozygous) genotype was found to be overrepresented in cases (8%) when compared to controls (2%). CYP2E1*5B (mutant) genotype of CYP2E1 was observed in 7% of cases. GSTT1 (null) was slightly increased in cases (18%) than the controls (13%). Similarly, polymorphism in DRD2, DAT and MAO-B are also found to be increased in the cases as compared to the controls. Our result suggests that CYP2D6, 2E1, GSTT1, DRD2, MAO-B and Parkin gene polymorphisms alone or in combination may modulate the susceptibility to PD.

## P3-j06 Nurr1 is essential for maintenance of the dopaminergic phenotype in the nigro-striatal dopaminergic neurons

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Nurr1 is mainly expressed within the central nervous system, both during development and in adult. To clarify the role of Nurr1 in survival and maintenance of the nigro-striatal dopaminergic neurons in the adult mouse brain, we generated *Nurr1*$^{flox/flox}$ mice harboring *loxP* sites into the second and third intron of the *Nurr1* gene, and used AAV-Cre. Focal injection of AAV-Cre into the substantia nigra of *Nurr1*$^{flox/flox}$ mice results in efficient ablation of the *Nurr1* gene within the neurons infected with AAV-Cre. The ablation of the *Nurr1* gene reduced the expression of TH, AADC, and GCH. These results suggest that Nurr1 is essential to maintain the dopaminergic phenotype of the nigro-striatal dopaminergic neurons in the adult mouse.

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## P3-j07 Protective effect of water-soluble coenzyme $Q_{10}$ on dopaminergic neurodegeneration in MPTP-treated C57BL/6 mice

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We demonstrated the neuroprotective effect of water-soluble $CoQ_{10}$ in subchronic 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-treated C57BL/6 mice. Ten-weeks-old mice were received one i.p. injection of MPTP-HCl (30 mg/kg of free base) per day for 5 consecutive days and were killed at 3 days after the last injection. $CoQ_{10}$ (200 or 400 mg/kg per day) were orally administered at the day before the first MPTP injection and just before each injection. MPTP injections substantially decreased dopamine transporter protein and increased glial fibrillary acidic protein levels in the striatum as measured by immunoblotting. $CoQ_{10}$ administrations partly prevented the toxin-induced changes in both the protein levels. These results indicate that the water-soluble $CoQ_{10}$ can protect the nigrostriatal dopaminergic system from MPTP neurotoxicity in mice.

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## P3-j08 Enhanced aggregate formation by dopamine, L-dopa, and quinone in PC12 cells

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The association between protein aggregation and the pathogenesis of Parkinson's disease has been widely documented. In order to understand how Lewy bodies are formed in dopaminergic neurons, we investigated the effect of dopamine and L-dopa on the aggregate formation using neural-differentiated PC12 cells and Tet-off conditioned alpha-synuclein expressing cells. The tyrosine hydroxylase inhibitor, alpha-methyl tyrosine dramatically reduced the formation of aggregates under the proteasome-inhibited condition and upregulated proteasome activity. An in vitro aggregation assay for ubiquitin and alpha-synuclein demonstrated that dopamine and L-dopa enhanced the oligomerization of ubiquitin and alpha-synuclein, respectively. Moreover, a scavenger of quinone body cystein dramatically decreased oligomerization. These data suggest that dopamine and L-dopa enhance the formation of aggregates via quinone formation in dopaminergic neurons.

## P3-j09 Effects of zinc by food on adjuvant arthritis in Lewis rats

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We have reported that serum zinc (Zn) decrease after adjuvant arthritis (AA), however, there is no study to reveal an involvement of Zn in the expression of AA. To know whether supplement of Zn by food improve AA or did not, three types of diets, low-Zn (2.97 mg), Zn-standard (5.19 mg), Zn-sufficient (10.38 mg) diets, were used as food for male Lewis rats received a suspension of complete Freund' adjuvant (1.0 mg), injected intradermally into the tail. All AA rats showed signs of systemic inflammation after the 11th day after the inoculation. There were no significant differences in their food intakes and in their body weight changes among these groups in intact rats and also in AA rats. Hind paw volumes in AA rats of these 3 groups did not show any difference and it showed tendency to increase a hind paw volume according to amount of Zn including diets. Thus, the present results support our previous report that Zn is not involved in the expression of AA.

## P3-j10 Effects of 1-bromopropane on neurotrasmitter receptors, intracellular signalings and feedback inhibition in the CNS

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1-Bromopropane (1-BP), a new substitute for chlorofluorocarbons, could adversely affect the human nervous system. However the underlying mechanism still requires to be investigated. Here we report that 1-BP potentiated GABA-but inhibited ACh-evoked responses in *Xenopus* oocytes expressing $GABA_A$ and nicotinic ACh receptors, respectively. 1-BP also directly enhanced recurrent inhibition in the rat hippocampus. Moreover, in cultured astrocytes, 1-BP inhibited activity of NF-κB and expression of Bcl-xL. BDNF was also suppressed possibly through PKA pathway. Chronic inhalation disclosed hippocampal disinhibition in a concentration-dependent manner. Alteration of $GABA_A$ receptor subunit expression and reduction of BDNF and Bcl-xL expression were accompanied with the disinihibiton at 400 ppm concentration. These results clearly indicate that 1-BP has both chronic and acute neurotoxicity in the CNS.

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## P3-j11 Cellular damages on the CNS neurons treated with ethanol are alleviated by roasted rice bran and soybean extracts

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Roasted rice bran and soybean extracts have antioxidant activity to eliminate hydroxyl radical. We examined how the CNS neurons treated with 20% ethanol for 23 weeks were influenced with 1% rice bran or soybean extract in the ethanol. Immunohistochemically, expressions for neurofilament (NF) protein, antioxidant enzyme and lysosome/autophagy/apoptosis-related proteins were altered in ethanol-treated rats, but they were completely opposite in pattern with the extracts, similar in control rats drinking water alone. Immunoblottings produced results the same as those from the immunohistochemistry. Electron microscopy showed the increase in density of lipofuscin granules and axonal NFs in ethanol-treated rats, and the reduction to the normal level of such organelles with the extracts. These suggest that some components in the extracts suppress the ethanol-induced impairment in the CNS neurons.

## P3-j12 Comparison research on excitatory amino acid transporters between astrocyte and microglia

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Glutamate-induced excitotoxicity is considered as a major cause of neurodegenerative disease. Excitatory amino acid transporters (EAATs) on glial cells are responsible for homeostasis of extracellular glutamate in the central nervous system, which may contribute to prevention of excitotoxicity. However, the difference of EAATs expression in astrocyte and microglia is not fully understood. Thus we compared EAATs expression in astrocyte and microglia. We assessed the neuroprotective function of astrocyte and microglia by neuron/astrocyte/microglia co-culture. Transcripts for EAAT1-5 were detected by RT-PCR in astrocytes, but microglia lose EAAT4 mRNA expression. Western blot analysis demonstrated that the expression of membranous EAATs in astrocytes is higher than that in microglia. The observation of co-culture system revealed that astrocyte reduce excitotoxicity. These results showed that astrocytes prevent excito-neurotoxicity via abundant EAATs.

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## P3-j13 The effect of acetylcholine receptor activation on the epileptogenesis in rat hippocampal slices

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We studied the effect of the activation of acetylcholine receptor on the epileptogenesis using rat hippocampal slices. Carbachol(30-100μM) can induce β oscillation in the slices. Picrotoxin(PTX)-induced epileptiform discharges were disappeared and β oscillation was induced when carbachol(30μM) was applied. Similar phenomena was observed when carbachol was applied to $GABA_A$ receptor antagonist SR95531(10μM)-induced epileptiform discharges. PTX-induced epileptiform discharges were not disappeared by application of simultaneous application of atropine sulfate(30μM) with carbachol. The frequency of PTX-induced epileptiform discharges was increased and the amplitude was decreased by carbachol(1-5μM). Population excitatory postsynaptic potential(pEPSP) was decreased by carbachol(1-5μM). These results suggest that the activation of acetylcholine receptors by carbachol decreased pEPSP, the activation of the pyramidal cells were desynchronized by the decrease in the results carbachol suppresses the hippocampal epileptiform discharges.

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## P3-j14 Neuronal activities in the parietal cortex of an EL and DDY mouse

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To elucidate the precise neuronal activities in the parietal cortex, an electrophysiological study was done with adult EL and DDY (14 each) mice, both of which were kept as inbred strains and the former ones were developed as seizure-prone in our laboratory. The animals were fixed with a stereotaxic apparatus under deep anesthesia by pentobarbital. After removing the skull thin tungsten microelctrode was penetrated into the parietal cortex. Unit firings of neurons were recorded with a digital tape recorder and analyzed by the Spike2. The contralateral gastrocnemius muscle was stimulated by the electric current at a supramaximal strength with thin paired stainless steel wires. The frequencies of unit firings of EL cortical neurons were lower than those of DDY. The responses to the muscle stimulation in the EL cortical neurons were significantly more than those of DDY. The latencies of those neuronal responses of EL were remarkably shorter than those of DDY. The parietal cortical neurons of the EL are rather larval at the resting stage and more excitable to the input.

## P3-j15 Scn1a mutant rats established by gene-driven ENU mutagenesis

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Chemical mutagenesis is a powerful tool to produce genetically modified mutations in many species, especially where no functional embryonic stem cells exist, such as in rats. However systematic approaches for the application of the mutagenesis to this species are required because large space and high breeding costs are need in the rat. Here we report an efficient approach to produce gene-targeted mutant rats by gene-driven ENU mutagenesis combining Mu-transposition based on high-throughput screening (MuT-POWER) and intracytoplasmic sperm injection (ICSI). Mutation screening on 1600 progeny of ENU-treated males in the rat *Scn1a* gene identified two missense mutations, of which one is included in the pore region of the sodium channel, similar to the human case of generalized epilepsy with febrile seizures plus (GEFS+).

## P3-j16 Pathway-specific enhancement of NMDA-mediated currents in $Ca^{2+}$-channel mutant mice, *tottering*

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The *tottering* (*tg*) mice have a mutation in the $Ca_V2.1$ (P/Q-type) voltage-dependent $Ca^{2+}$ channel $\alpha_1 2.1$ subunit gene, and are one of genetic models for absence epilepsy. Absence epilepsy is characterized by highly synchronized oscillations in the thalamocortical (TC) network. Although an integral part of the modulation and regulation of epileptic rhythmicity is thought to result from glutamate-mediated excitation, the effect of the mutation on glutamate-mediated excitation remains unknown in the TC network. To explore this, we studied TC and intracortical (IC) glutamatergic transmission of somatosensory layer IV neurons using brain slice preparation. The *tg* mice showed an enhancement of NMDA-mediated, but not AMPA-mediated, synaptic currents evoked by TC stimulation. In contrast, this enhancement of NMDA-mediated synaptic currents was not seen by IC stimulation. Theses results indicate TC-specific enhancement of NMDA-mediated currents in *tg* mice.

## P3-j17 Phenotype analysis of PRIP1-Knock out mouse

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PRIP1 was first identified as a novel inositol 1,4,5-triphosphate binding protein. PRIP1 also functioned as a transporter protein of GABAA receptor to plasma membrane. Although many experiments in vitro has revealed that PRIP1 possessed variety of function, PRIP1-KO phenotype analysis were not thoroughly performed. We found that PRIP1-KO mouse showed spontaneous ictal seizure waveform by encephalogram. We investigated the effect of benzodiazepine and the effect of IP3 modulator on ictal and interictal discharges using enchephalogram, microdialysis and patch-clamp technique, indicating dysfunction of Ca2+ mobilization might be involved in epileptogenesis.

※KAKENHI(18300137)

## P3-j18 A higher sensitive pentylenetetrazole-indued seizures in nNOS deficient mice

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We previously reported that excessive nitric oxide (NO) generating by neuronal NO synthase (nNOS) induces tonic-clonic convulsions following treatment with convulsive dose (60 mg/kg) of pentylenetetrazole (PTZ) in mice. To confirm this result, we used the nNOS deficient ($nNOS^{-/-}$) mice. After injection of convulsive dose of PTZ, $nNOS^{-/-}$ mice unexpectedly died following tonic convulsions. More surprisingly, $nNOS^{-/-}$ rather than $nNOS^{+/+}$ mice exhibited more severe tonic-clonic seizures with subconvulsive dose (40 mg/kg) of PTZ. In addition, we assessed the effects of various NOS inhibitors. $nNOS^{+/+}$ mice pretreated with Nω-Nitro-L-arginine exhibited clonic sizures by subconvulsive dose of PTZ. Moreover 1-[2-(Trifluoromethyl)phenyl]imidazole, a potent inhibitor of nNOS, dose-dependently induced the tonic-clonic convulsion in $nNOS^{+/+}$ mice. These behavioral changes in $nNOS^{+/+}$ mice pretreated with nNOS inhibitor closely consistent with a higher sensitive PTZ-induced seizures in $nNOS^{-/-}$ mice. These results suggest that NO generating by nNOS has reciprocal effects.

※JSPS, Tokushima Bunri

## P3-j19 Genetic analysis of an ENU-induced mutant that shows convulsive seizure and immobility

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In RIKEN mouse mutagenesis project, we are carrying out a large-scale screening for behavioral mutants induced by a chemical mutagen ENU. Mice were screened by open field test, home cage activity test and passive avoidance test. In the screening for dominant phenotypes, we detected a mutant line M-200, which exhibits defect of passive avoidance. M-200 showed not only learning deficit but also convulsive seizure, immobility and body weight loss. Fine linkage analysis narrowed the causative region to a 0.9cM interval. We explored candidate genes located to this region by using gene search databases. Sequencing of Slc2a1 of M-200 detected a missense mutation. Slc2a1 is exclusive glucose transporter at the blood-brain barrier, and deficiency of this gene causes human refractory infantile epilepsy. Thus, all results suggested that Slc2a1 is a strong candidate for the causative gene.

## P3-j20 Effects of electrical coagulation on the threshold of electrically induced paroxysmal discharge in the rabbit hippocampus CA1

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Previous study demonstrated that low power laser irradiation can increase the threshold of electrically induced paroxysmal discharge (PADT) in rabbit hippocampus CA1. The present experiment was designed to reveal effects of electrical coagulation procedure. Using 14 adult rabbits, we measured PADTs elicited by stimulation of 1-ms biphasic pulses of 50 Hz for 1 s before and after electrical coagulation. It was made by direct current of 0.2 (case A), 0.4 (B), 0.6 (C), and 0.8 mA (D) for 2 min. Damaged or infarcted area was measured on the histological sections. Each PADT magnitude to control was 2.3±0.3 (case A: n=3), 1.7±0.1 (B: n=3), 1.7±0.4 (C: n=3), and 1.5±0.1 times (D: n=2), whereas each diameter of damaged area was 1.7±0.5 (A), 2.1±0.4 (B), 1.7±0.3 (C), and 2.0±0.6 mm (D), respectively. A negative correlation between them was obtained (R=-0.6068). In comparison with laser effects, electrical coagulation produced smaller PADT increment and lager damage, which showed a disadvantage to the laser irradiation.

※MEXT HAITEKU (2004-08)

## P3-j21 EFHC1 short isoform-specific mutations in patients with epilepsy

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Juvenile myoclonic epilepsy (JME) accounts for ~20% of all epilepsies. Previously we reported *EFHC1* as one of the responsible genes for JME. Human *EFHC1* encodes 640 amino acids (a.a.) protein (isoform A) and 278 a.a. protein (isoform B). The N-terminus of the isoform B is common to the N-terminus of isoform A, but 36 a.a. of the isoform B's C-terminus is unique. Mouse has the isoform A but does not have the isoform B. Previously we reported multiple missense mutations in the isoform A, but recently we also identified several frameshift and nonsense mutations in the isoform B-specific C-terminus region in JME patients. We isolated monkey *Efhc1* cDNAs and found that these contained several isoforms including the isoform B. The amino acid sequence of the isoform B-specific C-terminus region is highly conserved between human and monkey, suggesting that the isoform B is actually functioning. Identification of the isoform B-specific mutations may contribute to the understanding of the etiology of JME.

## P3-j22 The frequency of interictal activity was modulated by the generation of the activity per se

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We studied the frequency of bicuculline-induced interictal epileptic discharges in guinea-pig hippocampal slices. In our previous study, we showed the synaptic efficacy can determine the steady frequency of the discharges. It was confirmed that the synaptic potentiation induced by the theta-burst stimulation during the generation of the epileptic discharges accelerated the frequency of the interictal discharges. There is a possibility that the epileptic discharges cause to potentiate the synaptic efficacy. In this study, we check the possibility, The stimulation which induces long-term depression (1 Hz, 15 min) was done during the generation of the discharges and it diminished the discharges and also decreased the frequency for a while. After that the decreased frequency gradually increased and recovered. These results suggest that that the generation of the spontaneous interictal discharges can modulate its own frequency through the synaptic potentiation.

## P3-j23 Detection and classification of epileptic seizure using continuous wavelet transform and artificial neural network

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In this study, a new approach based on continuous wavelet transform and artificial neural network techniques was used to analyze electroencephalography (EEG) for epileptic rats. We examined EEG with the animal model for epilepsy, the Wakayama epileptic rat (WER). WER is a new mutant exhibiting both spontaneous absence-like behavior and tonic-clonic convulsions. A small telemetry transmitter and a receiver system recorded the EEG signals on the vibrissal somatic sensory region. The recorded EEG waveforms were band-passed between 1.5 and 30Hz. The seizure patterns in EEG were analyzed to focus on absence-like seizure. The accuracy of classifying epileptic waveforms for 24 hours with a new hybrid system was about 88%. It was suggested that the system comprising continuous wavelet transform and artificial neural network is effective for autonomic absence-like seizure detection in long-term epilepsy monitoring.

## P3-j24 Erythropoietin prevents the epileptogenesis and generalization in the epilepsy model rats

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Erythropoietin (EPO) has neuroprotective effects in brain injury in adult rodents. To determine whether EPO affects Kainic acid-induced seizure patterns or the hippocampal cell death by seizure, before KA i.p. administration, adult male Sprague-Dawley rats were continuously infused into the lateral ventricle with EPO, rat serum albumin (as control), anti-EPO antibody, control IgG using osmotic pump for 6 days. Four days after injection of KA, the rats were sacrificed. As a result, the seizure severity in EPO-infused rats decreased significantly than in control rats. Furthermore, the loss of NeuN-positive cells within the CA1 subfields in EPO-infused rats was less than that seen in control rats. In anti-EPO antibody-infused rats, the seizure became progressively more severe than in control IgG-infused rats. The finding suggests that EPO may modulate seizure severity and protect the hippocampal neurons from the damage seizures produce.

## P3-j25 PQBP1 and cell cycle arrest

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Polyglutamine tract binding protein-1 (PQBP1) was isolated as a binding protein to polyglutamine tract (polyQ) with an affinity to polyQ. Mutations deleting C-terminal domain (CTD) of PQBP1 are known to cause X-linked mental retardation associated with microcephaly. CTD interacts with U5-15KD/dim1, a component of splicing complex U5, whose deletion in yeast produces a lethal G2 arrest phenotype. Moreover, PQBP1 is expressed in the neuroproliferative regions of the embryonic brain. These results prompted us to study the role of PQBP1 in cell cycle of neural stem cells.

We performed knockdown of PQBP1 with shRNAs, and assessed the effect of shRNAs on cell cycle by cell proliferation and FACS analysis with neural stem cells and P19 cells equivalent to epiblast. We found that cell proliferation and G2/M progression were disturbed by reduction of PQBP1. These results suggest the impaired proliferation of neural stem cells as a possible mechanism underlying microcephaly.

KAKENHI (16390249, 16650076, 18390254, 18650097)

## P3-j26 Involvement of environmental factors in the expression of psychomotor abnormalities in PACAP knock-out mice

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Mice lacking the neuropeptide PACAP (PACAP-KO mice) display psychomotor abnormalities such as hyperlocomotion and jumping behavior in a novel environment, and deficits in prepulse inhibition (PPI). The present study examined the effects of rearing in isolation or enriched conditions on behaviors in wild and PACAP-KO mice. These mice were obtained from the intercross of heterozygous animals. Isolation rearing (for 2 weeks from 4-week-old) showed more pronounced abnormal behaviors such as hyperactivity, PPI deficits, and aggression in PACAP-KO mice than in the control. In contrast, rearing in enriched conditions (for 4 weeks from 4-week-old) attenuated the hyperlocomotion and jumping behavior in PACAP-KO mice. These results suggest that abnormal behaviors in PACAP-KO mice depend on environmental factors.

## P3-j27 Attention deficits induced by prenatal alcohol exposure in rats

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We investigated whether attention deficits induced by prenatal alcohol exposure (PAE) in rats were ameliorated by treatments of several psychotropic drugs. Pregnant Wistar rats were treated with 20% ethanol (6g/kg/day), 30% sucrose (7.5ml/kg/day) or saline (7.5ml/kg/day) from gestation day 8 through 20 via intragastric intubation. Ability of attention in these offspring was evaluated using choice reaction time (CRT) and percent correct of operant behavior tests. PAE resulted in a significantly longer CRT than those of the other two control groups, however, there were no effects on percent correct. Moreover, in PAE rats treated orally with inhibitors of dopamine or serotonin transporters, there was a significantly shortened CRT compared with solvent treatments 20 min before the tasks. These findings suggest that PAE rats likely suffer impairment in their dopaminergic and serotoninergic neuronal systems and alterations of these neuronal systems may cause the attention deficits of PAE rats.

## P3-j28 The role of nicotinic acetylcholine receptors on attentional dysfunction of an animal model for attention-deficit/hyperactivity disorder

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In the present study, we investigated the role of nicotinic acetylcholine receptor (nAChR) for attentional dysfunction and its related physiological responses in an animal model for attention-deficit/hyperactivity disorder. Male 6-week-old SHRSP/Ezo were bred in our laboratory. SHRSP was shown both impairment of alternation behavior (Y-maze task) and a decrease in choice accuracy rate (five-choice serial reaction time task), which was improved by $\alpha 4\beta 2$ nAChR agonist RJR-2403 (RJR). Reduced blood flow in the prefrontal cortex (PFC) of SHRSP was significantly increased by nicotine and RJR but not by $\alpha 7$ nAChR agonist AR-R17779 (ARR). However, dopamine and serotonin release in the PFC was elevated by ARR but not by nicotine and RJR. These findings demonstrated that attentional dysfunction in adolescent SHRSP are mediated through the nAChergic neuronal system via the $\alpha 4\beta 2$ nAChR but not $\alpha 7$.

## P3-j29 Eye movements in Pervasive developmental disorder (PDD)

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Brain mechanisms that cause PDD are still unknown. Examination of eye movements may reveal brain areas that are involved in PDD. In addition, we can compare the results with large samples of normal development. We examined the voluntary control of saccadic and smooth pursuit eye movements in 13 subjects with PDD (6 autistic, 7 Asperger's disorders) and compared the results with the performance of 13 normal controls. The following eye movement tasks were tested: visually guided saccade, anti-saccade, memory guided saccade, sinusoidal pursuit and step-ramp pursuit. Eye movements were recorded using an infrared system. The subjects with PDD showed higher error rates in the anti-saccade task. They showed lower gains in vertical smooth pursuit. They also showed longer latency in step-ramp pursuit. The abnormalities in the antisaccade task and smooth pursuit tasks were significantly correlated in PDD. These results suggest a possible dysfunction of the frontal eye movement related areas in PDD.

KAKENHI 18023004

## P3-j30 Behavioral and histological examinations in TR-alpha or TR-beta deficient mice

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Thyroid functions are well known to participate in neuronal development after birth in mice. The drug-induced hypothyroidism in a early period of the neuronal development results in permanent behavioral abnormality, including memory disturbance and hyperactivity. However, the relevance of the memory disturbance and the hyperactivity to thyroid hormone receptors, TR-alpha and TR-beta, remains unknown. To address this issue, we performed histological examination and behavioral experiments, including open field and radial maze in TR-alpha and TR-beta deficient mice. In open field experiment, the TR-alpha deficient mice exhibited the tendency of hyperactivity, but unexpectedly we did not observed any gross behavioral abnormalities in TR-beta deficient mice. In this study, we explored the possible involvement of the thyroid hormone signaling in the development of the brain functions after birth.

## P3-j31 TSC2-Rheb-mTOR pathway regulates dendritic spine morphology

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Tuberous sclerosis complex (TSC) is a hereditary autistic disorder caused by mutations in *tsc2* gene. TSC2 has GAP activity towards small GTPase Rheb, and TSC1/2 antagonizes mTOR pathway by stimulation of GTP hydrolysis of Rheb. We used Eker rats that heterozygous for a mutation in the *tsc2* to examine an involvement of TSC pathway in synaptogenesis. TSC2 mutation exhibited an increase in spine length and density, and a decrease in head width. Rapamycin reversed these changes, suggesting the effects of TSC2 mutation were mediated via mTOR. As enhanced synaptic protein synthesis may be associated with abnormal spine morphology in Eker, we have undertaken proteomic study to define protein diversity in synaptosomal fraction between wild and Eker rats. Thus, perturbations of TSC pathway may cause autistic symptoms of TSC by changing spine morphology.

## P3-j32 CtBPs are binding proteins of the novel isoform of FoxP2 lacking forkhead domain

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The mutations of FOXP2 have been detected in the family of the speech/language disorder. Recenly, we identified the forkhead nuclear localization domains that contribute to the cellular distribution of FOXP2. Nuclear localization of FOXP2 depended on two distally separated nuclear localization signals in the forkhead domain. In contrast with wildtype, the mutated FOXP2s are located in the cytoplasm. However, we also identified the novel isofrom lacking forkhead domain located in the cytoplasm of Perkinje cells (P2-P10). To examine the biological role of the novel isoform, we tried to isolate the proteins binding with the novel isoform of FOXP2 by yeast-two-hybrid system. Here we show that this isoform specifically binds with CtBPs with multifuctions such as repressor, golgi fission, and synapticvesicle fusion. The novel isoform may be involved in the synaptic function via binding with CtBPs.

KAKENHI(18700333)

## P3-j33 Identification of a novel isoform of Foxp2 located in the cytoplasm of mouse developing Purkinje cells

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FOXP2 belongs to a newly identified subfamily of Forkhead box (FOX)-transcription factors. The mutations of FOXP2 have been detected in the family of the speech/language disorder, suggesting that FOXP2 is involved in acquiring human speech/language ability. In contrast with wild type, the mutated FOXP2s are located in the cytoplasm. In the present study, we identified the forkhead nuclear localization domains that contribute to the cellular distribution of FOXP2. Nuclear localization of FOXP2 depends on two distally separated nuclear localization signals in the forkhead domain. Furthermore, we identified the novel isofrom lacking forkhead domain located in the cytoplasm during mouse brain development. In the mouse cerebellum, this novel isoform was mainly detected in the cytoplasm of the Purkinje cells (P2-P10), but not in those of P15-P22. This novel isoform may be involved in the acquiring mouse "singing" ability related to the human speech/language ability.

## P3-j34 Brain Proteins with PDZ domains associated with RA175/SynCAM

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Association of neuroligins with PSD-95 and Shank controls the balance between neuronal excitation and inhibition. Mutations of neuroligin-3 and Shank-3 have been detected in the patient of Autism. RA175/SynCAM, homophilic cell adhesion molecule with PDZ binding domain at C-terminal, is also involved in the formation of functional synapse. RA175/SynCAM interacts with CASK at presynapse, but little is known about the binding molecules at postsynapse. We examined the RA175C-terminal binding protein in mouse brain by yeast two hybrid analysis and pull-down assay. Unlike neuroligin, RA175 directly band with Par-3 and RA175 had a complex with Shank but not with PSD-95. RA175 may be associated with Shank via other scaffolding molecues with PDZ domain. We will discuss this putative complex associated with the autism.

KAKENHI(18700333), Research for Nervous and Mental Disorders(17A-1)

## P3-j35 Perinatal BPA exposure impairs synaptic architectures and functions in the hippocampus of offspring

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Humans are routinely exposed to the environmental estrogen bisphenol A (BPA) leaking from food and beverage containers. Exposure to low doses of BPA are kown to affect a variety of animal behaviors including memory and learning. Here we report that perinatal exposure to a low dose of BPA (100 ng/kg) impaired the maze learning, and increased the spontaneous motor activities of 4 weeks male offspring. We investigated the neuronal functions and ultra-structures of the hippocampal CA1 region in these animals. Basal synaptic transmission and population spikes were significantly enhanced, whereas tetanic LTP was impaired without changes in the paired-pulse facilitation. Although the density and spatial configuration of pyramidal cells looked normal, their spines and synaptic contacts at apical dendrites were morphologically altered. These results suggest that perinatal exposure to BPA will alter the hippocampal synaptic architectures and functions, resulting in behavioral abnormalities of the offspring.

## P3-j36 Effects of Mitochondrial ATP-sensitive K Channel Opening during ischemia/reperfusion in neurons

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We investigated the effect of mitochondrial ATP-sensitive K ($mitoK_{ATP}$) channel opening on the ability of oxygen glucose deprivation (OGD) to prevent intracellular $Ca^{2+}$ ($[Ca^{2+}]i$) overload or $\Delta\Psi m$ depolarization in rat hippocampal CA1 pyramidal cells. Acute hippocampal slices were preloaded with $[Ca^{2+}]i$ indicator or $\Delta\Psi m$ indicator. Simulated ischemia was performed by subjecting to a 10-min OGD, then slices were recovered for 5 minutes. Relative changes in the $[Ca^{2+}]i$ and the $\Delta\Psi m$ were measured by confocal system. After OGD the $[Ca^{2+}]i$ increased and the $\Delta\Psi m$ deceased, but both deceased after recovery phase. Ischemic preconditioning(1-min OGD) and anti-anginal drug nicorandil dramatically reduced the $[Ca^{2+}]i$ overload and $\Delta\Psi m$ depolarization resulting from OGD. These results suggest that $mitoK_{ATP}$ channel opening might be attributed to the mechanism of neuroprotection.

## P3-j37 Neuroprotective effects of ethyl pyruvate on brain energy metabolism after ischemia depend on glial cells: a $^{31}$P-NMR study

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Neuroprotective effects of ethyl pyruvate (EP), a stable derivative of pyruvate, on energy metabolism of rat brain exposed to ischemia were investigated by $^{31}$P-nuclear magnetic resonance ($^{31}$P-NMR) spectroscopy.Brain slices (400 μm) were incubated in standard artificial cerebrospinal fluid (ACSF) with 2mM EP (EP+) or ACSF only (EP-) at 25°C. Brain slices were exposed to ischemia by halting the perfusion for 1 h. Levels of high-energy phosphates, phosphocreatine (PCr) and γ-ATP, relative to pre-ischemic levels were measured by $^{31}$P-NMR. Recovery of PCr after reperfusion was significantly greater in EP+ than in EP-. When brain slices were pretreated with 100μM fluorocitrate, an astrocytic poison, such difference in recovery of PCr was not observed.These results indicated that neuroprotective effects of EP depend on the presence of astrocytes. EP might be neuroprotective via functions of astrocytes as a neuro-protectant from oxidative stress and/or a provider of lactate by glycolysis.

※KAKENHI (17591639)

## P3-j38 Mitochondrial permeability transition: evaluation of putative "pore blockers"

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The mitochondrial permeability transition (mPT) is a feasible target for neuroprotection. In a recent screening study of FDA-approved drugs were found to be some of the most potent mPT inhibitors tested. In addition, several clinically available drugs have been proposed to afford cytoprotective effects through mPT inhibition. It has also been suggested that mPT in brain mitochondria can be inhibited by the $IP_3$-receptor modulator 2-APB. Here, we (re)evaluate the effects of putative inhibitors of calcium-induced mPT in brain mitochondria in an attempt to address two basic and important questions: (i) How should mPT inhibition be defined? and (ii) How can mPT-modulation reliably be detected in vitro? We conclude that uncoupling and inhibition of respiration are important possible confounding factors in the search for mitochondrial "pore blockers" with clinical relevance.

## P3-j39 Identification and characterization of protein marker(s) for diagnosis and prognosis of acute Ischemic stroke patients

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Acute ischemic stroke (AIS) is the most common devastating neurological disease worldwide .An early diagnostic and prognostic marker would aid in improvement of clinical outcome. One and 2D PAGEwas performed in sera of AIS patients at different time interval and compared with controlssubjects. The result shows absence of 120-kDa protein band in AIS patients, which is consistently present in the control group. Protein band of 120 kDa was excised from gels and digested by trypsin and peptides were analyzed by LC-MS methods. The 120-kDa bands representing the human inter alpha trypsin inhibitor heavy chain (ITIH4). Antibodies against the protein was generated and evaluated in the serum samples obtained from AIS patients by ELISA and western blotting. Results were compared with S-100, NSE, IL-2, IL-10 and ADA. In conclusion, this report summarizes the significance of ITIH 4 protein in the AIS patients and the initial clinical testing designed to define better the role that it may play in the management of AIS disorder.

※CIIMS project

## P3-j40 Expression of the proteoglycan in rat hippocampus with hypoxia-ischemia insult

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Hypoxia-ischemia (HI) is one of the main mechanisms of the cerebral palsy. In our previous study, hippocampus CA3 was a major damage partin 7day old Levine-Rice model. We sought to investigate effects of HI insult to proteoglycan expression. (Materials and methods) Left carotid artery of the seven day old rats were ligated and exposed to hypoxic insult. In 14th day, rats were sacrificed. Each proteoglycan (N-syndecan, neurocan, neuroglycan-C and phosphacan) was stained with immunohistochemical method. A part of brain tissue was homogenized and digested with heparitinase. N-syndecan was analyzed with western blot. (Results) With HI insults, brain lesions were observed in cortex (CX) and hippocampus. N-syndecan was positive in neuronal cell of CX and hippocampus, especially hippocampus CA3. Phosphacan was positive in both hippocampuses. N-syndecan was up regulated in ipsilateral side brain tissue when compared with contralateral side with western blot analysis. (Conclusions) Increased expression of the N-syndecan was observed in neuronal cell of the injury site.

## P3-j41 The expression of high mobility group box 1 (HMGB1) after acute cerebral infarction in rats

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The high mobility group box-1(HMGB1) was identified as a novel cytokine-like mediator linking acute neuronal death and delayed neuroinflammation in the postischemic brain.We examined the expression and localization of HMGB-1 using experimental models of rat cerebral infarction induced by 90-min MCA occlusion using an intraluminal filament.Immunohistchemical staining of HMGB1 expression at 2 hours, 12 hours and 24 hours after reperfusion.HMGB1 was localized in the nucleus of neurons of the normal area.In the core, a large number of cells expressed HMGB1 in cytoplasm immediately after infarction. At 12 hours, the expression of HMGB1 was weaker than 2 hours, and in 24 hours, that was faded out in the core.In this study, we verified the expression of HMGB1 in the cytoplasm and secretion of neuronal cells from nucleus triggered with ischemia.

## P3-j42 Expression of heparanase in rat brain after transient middle cerebral artery occlusion

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Heparanase is an enzyme that cleaves heparan sulfate proteoglycans leading to the remodeling of the extracellular matrix and the basement membrane particularly during cancer metastasis. Heparanase has been also known to serve multiple functions in normal tissues including the central nervous system. In this study, we analysed the expression of heparanase in rat brain after transient middle cerebral artery occlusion. RT-PCR and Western blot analyses revealed that heparanase expression was upregulated during the subacute phase of ischemia. Heparanase was localized in nestin-positive reactive astrocytes located in the peri-infarct region by immunohistochemical study. Infiltrated neutrophils were weakly heparanase+. The time course of heparanase expression in astrocytes may correlate with the time of migration of reactive astrocytes toward the ischemic core, which may result in astrogliosis. These results suggest a novel role of heparanase in the pathophysiology of brain ischemia.

## P3-j43 Lycopene uptake to Mongolian gerbil attenuates apoptosis in hippocampus induced by ischemia

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Lycopene from plant origin is known to have an antioxidant action in tissues. Application of transient ischemia/recirculation to gerbil produced apoptosis in hippocampus after several days. We examined antioxidant effect of lycopene administration to the animal, expecting to find effect of lycopene to attenuate the progress of apoptosis caused by ischemia. Weanings at 21st days after birth were fed on lycopene containing MF food (5mg/100g) freely, and then we burdened cerebral ischemia to the animal. Amount of bcl-2 was decreased in the animal without lycopene intake, whereas it was increased clearly in lycopene administered animal after surgery. Increased TUNEL positive cells after ischemia reduced in lycopene treated animal after a week. These results strongly suggest that lycopene can act as an inhibitory modulator against apoptosis in neurons caused by ischemia even at genetic level.

## P3-j44 Roles of macrophage-like cells expressing NG2 proteoglycan in ischemic rat brain

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Macrophage-like cells accumulate in ischemic core in the rat brain after transient middle cerebral artery occlusion (90 min) from 3 to 14 days post-reperfusion (dpr). Because such macrophage-like cells expressed NG2 chondroitin sulfate proteoglycan (NG2) as well as a macrophage-marker Iba1+, they were termed BINCs (Brain Iba1+/NG2+ Cells). BINCs were highly proliferative as revealed by labeling with 5-bromodeoxyuridine (BrdU) at 2 dpr. In this study, to gain insights as to whether BINCs play favorable roles in the ischemic events, 5-fluorouracil (5FU) was intraperitoneally injected to ischemic rats at 2 dpr to kill BINCs specifically. This treatment caused marked loss of BINCs in ischemic core. Nearly half of the 5FU-injected ischemic rats died by 10 dpr. Ischemic region of the survived rat brains markedly enlarged. These results indicate that BINCs play protective roles in the ischemic events.

## P3-j45 Involvement of tyrosine phosphorylation of the NMDA receptor via metabotropic glutamate receptor 1 in infarction after middle cerebral artery occlusion and reperfusion

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To determine whether activation of metabotropic glutamate receptor 1 (mGluR1) is involved in cerebral infarction after transient focal ischemia, we examined the effect of mGluR1 antagonist LY367385 on cerebral injuries. Transient focal ischemia was produced in the rat by intraluminal middle cerebral artery occlusion for 90 min with a surgical monofilament nylon suture. The intravenous administration of LY367385 reduced the infarct size at 24 hr of reperfusion. Transient ischemia increased tyrosine phosphorylation of Src and NMDA receptor subunits NR2A and NR2B in the cerebral cortex. The administration of LY367385 attenuated phosphorylation of Src in the PSD fraction. Furthermore, tyrosine phosphorylation of NR2A, but not of NR2B, was attenuated by LY367385 treatment. These results suggest that the increase in the tyrosine phosphorylation of NMDA receptor subunits through mGluR1 may be involved in cerebral infarction after transient focal ischemia.

※KAKENHI (17700357)

## P3-j46 Change and translocation of HSP60 in gerbil hippocampal CA1 region induced by transient ischemia and its protective effect against ischemic damage

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We observed changes in heat shock protein 60 (HSP60) in the gerbil hippocampal CA1 region after transient forebrain ischemia and its protective effect against ischemic damage. HSP60 immunoreactivity was increased at 3 h after ischemia/reperfusion (I-R) and peaked 6 h after I-R. At these time points, HSP60 immunoreactivity was detected in the mitochondria. At 24 h after I-R, HSP60 immunoreactivity was detectable in the cytoplasm of hippocampal CA1 region. Thereafter, HSP60 immunoreactivity was nearly disappeared in the hippocampal CA1 region. The HSP60 transfection via the adenovirus significantly increased the neuronal survival after transient forebrain ischemia. This suggests that the translocation and decrease of HSP60 may be associated with neuronal cell death and the induction of HSP60 protects neurons from ischemic damage.

※BK21 Program for Veterinary Science, Seoul National University.

## P3-j47 Time dependent progesterone-nouroprotection against ischemic brain damages

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Ovarian hormone progesterone (P4) is a particularly interesting candidate agent in treating stroke because of its wide time-window for administration to protect neurons from ischemic neuronal damages. We previously reported in a rat model that a single administration of P4 (4mg/kg) at either 1 hr or 48 hr before middle cerebral artery occlusion (MCAO) was able to prevent the ischemia-induced pyramidal cell loss and long-term potentiation (LTP) in the hippocampus as well as the deficits in spatial cognition. Herein, we report that the delayed (48 hr) neuroprotection was mediated by P4 receptor, a nuclear transcription factor receptor, followed by Src/ERK signaling. On the other hand, the acute (1 hr) neuroprotection seemed to be mediated by an antagonism of a sigma $_1$ receptor by P4. These results indicate that the wide time-window of P4-neuroprotection against ischemic brain damages includes two independent processes, a sigma $_1$ receptor-mediated non-genomic and a P4 receptor-mediated genomic signal pathways.

## P3-j48 Inducible expression of retrograde transynaptic genetic tracer in mice

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A key step toward understanding of neural function is characterizing the neuronal connectivity. Tetanus toxin C fragment (TTC) is transynaptically and retrogradely transported without the toxin's pathogenic effect. We introduce a new fusion construct, APTTC, consisting of the truncated human placental alkaline phosphatase with TTC, and generated the transgenic mouse line, tetO-APTTC, for inducible expression of APTTC regulated by tetracycline operator (tetO). In order to examine the connectivity of striatal medium spiny neurons (MSNs), we generated double transgenic mice by mating the tetO-APTTC transgenic mice to the PDE10A2-tTA knock-in heterozygote mice ($PDE10A2^{tTA/+}$), in which tTA is selectively expressed in striatal MSNs. In the double transgenic mice, we found alkaline phosphatase (AP) staining in the tTA expressing regions and other specific regions whose axons are reported to terminate in striatal MSNs with previous tracing studies. Our data indicate that APTTC works as a retrograde transynaptic genetic tracer in mice.

CREST

## P3-k01 Functional transplant of photoactivated adenylyl cyclase into *Aplysia* mechanosensory neurons

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In the present experiments we attempted functional transplant of a photoactivated adenylyl cyclase (PAC) isolated from *Euglena* into mechanosensory neurons in *Aplysia* pleural ganglia. When cAMP was injected into the neurons, spike amplitude temporarily decreased while spike width temporarily increased. This effect was sustained longer in a bath solution containing IBMX, the phosphodiesterase inhibitor. We, therefore, explored these changes as indicators of appearance of the PAC function. PAC or the PAC expression vector (pNEX-PAC) was injected into cell bodies of neurons. Spike amplitude decreased in both cases and spike width increased in the PAC injection when the neurons were stimulated with light, suggesting that the transplanted PAC works well in *Aplysia* neurons. These results indicate that we can control cAMP production in specific neurons with light by the functional transplant of PAC.

## P3-k02 Identification and characterization of transcripts selectively expressed in the mouse locus coeruleus (LC)

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The LC is the major source of noradrenergic inputs to almost entire brain regions. The catecholaminergic (noradrenergic) neurons in the LC were collected selectively from fetal brain of the tyrosine hydroxylase (TH)-green fluorescence protein (GFP) transgenic mouse (TH-GFP mouse) by flow cytometry (FACS) as was reported previously. In this experiment, we made comprehensive analysis of genes expressed in the LC by GeneChip™ for the purpose of identifying genes expressed specifically in the LC. The transcripts identified in the LC by GeneChip™ were verified by RT-PCR and *in situ* hybridization. Thus, we obtained an array of transcript species that characterizes the LC. Interestingly, the majority of these transcripts were also identified in the adult LC, consistent with the knowledge that the differentiation of the LC takes place at an early embryonic stage.

## P3-k03 Simple, easy and long-term protein ablation method: an application of fluorophore-assisted light inactivation to cell. explant and organotypic cultures of the nervous system

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To exploit advances in methodology for analysis of protein function, direct target validation of proteins in living cells is the most useful approaches. We characterized fluorophore-assisted light inactivation (FALI) which uses coherent light and fluorescein- conjugated antibody. We developed a simple and easy FALI-based protein ablation method (SELT-FALI). FALI of Neuropilin-2 (Np2), a receptor for Semaphorin-3F (Sema3F) resulted in a complete reduction of Sema3F affinity in Np2-expressed COS7 cells. The FALI-mediated damage of Np2 was dependent on the quantity of light. We applied it to organotypic culture of developing mouse telencephalon. FALI of Np2 for 24 hours in culture resulted in a reduction of Sema3F affinity in Np2-expressed neurons. We report a useful methodology for identification of the protein function in living cells and tissues.

CREST, JST

## P3-k04 Dual viral transneuronal tracing using recombinant rabies virus vectors

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Currently, we developed a recombinant rabies virus vector which acts as a potential neurotracing tool. This virus vector infects neurons selectively and propagates along neurocircuit retrogradely via synapses. By using two vectors, each of which expresses different reporter protein, we tried dual viral tracing. Since the two neuronal groups constituting different neurocircuits would be labeled with different reporter proteins, we can easily detect and visualize the relationship of these circuit, e.g., overlapping of the two circuits. This method is expected as a powerful tool to analyze the interactions of multiple neurocircuits in the central nervous system. In this study, we demonstrated dual viral transneuronal tracing in the rat hippocampal system, and showed the manner of multisynaptic connections between the CA3 region and the bilateral entorhinal cortex.

## P3-k05 A novel cell count method with multiple thresholds

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In automated cell counts, we separate cell bodies from the background by thresholding digitized section images with a single threshold. However, the conventional method tends to underestimate the number of cells especially when they are densely packed. In this study, we developed a new method for detecting immunopositive cells one by one, by gradually changing the threshold level from high to low. By lowering the threshold from the highest, we firstly detected the densest cell, then the second densest and finally the least dense cell body. As an example, we applied the new method to sections of a mouse brain that were immunostained with NeuN antibody. Automated cell counts with our new method were almost identical ($r^2 = 0.94$) to manual cell counts. The linearity was unchanged over a wide range of cell density from 0 to 5000 /mm$^2$. In contrast, conventional automated cell counts with a single threshold yielded much worse correlation ($r^2 = 0.76$) due to saturation at ~1000 /mm$^2$. The results show that our new method is most advantageous when the cell density is higher than 1000 /mm$^2$.

Indust-Univ Joint Res, JST

## P3-k06 An efficient gene delivery system into cells using a nano-size silicon needle

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The transfer of DNA molecules into cells is performed by several different techniques, including lipid- or viral-mediated transfection, electroporation, and microinjection. One of the advantages of this microinjection technique over the others is the capability of pinpointing cells to be transfected. Currently, microinjection through glass pipettes requires much experience and effort, and often causes cell damage. Here we have developed a user-friendly and efficient microinjection technique using a fine needle on an etched AFM cantilever. A standard silicon needle, AC160BN (Olympus) etched by focused ion beam, can be manipulated under the control of a piezoelectric transducer. When a needle penetrates a cell, a small gap is created in the plasma membrane, through which DNA molecules go enter the cell. This microinjection technique offers a means for transfecting any two adjacent cells with cDNAs for different fluorescent proteins.

## P3-k07 Subtype-specific ligand sensors: utilization of neurotransmitter binding proteins

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Venom from Elapidae snake and Conus snail contains molecularly and physiologically diverged neurotoxins, causing neurogenic symptoms such as paralysis, convulsion, hallucination etc. $\alpha$-neurotoxins, which specifically interact with nicotinic acetylcholine receptors, are invaluable molecular tools to investigate signal transduction in the cholinergic synapses. We cloned a cDNA encoding acetylcholine binding protein (AChBP) from the sea molluscs nervous system. Recombinant AChBP was conjugated to SPR sensor chips, and they showed high affinty ($K_D$ = 2.1 nM) to a neuronal type $\alpha$-conotoxin, while lower affinity to the neuromuscular junction (NMJ)-type $\alpha$-conotoxins ($\mu$M to sub-$\mu$M). These results suggest that the AChBP isolated from the sea molluscs can be used for sensing cholinergic ligands discriminating the neuronal type from the NMJ type. We also prepared an amino-terminal half, extracellular domain, of the serotonin receptor 3 (5-HT3), and found its affinity to 5-HT ligands.

## P3-k08 The morphogenesis and trans-neuronal transfer of swine hemagglutinating encephalomyelitis virus in the central nervous system

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Swine hemagglutinating encephalomyelitis virus has recently been introduced as a potential tool for multisynaptic neural circuit tracing. The present study used electron microscopy to characterize pathways of viral replication and egress that lead to transneuronal infection. Six-week-old rats were inoculated with 67N HEV in the right hind footpad. HEV particles were found in the pyramidal cells in the motocortex on the 4th day after infection. Morphogenesis of HEV took place in the cytoplasm of the infected neurons, with the participation of different membranous compartments.The acquisition of viral spikes occurred mainly in the ER lumen. ER-derived uncoated vesicle-enclosed virions can be transferred by microtubules. In Golgi trans-aspects, viral paticles were assembled into coated vesicles, which conferred virions the ability to penetrate the synaptic terminals.

## P3-k09 Investigate the function of neural circuits in Drosophila using light-activating ion channels

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For studying the function of specific neurons in their native circuitry, it is desired to precisely control their activity. So, we expressed channelrhodopsin-2(ChR2), a directly light-gated cation channel from the green alga Chlamydomonas reinhardtii, in tissue-specific pattern in the Drosophila, to trigger specific behaviors, simply by illumination. ChR2 are 7-transmembrane proteins, which utilizes the chromophore all-trans retinal, to open an intrinsic cation pore. In transgenetic flies which carry ChR2 in different tissues, blue light can depolarize the neurons and evoke action potentials. When ChR2 was expressed in nociceptive neurons, light evoked avoidance responses that are normally elicited by nociceptive stimuli. This effect can be used to confine the larvea in a small area or transfer them along a pre-designed pathway. ChR2 also function well in adult fly, it can activate sensory neurons, modulatory neurons and motor-output neurons.

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## P3-k10 Non-parametric resampling-based methods for functional NIRS studies

Archana K. Singh[1], Masako Okamoto[1], James B. Cole[2], Ippeita Dan[2]

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The distribution of functional near-infrared spectroscopy (fNIRS) data is still under exploration and is uncertainly known. The traditional parametric models that rely on strong assumptions regarding the data distribution may be too conservative or unreliable on such data, particularly in group studies that employ few subjects. Nonparametric resampling-based bootstrap and permutation tests can relax most of the assumptions required by the parametric tests in statistical testing of multichannel fNIRS data. We will evaluate the scope of these tests in functional analysis of NIRS studies, and present a resampling-based framework for hypothesis testing. We have found that such model is easily applicable with fNIRS data and produces more significant results in comparison with the conventional parametric model.

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## P3-k11 What was the brain for historical Japanese or Chinese; Examination of the Chinese letter (Kanji) of the brain in literature

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At present we understand that the brain is the organ to process various information, spinning mind from neural activity. What organ was the brain for old Japanese or Chinese people? When did the idea of the brain to be a seat of mind emerge? We tried to answer such questions by inspecting meaning of the Kanji of the brain appearing in old medical literature (I-shin-poh), or sample sentences in old Chinese literature recorded in the Morohashi's biggest Chinese-Japanese dictionary. Various Kanji's were used to express the brain (Fig). All of them mainly mean substance, or marrow, within the skull, but an example is shown that a Chinese writer used the brain as a meaning of heart or spirit in about 300 A.D.



## P3-k12 Independent component analysis applied to the removal of artifacts in combined TMS/EEG recordings

Shigeki Takauchi, Yasoichi Nakajima, Hiroshi Kadota, Hirofumi Sekiguchi, Kenji Kansaku

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In recent years, short latency evoked potentials induced by TMS have begun to be recorded, but it has difficulties in that there exist large magneto-electrical artifacts induced by the stimulation. To overcome the shortcomings, we first recorded the TMS induced artifacts by using an artificial circuit (5 kΩ, 150 pF), and prepared a template data. We then obtained TMS/EEG data by stimulating the left primary motor cortex (60% RMT, FDI), and applied independent component analysis to the TMS/EEG data (n=18). The decomposed independent components were cross-correlated with the template data that we prepared, and independent components that showed r values higher than 0.8 were regarded as artifact related components. The reconstructed signals after removal of the artifact related components showed clear positive potential at about 25ms over the prefrontal areas. These methods may be useful in removing artifacts in combined TMS/EEG recordings and allow us to obtain better short latency evoked potentials induced by TMS.

HLSRG (H15-0003)

## P3-k13 Alkmaion, ca 500 BC, discovers that mind is in the brain

Robert W. Doty

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Data show that the first recorded recognition of the brain as origin of thought came from Alkmaion, writing at Kroton, the famous medical school of southern Italy. The Dorian dialect of the Greek language linked Kroton with the still more famous school of Hippokrates on the island of Kos. From there the idea was promulgated in eloquent terms, quite comparable to those we use today, that all our joys and fears, prejudices and desires, emanate from actions in the brain. This is in striking contrast to contemporary Egyptian usage where, in punctilious funereal practice, the brain was heedlessly discarded. More than 2000 years ensued, however, before it was recognized that the mind derived from "gray matter" rather than the "air" (pneuma) in the cerebral ventricles.

## P3-k14 Effects of methycobal(MTB) on rat embryos cultured in vitro

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We have developed the screening system of the neuraltube development of drugsby the use of rat whole embryo culture. The advantages of whole embryo culturesare to examine the direct effects of drugs on embryos and also to find the teratogenic agents of brain. As the testing agent, methycobal(MTB) was examind in the presentstudy using the cultures of the rat embryos of day 11 to day 13 gestation . Rat embryos were on day 11 of gestation were cultured in vials containing 5ml of rat serum usingimproved rotator for 48hrs. The group of embryos treated with MTB was not changed in the heart beat, the crowm-rump length, the embryo weight and the embryonic totalsomites from control embryos cultured without MTB. The malformation was also notobserved in the embryos cultured with MTB. The head length was increased from control group. On the other hand, the neuraltube was observed to the growth in the cultured rat embryos with MTB.

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## ◆ 助成

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TWO PHOTON P2-e45 S2A-D1 S2A-D3 S2A-D4 S2A-D5
TWO PHOTON LASER MICROSCOPY P3-c18
TWO-DIMENSIONAL ELECTROPHORESIS P3-c29
TWO-PHOTON EXCITATION MICROSCOPY P2-d27
TWO-PHOTON MICROSCOPE P2-b05
TWO-PHOTON MICROSCOPY S3P-G6
TYPE2 DIABETES P3-g34
TYROSIN HYDROXYLASE O2P-D15
TYROSINE HYDROXYLASE P2-b30 P2-b40 P3-j06
TYROSINE KINASE P1-j26 P3-c05
TYROSINE PHOSPHATASE O1P-D12
TYROSINE PHOSPHORYLATION O1P-K07 P3-j45 S1A-G2

## U

U-SHAPED O2P-A02
UBIQUITIN O1P-D15 O1P-D16 O1P-F01 O1P-K05 P1-h46 P1-j08 P2-g27 P3-j03
UBIQUITIN-POSITIVE INCLUSION P1-h39
UBIQUITIN-PROTEASOME P1-c06
UBIQUITINATION P2-h36
UCH-L1 P1-c16
UDP O1P-H13
ULTRASONIC P3-d14
ULTRASTRUCTURE O2P-B06
UMAMI P3-f20
UNC-51 P1-d16
UNC-76 P1-d16
UNC5H4 P3-c39
UNCONDITIONED FEAR S1A-G3
UNEXPLAINED SYMPTOMS P1-j46
UNFAMILIAR O2P-G03
UNILATERAL LABYRINTHECTOMY P1-e46
UNIT RECORDING P2-e16
UNITARY EVENT O2P-C08
UPREGULATION P1-h27
URINARY BLADDER P3-f32
UVULA P2-f12

## V

V-ATPASE P3-e42
V1 O1P-C07 P2-e29
V1 LESION P1-e21
VACALIZATION P1-f02
VAGUS AFFERENT P1-g02
VALPROATE P1-j44
VALPROATE ANALOG P1-c19
VAMP2 P2-c14
VARIATIONAL BAYESIAN P2-k16
VASOPRESSIN P1-b48 P3-d11 P3-h24
VASOTOCIN/ISOTOCIN P3-f37
VENTILATION P1-f48
VENTRAL ANTERIOR-VENTRAL LATELAL THALAMIC NUCLEUS O1P-B12
VENTRAL HIPPOCAMPUS P1-j25
VENTRAL PALLIDUM O2P-J12
VENTRAL TEGMENTAL AREA P1-j36
VENTRAL VISUAL SYSTEM O1P-C12
VENUS P3-g09
VERBAL COMPREHENSION P2-h17
VERBAL LEARNING P3-g35
VERGENCE P3-f14
VERGENCE EYE MOVEMENT P3-e44
VESICLE P3-b41
VESICULAR GABA TRANSPORTER P3-b40
VESICULAR GLUTAMATE TRANSPORTER 2 P2-g01
VESTIBULAR P1-e23 P2-f10
VESTIBULAR NUCLEUS P1-e46 P1-e48
VESTIBULAR NUCLEUS COMPLEX P2-f26
VESTIBULAR SIGNALS P1-e47
VGAT P1-b18
VGLUT P1-b20 P1-b27 P1-b29 P2-f03
VIBRO-DISSOCIATION P1-e43
VIEW INVARIANCE O1P-C12
VINCRISTINE P1-k12
VIP P3-c30
VIRTUAL SCREENING P1-c16
VIRUS P3-k04 P3-k08
VISCERAL SENSATION P3-f34
VISION O1P-C11 O2P-C10 O2P-F03 S1A-B3
VISUAL O1P-C05 S2A-D5
VISUAL ACUITY P2-e48
VISUAL CATEGORIZATION TASK O2P-C01

VISUAL CORTEX O1P-C06
O1P-C07
O1P-C09
O1P-C10
O1P-C14
O1P-C16
O1P-F07
O2P-C12
O2P-G06
P1-d24
P1-e20
P1-e29
P1-e30
P1-e35
P1-e38
P2-e33
P2-e34
P2-e44
P2-f35
P3-f03
S1A-A4
VISUAL DOMINANCE O1P-B07
VISUAL EXPERIENCE P2-e41
VISUAL EXPLORATION P1-g22
VISUAL FEEDBACK P1-e09
VISUAL FUNCTION P3-e43
VISUAL GROUPING O2P-C11
VISUAL IMAGE RECONSTRUCTION O2P-C12
VISUAL IMPRINTING P3-f08
VISUAL MEMORY P3-g32
VISUAL MOTION P1-e31
P3-f06
P3-f12
VISUAL PERCEPTION O2P-F01
P1-e42
VISUAL RECONSTRUCTION P1-h19
VISUAL RESPONSE O1P-G11
VISUAL SALIENCY P1-e28
VISUAL SEARCH P1-e33
P1-h11
P3-e45
VISUAL STABILITY P1-e17
VISUALIZATION P2-k02
P2-k03
VISUO-MOTOR INCONGRUENCY P1-e03
VISUO-MOTOR TRANSFORMATION, MOTOR LEARNING, THROWING MOVEMENT P1-f31
VISUO-TACTILE BIMODAL NEURON P1-h14
VISUOMOTOR ADAPTATION P3-e38
VISUOMOTOR CONTROL P3-e39
S3P-B2
VISUOMOTOR IMAGERY P1-f37
VISUOMOTOR TRANSFORMATION O1P-B14
VISUOSPATIAL WORKING MEMORY S3P-A2
VLDLR P3-d05
VLSI P3-f14
VMAT2 P1-b36
VOCAL LEARNING P2-h19
VOCALIZATION P1-g12
S3A-K3
VOICE P3-h05
VOLATILE ORGANIC COMPOUNDS P1-f18
VOLTAGE SENSITIVE DYE IMAGING P1-f12
VOLTAGE SENSOR P2-k07
VOLTAGE SENSOR DOMAIN O1P-E12
VOLTAGE-DEPENDENT CALCIUM CHANNEL P3-a01
P3-a15
P3-a16
P3-b04
VOLTAGE-DEPENDENT CALCIUM CHANNELS P3-b01
VOLTAGE-GATED P3-a11
VOLTAGE-GATED CALCIUM CHANNEL O1P-E03
VOLTAGE-GATED CHANNEL P2-k21
VOLTAGE-GATED ION CHANNELS P3-b06
VOLTAGE-GATED POTASSIUM CHANNELS O1P-E01
VOLTAGE-GATED SODIUM CHANNEL P1-e44
P3-a09
VOLTAGE-SENSITIVE DYE P2-a13
P2-b06
P2-k04
VOLUNTARY INHIBITION O1P-B16
VOLUNTARY MOVEMENT O1P-B05
P2-e18
P2-e20
P2-e21
VOMERONASAL P1-f25
VOMERONASAL NEURONS P1-f27
VOMERONASAL PROJECTION P1-f15
VOMERONASAL RECEPTOR P1-f14
VOWEL P1-f10
VOXEL-BASED MORPHOMETRY P2-h15
VPAC2 P3-c30
VPM P2-f16
VRG P1-f46
VSD P2-h12
VTA P1-g21

## W

WARM P3-b42
WDR P2-j25
WEB2.0 P1-k22
WFV CELLS P2-e40
WHISKER P2-f15
WHISKER SYSTEM P2-f30
WHITE MATTER O2P-G04
WHITE MATTER REGION P2-h42
WHOLE EMBRYO CULTURE P3-k14
WHOLE-CELL PATCH-CLAMP P2-e34
WHOLE-CELL RECORDING P3-a09
WIGGLING O1P-J07
WILSON DISEASE P1-k19
WNT O2P-D01
P1-c30
P2-h39
WNT SIGNALLING O2P-D03
WNT5 P2-d05
WORD O1P-G14
WORKING MEMORY O1P-G07
P1-g23
P1-j21
P3-b20
P3-h10
WRIST MOVEMENT P1-d47
WRITTEN WORD P2-h22

## X

X-LINKED ADRENOLEUKODYSTROPHY (ALD) P1-b26
X-RAY P1-b27
X-RAY MICRO-CT P2-k10
X11 P1-h25
X11L P1-g09
XENOBIOTIC METABOLISM O2P-J04
XENOPUS P1-f14
XERODERMA PIGMENTOSUM P1-d39
XERODERMA PIGMENTOSUM(XP) P2-j29
XPA P1-j17
XPG P2-j29

## Y

YAWNING P2-f43

## Z

ZEBRA FINCH P1-f07
P2-h24
ZEBRAFISH O2P-H14
P1-c24
P1-c25
P1-c26
P1-d33
P1-f33
P2-d29

P2-e19
S2A-K3
ZEBRIN O1P-B09
P2-b35
ZENK P3-g38
ZIF268 O2P-K04
ZINC O1P-H07
P2-g07
P3-e06
P3-j09
ZINC DEFICIENCY P3-g10
ZN FINGER P1-c40
ZONISAMIDE O2P-E07

## Number

1-BROMOPROPANE P3-j10
1-METHYL-4PHENYLPYRIDINIUM (MPP+) P3-j01
2-ARACHIDONOYL-GLYCEROL P1-b15
2AFC P1-e42
3-D STRUCTURE PERCEPTION FROM MOTION P2-e36
$^{31}$P-NMR P3-j37
3D RECONSTRUCTION P1-k22
P2-k10
5-HT P2-j36
P3-b37
5-HT TRANSPORTER P1-c15
5-HT3 RECEPTOR P3-b28
5HT P2-b33
5HT1A RECEPTOR P2-b33
5HTTLPR POLYMORPHISM P1-g07
6-HYDROXYDOPAMINE (6-OHDA) P3-j01
6-OHDA O2P-D07
8-OH DPAT P2-g02

## Other

α-SYNUCLEIN P2-j34
γ-SECRETASE P1-h26

# 学会掲示板

## 【 助成・褒章案内 】

### 財団法人ノバルティス科学振興財団
### 平成20年度（2008年度）研究集会助成候補推薦要領

1．助成対象研究集会：
生物・生命科学およびそれに関連する化学の領域において、わが国で開催される研究集会に対し、運営経費の一部を助成する。助成対象は下記の選考基準に基づく。

①かなりの数の国外からの参加者を含み、国際性豊かで、学術的意義の大きい研究集会であること。
②1,000名を越す大規模な研究集会は、原則として助成の対象としないが、国内の学会および機関が主催する集会において行われるシンポジウム、招待講演、セミナーなどは助成対象とする。
③2国間の研究集会は、原則として助成の対象としない。
④他の財源から多額の収入が得られると予想される研究集会よりも、ノバルティス科学振興財団からの助成が有効となるような研究集会を優先する。
⑤平成20年4月以降平成21年3月末までに開催される研究集会であること。

2．推薦件数：　1申請者から1件に限る。
なお、ノバルティス科学振興財団の現理事、評議員、選考委員は候補者となれない。

3．助成金額：　助成金は1件5万円を限度とし、予算範囲内で助成を行う。

4．申請方法：　ノバルティス財団ホームページ（www.novartisfoundation.jp）からダウンロードした所定の用紙またはそのコピーに必要事項を記入し、ノバルティス科学振興財団事務局に提出する。これとは別に同書類を電子メールで下記アドレスへ送付する。
E-Mail：novartisfound.japan@novartis.com

5．申請受付期間：平成19年（2007年）7月より受付を開始し、平成19年（2007年）9月末にて締切る。なお、推薦締切り後も、助成総額が本年の予算額に達しない場合は、推薦の受付けを継続する。
※日本神経化学会からの推薦を必要とする場合は、平成19年8月31日（金）必着で日本神経化学会事務局宛までお送り下さい。

6．ノバルティス科学振興財団による選考方法：
選考委員会（平成19年12月開催予定）において選考した後、評議員会の承認を求め、理事会で決定する。

7．採否の通知：平成20年2月中旬に採否を申請者に通知する。

8．助成金の交付：集会開催の約2ヶ月前。

9．助成金の使途：会場使用料、外国人講演者の招聘費用、抄録集の印刷費など研究集会の開催に直接必要な経費に限る。飲食費などに使用してはならない。助成金は申請書記載の通りに使用することを原則とする。万一使途を変更する場合には、あらかじめノバルティス科学振興財団理事長の承認を求めること。

10．助成金を受けた表示：ノバルティス科学振興財団の助成を受けて行われるシンポジウム、招待講演、セミナーなどにはノバルティス科学振興財団（英文の場合にはThe Novartis Foundation (Japan) for the Promotion of Science）の名を明らかにすること。
研究集会のプロシーディングを刊行する場合には、財団法人ノバルティス科学振興財団から助成を受けた旨を明記すること。

11. 成果の報告：研究集会の成果及び会計報告を集会終了後2ヶ月以内にノバルティス科学振興財団に報告するものとする。報告書作成方法等については、集会終了後にノバルティス科学振興財団事務局より連絡する。
なお、採択された場合は、申請者氏名、所属機関、研究集会名は報道機関、ノバルティス科学振興財団ホームページ、年報で公表されます。

12. 日本神経化学会からの推薦の場合：
応募先：　日本神経化学会事務局（研究助成金等候補者選考委員会）
〒160-0016　東京都新宿区信濃町35信濃町煉瓦館
財団法人　国際医学情報センター内
電話：03-5361-7107　　FAX：03-5361-7091
E-mail：jsn@imic.or.jp

## 第39回（2007年度）内藤記念科学振興賞候補者推薦要領

1．趣旨：　人類の健康の増進に寄与する自然科学の基礎的領域において進歩発展に顕著な功績のあった研究者に対してほう賞を授与するものである。

2．候補者資格：①人類の健康の増進に寄与する自然科学の基礎的研究において独創的テーマに取り組み、進歩発展に顕著な功績を挙げた研究者
②候補者は単独とするが、異なる研究グループによる共同研究の場合には、連名であっても良い。
③候補者の再度の推薦は差支えない。

3．推薦者：　①指定学会の代表者
②内藤記念科学振興財団の役員および評議員

4．推薦方法：　所定の用紙（候補者推薦書、宛名シール）に必要事項を記入し、日本神経化学会事務局（研究助成金等候補者選考委員会）宛に送付。（応募者多数の場合は選考）

5．締切日：　2007年8月31日（金）日本神経化学会事務局必着
※学会推薦が不要で直接ご応募される場合は、2007年10月1日（月）内藤記念科学振興財団必着。

6．授賞式・顕彰：　授賞式は受賞者夫妻を招いて2008年3月19日（水）に行う。
受賞者には内藤記念科学振興賞（正賞：金メダル、副賞：500万円）を贈呈する。

7．その他：　①候補者推薦書は、できるだけワープロでご記入ください。（切り貼り可）
②推薦書用紙は両面コピーで、宛名シールは同型のシールで申請可能です。（用紙の入手は日本神経化学会事務局までご連絡ください）

## 第39回（2007年度）内藤記念海外学者招へい助成金申請要領

1．趣旨：　人類の健康の増進に寄与する自然科学の基礎的研究を行う外国の研究者を招へいする際の費用を補助するものである。

2．申請者資格：①人類の健康の増進に寄与する自然科学の基礎的研究に独創的・意欲的に取り組み国際的に高い評価を得ている外国の研究者を招へいする際の当該学術集会組織委員長
②同一年度の同一学術集会に招へいする場合の申請は1件とする。
③招へい時期が下記の期間内であること。

| 招へい時期 | 申請区分 |
|---|---|
| 2008年1月1日～2008年6月30日 | 前期 |
| 2008年7月1日～2008年12月31日 | 後期 |

④申請招へい学者が来日中止の場合は対象から除外する。

3．推薦者：　①大学関係・・・大学院：研究科長、学部：学部長、研究所：研究所長、大学共通組織（研究センター、研究施設等）：学長
ただし医学研究科・医学部がある場合などは、どちらか一方の推薦者とする。
注）センター長、施設長、病院長は推薦者となることができない。
②大学以外の研究機関・・・当財団の理事会が承認した基礎研究機関の代表責任者
ア）内藤記念科学振興財団の理事・監事および評議員
（ホームページ参照URL：http://www.naito-f.or.jp）
イ）内藤記念科学振興財団の指定した学術集会の代表者

4．推薦件数：　1推薦者につき1件（前期・後期　各々1件）

5．申請方法：　希望者　⇒　大学・研究機関事務　⇒　内藤記念科学振興財団
所定の用紙(申請書、宛名シール)に必要事項を記入し、内藤記念科学振興財団宛送付する。

6．締切日：　前期：2007年6月1日（金）　後期：2007年10月1日（月）
いずれも内藤記念科学振興財団必着。

財団法人　内藤記念科学振興財団
〒113-0033　東京都文京区本郷3-24-6ＮＫＤビル8階
電話　：03-3813-3005
ＦＡＸ：03-3811-2917
E-mail：info@naito-f.or.jp

7．選考方法・採択件数：選考委員会で審査し、評議員会の同意を得て、理事会で決定する。

8．採択件数：　前期・後期各10件
採否の結果は、前期：2007年10月、後期：2008年2月に申請者および推薦者に通知する。

9．助成額：

| エリア | 助成額（万円） |
|---|---|
| 中東・アフリカ | 80 |
| 米国・カナダ（西海岸除く）、ヨーロッパ | 60 |
| 米国・カナダ西海岸、オーストラリア、ニュージーランド | 50 |
| 東南アジア、インド | 30 |
| 中国、台湾、韓国 | 20 |

10．送金時期：　前期：2007年12月、　後期：2008年3月

11. 所定用紙の注意事項：
・申請書はできるだけワープロでご記入下さい。(切り貼り可)
・申請書用紙をコピーして使用する場合は普通紙に両面コピーして下さい。宛名シールをコピーして使用する場合は、シール用紙をご使用下さい。

12. 報告の義務：
①スポンサーについて：当該学術集会で海外学者による招待講演が行われる場合は、プログラム等に内藤記念科学振興財団（英文：The Naito Foundation）の助成によるものであることを明記する。なお、プログラム等を一部送付ください。
②研究結果報告について：組織委員長は招へい終了1ヶ月以内に所定用紙にて報告する。
③来日中止の場合について：申請招へい学者が来日中止の場合は対象から除外しますので連絡ください。

◎申請書類は所属大学及び所属研究機関の研究協力課等、助成金担当部署にお問合わせください。
無い場合　⇒　大学・研究機関事務　⇒　財団

## 第48回（平成19年度）東レ科学技術賞候補者推薦要領

1．候補者の対象：　日本神経化学会が関与する分野で、下記のいずれかに該当するもの
(1) 学術上の業績が顕著なもの
(2) 学術上重要な発見をしたもの
(3) 重要な発明をして、その効果が大きいもの
(4) 技術上重要な問題を解決して、技術の進歩に大きく貢献したもの
年齢は問いません。

2．科学技術賞：1件につき、賞状、金メダルおよび賞金500万円（2件前後の予定）

3．推薦者：　東レ科学振興会指定の学協会の代表者および東レ科学振興会の推薦委員（非公開）

4．候補者推薦件数：1学協会から2件以内および1推薦委員から1件以内

5．推薦手続：　所定の推薦書用紙に選考に必要な事項を正確に記載し、東レ科学振興会あて1部ご送付願います。
※日本神経化学会より推薦をご希望の方は、8月31日（金）までに応募書類を日本神経化学会事務局 研究助成金等候補者選考委員会あてでお送り下さい。

6．推薦締切期日：平成19年10月10日（水）必着

7．選考方法：　下記委員からなる選考委員会において選考し、評議員会において受賞者を決定します。

| | | | |
|---|---|---|---|
| 選考委員 | 浅島　誠（委員長） | 末松　安晴 | 新宮　秀夫 |
| | 清水　孝雄　本河　光博 | 奈良　坂紘一 | 竹縄　忠臣 |
| | 本島　修　松本洋一郎 | | |

選考結果は平成20年2月下旬に推薦者（日本神経化学会の代表者および東レ科学振興会の推薦委員）に通知します。

8．科学技術賞の贈呈：平成20年3月の予定

9．日本神経化学会からの推薦の場合：

応募先：　日本神経化学会（研究助成金等候補者選考委員会）
〒160-0016　東京都新宿区信濃町35信濃町煉瓦館
財団法人　国際医学情報センター内
電話：03-5361-7107　　FAX：03-5361-7091
E-mail：jsn@imic.or.jp

（注）a．候補者は、必ずしも日本神経化学会会員であることを要しません。
b．推薦を受けた候補者は、その年度および次の年度にわたって選考の対象としております。したがって平成18年度に推薦された候補者については、平成19年度候補者として再度推薦の必要はありません。ただし平成18年度の推薦以降において、追加すべき顕著な業績又は発表や受賞があり加筆が必要な場合は、再提出いただいても結構です。
c．受賞者は、原則として1件1人とします。特に複数であることを必要とするときはそれらの研究者の寄与が同等であることを示して下さい。ただし、この場合でも1件として取り扱います。
d．この科学技術賞は、所得税法（第9条第1項第13号ニ）の規定により非課税となっています。
e．推薦書の用紙は、下記ホームページからダウンロードできます。
財団法人　東レ科学振興会　http://www.toray.co.jp/tsf/index.html

## 第48回（平成19年度）東レ科学技術研究助成候補者推薦要領

1．候補者の対象：　日本神経化学会が関与する分野で国内の研究機関において自らのアイディアで萌芽的研究に従事しており、今後の研究の成果が科学技術の進歩、発展に貢献するところが大きいと考えられる若手研究者（原則として推薦時45才以下）従来の研究の多くが海外で行われていても差し支えありません。

2．研究助成金：　総額1億3千万円。1件3千万円程度まで10件程度とします。

3．推　薦　者：　東レ科学振興会指定の学協会の代表者および東レ科学振興会の推薦委員（非公開）

4．候補者推薦件数：　1学協会から2件以内および1推薦委員から1件以内

5．推薦手続：　所定の推薦書用紙に、選考に必要な事項を正確に記載し、東レ科学振興会あて1部ご送付願います。
※日本神経化学会より推薦をご希望の方は、8月31日（金）までに応募書類を日本神経化学会事務局（研究助成金等候補者選考委員会）宛お送り下さい。

6．推薦締切期日：　平成19年10月10日（水）必着

7．選考方法：　下記委員からなる選考委員会において、提出された書類により一次選考を行い、さらに面接による二次選考を経て、評議員会において決定します。
なお、二次選考対象の助成候補者の方は、平成19年12月下旬に面接をさせていただきます。

| | | | |
|---|---|---|---|
| 選考委員 | 浅島　誠（委員長） | 末松　安晴 | 新宮　秀夫 |
| | 清水　孝雄　　本河　光博 | 奈良　坂紘一 | 竹縄　忠臣 |
| | 本島　修　　松本洋一郎 | | |

選考結果は平成20年2月下旬に推薦者（日本神経化学会の代表者および東レ科学振興会の推薦委員）に通知します。

8．研究助成金の贈呈：　平成20年3月の予定

9．日本神経化学会からの推薦の場合：

応募先：　日本神経化学会事務局（研究助成金等候補者選考委員会）
〒160-0016　東京都新宿区信濃町35信濃町煉瓦館
財団法人　国際医学情報センター内
電話：03-5361-7107　　FAX：03-5361-7091
E-mail：jsn@imic.or.jp

（注）a．候補者は、必ずしも日本神経化学会会員であることを要しません。
b．東レ科学振興会では専門分野の異なる選考委員全員が選考に関与します。したがって、日本神経化学会内での選考過程で用いられた推薦書の内容が異なる分野の研究者にわかりにくいと感じられる場合は、候補者が、被推薦決定後に日本神経化学会代表者の了解を得て、推薦書の表現を手直しされても結構です。
c．この研究助成は、研究目的を達成するために有効に使用されるものであれば、どのような使途で申請されても結構です。ただし、原則として間接経費、管理経費、共通経費は助成の対象になりません。また、助成金受領決定後、その使途を変更される場合は、事前に東レ科学振興会の承諾を得ていただきます。
助成研究の期間は、特別の事情がない限り助成金受領決定の次年度から3年以内とします。
d．助成金の受領者は、研究終了時まで毎年「助成研究年次報告書」を、また研究終了時に、「助成研究終了報告書」を提出していただきます。
e．この助成金は、所得税法（第9条第1項第13号ニ）の規定により非課税となっています。
f．推薦書の用紙は、下記ホームページからダウンロードできます。
財団法人　東レ科学振興会　http://www.toray.so.jp/tsf/index.html

## 2008年度　女性科学者に明るい未来をの会　「猿橋賞」受賞候補者募集

1．本賞は自然科学の分野で、顕著な研究業績を収めた女性科学者（ただし、下記の推薦締切日で50才未満）に贈呈します。

2．本賞は賞状とし、副賞として賞金（30万円）を添えます。

3．本賞の贈呈は、1年1件（1名）です。

4．所定の用紙に受賞候補者の推薦対象となる研究題目、推薦理由（800字程度）、略歴、推薦者（個人・団体）、及び主な業績リストを記入して、主な論文別刷10編程度（2部ずつ、コピーも可）を添え、7．の送付先までお送り下さい。推薦書類はhttp://www.saruhashi.net/からもダウンロードできます。

5．締切は2007年11月30日（金）必着です。

6．第28回の贈呈式は、2008年5月、東京において行う予定です。

7．推薦書類送付先：　〒168-0071
杉並区高井戸西3-6-26　古在由秀方
女性科学者に明るい未来をの会
（封筒には、「猿橋賞推薦書類」と明記して下さい。書類は、猿橋賞選考のために選考委員会などで用いられます。書類は返却いたしませんのでご了承下さい）

なお、この件についての問い合わせは、下記に電子メールでお願いいたします。
Saruhashi2006@saruhashi.net

※日本神経化学会より推薦をご希望の方は、選考の上推薦いたしますので、10月31日（水）までに下記の住所へ申請書類をお送り下さい。
〒160-0016
東京都新宿区信濃町35信濃町煉瓦館
財団法人　国際医学情報センター内
日本神経化学会事務局（研究助成金等候補者選考委員会）宛

## 【 セミナー・講習会 】

### 第57回科学講演会

主　　催：財団法人　東レ科学振興会
と　　き：平成19年9月21日（金）
開　場：16時30分
開　演：17時00分　　終　演：20時00分
と こ ろ：有楽町朝日ホール
東京都千代田区有楽町2-5-1　有楽町マリオン11階（JR有楽町駅前）
テ ー マ：プレートの沈み込みとその行方
Ⅰ．日本の巨大地震と超巨大地震
東京大学地震研究所教授　　島崎　邦彦
Ⅱ．沈み込んだプレートはどこへ？
独立行政法人 海洋研究開発機構
地球内部変動研究センター長　深尾　良夫
そ の 他：＊入場無料・予約不要・当日会場先着順（定員630名）
・ご来聴の方には、後日講演記録を進呈いたします。

問合せ先　：財団法人　東レ科学振興会
千葉県浦安市美浜一丁目8番1号
Tel：047-350-6103　　Fax：047-350-6082
E-mail：torayzaidan@tgnts.toray.co.jp
URL：http://www.toray.co.jp/tsf/index.html

### 第21回国際哺乳類ゲノム会議
### —21st International Mammalian Genome Conference (IMGC2007)—

日　　時：平成19年10月28日（日）～31日(水)
場　　所：京都テルサ（京都勤労者総合福祉センター）
主　　催：国際哺乳類ゲノム学会（IMGS）、独立行政法人理化学研究所
開催責任者：林崎良英（独立行政法人理化学研究所ゲノム科学総合研究センター
遺伝子構造・機能研究グループプロジェクトディレクター）
※詳細は下記URLをご参照ください。
http://www.imgc2007.com/

## 千里ライフサイエンス第100回記念セミナー
## 「細胞生物学・免疫学における日本発オリジナル研究―発見の発端と展開」

趣旨：今年は、この3月に退任した岡田善雄当財団前理事長（現特別顧問）が細胞融合に関する論文を発表してから50年、さらに当財団主催のセミナーが創立以来100回目となります。そこでこれらを記念して本特別セミナーを企画しました。岡田前理事長をはじめ細胞生物学・免疫学分野で大きな業績を残された世界的に著名な5名の研究者を講師にお招きし、それぞれの研究について、発見のきっかけ、重要性、発見当時の位置づけ、その後の発展、今日的意義付け等を講演していただきます。科学あるいは研究の本質がどういうものであるかを特に若い研究者に考えていただく手がかりになれば幸いです。

プログラム：
1. 開会挨拶
　千里ライフサイエンス振興財団　理事長　岸本忠三
2. 体細胞培養と細胞融合のドッキング時代―昔を回顧して
　千里ライフサイエンス振興財団　特別顧問　岡田善雄
3. 多細胞体制の構築と崩壊
　理化学研究所発生・再生科学総合研究センター　センター長　竹市雅俊
4. サイトカインの研究－遺伝子の発見から分子免疫学の展開へ
　東京大学大学院医学系研究科　教授　谷口維紹
5. 病原体認識受容体 Toll-like receptors 研究への道
　大阪大学微生物病研究所　教授　審良静男
6. Bリンパ球分化因子から抗体医薬へ―IL-6と共に35年
　大阪大学大学院生命機能研究科　教授　岸本忠三
7. おわりに
　千里ライフサイエンス振興財団　理事長　岸本忠三

日時：平成19年11月13日（火）10:00 － 17:00

場所：千里ライフサイエンスセンタービル5階ライフホール

主催：（財）千里ライフサイエンス振興財団

定員：300名

参加費：無料

申込要領：氏名(ふりがな)、所属機関、部署、〒所在地、電話番号、Eメールアドレスを明記の上、Eメールで下記宛お申し込み下さい。

申込先：Eメール sng@senri-life.or.jp
（財）千里ライフサイエンス振興財団　村上和久
〒560-0082　大阪府豊中市新千里東町1-4-2
千里ライフサイエンスセンタービル8階
TEL：06-6873-2001　　URL：http://www.senri-life.or.jp

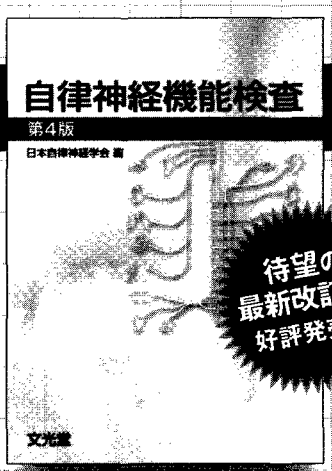
# MEMO

自律神経機能検査のスタンダードが7年ぶりに大改訂！

# 自律神経機能検査

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学会編集による自律神経機能検査法のスタンダードの最新版．7年ぶりとなる今回の改訂では，新規15項目を追加しただけでなく，7割の項目で執筆者を新たにし，現状に沿った内容に刷新した．総論については，最新の動向を記して自律神経学のテキストとしても役立つように編集．各論については，検査を実施するときのマニュアルとして「原理・目的」「方法」「基準値・判定」「適応と禁忌」「注意点」を記し，役立つ内容になっている．神経内科医のみならず，検査技師にもおすすめの1冊．

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2. 自律神経系の中枢制御
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5. 自律神経反射
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7. 自律神経機能の地域性
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5. CASS

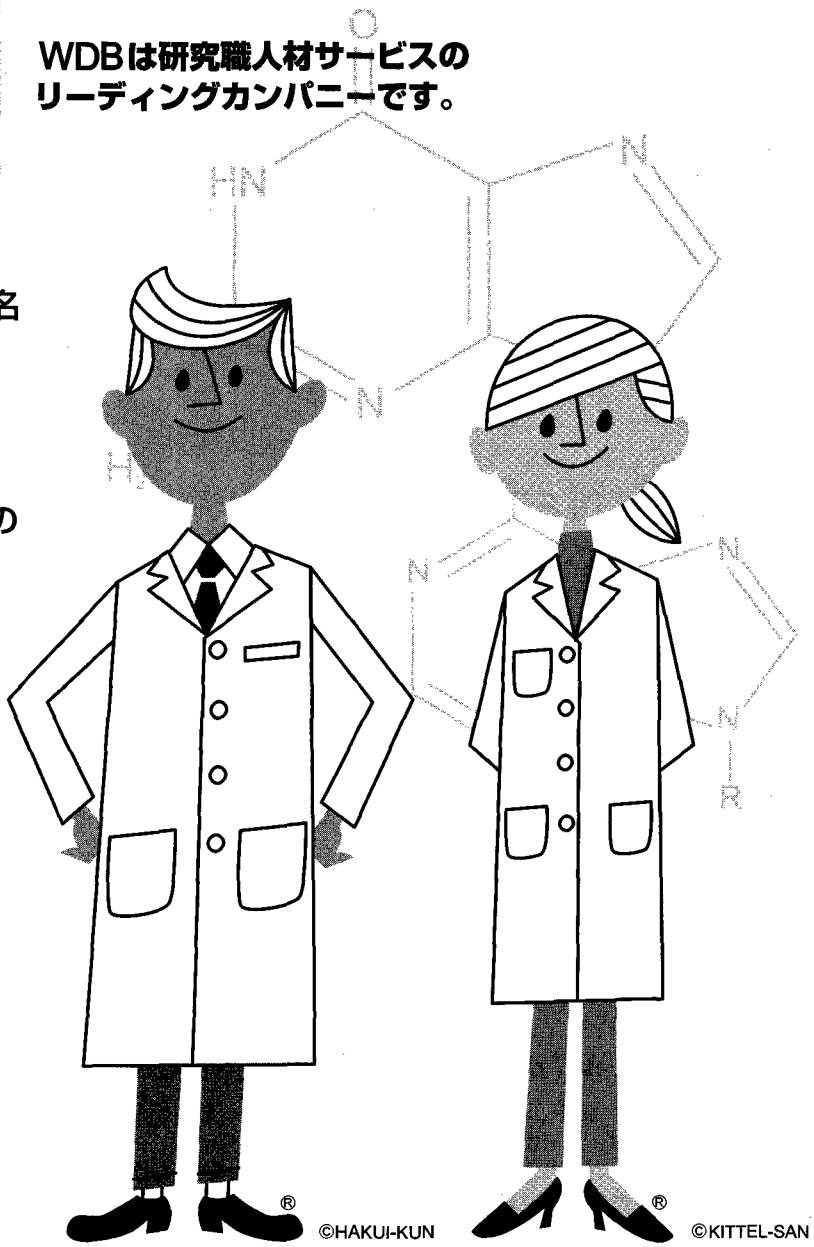
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3. 機能的核磁気共鳴法
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- ■神経蛋白制御研究
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# BRAIN and NERVE 神経研究の進歩

## 『脳と神経』と『神経研究の進歩』の統合誌として2007年発刊




### 統合誌の特徴

『脳と神経』誌と『神経研究の進歩』誌は、2007年1号より統合し、『BRAIN and NERVE—神経研究の進歩』として新たにスタートしました。

①「大特集・特集・総説・投稿論文掲載」を柱とした、質・量ともに専門医レベルに相応しい「和文神経科学専門誌」。

②『神経研究の進歩』のレビュー方式の踏襲、また『脳と神経』の臨床志向も継承し、基礎から臨床にわたる「神経科学の現在」を理解するのに欠かせないテーマを取り上げる本格的なレビュー誌。

③日本の有益な原著論文を積極的にリクルートして掲載。"これ1冊で神経科学領域における必要かつ十分でupdateな情報"を獲得。

### 2007年7月号 (Vol.59 No.7) 増大号

**特集 情報伝達処理におけるグリアの機能と異常**

グリア研究の新しい展開 ……工藤佳久
小脳プルキンエ細胞の興奮性シナプス伝達における
グリア型グルタミン酸トランスポーターの役割 ……小澤瀞司
アストロサイトにおけるグルタミン酸トランスポーターの機能
……田中光一
介在ニューロン付随性グリア細胞による神経活動の修飾
……山崎良彦・他
アストロサイトにおける神経伝達物質の開口放出 ……高橋正身・他
ATPを介したニューロン・アストロサイト相互調整 ……小泉修一・他
グリア細胞におけるG蛋白質共役型受容体の機能 ……古田晶子・他
哺乳類中枢神経系におけるD-セリンの役割
……Zhao Ying-Luan・他
アストロサイトにおけるアミノ酸代謝異常と神経疾患
—ホモシスチン尿症を手がかりとして……榎戸 靖
神経因性疼痛におけるミクログリアの関与 ……井上和秀
グリア細胞機能異常動物の開発 ……田中謙二・他
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ミクログリア可視化トランスジェニックマウスの作製
……平澤孝枝・他
イン・ビボにおけるアストロサイトの動態計測 ……平瀬 肇・他

8月号 特集 パーキンソン病の分子遺伝学—最近の進歩
9月号 特集 パーキンソン病の認知障害

### 2007年バックナンバー 特集テーマ

1号 (Vol.59 No.1)
**高次視覚研究の最近の進歩**
2号 (Vol.59 No.2)
**進行性多巣性白質脳症の新しい展開**
**—PMLが治る時代に向けて**
3号 (Vol.59 No.3)
**分子イメージング**
4号 (Vol.59 No.4) 増大号
**最近注目される脳神経疾患治療研究**
5号 (Vol.59 No.5)
**脳画像最前線**
6号 (Vol.59 No.6)
**職業性神経障害の新しい展開**

- 判型：A4変型
- 発行：月刊（3冊の増大号を含む年12冊）
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- 年間購読料：34,860円（税込）

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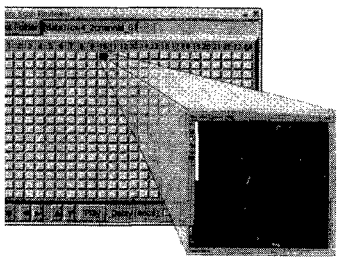
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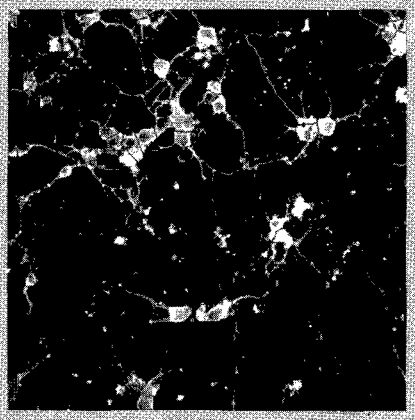


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混在培養におけるニューロンのブランチポイント、神経突起伸張、その他形態学的な特徴を測定します。また神経伝達物質の違いによる「subpopulation」での伸張測定値についてレポートできます.

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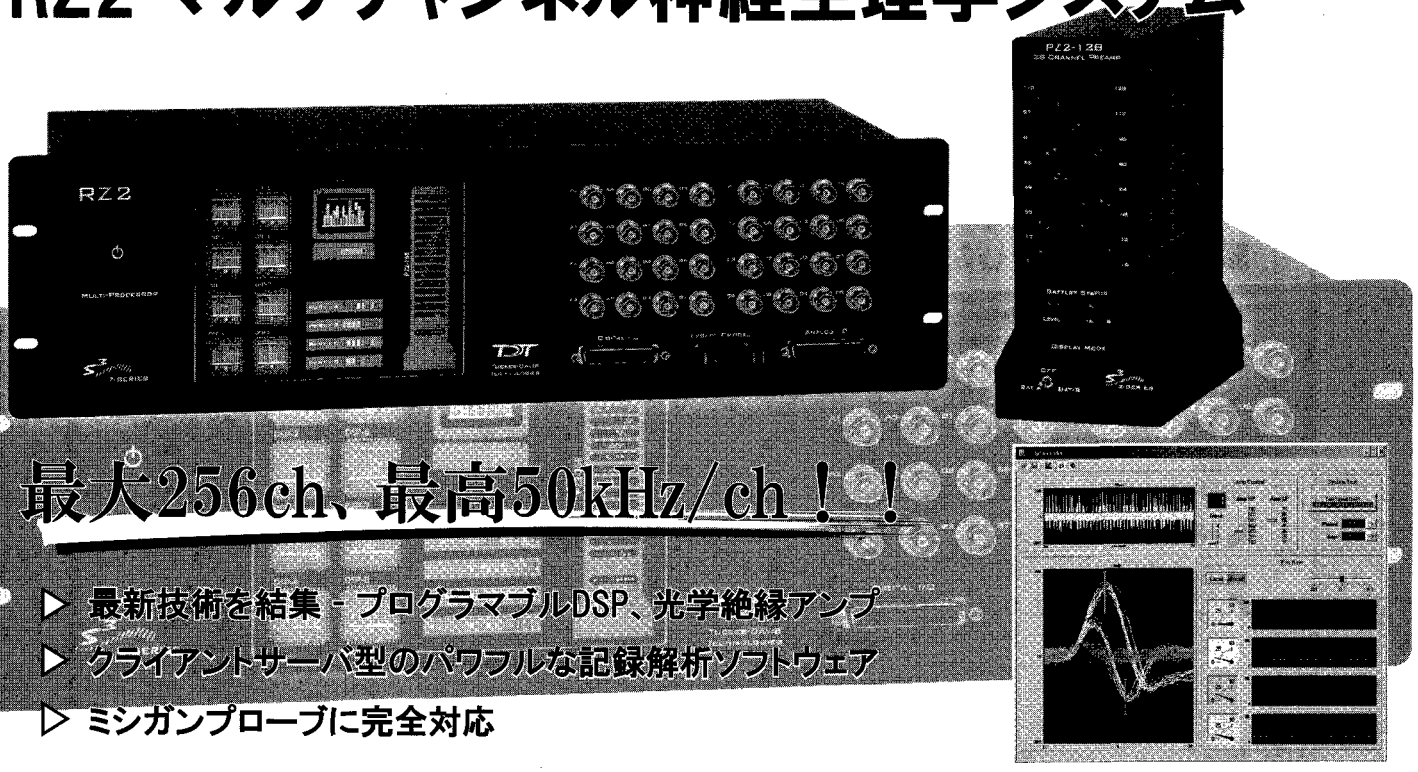
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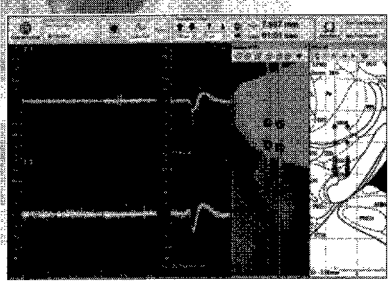
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## 超高感度・高安定性 微量生体試料分析システム EiCOM

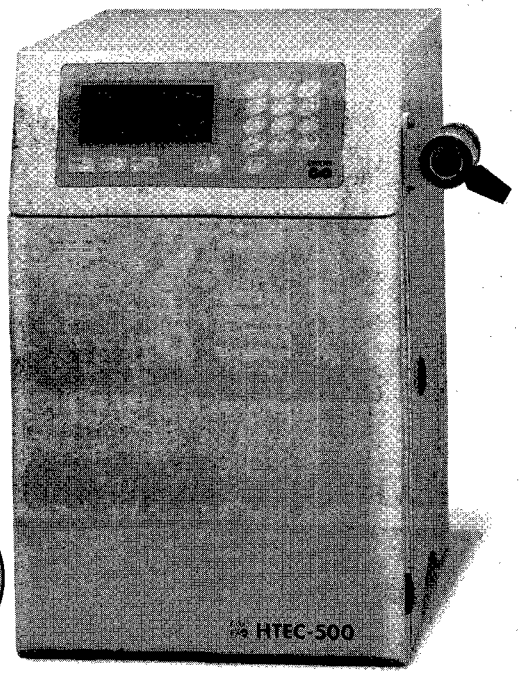
# HTEC-500

**生体内神経伝達物質の高感度分析に
多用されているエイコム300シリーズ。
HTEC-500はさらに安定性・
感度が上がりました。**

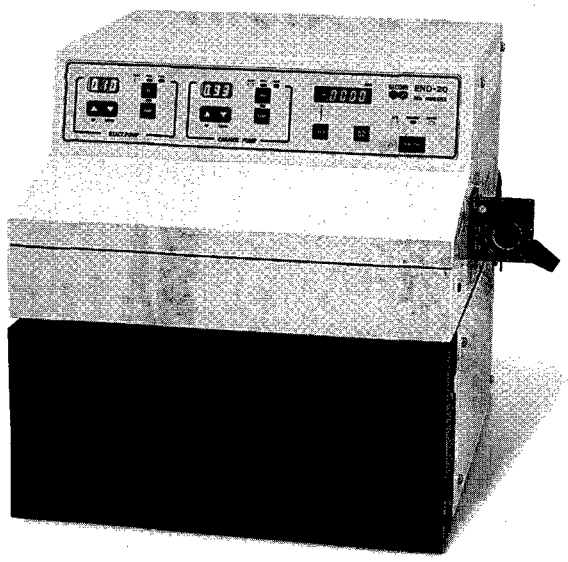
生体内神経活動をリアルタイムで観察することは、
神経科学的研究を行う上で極めて大きな目標の一つです。
エイコムでは今以上の高感度分析には一体化が
必要だと考え分析に必要なユニットを
すべてまとめてHTEC-500型を完成しました。
専用器的な高感度分析計として、限りなく
目標に近づくことを目指します。

- 感度はドーパミン・セロトニンで30fgを実現。
- 設置面積はB4サイズ。

マイクロダイアリシスの分析計としても最適です。




## 酸化窒素分析システム EiCOM




# ENO-20

**生体液中の窒素酸化物測定
のために開発されました。**

液体クロマトグラフとグリース試薬法の
ドッキングで大きな特長を得ました。

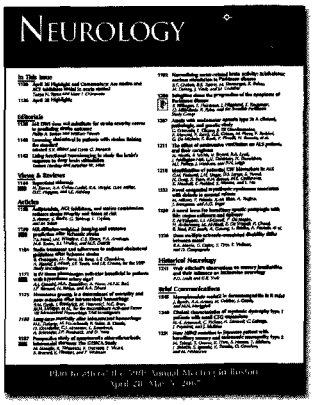
- 0.1ピコモルの他に例を見ない高感度。
- $NO_2^-$、$NO_3^-$を高精度で分離測定。
- 安定性と再現性が良い高稼働率測定器。

$NO_2^-$、$NO_3^-$の測定から生体内NOの動態を知ることができます。

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# 神経科学関連雑誌 from Lippincott Williams & Wilkins



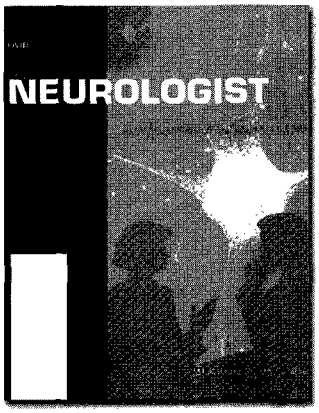

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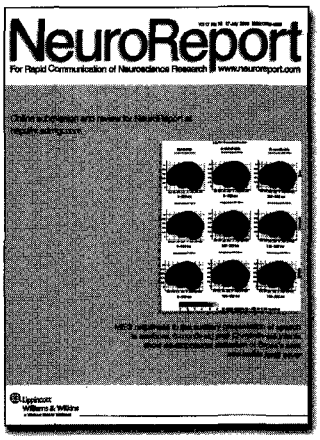

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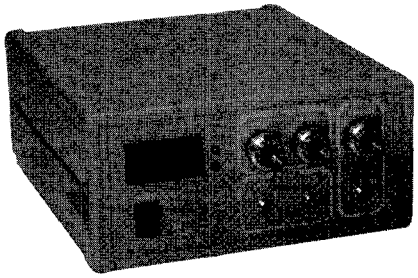
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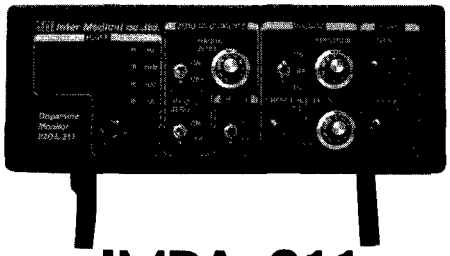
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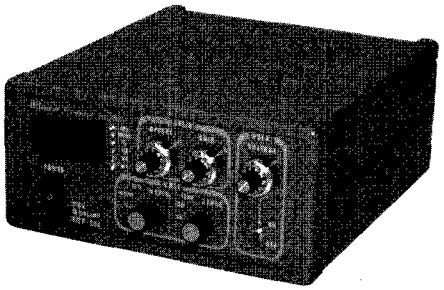
## IMN-101
## NO Monitor



新発売

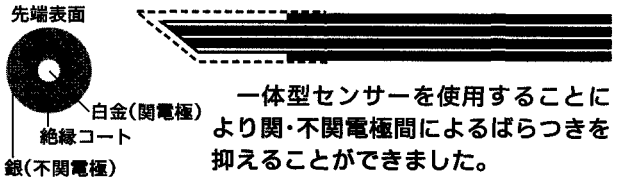


## IMP-201
## PO2 Monitor

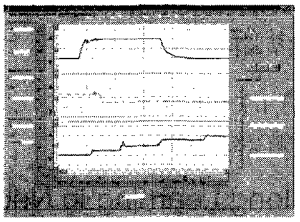


## IMDA-311
## Dopamione Monitor

コンビネーション電極

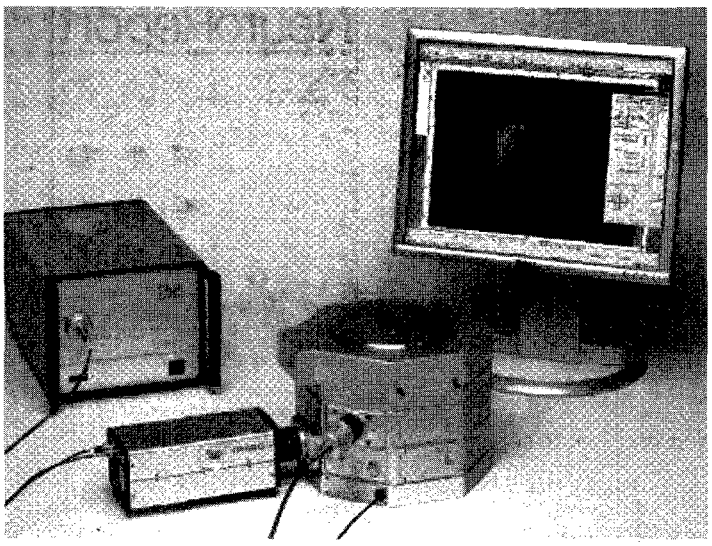



一体型センサーを使用することにより関・不関電極間によるばらつきを抑えることができました。

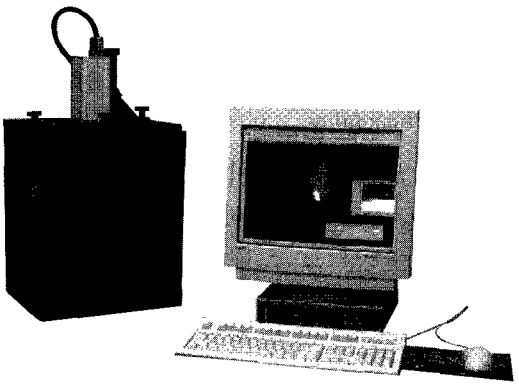


I-REC
データ取得ソフト

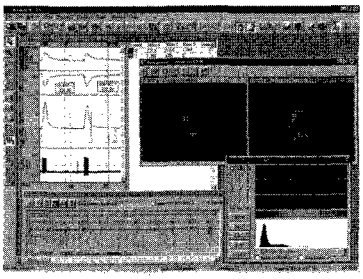
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蛍光画像取得解析ソフトウェア ●蛍光画像と電気生理を同時に取得可能

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生細胞タイムラプス計測向け光学顕微鏡システム

# DeltaVision® Core

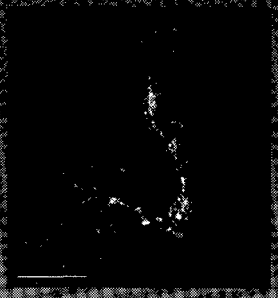
**生細胞計測に最適な機能搭載**

生細胞ダイナミクスの自動補足機能を標準装備
（Cell Tracking機能 | Auto Focus機能）
リアルタイムデコンボリューション機能
リアルタイムZ軸スイープ　新機能（OAI）搭載

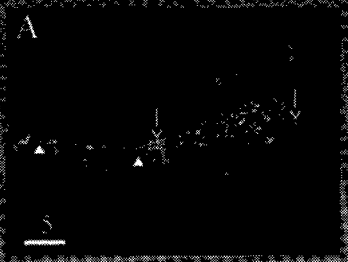
**優れた画像分解能**

3Dデコンボリューション採用による微細構造を
補える解像度を実現（0.2μm以下の分解能）

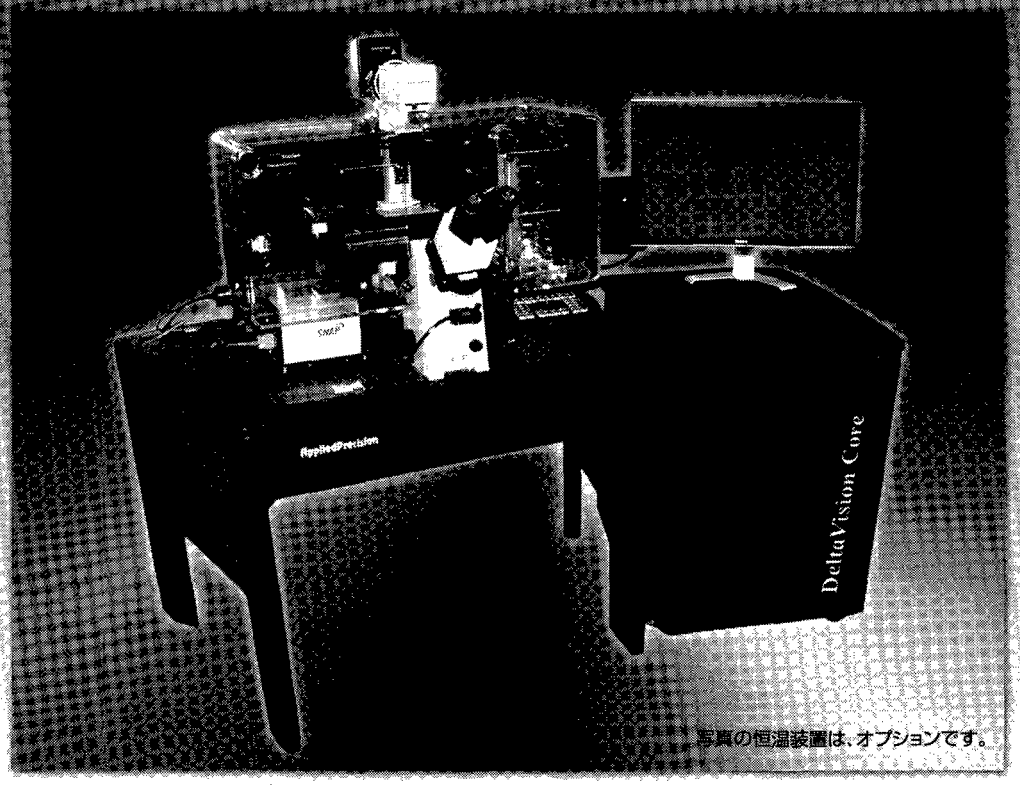
神経系画像



神経突起画像
（Rat：Growth Cone）




DCG顆粒（tPA-EGFP染色）




写真の恒温装置は、オプションです。

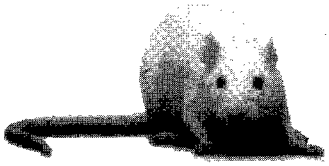
★姉妹品（Personal DV）や冷却CCDカメラ（SPOT）シリーズ等を取扱っています。

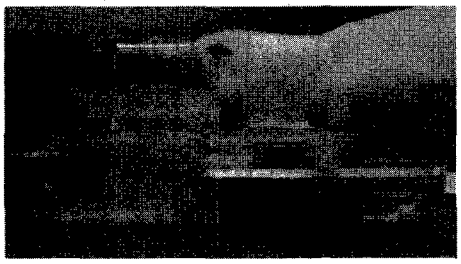
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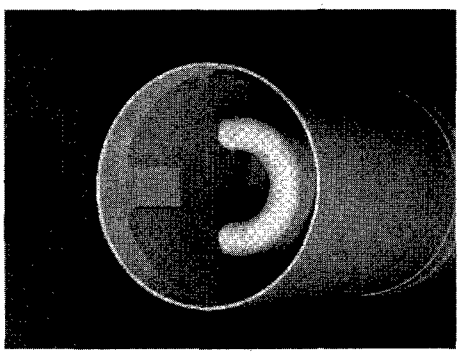
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バイオ計測G
〒135-0042　東京都江東区木場5-6-30
TEL.03(3820)1712　FAX.03(3820)1728
URL http://www.sekitech.co.jp

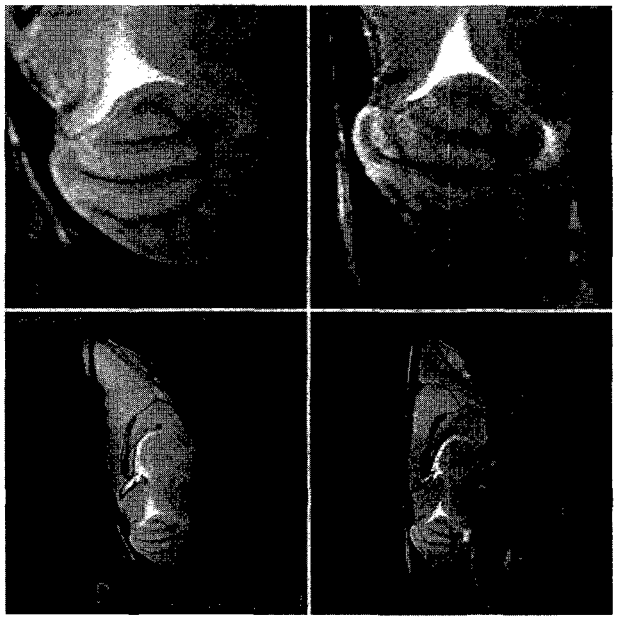
# 高感度・高解像度MRI CryoProbe™











**RARE (8 echoes)**
**TR: 4200 ms**
**TE: 39.6 ms**
**47 x 52 μm² in plane**
**0.5 mm slice thickness**

**測定時間:6分40秒**

**CryoProbe　　QD Coil**

**Bruker BioSpin**

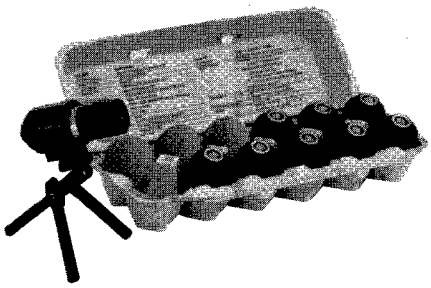

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ホームページアドレス：http://www.bruker-biospin.com　　メールアドレス：info@bruker-biospin.jp

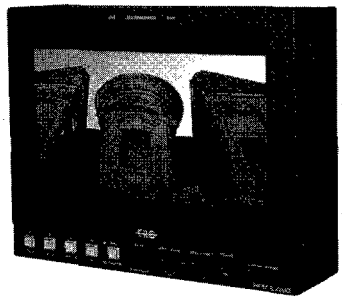
# ハイビジョン

## のシステムならおまかせ下さい!

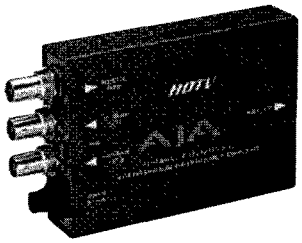
- ハイビジョン小型のカメラをお探しなら?
- ハイビジョン高精細のモニターをお探しなら?
- ハイビジョン信号変換器をお探しなら?



超小型ハイビジョンカメラ



高精細小型ハイビジョン液晶モニタ



ハイビジョン信号変換機

## 今すぐご連絡下さい!

↓

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- Eメールはコチラ Email erg@erg-ventures.co.jp
- お電話はコチラ TEL 03-3760-8161

詳しくは弊社ウェブサイトをご覧下さい。 株式会社 エルグ ベンチャーズ 検索

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Email : erg@erg-ventures.co.jp

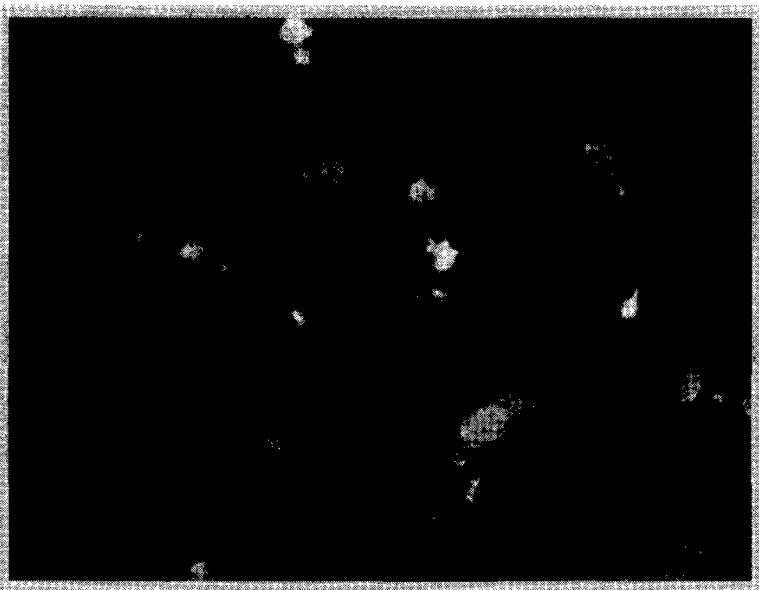
Wako

# Anti Iba1, Rabbit
## (for Immunocytochemistry)

**ミクログリアの二重染色に最適!!**

Iba1はマクロファージ/ミクログリアに特異的に発現している分子量17,000のカルシウム結合タンパク質です。本品はミクログリアと特異的に反応するウサギポリクローナル抗体ですので、アストロサイトに特異的なGFAPモノクローナル抗体などとの二重染色に適しています。

- ■形　　状：TBS溶液(0.5mg/ml)
- ■免 疫 原：Iba1のC末端に相当する合成ペプチド
- ■特 異 性：ミクログリア、マクロファージに特異的に反応する。ニューロン、アストロサイトとは交差反応しない。ヒト、マウス、ラットIba1と反応する。
- ■精　　製：抗原アフィニティ精製
- ■使用濃度：Immunocytochemistry　1～2μg/ml



rat primary mixed culture cellsの二重染色像

データ提供:国立精神・神経センター神経研究所代謝研究部

**参考文献**


1) Imai, Y., Ibata, I., Ito, D., Ohsawa., K. and Kohsaka, S.: *Biochem. Biophys. Res. Commun.*, **224**, 855(1996).
2) Ito, D., Imai, Y., Ohsawa, K., Nakajima, K., Fukuuchi, Y. and Kohsaka, S.: *Brain Res. Mol. Brain Res.*, **57**, 1(1998).
3) Ohsawa, K., Imai, Y., Kanazawa, H., Sasaki, Y. and Kohsaka, S.: *J. Cell Sci.*, **113**, 3073(2000).
4) Sasaki, Y., Ohsawa, K., Kanazawa, H., Kohsaka, S. and Imai, Y.: *Biochem. Biophys. Res. Commun.*, **286**, 292(2001).
5) Kanazawa, H., Ohsawa, K., Sasaki, Y., Kohsaka, S. and Imai, Y.: *J. Biol. Chem.*, **277**, 20026(2002).


**019-19741 Anti Iba1, Rabbit (for Immunocytochemistry) 免疫化学用 50μg (100μl)**

この他、Anti Iba1, Rabbit (for Western blotting)、016-20001、50μg (100μl)もございますので、ご照会下さい。


**和光純薬工業株式会社**

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HITACHI

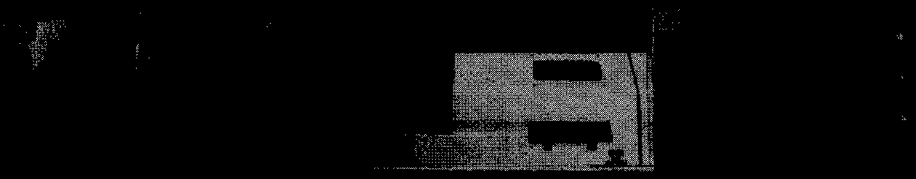
日立ハイテク

## 日立液体クロマトグラフ質量分析計

# NanoFrontier LD

高性能な NanoFrontier LD は
低分子化合物からタンパク質などの高分子化合物の構造解析
まで、さまざまな分野でのニーズにお応えします。

【応用分野】
天然物　合成物　代謝物　メタボローム　タンパク質同定
バイオマーカ探索　比較解析　精密質量分析など



特長

- リニアイオントラップを使用しているため、高感度で実用的なMS$^n$分析が可能
- 目的サンプルに応じて選択できる2種類のCIDモードを搭載
  - ・リニアイオントラップでのCID ⇒ MS$^n$分析が可能
  - ・コリジョンチャンバーでのCID ⇒ 低質量側のフラグメントイオンを検出
  - ＊イオントラップで問題となる低質量側のカットオフがありません
- 新規自社開発ADCの搭載により広いダイナミックレンジを実現
- 日立独自のイオン光学設計のTOFを搭載することで、安定した質量精度・高分解能の分析を実現
- 構造解析に有効なMS$^n$分析が可能（タンパク質同定、低分子構造解析）
- 精密質量分析（組成式の推定）が可能

株式会社日立ハイテクノロジーズ　〒105-8717　東京都港区西新橋一丁目24番14号　電話 ダイヤルイン（03）3504-7301
URL http://www.hitachi-hitec.com/science/

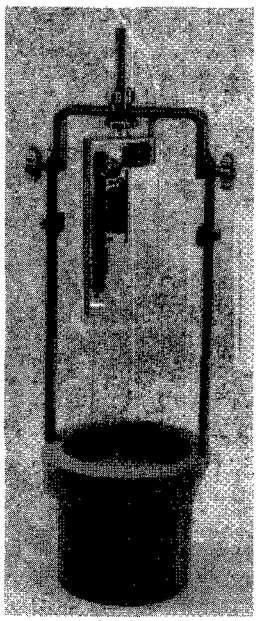
# Thomasの実力を御存知ですか？

## ドイツ技術の精緻をあなたの研究室へ

### 超小型マイクロマトリックスシステム

**超軽量システム誕生、わずか63g!!**

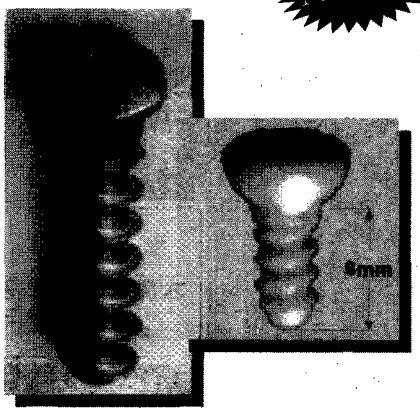
NEW!!



最新超軽量マイクロマトリックスシステムは、MRI計測に対応した埋め込み型グリッドアレイチェンバーと、超小型モータードライブのマイクロマトリックスから成り立っています。電気生理とMRI計測を両立させることが可能です。グリッドアレイ採用により精確な位置再現性が確保されます。掌にのる大きさを、是非実感して下さい（サイズ：チェンバーφ38mm：ドライブ53mm）

### セラミックネジとチタンネジ

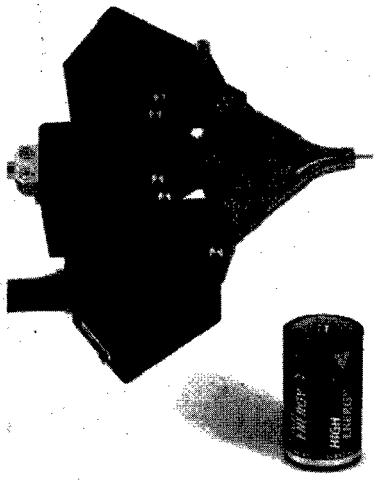
NEW!!




チャンバー等の埋め込み、固定用にセラミックとチタン製のネジを御用意致しました。セラミックネジはMRI計測に最適です（消毒済み製品を、お届け致します。）

### 48チャネルミニマトリックスドライブ

NEW!!



**ミニマトリックス**は、霊長類脳に固定可能な多電極用小型軽量マニピュレータで、最小軸径25μmのワイア電極を扱うことが可能です。Thomasのオリジナル（Eckhorn）回路採用により、ヒステリシス皆無の脳内マニピュレーション動作環境をお届けします。**最新型の48チャネルモデルでは、3台のミニマトリックスドライブを組み合わせることにより、12本の独立電極、または12×4本電極（48チャネル）が操作可能となっております。**また御使用に当たっては、頭蓋固定用チャンバーや固定器具（MRI対応製品）のカスタマイズ化にも対応致しております。今までにない小型システムを是非お試し下さい（単一電池と大きさをお比べ下さい！！！）

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弊社ホームページで確認下さるか、または弊社まで直接にお問い合わせ下さるよう、是非、お願い致します。


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BIO MATRIX RESEARCH

# ご希望の抗体を、いち早く製品化します！

## BMR 抗ヒト転写因子モノクローナル抗体シリーズ

ヒトゲノムプロジェクトが2003年に終了し、ヒトの遺伝子は約2万2千個に過ぎないと推定されています。
しかしながらヒト細胞から見つけられるタンパク質は10万個以上と推定され、このギャップを埋めるためにハイスループットなタンパク質解析が求められております。
そこでBMRでは、1st ターゲットとして「ヒト転写因子」に対するモノクローナル抗体を網羅的に作製し、迅速にお客様へご提供いたします。

**抗ヒト転写因子マウスモノクローナルを網羅的に作製！**
モノクローナル抗体を網羅的に作成中です。第一弾はヒト転写因子がターゲット

**同一容量！ 同一価格！** 価格 = ￥39,000 均一
濃度=100ng/μL 液量=500μL 各種マウスから作製

**ヒト由来細胞を用いて確認！**
ヒト由来細胞を用いたウエスタンブロッティングなどにより、確認を行っています

● 最新抗体ラインナップにつきましては弊社ホームページをご覧ください。
http://www.iwai-chem.co.jp/products/bmr/antibody.htm

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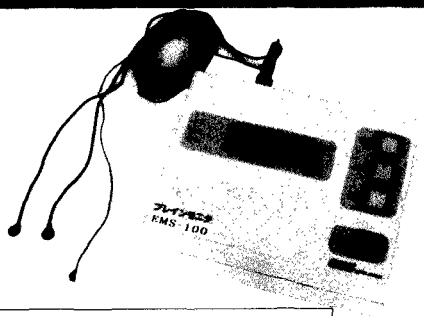
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# 前頭前野脳活動状態 を どこでも、いつでも 観察できる

# ブレインモニタ

1チャンネル EMS-100
2チャンネル（右脳・左脳同時測定） EMS-200 （保険適用機種）

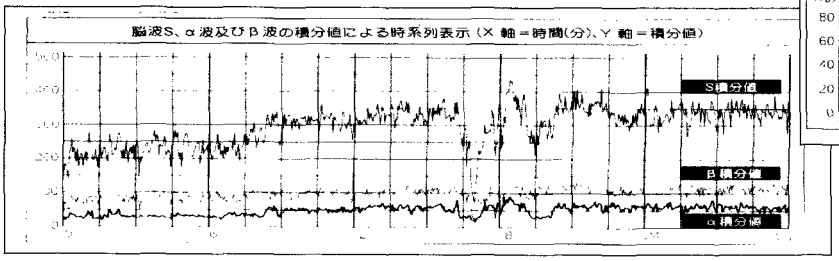


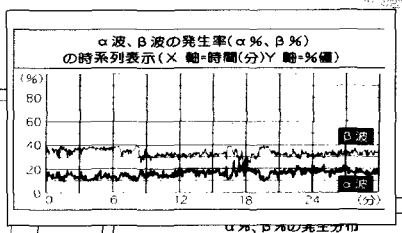

●前頭前野脳波を数値積分し、専用ソフトで数画面の解析表示し収納記録します。

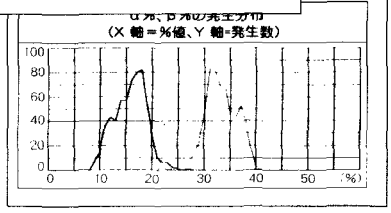
●α波とβ波を主とした時系列及びヒストグラム表示で容易に脳活動の解読と解析ができます。

●オランダ・ノルダス社「行動解析ソフト」（オプション）の生体情報として同期できます。

●脳波原波形をパソコンに収納表示記録しファイル化できます。
（オプション：別途デジタル記憶装置を使用）




[用途] ・行動中の脳波計測（読み、書き、会話、散歩、マラソン、車両運転・・・・・）
・情 動 計 測（精神神経科、小児科、歯科、心理、看護、教育・・・）等

総発売元/資料請求先 株式会社アスク
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# 研究開発の人材派遣
# 東京ビジネスサービス

私共は当分野のフロントランナーとして、数々の大学や独立行政法人、国立研究機関および民間企業の研究施設にスタッフを派遣して参りました。
科学研究費は勿論のこと、その他の研究資金を有効活用するにあたっても人材派遣を利用すれば、煩わしい雇用管理や事務作業の手間が省け、研究に専念していただくことができます。
まずはお問い合わせをお待ちしております。


**東京ビジネスサービス**

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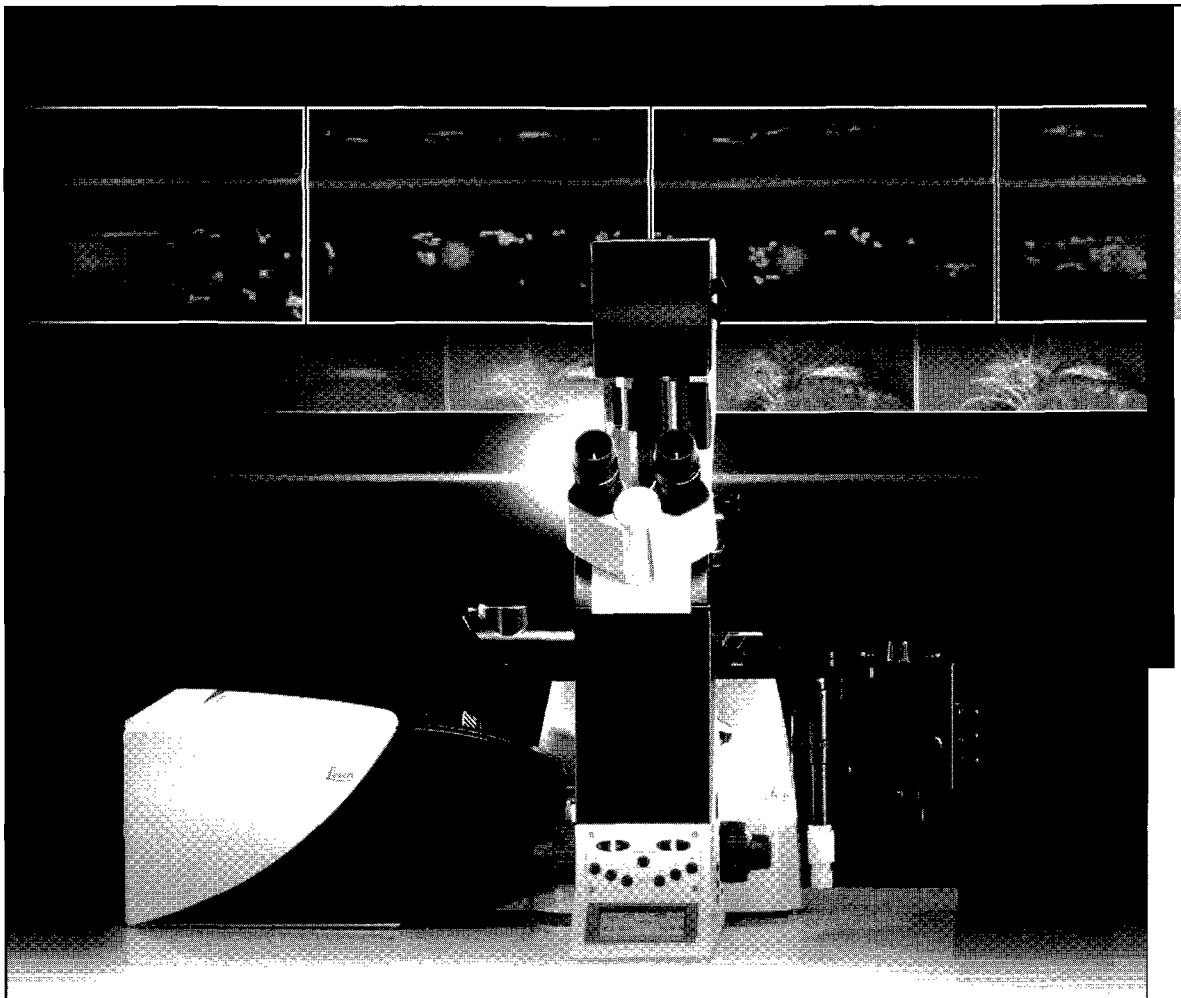
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**優秀な研究スタッフ、登録受付中！**

ただいま、意欲あふれるスタッフの登録も受付中です。詳しいことは、お気軽にお問い合わせください。

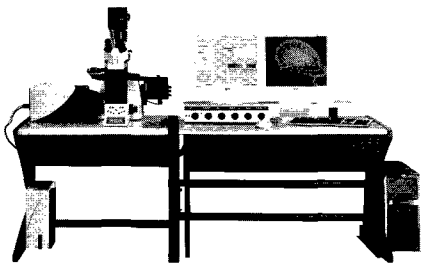
※ホームページでも登録可能です。



# 速さと美しさ、高次に兼備。
# ライブイメージングの理想鏡。

タンデムスキャナー共焦点顕微鏡

## ライカ TCS SP5



**ライカの歴史と技術、ここに凝縮。比類なき共焦点顕微鏡**

- 250画像／秒（512×16画素）および25画像／秒（512×512画素）のライブイメージング。共振ガルバノ方式で分解能を下げず、多重染色サンプルの鮮明画像を高速取得。
- 高速ライブ画像と高分解能画像を高度に両立させる世界初、タンデム（2組）走査方式。
- 効率の良い蛍光検出が行え、波長の設定が自由なプリズム分光スリット方式。
- ライカ独自のモジュラー方式を採用、エントリーレベルからアップグレード可能。

Leica
MICROSYSTEMS


ライカ マイクロシステムズ 株式会社
〒108-0072 東京都港区白金1-27-6 白金高輪ステーションビル6F Tel.03-5421-2814 Fax.03-5421-2891
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●E-mail:marketing@leica-microsystems.co.jp

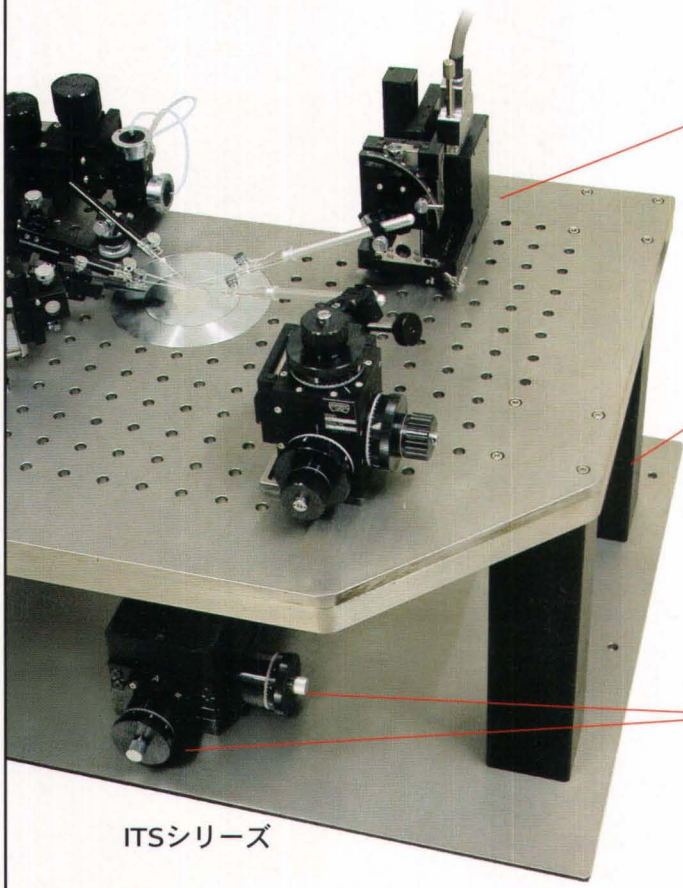

製品に関する詳しい情報はWebサイトでご覧いただけます。 http://www.leica-microsystems.co.jp

NARISHIGE JAPAN NARISHIGE

Electro-physiology

# 安定が一番

## パッチクランプでの安定性と操作性を向上させます
## ナリシゲのアイソレーションシステム



ITSシリーズ

### マルチセットが可能な広いテーブル

広く厚みのある孔付きのステンレス製テーブルを採用。複数のマニピュレーターをマグネットやネジで強固に取り付けることができます。

### 水平で安定したテーブル

テーブルの高さ調整にスペーサー方式を採用。高さ調整時の水平出しが容易です。また、テーブルを支える支柱も太く強固なものを採用、梁を加えたことで耐震性、安定性を更に高めています。

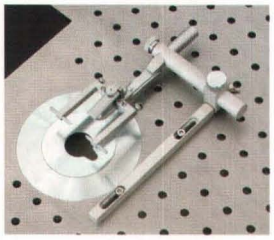
### 大型ハンドルで滑らかなXY移動

顕微鏡の移動が楽に行える大型ハンドルを採用。きめ細かな操作が行えるので、容易に観察ポイントを変えられます。また、高精度に加工された強固なスライダーにより滑らかな動きを実現しています。



### 固定器を使った小動物実験に対応

電動、液圧、手動など多彩なマニピュレーターをラインアップしています。マウスなどの小動物を用いた実験のために固定器を取り付けることも可能です。



詳しくは当社担当までお問い合せください。

インターネットホームページなら、他の各種製品の詳細も手にとるように判ります。 http://www.narishige.co.jp

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