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第47回日本神経化学会（大阪）大会

抄録号

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第47回日本神経化学会大会

Neuro2004

第27回日本神経科学大会

合同大会

プログラム・抄録集

会　期：2004年9月21日（火）～23日（木）

会　場：大阪国際会議場
〒530-0005　大阪市北区中之島5-3-51
TEL：06-4803-5555（代表）

主　催：日本神経化学会
日本神経科学学会

共　催：（財）ルイ・パストゥール医学研究センター

第47回日本神経化学会大会　　大会長　　遠山　正彌

第27回日本神経科学大会　　大会長　　村上富士夫

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Neuro 2004

Neuro2004 委員一覧

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和中　明生　奈良県立医科大学

Neuro2004大会事務局

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TEL：06-6227-0157　FAX：06-6229-2556
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理事会議事録

2004年度第1回理事会

日　時：2004年3月3日（水）13:00～17:00
会　場：主婦会館プラザエフ「パンジー」
出席者：御子柴克彦、熊倉鴻之助、岡野　栄之、神庭　重信、高坂　新一、佐藤　　真、島田　昌一、
高松　　研、辻　　省次、遠山　正彌、鍋島　俊隆、馬場　明道、柳澤　勝彦
植村　慶一、宮武　　正（監事）
池中　一裕（国際対応委員長）
欠席者：井上　和秀、野村　靖幸（理事）
西澤　正豊（奨励賞選考委員長）

議　題

議事に先立ち、御子柴理事長より挨拶がなされた。

Ⅰ．報告事項

1．庶務報告

岡野庶務担当理事より、庶務報告が行われた。

・総会員数1,659名（2003年12月31日現在）

名誉会員	15名
一般会員	1,355名
学生会員	254名
賛助会員	22名
団体会員	13名

・会員異動状況（2003年1月1日～2003年12月31日）

新入会者	138名
退 会 者	191名

尚、入退会者の会員種別内訳は新入会員138名（正会員60名・学生会員76名、賛助会員2社）、退会会員191名（正会員139名・学生会員50名、賛助会員2社）だった。

2．出版・広報報告

柳澤出版・広報担当理事より以下の報告がなされた。

学会誌「神経化学」43巻1号は原稿を印刷会社に入稿し予定通りに発行が行われる。

学会Webサイトでは会員検索を行うことができるが、英語版のWebサイトでも同様の事ができるかを検討した。システム自体を英語化することは可能だが、実際の運用面や海外からの利用者のニーズにどの程度対応できるのかという問題もあり、本学会の国際化の問題とあわせて今後検討していく事とした。

3．委員会報告

(1) 国際委員会

池中委員長の報告を受け議論がなされた。

2003年2月に香港でAPSNが開催された。ISNはAPSNがきちんと組織化されなければAPSNへのサポートを打ち切るという意向を出しており、今後、APSNの急速な組織改革が必要となり対応する委

員会が池中氏を委員長として発足された。そのためJSNとして池中氏をサポートすることが確認された。

現在のAPSNは国単位で加盟し会費も各国の学会から徴収され、その国の学会員は自動的にAPSNの会員になっている。そのため各会員が「自分がAPSNの会員である」という意識が薄かった。今後、各個人から会費を徴収し会員であるという意識を持っていただく方向を検討している。次回のAPSNは2006年にシンガポールで行われる。

今後のISNの予定は2005年インスブルグ、2007年メキシコ（カンクーン）となっており2009年はアジアでの開催となる。今年の合同大会の前にISNのカウンシル・ミーティングがあり、日本の現状を見ていただくいい機会でもあるので2009年に日本が立候補するかについてはその時までに決めておく必要がある。

(2) シンポジウム企画委員会

鍋島委員長より以下の報告がなされた。

今年の合同大会でのオープンシンポジウムは前回理事会で承認された「論文投稿だけでなく、研究成果を社会にもっと還元しよう！；研究成果が特許申請・臨床治験・創薬・起業に結びついた例」を行う予定であり、最終案が報告された。

また学会活性化のために他学会と積極的に合同大会・合同シンポジウムを検討することが理事会で承認された事を受け、今年の合同大会で日本生物学的精神医学会との合同シンポジウム「精神神経疾患の分子基盤と治療戦略」を提案し採択された。

(3) 研究助成金等候補者選考委員会

馬場委員長より平成15年度の各財団の褒賞・助成に対する採択状況、学会内の応募状況の報告がなされた。また褒賞については応募のあった方をそのまま推薦するのではなく各財団褒賞の趣旨にあった方を推薦するという方針が了承された。

(4) 脳研究推進委員会

辻委員長より以下の報告がなされた。

日本の脳研究の問題点として「研究費のあり方」「脳関係の研究所、学会の足並みが揃っていないため対応がばらばらである」「日本学術会議の脳神経学研連が現況に対応した働きをしていないように見受けられる」といった、学会の外にある問題が山積しているため、各学会間のネットワークが必要と思われる。本件に関し評議員を対象にアンケートを行う事を検討している。

(5) 将来計画委員会

前回の評議員に対して行ったアンケートの回答を現実に実行に移すことの重要性が論じられた。

関連して特に最近の学会ではシンポジウムが多過ぎプログラム編成がパターン化しすぎているのではないかという意見が出された。

大会長・大会組織委員会の自主性も重要だが、学会としてのポリシーと継続性を持たせる意味から前回の理事会で承認された理事会管轄のプログラム委員会を設置する重要性が再確認された。

また、オープンシンポジウムが企画されることになった経緯として、以前はシンポジウムの企画が大会長に一任されていたため、学会としての方向性を示すシンポジウムを行う必要性があった。現在では学会の冠シンポジウムも増え、半数のシンポジウムが公募されている現状と大会マニュアルが整備されていく事を考慮すると、オープンシンポジウムは役割を終えたのではという意見や、この学会の大会規模のプログラムとしてプレナリー・レクチャーの数が適正かという疑問も出された。

上記の議論を踏まえ大会マニュアル、「大会プログラムに対して理事会が要望する基本的立場と方針」を将来計画委員長及び島田理事で文書化する事とした。

Ⅱ.審議事項

1. 平成15年度決算及び監査の件

高坂会計担当理事より平成15年度（平成15年1月1日～12月31日）の決算報告がなされた。引き続き植村監事より監査報告があり、了承された。高坂理事より会員数減少により収入が予算を下回っているため、会員を増やす努力が必要ではないかという提案がなされた。

2. 日本神経化学会奨励賞の件

2003年11月29日に奨励賞選考委員会が開催され委員の互選で西澤正豊委員長が選出された。委員会で検討された奨励賞規定改定案と奨励賞内規規定案が提案され理事会で承認された。

日本神経化学会奨励賞規定

規定（現行）

1. 申請者は会員歴3年以上、研究歴3年以上で申請の暦年度の4月1日現在で満40歳未満の者とする。

規定（改定案）

1. 申請者は会員歴3年以上、研究歴3年以上で**本学会で積極的に発表した実績を持ち**申請の暦年度の4月1日現在で満40歳未満の者とする。

（ゴシック体の箇所を条文に追加）

日本神経化学会奨励賞内規

規定（現行）

5　その他の申し合わせ事項

選考委員の所属する研究室から自薦者が出た場合は、選考委員を辞退すること。

規定（改定案）

5　その他の申し合わせ事項

選考委員の所属する研究室から自薦者が出た場合は、選考に加わらない。

評議員会の活性化のため若手研究者を積極的に評議員に推薦し学会で活躍をしていただくようにしている。その一環として昨年度の総会で理事会として奨励賞受賞者を評議員に推薦した。平成15年度奨励賞受賞者4名も今年の理事会として総会で評議員に推薦することが了承された。

3. 科研費関連事項の件

御子柴理事長宛に脳・神経学研究連絡委員会の金澤委員長（第一段細目「神経化学・神経薬理学」・第二段分科「神経科学」）及び細胞生物学研究連絡委員会の室伏委員長（第一段細目「細胞生物学」・第二段分科「生物科学」）より平成17年度科学研究費補助金の審査委員選任にかかわる研究者情報の依頼が届いた。

学協会に対する科研費審査委員候補者推薦については平成17年度分より変更が行われ、従来の「候補者推薦依頼」ではなく「研究者情報提供」という位置づけとなり、候補者の前年度継続審査委員や他の細目・分科での重複を確認する必要がなくなった。

本学会では各分科・細目ごとに2年分の候補者を選挙で選出しており、選挙結果の順位通りに各委員長に推薦した。

尚、今回の変更で本学会の「科学研究費審査委員候補者選挙に関する内規」「科学研究費審査委員候補者推薦方法」が現状とは合わない部分が生じたが、科研費審査委員候補者の推薦方法については現在過渡期という事もあり、早急に内規を改定せず、当面は様子を見てから対応を協議することが了承された。

4. 第47回日本神経化学会の件

遠山正彌理事より大会の進捗状況が報告された。今大会でのISNシンポジウムは国際対応委員会が担当し、池中委員長が取りまとめて遠山大会長に提案し、他のプログラムとのバランスを考慮して取りまとめることとした。カウンシルミーティングで来日するISNの理事には積極的にシンポジウムの演者になっていただく事とした。

5. 第48回日本神経化学会の件

2005年度の第48回日本神経化学会大会の大会長に神庭重信教授（九州大）が推薦され了承された。

2006年度の第49回日本神経化学会大会の大会長に鍋島俊隆教授（名古屋大）が推薦され了承された。第49回大会は日本生物学的精神医学会／日本神経精神薬理学会合同大会との合同大会を念頭において準備を進める事が了承された。

2007年度の第50回日本神経化学会大会の大会長として高坂新一所長（国立精神・神経センター）が推薦され了承された。2007年は3年に一度開催される日本神経科学学会との合同大会の年にあたる。双方が50回大会・30回大会という節目の学会に当るが、日本神経科学学会側は合同大会を希望しているため合同として大会を行うが、会期を1日ずらして合同大会を行う案が出され先方に提案することとした。

ご 挨 拶

平成16年9月21日から23日まで第47回日本神経化学会大会と第27回日本神経科学大会の合同大会をNeuro2004として大阪国際会議場で開催するはこびとなりました。本学会では両学会の特徴を生かしつつ将来に向け 1）若手研究者の育成、2）国際化、3）研究者の交流を柱として企画が進められました。若手の育成につきましては口頭発表をできるだけ取り入れ、自分の言葉で論理的に内容を人に理解してもらう場といたしました。また国際化につきましてはシンポジウムを英語で行うこととし、国際化に向けての一つの場と位置づけました。また研究者の交流の場として、特に若手の研究者間の研究交流、情報交換の場として多くの若手の研究者の参加を期待して、きわめて安い参加費での懇親会を設定しております。プレナリーレクチャー（3題）、特別講演（3題）、シンポジウム（36セッション）に加えて両学会員の先生方のご協力もあり、一般演題1,290題のご発表が予定されております。さらにランチョンセミナーの企画もございます。分子・ナノから行動・病気の機序、治療まで最先端の発表が必ずや参加される皆様方の知的好奇心を満足させてくれるものと確信しております。

また20日午後には「知られざる脳機能～その解明と応用～」と題して市民公開講座が開催され、脳の、心の不思議を様々な角度からやさしく解き明かしていただきます。一般市民の方々のみならず研究者の方々にも十二分にご満足頂ける内容です。こちらにもぜひご参加をお願いする次第です。

まだ残暑厳しき折とは存じますが、一人でも多くの先生方にご参加いただき合同学会を盛り上げ次回へと繋ぎうる学会にしていただけますようお願いいたします。

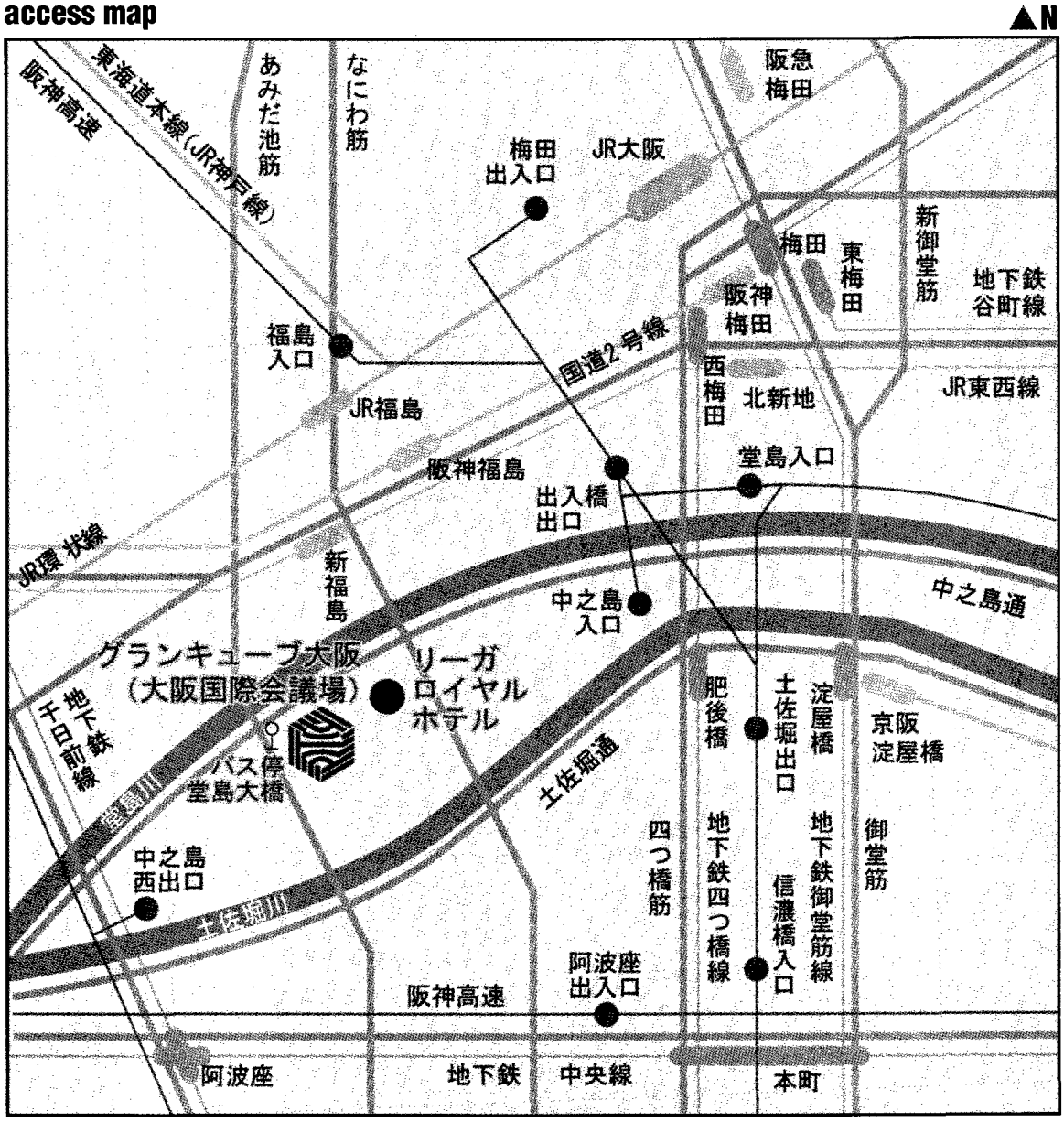
第27回 日本神経科学大会
大 会 長 村上富士夫
（大阪大学大学院生命機能研究科 脳神経工学講座）

第47回 日本神経化学会大会
大 会 長 遠山 正彌
（大阪大学大学院医学系研究科 ポストゲノム疾患解析学講座）

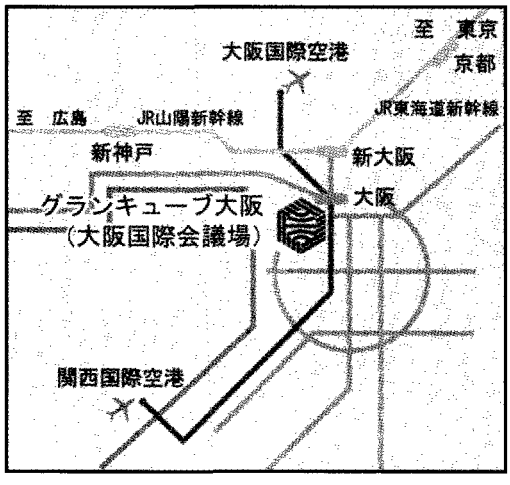
会場のご案内

大阪国際会議場

access map



from airport



access:

■所在地：〒530-0005 大阪市北区中之島5丁目3番51号
TEL：06-4803-5555（代表） FAX：06-4803-5620
URL：http://www.gco.co.jp/

■関西国際空港から
- JR「大阪駅」まで約55分。
- 南海電鉄で「難波駅」まで約30分。
- 空港リムジンバスでJR「大阪駅」まで約60分。

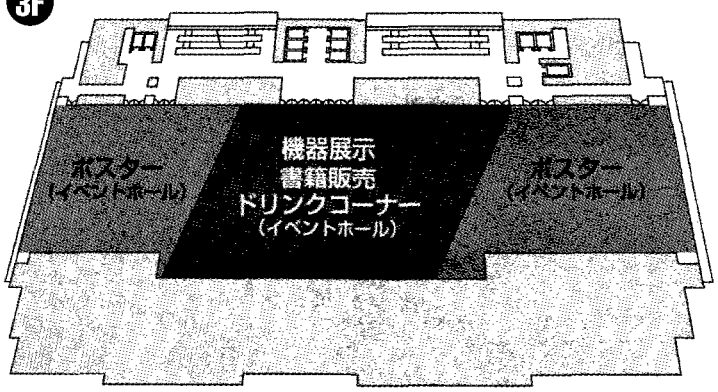
■大阪国際空港（伊丹）から
- 空港リムジンバスで「大阪駅」前まで約30分。

■新幹線（新大阪駅）から
- JR在来線で「大阪駅」まで約5分。

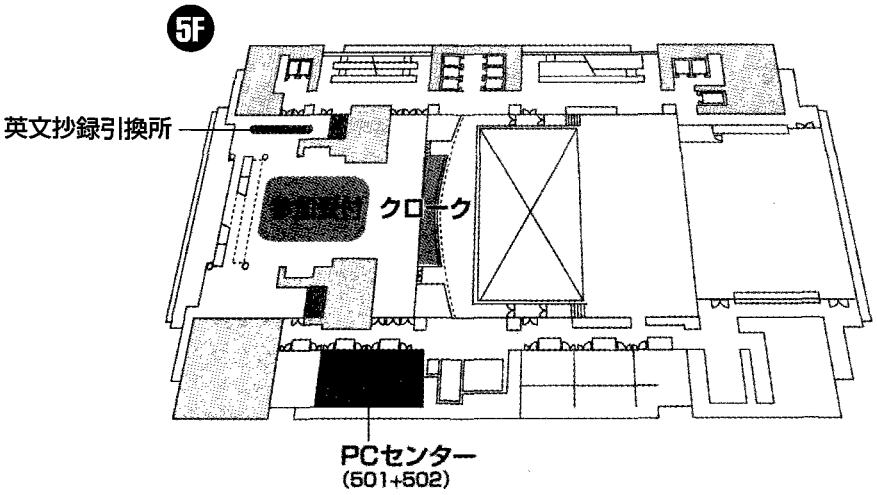
■周辺アクセス
- JR「大阪駅」駅前バスターミナルから、大阪市バス（53系統 船津橋行）または（55系統 鶴町四行）で約15分「堂島大橋」バス停下車すぐ
- JR大阪環状線「福島駅」から徒歩約10分
- JR東西線「新福島駅」（2番出口）から徒歩約10分
- 阪神電鉄「福島駅」から徒歩約10分
- 大阪市営地下鉄「阿波座駅」（中央線1号出口・千日前線9号出口）から徒歩約10分
- シャトルバスが、「リーガロイヤルホテル」と各ターミナル（JR「大阪駅」中央北口、地下鉄・京阪「淀屋橋駅」西詰）の間で運行しており、ご利用いただけます（但し定員28名）

会場案内図

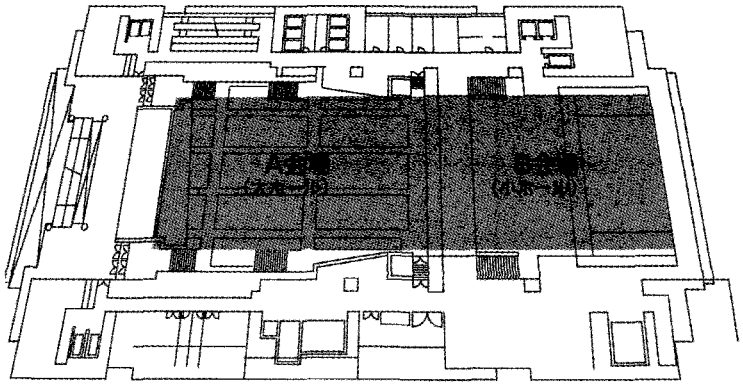
3F



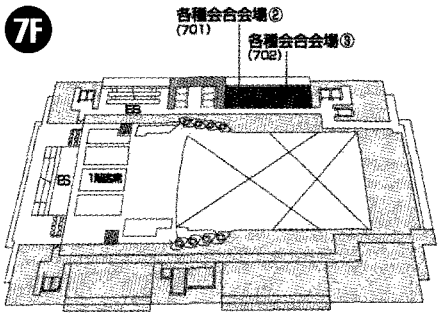
5F



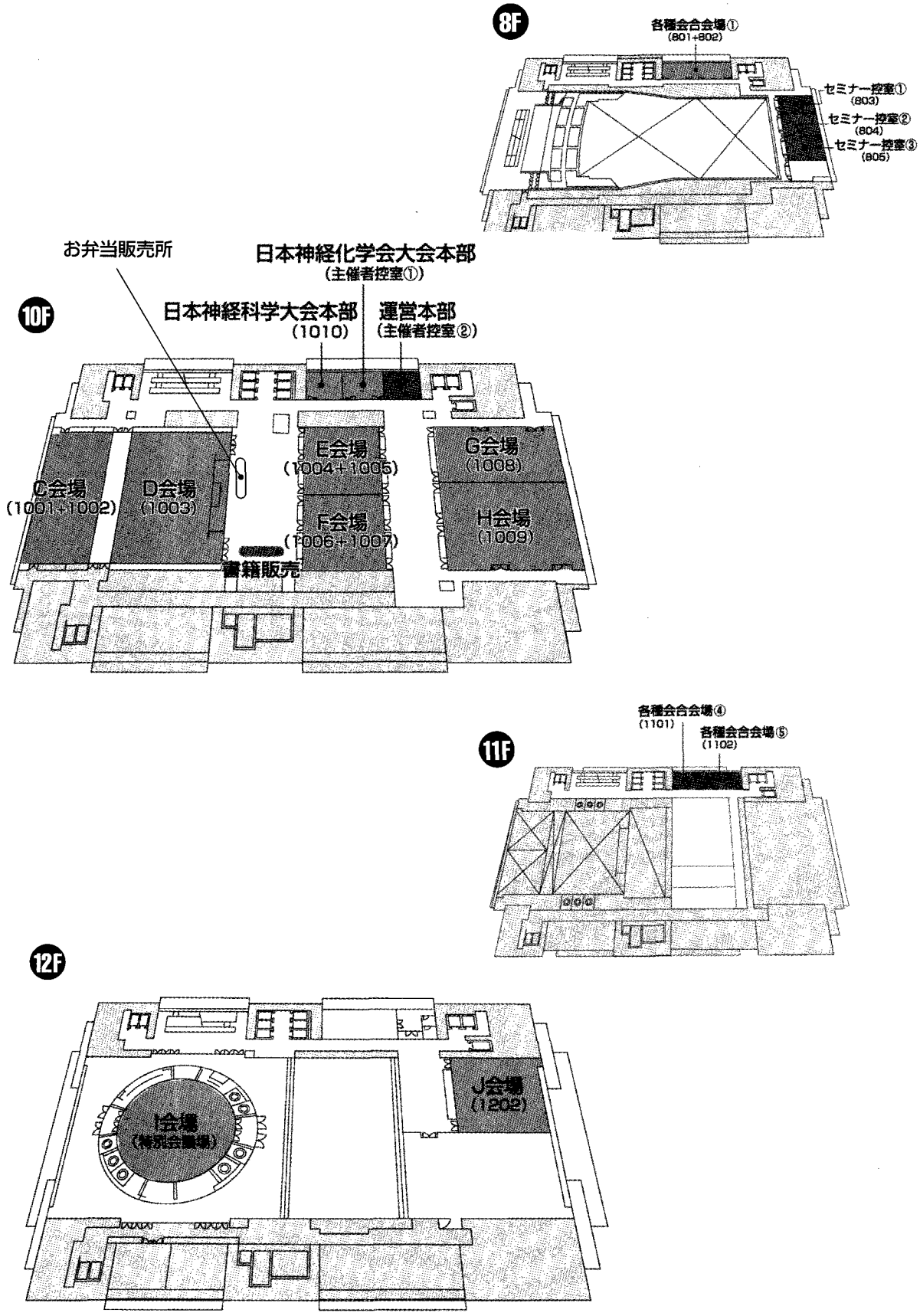
6F



7F



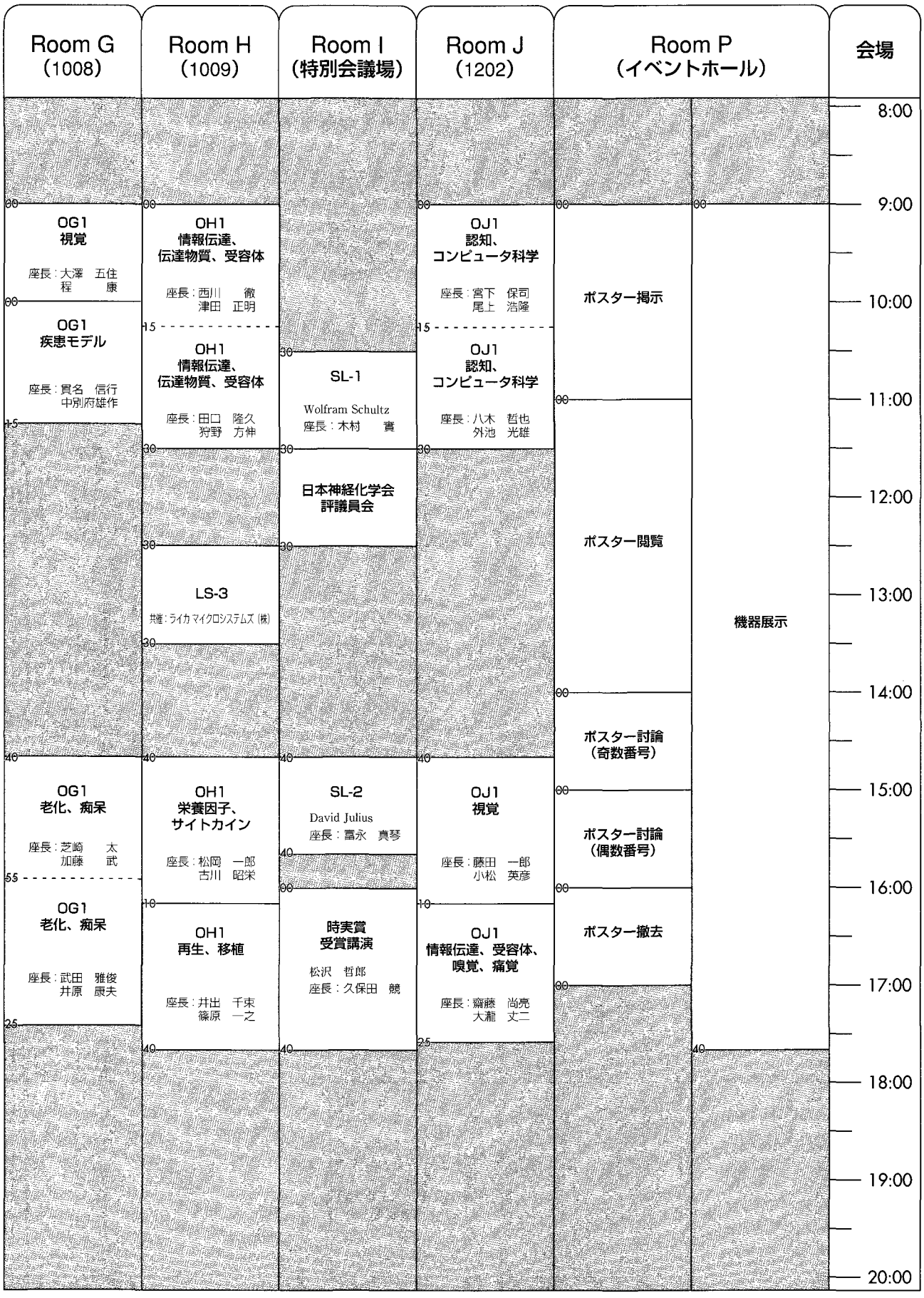
会場案内図


8F
各種会合会場①
(801+802)
セミナー控室①
(803)
セミナー控室②
(804)
セミナー控室③
(805)
お弁当販売所
日本神経化学会大会本部
(主催者控室①)
10F
日本神経科学大会本部
(1010)
運営本部
(主催者控室②)
C会場
(1001+1002)
D会場
(1003)
E会場
(1004+1005)
F会場
(1006+1007)
G会場
(1008)
H会場
(1009)
書籍販売
11F
各種会合会場④
(1101)
各種会合会場⑤
(1102)
12F
I会場
(特別会議場)
J会場
(1202)

大会日程概要

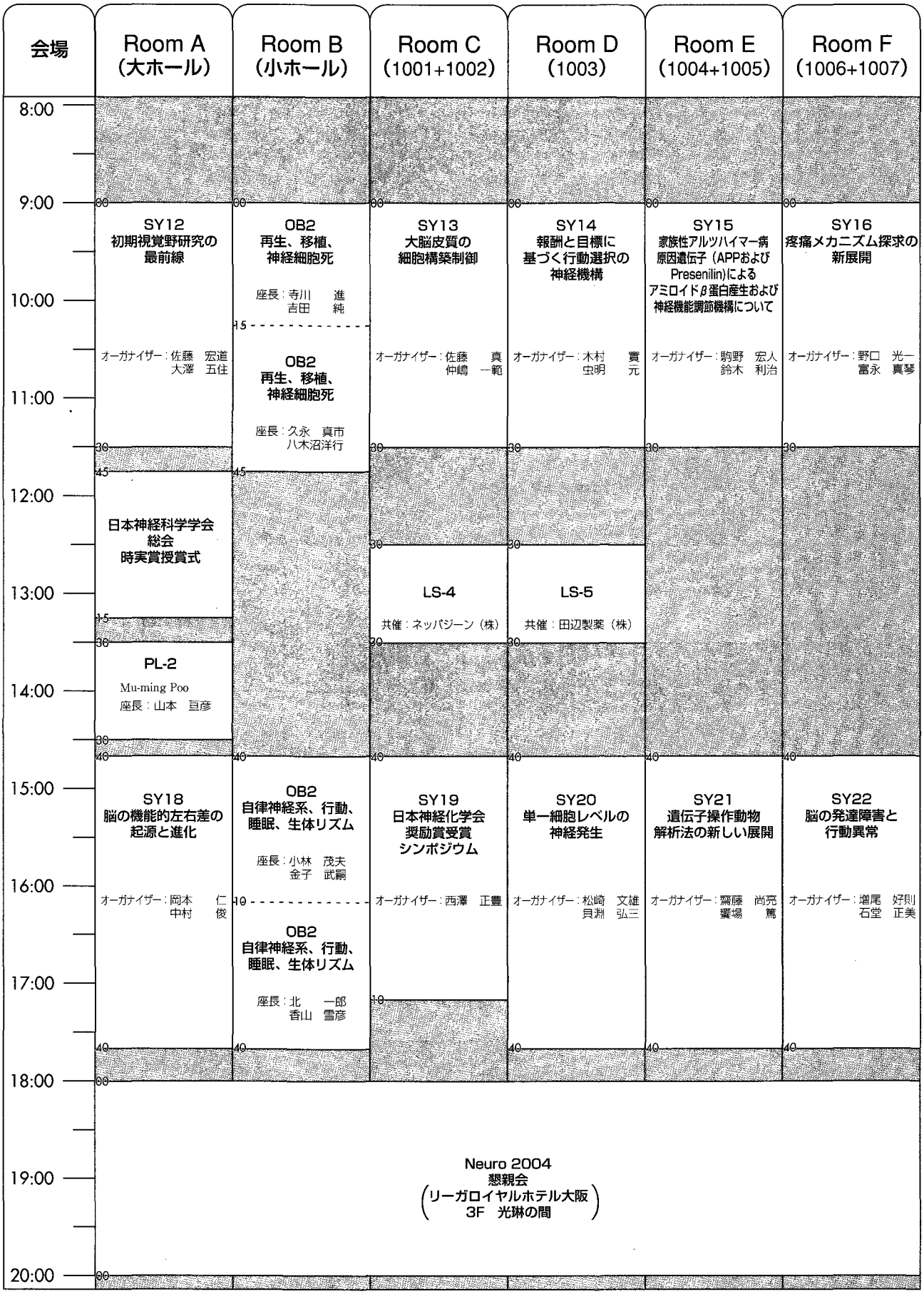
●Sep 21 (Tue)

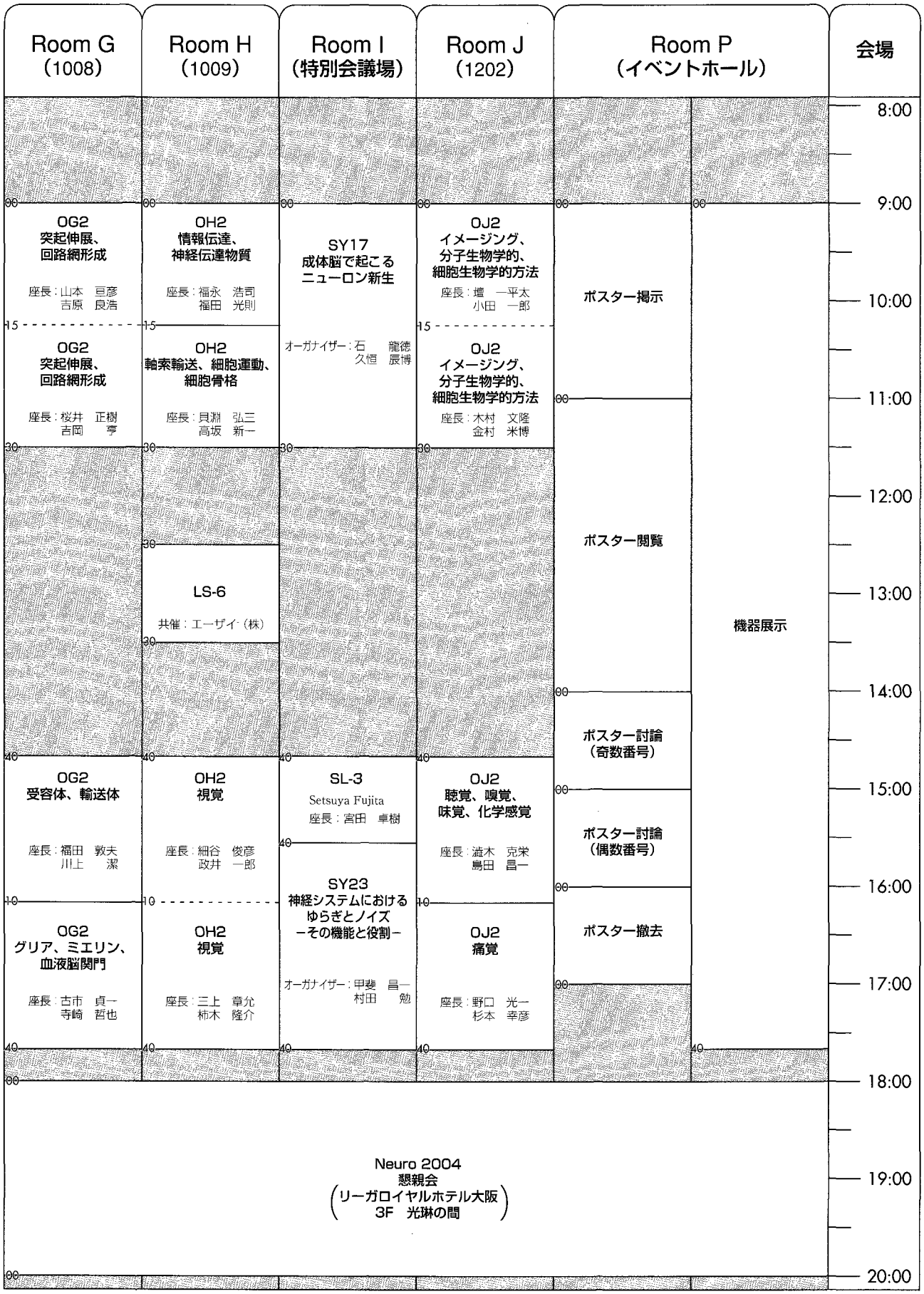
会場	Room A (大ホール)	Room B (小ホール)	Room C (1001+1002)	Room D (1003)	Room E (1004+1005)	Room F (1006+1007)
8:00						
9:00	SY01 行動の多様性の分子基盤 オーガナイザー：山元　大輔 八木　健	OB1 神経疾患 I 座長：馬場　明道 功刀　浩	SY02 脳機能発達における遺伝子発現とその障害：自閉症、LD、ADHDなどの原因研究と内分泌かく乱物質 オーガナイザー：田代　朋子 黒田洋一郎	SY03 シナプス可塑性研究の新しい潮流 ―短期可塑性から長期可塑性へ オーガナイザー：小倉　明彦 藤井　聡	SY04 脳と生殖：生殖の中枢制御機構をめぐる神経生物学 オーガナイザー：岡　良隆 前多敬一郎	SY05 神経変性疾患克服へ向けて ―若手研究者たちの挑戦 オーガナイザー：永井　義隆 小野寺　理
10:00		OB1 神経疾患 I 座長：内田　さえ 山形　要人				
11:00						
12:00						
13:00			LS-1 共催：カールツァイス（株）	LS-2 共催：アルファメッドサイエンス（株）		
14:00	PL-1 Peter St. George-Hyslop 座長：今泉　和則					
15:00	SY06 脳機能イメージングにおけるNeurovascular Couplingの意義 オーガナイザー：精山　明敏 関　淳二	OB1 神経疾患 II 座長：川野　仁 内山　安男	SY07 情動と社会性のニューロイメージング オーガナイザー：加藤　進昌 川島　隆太	SY08 RNA 神経生物学：ニューロンにおける転写後の世界 オーガナイザー：武井　延之 岡野ジェイムス洋尚	SY09 局所回路ネットワークとしての孤束核複合体 ―発生・発達から機能分子まで オーガナイザー：加藤　総夫 佐藤　容子	SY10 モデル脳領域(小脳とキノコ体)に着目した分子から行動までの脳機能解析 オーガナイザー：伊藤　悦朗 川原　茂敏
16:00						
17:00		OB1 神経疾患 II 座長：戸田　達史 岡野　栄之				
18:00				SY11 論文投稿だけでなく、研究成果を社会にもっと還元しよう！：研究成果が特許申請・臨床治験・創薬・起業に結びついた例 オーガナイザー：渡辺　恭良 鍋島　俊隆 （日本神経化学会オープンシンポジウム）		
19:00						
20:00						


Room G (1008)
Room H (1009)
Room I (特別会議場)
Room J (1202)
Room P (イベントホール)
会場
8:00
9:00
10:00
11:00
12:00
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16:00
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18:00
19:00
20:00
OG1 視覚
座長：大澤 五住
程 康
OG1 疾患モデル
座長：貫名 信行
中別府雄作
OG1 老化、痴呆
座長：芝崎 太
加藤 武
OG1 老化、痴呆
座長：武田 雅俊
井原 康夫
OH1 情報伝達、伝達物質、受容体
座長：西川 徹
津田 正明
OH1 情報伝達、伝達物質、受容体
座長：田口 隆久
狩野 方伸
LS-3
共催：ライカ マイクロシステムズ (株)
OH1 栄養因子、サイトカイン
座長：松岡 一郎
古川 昭栄
OH1 再生、移植
座長：井出 千束
篠原 一之
SL-1
Wolfram Schultz
座長：木村 實
日本神経化学会評議員会
SL-2
David Julius
座長：富永 真琴
時実賞受賞講演
松沢 哲郎
座長：久保田 競
OJ1 認知、コンピュータ科学
座長：宮下 保司
尾上 浩隆
OJ1 認知、コンピュータ科学
座長：八木 哲也
外池 光雄
OJ1 視覚
座長：藤田 一郎
小松 英彦
OJ1 情報伝達、受容体、嗅覚、痛覚
座長：齋藤 尚亮
大瀧 丈二
ポスター掲示
ポスター閲覧
ポスター討論（奇数番号）
ポスター討論（偶数番号）
ポスター撤去
機器展示

大会日程概要

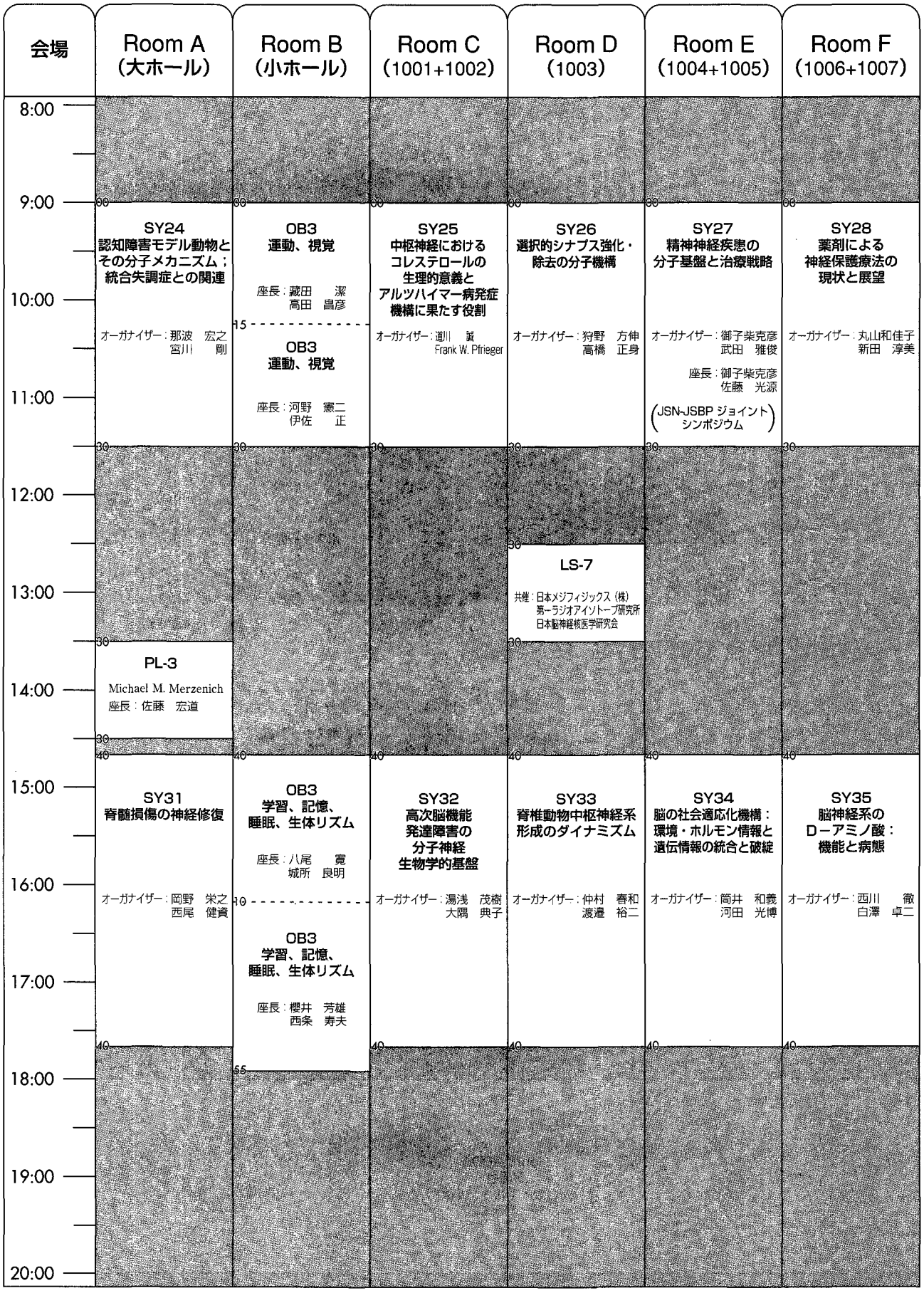
●Sep 22 (Wed)

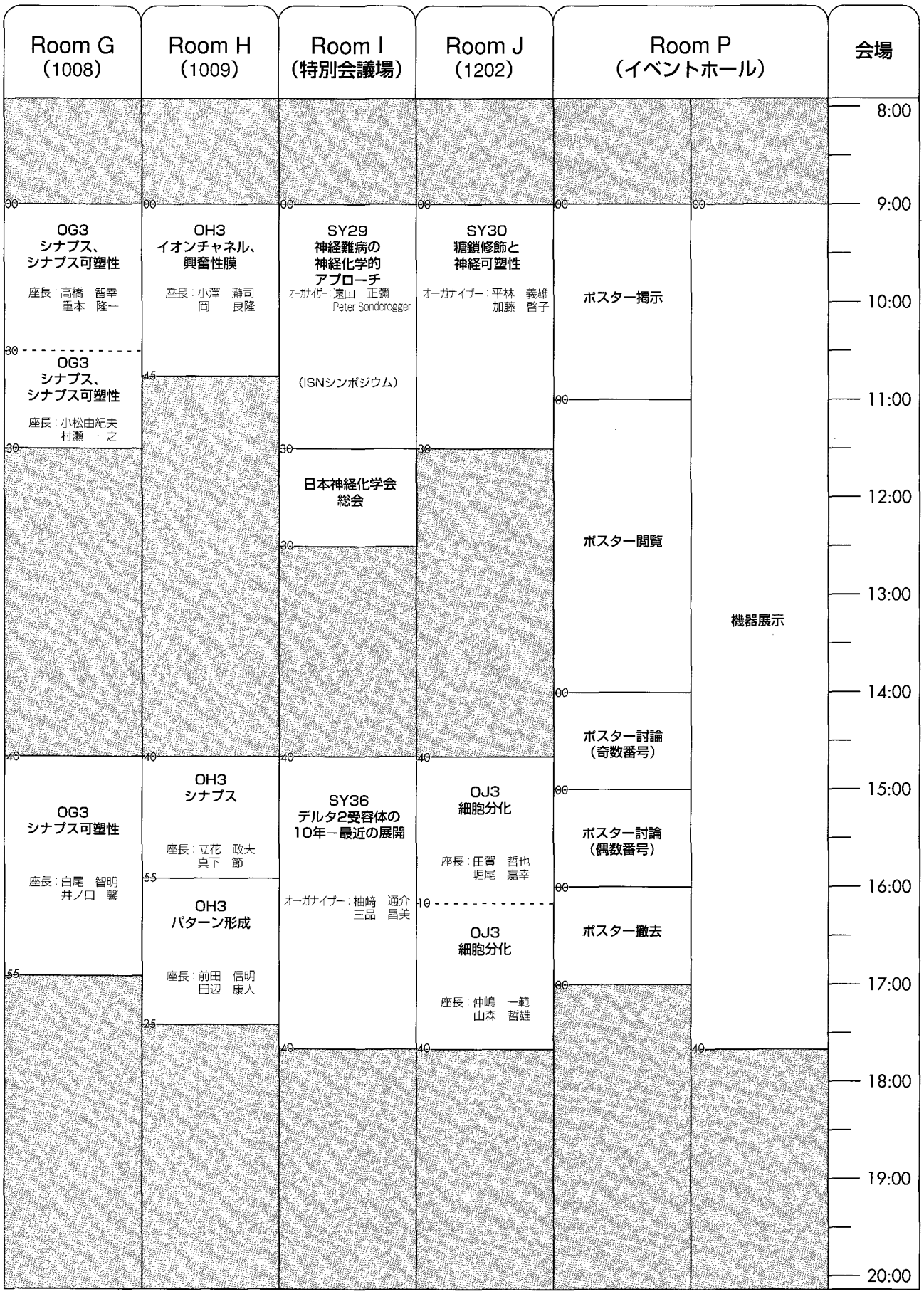



Room G
(1008)
Room H
(1009)
Room I
(特別会議場)
Room J
(1202)
Room P
(イベントホール)
会場
8:00
9:00
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18:00
19:00
20:00
OG2
突起伸展、
回路網形成
座長：山本 亘彦
吉原 良浩
OG2
突起伸展、
回路網形成
座長：桜井 正樹
吉岡 亨
OG2
受容体、輸送体
座長：福田 敦夫
川上 潔
OG2
グリア、ミエリン、
血液脳関門
座長：古市 貞一
寺崎 哲也
OH2
情報伝達、
神経伝達物質
座長：福永 浩司
福田 光則
OH2
軸索輸送、細胞運動、
細胞骨格
座長：貝淵 弘三
高坂 新一
LS-6
共催：エーザイ（株）
OH2
視覚
座長：細谷 俊彦
政井 一郎
OH2
視覚
座長：三上 章允
柿木 隆介
SY17
成体脳で起こる
ニューロン新生
オーガナイザー：石 龍徳
久恒 辰博
SL-3
Setsuya Fujita
座長：宮田 卓樹
SY23
神経システムにおける
ゆらぎとノイズ
－その機能と役割－
オーガナイザー：甲斐 昌一
村田 勉
OJ2
イメージング、
分子生物学的、
細胞生物学的方法
座長：壇 一平太
小田 一郎
OJ2
イメージング、
分子生物学的、
細胞生物学的方法
座長：木村 文隆
金村 米博
OJ2
聴覚、嗅覚、
味覚、化学感覚
座長：滝木 克栄
島田 昌一
OJ2
痛覚
座長：野口 光一
杉本 幸彦
ポスター掲示
ポスター閲覧
ポスター討論
（奇数番号）
ポスター討論
（偶数番号）
ポスター撤去
機器展示
Neuro 2004
懇親会
（リーガロイヤルホテル大阪
3F 光琳の間）

大会日程概要

●Sep 23 (Thu)




Room G
(1008)
Room H
(1009)
Room I
(特別会議場)
Room J
(1202)
Room P
(イベントホール)
会場
8:00
9:00
10:00
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15:00
16:00
17:00
18:00
19:00
20:00
OG3
シナプス、
シナプス可塑性
座長：高橋 智幸
重本 隆一
OG3
シナプス、
シナプス可塑性
座長：小松由紀夫
村瀬 一之
OG3
シナプス可塑性
座長：白尾 智明
井ノ口 馨
OH3
イオンチャネル、
興奮性膜
座長：小澤 瀞司
岡 良隆
OH3
シナプス
座長：立花 政夫
真下 節
OH3
パターン形成
座長：前田 信明
田辺 康人
SY29
神経難病の
神経化学的
アプローチ
ｵｰｶﾞﾅｲｻﾞｰ：遠山 正彌
Peter Sonderegger
(ISNシンポジウム)
日本神経化学会
総会
SY36
デルタ2受容体の
10年－最近の展開
オーガナイザー：柚崎 通介
三品 昌美
SY30
糖鎖修飾と
神経可塑性
オーガナイザー：平林 義雄
加藤 啓子
OJ3
細胞分化
座長：田賀 哲也
堀尾 嘉幸
OJ3
細胞分化
座長：仲嶋 一範
山森 哲雄
ポスター掲示
ポスター閲覧
ポスター討論
(奇数番号)
ポスター討論
(偶数番号)
ポスター撤去
機器展示

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

予め送付しました参加登録証（ネームカード）を必ずご持参のうえ、ご着用ください。参加登録証（ネームカード）のない方の入場はお断りいたします。ご協力をよろしくお願いいたします。

当日参加の方へ

大会当日参加登録される方は、大阪国際会議場5F当日参加受付にてご登録ください。お支払いは、すべて現金でお願いいたします。参加費と引き換えに参加登録証（ネームカード）・プログラム集・CD-ROM抄録集をお渡しいたします。会場内では、必ず参加登録証（ネームカード）をご着用ください。

懇親会にご参加の方へ

懇親会当日参加を希望される方は、大阪国際会議場5F当日参加受付にてご登録ください。お支払いは、すべて現金でお願いします。参加費と引き換えに参加シールと領収書をお渡しいたします。

懇親会：日時／9月22日（水）　午後6時00分　～
場所／リーガロイヤルホテル大阪　3F　光琳の間

＜受付場所・時間＞

大阪国際会議場　5F　ホワイエ
9月21日（火）午前8時00分～午後6時00分
9月22日（水）午前8時15分～午後6時00分
9月23日（木）午前8時15分～午後4時00分

＜当日参加費＞

会　　員	12,000円	
非 会 員	13,000円	
学 生 会 員	3,000円	（学生証をご提示ください。）
学生非会員	4,000円	（学生証をご提示ください。）
懇親会費（一般）	5,000円	
懇親会費（学生）	3,000円	（学生証をご提示ください。）

2. 英文抄録

日本神経科学学会員として演題を投稿された方の英文抄録は、「Neuroscience Research誌のSupplement（Elsevier）」に掲載されます。抄録代を支払われた方には事前に引換券を送付致しますので、英文抄録引換所（大阪国際会議場　5Fホワイエ）にて英文抄録集と引き換えてください。

3. 単位取得について

本大会への参加は、日本神経学会・日本小児神経学会および日本良導絡自律神経学会の認定医研修単位の対象となります。詳細はそれぞれの学会に直接ご照会ください。

4. 呼び出し、伝言

会場内スライドおよび館内放送での呼び出しは行いません。参加受付近くの会員伝言板をご利用ください。

5. クローク

大阪国際会議場5F　ホワイエにクロークを設けますのでご利用ください。尚、貴重品・傘などはお預かり出来ませんのであらかじめご了承ください。

9月21日（火）午前8時00分～午後8時00分
9月22日（水）午前8時15分～午後6時00分
9月23日（木）午前8時15分～午後6時15分

6. ドリンクサービス

大阪国際会議場3F　イベントホールのポスターおよび機器展示会場でドリンクサービスを行います。

7. 機器展示・書籍販売

大阪国際会議場3F　イベントホールにおいて機器展示・書籍販売を行います。書籍販売は10Fホワイエでも行います。

9月21日（火）午前9時00分～午後5時40分
9月22日（水）午前9時00分～午後5時40分
9月23日（木）午前9時00分～午後5時40分

8. 託児所

大阪国際会議場隣のリーガロイヤルホテル大阪ウエストウイング3Fのベビールームをご利用ください。（有料）詳しくはベビールームまで直接お電話にてお問合せください。

（お問合せ先）株式会社リトルメイト　TEL：06-6445-1343

尚、ご利用される方には、大会より料金の一部分を補助させて頂く予定ですので大会事務局（06-6227-0157）までご連絡ください。

9. 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（大阪国際会議場5Fホワイエ）にて発券いたします。

10. お弁当の販売について

会期中3日間（21～23日）、お弁当の販売を大阪国際会議場10Fホワイエで行います。お食事場は、E・F会場を開放いたします。

★お弁当販売時間（12:00～12:45）

11. 大会行事

●Neuro2004市民公開講座

日　時：9月20日（月）13:30～16:30　　**会　場：**大阪国際会議場　5F　A会場

テーマ：知られざる脳機能～その解明と応用～

1. 脳の情報処理の不思議　　川人　光男（ATR脳情報研究所）
2. 心の不思議　　武田　雅俊（大阪大学大学院医学系研究科）
3. 脳の不思議　　養老　孟司（北里大学大学院医療系研究科）

●時実賞受賞講演

日　時：9月21日（火）16:00～17:40　　会　場：大阪国際会議場　12F　I会場

座　長：久保田　競（日本福祉大）

チンパンジーにおける認知機能の実験的分析　　松沢　哲郎（京都大学霊長類研究所）

●日本神経化学会オープンシンポジウム

日　時：9月21日（火）17:40～19:40　　会　場：大阪国際会議場　10F　D会場

●日本神経化学会奨励賞受賞シンポジウム

日　時：9月22日（水）14:40～17:10　　会　場：大阪国際会議場　10F　C会場

●Neuro2004懇親会

日　時：9月22日（水）18:00～20:00　　会　場：リーガロイヤルホテル大阪　3F　光琳の間

●国際神経化学会シンポジウム

日　時：9月23日（木）　9:00～11:30　　会　場：大阪国際会議場　12F　I会場

●日本神経化学会・日本生物学的神経医学会　合同シンポジウム

日　時：9月23日（木）　9:00～11:30　　会　場：大阪国際会議場　10F　E会場

12. 関連行事

■日本神経科学学会関連

●日本神経科学学会理事会

日　時：9月20日（月）16:00～20:00　　会　場：大阪国際会議場　7F　702

●日本神経科学学会総会

日　時：9月22日（水）11:45～13:15　　会　場：大阪国際会議場　5F　A会場

総会に引き続いて授賞式を行います。

1．時実利彦記念賞授賞式

平成16年度受賞者

松沢　哲郎　教授（京都大学霊長類研究所）

受賞理由「チンパンジーの高次認知機能の実験的解析」

2．日本神経科学学会奨励賞授賞式

平成16年度受賞者

大須理英子（ATR脳情報研究所）・澤本　和延（慶応義塾大学医学部）・

中原　裕之（理化学研究所・脳科学総合研究センター）・

橋本　浩一（金沢大学大学院医学系研究科）・宮田麻理子（自然科学研究機構・生理学研究所）

3. 2003 Neuroscience Research 優秀論文賞発表

■日本神経化学会関連

●日本神経化学会理事会

日　時：9月20日（月）12:00～16:00　　会　場：大阪国際会議場　7F　701

●日本神経化学会評議員会

日　時：9月21日（火）11:30～12:30　　会　場：大阪国際会議場　12F　I会場

●日本神経化学会総会

日　時：9月23日（木）11:30～12:30　　会　場：大阪国際会議場　12F　I会場

●ISN理事会

日　時：9月19日（日）　9:00～18:00　　会　場：リーガロイヤルホテル大阪　6F　錦の間

●ISN総会

日　時：9月21日（火）18:00～19:30　　会　場：大阪国際会議場　12F　I会場

13. 各種委員会

9月20日（月）

会合名	開催時間	会場
Neuroscience Research 優秀論文賞選考委員会	12:00～13:00	大阪国際会議場　8F　801・802
Neuroscience Research 編集委員会	13:30～15:30	大阪国際会議場　8F　801・802

9月21日（火）

会合名	開催時間	会場
日本神経科学学会実験動物利用者会議	12:00～13:00	大阪国際会議場　11F　1101
日本神経化学会脳研究推進委員会	12:30～13:30	大阪国際会議場　7F　701

9月22日（水）

会合名	開催時間	会場
日本神経化学会将来計画委員会	11:30～12:30	大阪国際会議場　7F　701
日本神経化学会シンポジウム企画委員会	11:30～12:30	大阪国際会議場　7F　702
日本神経化学会国際対応委員会	11:30～12:30	大阪国際会議場　8F　801
日本神経化学会研究助成金等候補者選考委員会	11:30～12:30	大阪国際会議場　8F　802
日本神経化学会編集委員会	12:30～13:30	大阪国際会議場　11F　1102
日本神経化学会奨励賞選考委員会	16:30～18:00	大阪国際会議場　11F　1101

9月23日（木）

会合名	開催時間	会場
脳の世紀推進会議	12:30～13:30	大阪国際会議場　7F　701

オーガナイザー・座長へのご案内

・オーガナイザー、座長の方は、セッション開始45分前までに、大阪国際会議場5Fのオーガナイザー・座長受付にて時間をご確認ください。また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。

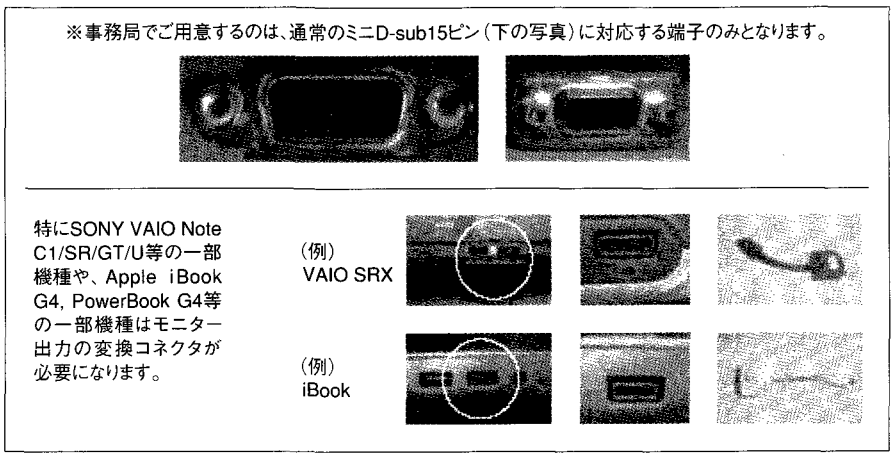
シンポジスト・一般演題(口演)発表者へのご案内

・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持参ください。
・発表言語につきましては、シンポジウムは英語、一般口演は日本語・英語のいずれでも可能です。
・開始予定時刻45分前までに大阪国際会議場5F PCセンター（501・502）へお越しください。
・受付終了後、ご自身のPCを持参のうえ、PCオペレーター立会いのもとで動作確認を行ってください。
・確認済みのPCは、各講演会場の舞台左手の演者席横のPCデスクに講演予定時刻の15分前までにお持ちになり次演者席にお着きください。
・発表時間は、シンポジウムはオーガナイザーの指示に従ってください。一般口演は、発表10分・討論5分となっております。当日は演台に計時回線をご用意しますので、時間内で完結出来るようご協力をお願いします。
・発表後、PCは会場内のPCデスクでお返しいたします。

PCプレゼンテーションのご案内

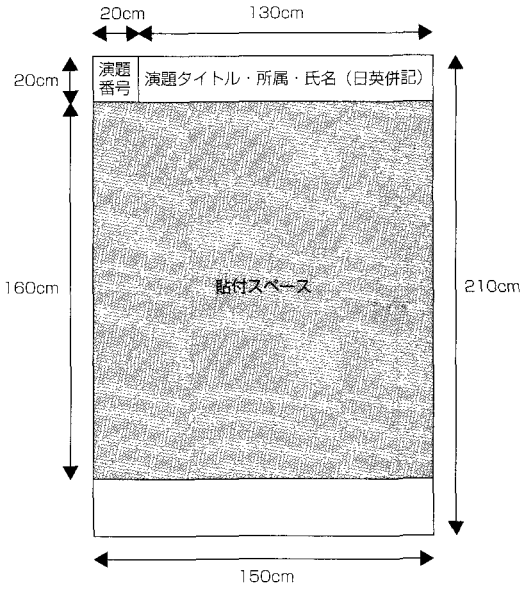
・発表中にスクリーンセーバーや省電力モードにならないよう事前に設定してください。
・発表機種は、Windows98またはMac OS 8.5以上が動作するPCをご使用ください。
・発表中またはその準備中にバッテリー切れになることが多々ございます。会場にはコンセント口をご用意しておりますので、コンセント用電源アダプタを必ずご持参ください。
・SONYのVAIOなど一部の薄型ノートパソコンで、モニター出力端子が、Dsub-15ピン（※1の写真）でないものが有ります。この端子がないものは本体のみではプロジェクタにつなぐことができません。アダプタが必要となりますので、必ずご用意ください。
・発表の45分前までにPCセンター（大阪国際会議場5F　501・502）にて出力をチェックしてください。
・演台上にモニターとリモートマウスをご用意しておりますので、発表時の操作はご自身でお願いいたします。
・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、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。
・動画データを使用の場合は、必ず発表データが作動するノートパソコンをお持込ください。
・スピーカーの用意はございません。必要な場合は事前に事務局にご連絡ください。

※1



一般演題（ポスター）発表者へのご案内

・ポスター展示は、大阪国際会議場3Fイベントホールで行います。まずポスター受付にて受付をお願いいたします。
・ポスターは所定の位置に貼り付け時間内に、ご自身で貼り付けてください。画鋲は各ポスターパネルに設置してあります。糊・セロテープは使用出来ません。（演題番号は事務局にて準備いたします。）
・ポスターパネルには右記のように縦160cm、横150cmのポスター貼付が可能です。
※演題名（日英）、所属（日英）、発表者名（日英）はご自身でご準備くださいますようお願いいたします。
・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。ただし日本語のサマリーも掲示してください。
・発表者の方には、ポスター受付でリボンをお渡ししますので、討論の際には胸につけて、下記の説明・討論の時間に各ポスターパネル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処分させていただきますのでご了承ください。



演題番号		P1-001～P1-350	P 2-001～P 2-350	P 3-001～P 3-344
発 表 日		9月21日（火）	9月22日（水）	9月23日（木）
貼付時間		9:00～11:00	9:00～11:00	9:00～11:00
閲　　覧		11:00～14:00	11:00～14:00	11:00～14:00
説明・討論	奇数番号	14:00～15:00	14:00～15:00	14:00～15:00
	偶数番号	15:00～16:00	15:00～16:00	15:00～16:00
撤去時間		16:00～17:00	16:00～17:00	16:00～17:00

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頂きますよう、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カ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ではなく、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。

Neuro 2004

展示案内

機器展示

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書籍販売

エルゼビアB. V.
大阪大学生活協同組合
オックスフォード大学出版局
NPO法人　脳の世紀推進会議　事務局　クバプロ
株式会社神陵文庫
丸善株式会社
Blackwell Publishing

（H16年7月28日現在）

演題番号の見方

■プレナリーレクチャー

PL-1

↓

プレナリーレクチャー番号

■特別講演

SL-1

↓

特別講演番号

■シンポジウム

SY01-01

↙ ↓

シンポジウム番号　シンポジウム内での発表順

■口演（一般演題）

OA1-01

↙ ↓ ↘ ↘

発表形式　会場番号　発表日　発表順

■ポスター（一般演題）

P1-001

↙ ↓ ↘

発表形式　発表日　ポスター番号

■ランチョンセミナー

LS-1

↓

ランチョンセミナー番号

プレナリーレクチャー
特別講演
時実賞受賞講演
シンポジウム
プログラム

◆◇◆ Plenary Lecture ◆◇◆

Sep 21 (Tue)

13:30～14:30 Room A

PL-1

座長：今泉 和則（奈良先端科学技術大学院大・バイオサイエンス）

Basic And Clinical Neurobiology of Alzheimer Disease

Peter St. George-Hyslop
Centre for Research in Neurodegenerative Diseases, Toronto Western Research Institute, Toronto Western Hospital, University of Toronto, Ontario, Canada

Sep 22 (Wed)

13:30～14:30 Room A

PL-2

座長：山本 亘彦（大阪大院・生命機能・脳神経工学）

Modification of Developing Neural Circuits by Sensory Experience

Mu-ming Poo
Division of Neurobiology, Department of Molecular and Cell Biology, University of California, Berkeley, USA

Sep 23 (Thu)

13:30～14:30 Room A

PL-3

座長：佐藤 宏道（大阪大・健康体育・運動生理）

Cortical plasticity contributing to child development and skill learning

Michael M. Merzenich
W.M. Keck Center for Integrative Neurosciences, University of California at San Francisco, USA

◆◇◆ Special Lecture ◆◇◆

Sep 21 (Tue)

10:30～11:30　Room I

SL-1

座長：木村　　實（京都府立大院・医・神経生理）

Reward, prediction and uncertainty

Wolfram Schultz
Department of Anatomy, University of Cambridge, UK

Sep 21 (Tue)

14:40～15:40　Room I

SL-2

座長：富永　真琴（岡崎統合バイオサイエンスセンター・細胞生理）

From Peppers to Peppermints: The Molecular Biology of Thermosensation

David Julius
Department of Cellular and Molecular Pharmacology University of California, San Francisco, USA

Sep 22 (Wed)

14:40～15:40　Room I

SL-3

座長：宮田　卓樹（名古屋大院・医・細胞生物）

マトリックス細胞と皮質形成の分子的細胞機構

The multipotent neural stem cell of the CNS, the matrix cell, and the molecular mechanism of corticogenesis

藤田　晢也
ルイ・パストゥール医学研究センター

◆◇◆ Tokizane ◆◇◆

Sep 21 (Tue)

16:00～17:40　Room I

Tokizane

座長：久保田　競（日本福祉大・人間福祉情報学科）

チンパンジーにおける認知機能の実験的分析

Experimental analysis of cognitive functions in chimpanzees

松沢　哲郎

京都大・霊長類研究所

◆◇◆ Symposium ◆◇◆

9:00～11:30 Room A

SY01-01～SY01-05 行動の多様性の分子基盤

Molecular basis for behavioral diversity

オーガナイザー：山元 大輔（早稲田大学・工・情報生命工学）
八木 健（大阪大院・工・生命機能研究科）

SY01-01 ショウジョウバエの寄主植物選択と種分化の遺伝子機構

Genetic basis of host plant preference and speciation in *Drosophila*

松尾 隆嗣[1]
[1]都立大・理・生物

SY01-02 中枢神経系の雄化と雄の性行動に関わるショウジョウバエのfruitless遺伝子

The Drosophila fruitless locus is required for male-typical development of the nervous system and male sexual behavior

山元 大輔[1]
[1]早稲田大学・理工・遺伝

SY01-03 子の神経内分泌系の発達に対する母親の影響

Maternal influence on neuroendocrine development of offspring

黒田 公美[1]、Michael Meaney[2]、加藤 忠史[1]
[1]理研・脳・精神疾患動態、[2]Psy. McGill, Montreal, Canada

SY01-04 マウス移動運動の系統差とその遺伝子基盤解明へのQTLアプローチ

QTL studies for understanding genetic mechanism of diversity on spontaneous activity in mice

小出 剛[1,2]
[1]国立遺伝学研究所・マウス開発、[2]総合研究大学院大学・生命科学研究科遺伝学専攻

SY01-05 幼少期社会経験による神経行動学的変容の解析

Neuro-behavioral changes induced by precocious weaning.

菊水 健史[1]、森 裕司[1]
[1]東京大院・農・動物行動

9:00～11:30 Room C

SY02-01～SY02-05 脳機能発達における遺伝子発現とその障害：自閉症，LD，ADHDなどの原因研究と内分泌かく乱物質

Gene expression profiles during functional development of the brain and their disorders; Are endocrine disruptors one of the causal factors of autism, LD, ADHD?

オーガナイザー：田代 朋子（青山学院大学・工・化学生命科学）
黒田洋一郎（東京都神経科学総合研究所）

SY02-01 Epidemiological trends in autism and developmental disorders

Eric Fombonne[1]
[1]Department of Psychaitry, McGill University, Montreal, Canada

SY02-02 環境化学物質の時空間的に異なる暴露による遺伝子発現、神経回路（シナプス）形成の攪乱がLD，ADHD、自閉症などの発症原因とするとcomorbidityが説明できる
COMORBIDITY OF LD, ADHD AND AUTISM SPECTRUM CAN BE CAUSED BY SPATIO-TEMPORAL DISRUPTION OF DEVELOPING CIRCUITS (SYNAPSE) FORMATION WITH CASCADES OF GENE EXPRESSION BY ENVIRONMENTAL TOXIC CHEMICALS.
黒田洋一郎[1]
[1]東京都神経科学総合研究所/CREST

SY02-03 活動依存的な脳由来神経栄養因子（BDNF）遺伝子の発現制御系と環境化学物質の与える影響
Effect of environmental disruptors on the activity-dependent expression of brain-derived neurotrophic factor (BDNF) gene in neurons
津田　正明[1]、今村　理佐[1]、安田　誠[1]、川原　正博[2]、福地　守[1]、田渕　明子[1]
[1]富山医薬大・薬・分子神経生物、[2]九州保健福祉大・薬・分析学

SY02-04 甲状腺ホルモンによる転写制御メカニズムとPCB類による阻害作用
Suppression of thyroid hormone receptor-mediated transcription by polychlorinated biphenyls.
鯉淵　典之[1]
[1]群馬大院・医・器官機能学

SY02-05 脳機能発達に対する環境化学物質の影響を評価するための定量的オリゴDNAマイクロアレイ「シナプトアレイ」の開発
Development of a quantitative oligonuceotide microarray "Synaptoarray" to monitor the effect of environmental chemicals on brain development
田代　朋子[1,2]
[1]青山学院大・理工・化学生命科学、[2]独立行政法人科学技術振興機構

9:00～11:30　Room D

SY03-01～SY03-05 シナプス可塑性研究の新しい潮流－短期可塑性から長期可塑性へ
New trends in synaptic plasticity research – Toward the long-term plasticity

オーガナイザー：小倉　明彦（大阪大院・工・生命機能研究科）
藤井　聡（山形大学・医・生理学）

SY03-01 LTP/LTD/Depotentiationにおける刺激パターン依存性
Properties of activity-dependent synaptic plasticity induced by 0.5-10 Hz low-frequency stimulations
藤井　聡[1,2]、黒田洋一郎[2]、山崎　良彦[1]、加藤　宏司[1]
[1]山形大・医・第2生理、[2]科学技術振興機構

SY03-02 刺激依存的なアクチン細胞骨格動員
Activity-driven changes in neuronal actin cytoskeleton
尾藤　晴彦[1,2]、古屋敷智之[3]、竹本－木村さやか[1]、石原奈津実[1]、藤井　哉[1]、岡村　理子[1]、奥野　浩行[1]
[1]東京大院・医・神経生化学、[2]SORST-JST、[3]京都大院・医・神経細胞薬理学

SY03-03 生細胞観察によるシナプス後肥厚部足場蛋白質の相互依存性の解析
Organization of PSD scaffolding proteins revealed by live cell imaging
岡部　繁男[1]
[1]東京医科歯科大院・医歯・細胞生物

SY03-04 長期シナプス可塑性のガラス器内再現
Two forms of long-term synaptic plasticity produced in cultured brain slice
小倉 明彦[1]、篠田 陽[1]、冨永 恵子[1]
[1]大阪大院・生命機能・脳神経工学

SY03-05 記憶固定化・再固定化・消失の分子機構
Molecular mechanisms of memory consolidation, reconsolidation and extinction
喜田 聡[1]
[1]東京農大・応生科・バイオ

9:00～11:30 Room E

SY04-01～SY04-05 脳と生殖：生殖の中枢制御機構をめぐる神経生物学
Neurobiology of Reproduction

オーガナイザー：岡 良隆（東京大院・理学系研究科）
前多敬一郎（名古屋大院・生命農学研究科）

SY04-01 Membrane Excitability in Hypothalamic GnRH Neurons: Roles of G Protein-Coupled Receptors and Second Messengers
Lazar Krsmanovic[1]、Lian Hu[1]、Keiko Wada[1]、Kevin Catt[1]
[1]Endocrinology and Reproduction Research Branch, NICHD

SY04-02 GnRHニューロンに発現する膜電位依存性カルシウムチャネル
Voltage-gated Calcium Channels in Rat GnRH Neurons
加藤 昌克[1]、佐久間康夫[1]
[1]日本医科大・生理

SY04-03 神経修飾作用をもつGnRHペプチドニューロンの生理学的特性
Physiological properties of neuromodulatory GnRH peptidergic neurons
岡 良隆[1]
[1]東京大学大学院・理学系研究科・生物科学

SY04-04 Novel hypothesis for sexual differentiation of the control of GnRH secretion: Why females have reproductive cycles and males do not
Douglas L. Foster[1]、Leslie M. Jackson[1]、Vasantha Padmanabhan[1]、Jane Robinson[2]
[1]Reproductive Sciences Program, University of Michigan、[2]Department of Preclinical Veterinary Studies, University of Glasgow

SY04-05 泌乳期のパルス状黄体形成ホルモン（LH）分泌抑制のメカニズム
Lactational anestrus: Mechanism mediating suppression of pulsaitle luteinizing hormone (LH) release in lactating rats
東村 博子[1]
[1]名古屋大学院・生命農

Sep 21 (Tue) Symposium

9:00～11:30 Room F

SY05-01～SY05-06 神経変性疾患克服へ向けて－若手研究者たちの挑戦
Toward establishing therapies for neurodegenerative diseases-Challenges by young investigators

オーガナイザー：永井 義隆（大阪大院・医・ポストゲノム疾患解析学ゲノム機能）
小野寺 理（新潟大学・脳研究所）

SY05-01 ポリグルタミン蛋白質の構造異常・凝集体形成を標的とするポリグルタミン病の治療戦略
Therapeutic strategy for the polyglutamine diseases targeting the toxic conformational transition and aggregation of the polyglutamine protein

永井 義隆[1]、ポピエル ヘレナ明子[1]、藤掛 伸宏[1]、乾 隆[2]、内木 宏延[3]、戸田 達史[1]
[1]大阪大院・医・ゲノム機能、[2]三重短大・生活・栄養、[3]福井大・医・2病理

SY05-02 プリオン病と蛋白質アンフォールデイング因子; Unfoldin
Prion disease and Unfoldin: A protein with robust ATP-dependent unfoldase activity and a novel grapple-like structure in vitro

八谷 如美[1,2]、定塚 昌子[1,2]、逆瀬川裕二[1]、金子 清俊[1,2]
[1]国立精神・神経センター 神経研究所 疾病研究第7部、[2]科学技術振興機構、CREST

SY05-03 アミロイドβの分解機構
Metabolic mechanism of amyloid-β peptide

岩田 修永[1]
[1]理研・脳科学・神経蛋白制御

SY05-04 神経変性を引き起こす蛋白 APTX はDNAを修復する
Neurodegenerative disease protein, APTX, is a new DNA repair protein.

小野寺 理[1]
[1]新潟大・脳研・リソース・分子神経疾患資源解析

SY05-05 遺伝子治療への応用を目指したsiRNA発現ベクターの開発
Development of siRNA expression vector for gene therapy

川崎 広明[1,2]、多比良和誠[1,2]
[1]東京大院・工・化学生命工学、[2]産総研・ジーンファンクション研究センター

SY05-06 HSVアンプリコンベクターの開発と応用
Development of HSV amplicon vector system: a versatile gene delivery tool for gene therapy and functional studies

佐伯 嘉修[1]
[1]Mass. General Hosp. / Harvard Med. Sch.

14:40～17:40 Room A

SY06-01～SY06-06 脳機能イメージングにおけるNeurovascular Coupling の意義
Functional Neuroimaging and Neurovascular Coupling

オーガナイザー：精山 明敏（大阪大院・医・ナノ生体科学）
関 淳二（国立循環器病センター研究所）

SY06-01 Posing a problem for neuroimaging: Relationship between changes in hemodynamic parameters and BOLD effects

Akitoshi Seiyama[1]、Toshio Yanagida[1]
[1]Division of Physiology and Biosignaling, Osaka University Graduate School of Medicine, Osaka, Japan

SY06-02 Examination of a biophysical model of the BOLD signal using MRI measurements and concurrent optical imaging spectroscopy and laser Doppler flowmetry.
John Mayhew[1]
[1]University of Sheffield. UK

SY06-03 神経シナプス活動と局所脳循環反応の線形的関係－機能的信号と刺激パラダイムへの依存性
Functional signal- and paradigm-dependent linear relationships between synaptic activity and hemodynamic responses
根本 正史[1]、星 詳子[1]、Arthur Toga[2]
[1]東京都精神研・脳機能解析、[2]UCLA Neurology, Los Angeles, USA

SY06-04 神経活動によって引き起こされる光散乱変化を利用した脳機能イメージング
Functional Brain Imaging with Light Scattering Changes Elicited by Neural Activities
谷藤 学[1]、角田 和繁[1,2]
[1]理化学研究所、[2]東京医療センター

SY06-05 神経活動で誘発される局所脳血流量を調節するモデル
A Model for the Regulation of Local Cerebral Blood Flow Evoked by Neuronal Activation
菅野 巌[1]、正勝 嬉[1]
[1]秋田脳研・放

SY06-06 光干渉断層法による脳微小循環の刺激応答の解析
Stimulus induced changes in cerebral microcirculation measured by optical coherence tomography
関 淳二[1]、里村 泰光[1]
[1]国立循環器病センター研究所・生体工学

14:40～17:40 Room C

SY07-01～SY07-06 情動と社会性のニューロイメージング
Imaging of Emotional and Social Brain
オーガナイザー：加藤 進昌（東京大院・医・精神医学）
川島 隆太（東北大学・未来科学技術共同研究センター）

SY07-01 情動刺激に対するサルの側頭葉と扁桃核のニューロンの応答
Responses of single neurons coding emotional expressions in the amygdala of monkeys
倉岡 康治[1]、中村 克樹[2]
[1]京都大・霊長類研究所・認知学習、[2]国立精神神経センター・神経研究所

SY07-02 情動に関するfunctional MRI－報酬に対する脳活動変化の文脈依存性
Context-dependent cortical activation in response to financial reward and penalty: An event-related fMRI study
秋月 祐子[1,2]、川島 隆太[1]
[1]東北大学・未来科学技術共同研究センター、[2]東北大院・医・精神神経学

Sep 21 (Tue) Symposium

SY07-03 内臓知覚、情動と過敏性腸症候群の脳機能画像
Functional neuroimaging in visceral perception, emotion and irritable bowel syndrome
福土　審[1]、濱口　豊太[1]、鹿野　理子[1]
[1]東北大院・医・行動医学

SY07-04 うつ病・双極性障害・心的外傷後ストレス障害における前頭葉機能の特徴－多チャンネル近赤外線スペクトロスコピーによる検討
Characteristics of frontal lobe function in depression, bipolar disorder, and posttraumatic stress disorder: A multichannel near-infrared spectroscopy study
福田　正人[1]、上原　徹[1]、井田　逸朗[1]、三國　雅彦[1]
[1]群馬大院・医・脳神経精神行動学

SY07-05 外傷性ストレス障害（PTSD）の脳画像解析
Structural and functional neuroimaging in posttraumatic stress disorder
笠井　清登[1]
[1]東京大院・医・精神医学

SY07-06 Neuroimaging and the traumatized brain: PTSD and emotion
Ruth A. Lanius[1]
[1]Department of Psychiatry, University of Western Ontario, London, Ontario, Canada

14:40～17:40　Room D

SY08-01～SY08-05 RNA 神経生物学：ニューロンにおける転写後の世界
RNA neurobiology: Posttranscriptional world in neurons
オーガナイザー：武井　延之（新潟大・脳研究所・分子神経生物）
岡野ジェイムス洋尚（慶應義塾大・医・生理学）

SY08-01 神経活動による新規なRNAスプライシングの制御機構
Alternative poly (A) site selection confers neural activity-dependent regulation of RNA splicing of vesl-1/homer-1
井ノ口　馨[1]
[1]三菱化学生命科学研究所 井ノ口グループ

SY08-02 IP3 receptor type 1 mRNA の3末端非翻訳領域に結合し、その樹状突起への局在化及び小脳運動学習に関わるRNA結合タンパク質Hzf
Hzf binds to IP3R1 mRNA and is required for dendritic mRNA localization and the motor learning in cerebellum
飯島　崇利[1,2]、今井　貴雄[1,2]、高月香菜子[3]、小倉　博雄[4]、宮崎　太輔[5]、岡野　洋尚[1,2]、川原　茂敬[3]、渡辺　雅彦[5]、桐野　豊[3]、岡野　栄之[1,2]
[1]慶應義塾大・医・生理、[2]科技団・CREST、[3]東京大院・薬・神経生物物理、[4]エーザイ筑波研究所、[5]北海道大院・医・生体構造解析、[6]サミュエルルーネンフェルド研究所

SY08-03 RNA結合タンパク質RNG105による神経シナプス刺激依存的な局所的翻訳制御
RNG105: A Novel RNA-Binding Protein in Neuronal RNA Granules, Regulatory Machinery for Synaptic Stimulation-Dependent Local Translation
椎名　伸之[1,2]、新倉　和美[1]、徳永万喜洋[1,2,3]
[1]遺伝研・構造遺伝学センター、[2]総研大、[3]理研・免疫センター

SY08-04 マウスLarkによる時計遺伝子Peiod1の転写後発現制御機構の解析
Mouse Larks activate post-transcriptional expression of the Period1 gene.
小島志保子[1]、広瀬　松美[1]、長野　　護[2]、重吉　康史[2]、榊　　佳之[3]、程　　肇[1]
[1]三菱化学生命研・ゲノム時間生物学、[2]近畿大・医・第二解剖、[3]理研・ゲノム科学総合研究センター

SY08-05 ニューロンにおけるBDNFによって誘起されるmTOR依存性の翻訳調節機構
mTOR-dependent translational control in neurons induced by BDNF and neural activity
武井　延之[1]、稲村　直子[1]、戚　　思華[1]、難波　寿明[1]、水野　　誠[1]、那波　宏之[1]
[1]新潟大学・脳研究所・分子神経生物

14:40～17:40　Room E

SY09-01～SY09-05 局所回路ネットワークとしての孤束核複合体－発生・発達から機能分子まで
The solitary complex network - from development to the molecular organization of synaptic transmission
オーガナイザー：加藤　総夫（東京慈恵医大・総合医科学研究センター）
佐藤　容子（東京医歯大院・医歯学総合研究科）

SY09-01 孤束核局所神経ネットワークの特徴と生後発達
The nucleus of tractus solitarius: The local network and its developmental regulation
河合　良訓[1]
[1]東京慈恵会医科大学解剖学講座第1

SY09-02 光学的計測法を用いた孤束核シナプス伝達および回路網形成の機能発生過程の解析
Optical analysis of developmental organization of functional synaptic networks in the NTS
佐藤　容子[1]
[1]東京医歯大院・医・機能協関システム医学

SY09-03 DOPAによる孤束核内シナプス伝達
DOPA, as a neurotransmitter candidate in the nucleus tractus solitarii
五嶋　良郎[1]、矢上　達郎[1]、青木　令奈[1]、内海　雄一[1]、小倉　顕一[1]、中村　史雄[1]
[1]横浜市立大院・医・分子薬理神経生物

SY09-04 孤束核における咳反射のシグナル伝達経路
Synaptic organization of cough reflex in the nucleus tractus solitarius
櫨　　彰[1]、大井　義明[1]、武田　龍司[1]
[1]富山医科薬科大・医・薬理

SY09-05 孤束核における神経伝達物質放出制御
Transmitter release regulation in the nucleus of the solitary tract
加藤　総夫[1]
[1]慈恵医大・神経生理

Sep 21 (Tue) Symposium

14:40～17:40　Room F

SY10-01～SY10-05　モデル脳領域（小脳とキノコ体）に着目した分子から行動までの脳機能解析
Simple system approaches to the higher brain functions

オーガナイザー：伊藤　悦朗（北海道大院・理学研究科）
川原　茂敬（東京大院・薬学系研究科）

SY10-01　「モデル脳領域（小脳とキノコ体）に着目した分子から行動までの脳機能解析」を開催するにあたって
Overview of the symposium: Simple system approaches to the higher brain functions
伊藤　悦朗[1]
[1]北海道大・院理・生物科学

SY10-02　ミツバチ脳のキノコ体をモデルとした脳分子生物学の未来
Future aspect of molecular neurobiology using honeybee mushroom body as a model system.
竹内　秀明[1]
[1]東大・院理・生物科学

SY10-03　What bees can teach us: Networks and dynamic molecular mechanisms underyling memory formation
Uli Mueller[1]
[1]Free University of Berlin and Saarland University, Saarbruecken, Germany

SY10-04　小脳依存性記憶を担う分子機構の解析
Molecular dissection of cerebellar-dependent memory
遠藤　昌吾[1]
[1]理研・脳センター・遠藤研究ユニット

SY10-05　瞬目反射学習を用いた高次中枢機能の解析
Eyeblink conditioning as a window for the higher nervous system
川原　茂敬[1]
[1]東京大院・薬・神経生物物理学

17:45～19:45　Room D　日本神経化学会オープンシンポジウム

SY11-01～SY11-05　論文投稿だけでなく、研究成果を社会にもっと還元しよう!：研究成果が特許申請・臨床治験・創薬・起業に結びついた例
Positive Link to the Society: Not only scientific papers but also patents, clinical trials, development of novel drugs, and foundation of venture compamies.

オーガナイザー：渡辺　恭良（大阪市立大院・医・システム神経科学）
鍋島　俊隆（名古屋大院・医・医学部附属病院薬剤部）

SY11-01　遺伝子発現データの解析ツールGeneFISの開発
Development of customized software GeneFIS for analysis of gene expression profiles
折原　　進[1]、本多　裕之[2]
[1]三井情報開発株式会社・バイオサイエンス本部・新事業開発部、[2]名古屋大学大学院工学研究科

SY11-02　グレリン（Ghrelin）の発見とその生理的意義
Discovery of ghrelin and its physiological significance
寒川　賢治[1]
[1]国立循環器病センター研究所・生化学部

SY11-03 球脊髄性筋萎縮症治療薬の開発
Therapeutic approach for spinal and bulbar muscular atrophy based on pathogenesis
祖父江 元[1]
[1]名古屋大院・医・神経内科

SY11-04 神経変性の mechanism- based 治療技術の開発:筋萎縮性側索硬化症および上位運動神経変性疾患の治療
Apoptosis based remedy for amyotrophic lateral sclerosis (ALS) and upper motor neuron dominant ALS
池田 穣衛[1]
[1]東海大学・総合医学研究所・分子神経科学部門

SY11-05 中枢神経系創薬のための行動科学研究ラインアップ
Line-up of behavioral methods in development of CNS drugs
鍋島 俊隆[1]、野田 幸裕[1]
[1]名古屋大院・医・医療薬学 附属病院薬剤部

Sep 22 (Wed) Symposium

9:00～11:30 Room A

SY12-01～SY12-05 初期視覚野研究の最前線

Leading edge of the research on early visual systems

オーガナイザー：佐藤 宏道（大阪大学・健康体育部）
大澤 五住（大阪大院・工・生命機能研究科）

SY12-01 一次視覚野における文脈依存的反応修飾のメカニズム
Contextual modulation in the primary visual cortex
佐藤 宏道[1]
[1]大阪大・健康体育部

SY12-02 視覚野における神経結合特異性
Organizational rules for the specificity of neuronal connections in visual cortex
吉村由美子[1]、Edward Callaway[2]
[1]名古屋大・環境医学研・視覚神経科学、[2]Salk Inst., La Jolla

SY12-03 初期視覚野における神経細胞の受容野特性
Additional receptive field properties of neurons in the early visual cortex
大澤 五住[1]
[1]大阪大学・生命機能研究科

SY12-04 V1における視覚情報の充填と補完
Bridging gaps at V1: neural responses for filling-in and completion
小松 英彦[1]
[1]生理学研究所・感覚認知情報

SY12-05 初期視覚野に見る高次脳機能：V2野における形の表現
Seeing higher cortical functions in early visual systems: Shape representation in area V2.
伊藤 南[1,2]
[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

9:00～11:30 Room C

SY13-01～SY13-05 大脳皮質の細胞構築制御

Cellular principles of neocortical development

オーガナイザー：佐藤 真（福井大学・医・形態機能医科学講座）
仲嶋 一範（慶應義塾大・医・解剖学教室）

SY13-01 高等生物における大脳皮質形成とモータータンパク・微小管の制御
Dynamic regulation of microtubules and cytoplasmic dynein in migrating neurons.
広常 真治[1]
[1]阪市大院・医・細胞機能制御学

SY13-02 アクチン結合タンパクフィラミンによる移動細胞の形態制御
Regulation of cell polarity and shape by Filamin A in the subventricular and intermediate zones during radial migration
佐藤 真[1]、永野 隆[1]
[1]福井大・医・形態機能医科学

SY13-03 大脳皮質形成のダイナミクス
Dynamics of cerebral cortical development
仲嶋 一範[1,2]、味岡 逸樹[1]、田畑 秀典[1]
[1]慶應義塾大学・医・解剖学、[2]東京慈恵会医科大学・医・DNA研・分子神経生物学

SY13-04 CORTICAL INTERNEURON MIGRATION
John Parnavelas[1]
[1]Dept. Anatomy, University College London, UK

SY13-05 The involvement of cortical hem in neocortical histogenesis through being a major source of Cajal-Retzius cells
Michio Yoshida[1]、Elizabeth A Grove[1]
[1]Dept. Neurobiol., Pharm., Physiol., Univ. of Chicago, Chicago, IL. USA

9:00～11:30 Room D

SY14-01～SY14-05 報酬と目標に基づく行動選択の神経機構
Neural correlates of action selection based on reward and goal

オーガナイザー：木村 實（京都府立医大院・医・神経生理）
虫明 元（東北大院・医・生体システム生理）

SY14-01 報酬に基づく意志決定と学習におけるドーパミン系の関与
Involvement of dopamine system in reward-based decision and learning
木村 實[1]、佐藤 武正[2]、松本 直幸[1]、榎本 一紀[1]
[1]京都府立医大院・医・神経生理、[2]鳥取大院・医・生命科学

SY14-02 ドーパミン神経細胞による文脈依存の報酬予測誤差
Dopamine neurons represent context-dependent reward prediction error
中原 裕之[1]
[1]理化学研究所・脳総研・脳数理研究チーム

SY14-03 前頭連合野内側部における行動の選択と評価
Selection and evaluation of actions in the medial prefrontal cortex
松元 健二[1]、松元まどか[1]、田中 啓治[1]
[1]理研・脳センター・認知機能表現

SY14-04 ゴール選択に関わる前頭前野の神経機構
Prefrontal cortical mechanisms for selecting behavioral goals
虫明 元[1]
[1]東北大学大学院・医・生体システム生理

SY14-05 Reward-based learning in dopamine neurons, striatum and orbitofrontal cortex
Wolfram Schultz[1]
[1]Department of Anatomy, University of Cambridge, Cambridge, UK

Sep 22 (Wed) Symposium

9:00～11:30 Room E

SY15-01～SY15-05 家族性アルツハイマー病原因遺伝子（APPおよびPresenilin）によるアミロイドβ蛋白産生および神経機能調節機構について

The roles of APP and presenilin in the generation of amyloid β-protein and neuronal function

オーガナイザー：駒野 宏人（国立長寿医療研究センター）
鈴木 利治（北海道大院・ゲノム機能学講座）

SY15-01 プレセニリン複合体のもつガンマセクレターゼ活性の調節機構
Mechanism underlying the regulation of presenilin-mediated γ-secretase activity
駒野 宏人[1]
[1]国立長寿医療センター・アルツハイマー病研究部

SY15-02 X11LとAlcadeinによるAPPの代謝と機能の調節
Regulation of APP metabolism and function by X11L and Alcadein
鈴木 利治[1]、荒木 陽一[1]、宮城 尚美[1]、加藤 直子[1]、佐久間めぐみ[1]、水丸 智絵[1]、中矢 正[1]
[1]北海道大院・薬・神経科学

SY15-03 APP細胞質領域における神経組織特異的リン酸化の解析
Analysis of neuron-specific phosphorylation of APP cytoplasmic domain in neural functions
中矢 正[1]、関 健二郎[2]、鈴木 利治[1]、糸原 重美[2]
[1]北大院・薬・神経科学、[2]理化学研究所、脳センター

SY15-04 プレセニリン1の生物学的機能
Biological roles of presenilin 1
中島 光業[1]、白澤 卓二[1]
[1]東京都老人研・分子老化

SY15-05 プレセニリンのカドヘリン依存シグナリングにおける役割
γ-Secretase associated and unassociated roles of presenilin in cadherin-dependent signaling pathways
塩井 純一[1]
[1]Dept. of Psychiatry, Mount Sinai School of Medicine

9:00～11:30 Room F

SY16-01～SY16-06 疼痛メカニズム探求の新展開

The frontier of research on pain mechanisms

オーガナイザー：野口 光一（兵庫医大・第2解剖学）
富永 真琴（三重大学・医・分子細胞生理学）

SY16-01 神経因性疼痛におけるミクログリアとATP受容体の関与
The involvment of microglia and ATP receptors in neuropathic pain
井上 和秀[1,2]、津田 誠[1]、小泉 修一[1]
[1]国立衛生研・代謝生化学、[2]九州大学・院・薬学・分子制御

SY16-02 神経因性疼痛の誘発分子としてのリゾホスファチジン酸
Lysophosphatidic acid as the initiator of neuropathic pain
植田 弘師[1]
[1]長崎大院・医歯薬・分子薬理学

SY16-03 ニューロパチックペインの新規メカニズムとしてのセリンプロテアーゼ及び細胞接着因子の変化
Changes in serine protease and cell adhesion molecule in nociceptive pathway as novel pathomechanisms of neuropathic pain
野口 光一[1]、山中 博樹[1]
[1]兵庫医科大学第2解剖学

SY16-04 疼痛下における情動変化：上行性神経伝達の可塑性
Change in the emotionality under a chronic pain state: Focused on the synaptic plasticity related to the ascending pain processing
成田 年[1]、矢島 義識[1]、鈴木 勉[1]
[1]星薬大・薬品毒性

SY16-05 脊髄浅層での長期増強とグリア細胞の関与：シナプス前終末及び後細胞電位のイメージングによる解析
Glial control of LTP induction in superficial dorsal horn revealed by optical imaging of pre- and postsynaptic excitation
村瀬 一之[1]、池田 弘[1]、楠堂 圭[1]、石田 将之[1]
[1]福井大・工・知能システム

SY16-06 In vivo パッチクランプ法による大脳皮質における疼痛情報処理の解析
In vivo patch-clamp analysis of the pain processing in the primary sensory cortex
吉村 恵[1]、土井 篤[1]、水野 雅晴[2]、古江 秀昌[1]、片渕 俊彦[1]
[1]九州大院・医学院・統合生理学、[2]九州工業大学大学院生命体工学研究科

9:00～11:30 Room I

SY17-01～SY17-06 成体脳で起こるニューロン新生
Neurogenesis in the adult brain
オーガナイザー：石 龍徳（順天堂大学・医学部・第二解剖）
久恒 辰博（東京大院・先端生命科学）

SY17-01 成体マウス大脳皮質脳室下帯細胞の由来
Origin of the subventricular neuron-progenitors in the adult mouse neocortex
玉巻 伸章[1]
[1]熊本大学院・医薬・脳回路構造学

SY17-02 成体マウス脳室下層におけるニューロブラストの移動方向の制御機構
Mechanisms for long-distance directional migration of neuroblasts in the adult mouse subventricular zone
澤本 和延[1,2]、岡野 栄之[1]、Arturo Alvarez-Buylla[2]
[1]慶應大・医・生理、[2]Department of Neurological Surgery, University of California, San Francisco, San Francisco, USA

SY17-03 成体マウス嗅球における匂い入力依存的な新生顆粒細胞の生存と死の解析
Sensory experience-dependent survival of newly-generated granule cells in the olfactory bulb of adult mice
山口 正洋[1]
[1]東京大院・医・細胞分子生理

SY17-04 成体脳のニューロン新生における初期分化過程の解析
The early steps in adult neurogenesis
久恒 辰博[1]
[1]東京大院・新領域・先端生命

SY17-05 成体海馬のニューロン新生で見られる細胞間相互作用について
Cell-cell interaction in hippocampal adult neurogenesis
石　龍徳[1]
[1]順天堂大学・医・解剖二

SY17-06 パーキンソン病モデルによる神経再生
Neurogenesis in a model of Parkinson's disease
望月　秀樹[1,2]、大泉　英樹[1]、吉見　健二[2]、山田　正典[2]、水野　美邦[1,2]
[1]順天堂大・医・脳神経内科、[2]順天堂大・老人性疾患病態・治療研究センター

14:40～17:40　Room A

SY18-01～SY18-07 脳の機能的左右差の起源と進化
The origin and evolution of functional lateralization of the brain
オーガナイザー：岡本　仁（理化学研究所脳科学総合研究所）
中村　俊（国立精神・神経センター）

SY18-01 脳機能の左右差の起源と進化
Origins and subsequent evolution of lateralization
Richard Andrew[1]
[1]Sussex Center for Neuroscience, School of Biological Sciences, University of Sussex, Brighton BN1 9QG, UK

SY18-02 ADVANTAGES AND DISADVANTAGES OF LATERALIZATION, AND ITS DEVELOPMENT
Lesley Rogers[1]
[1]Centre for Neuroscience and Animal Behaviour, University of New England, Armidale, Australia

SY18-03 ゼブラフィッシュの脳の左右差
Left-right asymmetry of habenular projection in zebrafish brain
相澤　秀紀[1]、Bianco Isaac[2]、浜岡　崇憲[1]、宮下　俊雄[1]、植村　修[1]、Concha Miguel[2]、Wilson Stephen[2]、岡本　仁[1]
[1]理研・脳センター・発生遺伝子制御、[2]Dep. Anat and Dev Biol, Univ. College London, London, UK

SY18-04 Role of midbrain cholinergic and dopaminergic systems in social modulation of forebrain activity
Neal A. Hessler[1]
[1]Riken Brain Science Institute, Wako-shi, Japan

SY18-05 ニワトリにおける非対称的視床投射路の発生機構
Development of asymmetric thalamofugal visual pathway in chick
中村　俊[1]、小柴満美子[1,2]
[1]国立精神・神経センター・神経研・診断、[2]東京農工大生命工

SY18-06 マウス海馬神経回路の構造的・機能的非対称性ーその分子基盤
Molecular basis for the structural and functional asymmetry of the brain: A study on mouse hippocampus
伊藤　功[1]、川上　良介[1]、篠原　良章[2]、Yue Wu[3]、重本　隆一[2,4]
[1]九州大院・理・生物、[2]生理学研究所、[3]総合研究大学院大学、[4]戦略科技団

SY18-07 Functional asymmetry in the inferior frontal cortex for verbal and nonverbal information processing.
中村　克樹[1]
[1]国立精神・神経センター神経研究所

14:40～17:10 Room C

SY19-01～SY19-04 日本神経化学会　奨励賞受賞シンポジウム
The Japanese Society for Neurochemistry Young Scientists Lectureship Award

オーガナイザー：西澤　正豊（奨励賞選考委員長／新潟大・脳研・神経内科）

SY19-01 オリゴデンドロサイトの発生制御機構の解析
Molecular Mechanisms Underlying Oligodendrocyte Development
鹿川　哲史[1]
[1]熊本大・発生医学研究センター・転写制御

SY19-02 神経軸索成長の分子機構の研究
Molecular Mechanisms of Axon Growth
上口　裕之[1]
[1]理化研・脳科学総合研究センター・神経成長機構

SY19-03 嗅覚受容体の構造と機能に関する研究
Studies on structure and function of olfactory receptors
東原　和成[1]
[1]東京大・院・新領域創成科学・先端生命科学・分子認識化学

SY19-04 神経ペプチドPACAPの分子薬理学的機能解析
Molecular and pharmacological analysis of neuropeptide PACAP functions
橋本　　均[1]
[1]大阪大・院・薬・神経薬理

14:40～17:40 Room D

SY20-01～SY20-05 単一細胞レベルの神経発生
Single Cell Neurogenesis

オーガナイザー：松崎　文雄（理化学研究所・発生・再生科学総合研究センター・非対称細胞分裂研究グループ）
貝淵　弘三（名古屋大学院・医・神経情報薬理）

SY20-01 神経発生における非対称分裂と細胞極性
Cell polarity and asymmetric divisions in neurogenesis
松崎　文雄[1]
[1]理化学研究所・発生・再生科学総合研究センター・非対称細胞分裂研究グループ

SY20-02 神経上皮細胞の非対称分裂時における細胞内因子の動態の解析
Dynamic movement of cellular components during asymmetric cell division of neuroepithelial cells
若松　義雄[1]
[1]東北大院・医・器官構築

SY20-03 神経細胞の極性制御機構
Neuronal polarity and axon elongation
貝淵　弘三[1]、有村奈利子[1]、西村　隆史[1]
[1]名古屋大学院・医・神経情報薬理

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SY20-04 樹状突起のパターン形成の調節
Extrinsic and intrinsic control of dendritic shaping
上村　匡[1,2]、島　康之[1]、杉村　薫[3]、佐藤　大祐[3]、山本　美暁[1]、木村　宏史[3]、
碓井　理夫[1]、Stephen Crews[4]、竹市　雅俊[5]
[1]京都大院・生命科学・細胞認識、[2]CREST, JST、[3]京都大院・理学、[4]University of North Carolina, Chapel Hill, USA、[5]理研 CDB

SY20-05 軸索内パターニング：その機構と意義
Intra-axonal patterning: its mechanism and implications
広海　健[1]
[1]国立遺伝学研究所、[2]SOKENDAI・遺伝学専攻

14:40～17:40　Room E

SY21-01～SY21-04 遺伝子操作動物解析法の新しい展開
New analytical techniques for transgenic/knockout mice

オーガナイザー：齋藤　尚亮（神戸大・バイオシグナル研究センター）
饗場　篤（神戸大院・医・細胞生物）

SY21-01 ノックアウトマウスに対する部位特異的発現トランスジェニックマウスによる脳機能解析
Rescue of knockout mice by introducing transgenes using a tissue specific promoter: mGluR1-rescue mice
饗場　篤[1]
[1]神戸大院・医・細胞生物

SY21-02 遺伝子変異マウスとウイルスベクターを組み合わせた脳機能解析
Analysis of neural function by virus-mediated gene transfer into mutant mice brain
平井　宏和[1]
[1]金沢大学・学際科学実験センター・革新脳科学

SY21-03 γ PKC-GFP トランスジェニックマウスを用いた小脳プルキンエ細胞における PKC トランスロケーションのイメージング
Propagation of γPKC translocation in the Purkinje cell dendrites revealed by γPKC-GFP transgenic mice
坪川　宏[1]、酒井　規雄[2]、齋藤　尚亮[3]
[1]東北大院・情報科学、[2]広島大院・神経精神薬理、[3]神戸大・バイオシグナル研究センター

SY21-04 テトラサイクリン制御システムを用いたマウスの行動解析
Behavioral analysis of the bitransgenic mice with a region-specific promoter in combination with the tetracycline transactivator.
谷口　泰造[1,2]、齋藤　尚亮[1]
[1]神戸大学・バイオシグナル研究センター、[2]行動医科学研究所

14:40～17:40　Room F

SY22-01～SY22-07　脳の発達障害と行動異常
Developmental disorders and behavioral abnormalities

オーガナイザー：増尾　好則（独立行政法人産業技術総合研究所）
石堂　正美（独立行政法人国立環境研究所）

SY22-01　小児自閉症：疫学的所見・基本症状の理解・神経生物学的背景などに関する最近の所見
Infantile autism: recent understanding on epidemiological data, basic clinical symptoms and neurobiological background

成瀬　浩[1]
[1]財団法人日本公衆衛生協会スクリーニング精度管理センター

SY22-02　小児自閉症モデル動物におけるドーパミン神経系とアミノ酸との関連性
The relationship between dopaminergic neurons and amino acids in an animal model of autism

水間　広[1]、野崎　聡[1]、小橋隆一郎[2]、渡辺　恭良[1]
[1]大阪市大院・医・システム神経科学、[2]コハシメディカルセンター

SY22-03　ADHDの臨床と研究の進捗
Clinical characteristics of ADHD - recent advance in the pathophysiological investigation

瀬川　昌也[1]、野村　芳子[1]、福田　秀樹[2]、星野　恭子[1]、八森　啓[1]
[1]瀬川小児神経学クリニック、[2]独立行政法人 産業医学総合研究所

SY22-04　Multiple evidences for the dopamine hypothesis of Attention Deficit Hyperactivity Disorder in two animal models

Adolfo G. Sadile[1]、Davide Viggiano[1]、Lucia A. Ruocco[1]、Daniela Vallone[1]
[1]Dept Exptl Med, Second Univ. Naples, Italy

SY22-05　注意欠陥多動性障害（ADHD）モデルラットの解析
Analysis of attention-deficit hyperactivity disorder（ADHD）model rats

増尾　好則[1]
[1]産総研・ヒューマンストレスシグナル研究センター

SY22-06　内分泌撹乱化学物質は高次脳機能の性分化を障害する
Endocrine Disrupters Impair Sexual Differentiation of Higher Brain Functions

粟生　修司[1]、久保　和彦[2]、藤本　哲也、荒井　興夫[3]
[1]九州工大院・生命体・脳情報、[2]千鳥橋病院耳鼻咽喉科、[3]独協医大・生理学

SY22-07　環境ホルモンによるラット多動性障害
Endocrine disruptors cause neurodevelopmental disorder, leading to hyperactivity in the rat.

石堂　正美[1]
[1]国立環境研究所・環境ホルモン・ダイオキシン重点研究プロジェクト

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15:40～17:40 Room I

SY23-01～SY23-05 神経システムにおけるゆらぎとノイズ－その機能と役割－

Fluctuations, noise and variability in neural systems - their functional roles -

オーガナイザー：甲斐 昌一（九州大院・工学研究院）
村田 勉（情報通信研究機構 関西先端研究センター）

SY23-01 視覚的意識における離散的確率過程と神経ゆらぎの役割
Discrete stochastic process and neural fluctuations underlying visual consciousness
村田 勉[1]
[1]情報通信研・関西先端・脳情報

SY23-02 知覚交替に関連した過渡的な同期活動の特性
Property of transient synchrony related to perceptual switching
中谷 裕教[1]
[1]理研・脳科学総合研究センター・認知動力学

SY23-03 ヒト脳波の雑音による引き込み:確率同期現象
Noise-induced entrainment of human brain waves: Stochastic Synchronization
猪本 修[1]、森 敏生[2]、福田 弘和[2]、甲斐 昌一[1,2]
[1]九州大院・システム生命・生命工学、[2]九州大院・工学・エネルギー量子工学

SY23-04 ノイズによるヒトの脳の活性化
Noise-induced sensitization of human brain
山本 義春[1,2]
[1]東京大院・教育学、[2]科学技術振興機構

SY23-05 神経系はなぜ束なのか? 神経細胞の熱雑音感受性と神経系の基本構造
Why nerves are bundles of fibers? - Thermal noise sensitivity of neuron and parallel structures of the nervous system
下澤 楯夫[1]
[1]北海道大学・電子研・神経情報

9:00～11:30 Room A

SY24-01～SY24-05 認知障害モデル動物とその分子メカニズム；統合失調症との関連
Animal modeling of cognitive impairments; Their molecular mechanisms and implication in schizophrenia

オーガナイザー：那波 宏之（新潟大学・脳研究所・分子神経生物学分野）
宮川 剛（京都大院・医・先端領域融合医学研究機構）

SY24-01 統合失調症候補遺伝子 *DISC1* の機能解析
Functional analysis of *Disrupted-In-Schizophenia 1*, novel candidate gene for schizophrenia

本田 章子[1,2]、片山 泰一[2]、遠山 正彌[2]
[1]田辺製薬・薬理研、[2]大阪大・院・医・ポストゲノム疾患解析学

SY24-02 ネトリンG欠損マウスのNMDA受容体不全を伴った異常
Netrin-G deficient mice have deficits associated with NMDA receptor hypofunction

糸原 重美[1]、西村 幸子[1]、関 健二郎[1]、渡辺 明子[2]、新美 君枝[1]、吉川 武男[2]
[1]理化学研究所・脳センター・行動遺伝、[2]理化学研究所・脳センター・分子精神科学

SY24-03 NMDA受容体機能障害による認知障害の分子機序：グルタミン酸作動性神経系の関与
Molecular mechanisms of cognitive dysfunction in schizophrenic animal models: approaches from glutamatergic systems

野田 幸裕[1]、鍋島 俊隆[1]
[1]名古屋大院・医・医療薬学 附属病院薬剤部

SY24-04 タンパク脱リン酸化酵素遺伝子ノックアウトマウスの認知機能障害
Cognitive dysfunction of the forebrain specific calcineurin knockout mice: Relation to schizophrenia pathogenesis

宮川 剛[1]
[1]京都大院 医 先端領域融合医学研究機構

SY24-05 脳発達期における神経栄養因子作用攪乱とその後の認知行動異常；統合失調症との関連性
Perinatal Neurotrophic Perturbation and Later Cognitive Deficits; Implication in schizophrenia

那波 宏之[1]、任海 学[1]、津田 法子[1]、渡部雄一郎[1,2]、水野 誠[1]
[1]新潟大学・脳研・分子神経生物、[2]新潟大学大学院医歯学総合研究科精神医学分野

9:00～11:30 Room C

SY25-01～SY25-05 中枢神経におけるコレステロールの生理的意義とアルツハイマー病発症機構に果たす役割
Roles of cholesterol in the central nervous system and in the pathogenesis of Alzheimer's disease.

オーガナイザー：道川 誠（国立長寿医療研究センター）
Frank W. Pfrieger（Max-Planck/CNRS）

SY25-01 The risk for Alzheimer's disease is related to a cluster of genes involved in brain cholesterol metabolism.

Andreas Papassotiropoulos[1]、M. Axel Wollmer[1]、Dominique de Quervain[1]、
Fabienne Brunner[1]、Roger M. Nitsch[1]、Christoph Hock[1]
[1]Division of Psychiatry Research, Zurich Univ

SY25-02 Glial lipoproteins promote axon growth of CNS neurons
Hideki Hayashi[1]、Robert B. Campenot[2]、Dennis E. Vance[3]、Jean E. Vance[1]
[1]Dept. of Med., Univ. of Alberta, Edmonton, Alberta, Canada、[2]Dept. of Cell Biol., Univ. of Alberta、[3]Dept. of Biochem., Univ. of Alberta

SY25-03 Cholesterol homeostasis and function in CNS neurons
Frank W. Pfrieger[1]
[1]Max-Planck/CNRS Group

SY25-04 中枢神経系におけるアポEとその受容体の機能
Roles of apoE and it's receptors in the central nervous system
藤野　貴広[1]
[1]愛媛大・総合支援センター・生物機能解析

SY25-05 中枢神経系におけるアポリポ蛋白Eアイソフォーム特異的コレステロール輸送の検討
ApoE-isoform-specific cholesterol transport in the central nervous system
道川　誠[1]
[1]国立長寿医療センター・アルツハイマー病

9:00～11:30 Room D

SY26-01～SY26-06 選択的シナプス強化・除去の分子機構
Molecular mechanisms of selective synapse strengthening and elimination
オーガナイザー：狩野　方伸（金沢大院・医・シナプス発達機能学分野）
高橋　正身（北里大学・医学部・代謝蛋白学講座）

SY26-01 アクチビンは樹状突起スパインの形態制御を通じてシナプス可塑性に関与している。
Activin, a TGF-β superfamily protein, regulates maintenance of hippocampal LTP through modulating the dendritic spine morphology
笠井　陽子[1]、池上　司郎[1]、井ノ口　馨[1]
[1]三菱化学生命科学研究所 井ノ口グループ

SY26-02 小脳登上線維－プルキンエ細胞シナプスの生後発達
Differentiation of Climbing Fiber Synapses during Cerebellar Development
橋本　浩一[1]、狩野　方伸[1]
[1]金沢大院・医・シナプス発達・機能学

SY26-03 中枢神経系での選択的シナプス強化の分子・細胞機構におけるNMDA受容体チロシンリン酸化の役割
The role of NMDA receptor tyrosine phosphorylation in molecular and cellular mechanisms of selective synaptic potentiation in the central nervous system
真鍋　俊也[1]
[1]東京大学・医科学研究所・基礎医科学部門・神経ネットワーク分野

SY26-04 ニューロリギンを裏打ちする蛋白質S-SCAMとPSD-95
Neuroligin-associated proteins, S-SCAM and PSD-95
畑　裕[1]
[1]東京医科歯科大学・院・病態代謝解析学

SY26-05 細胞接着分子ネクチンによるシナプス形成制御
Regulation of synaptogenesis by nectin cell adhesion molecule
溝口　明[1]
[1]三重大学医学部修復再生大講座神経再生学部門

SY26-06 AMPA受容体を含有したさまざまな小胞の解析
Characterization of AMPA receptor-containing membrane vesicles in rat brain
板倉　誠[1]、山森　早織[1]、稲村実穂子[1]、西宮聖一朗[1]、高橋　正身[1]
[1]北里大学・医・代謝蛋白学

9:00～11:30 Room E JSN-JSBPジョイントシンポジウム
SY27-01～SY27-06 **精神神経疾患の分子基盤と治療戦略**
Molecular Basis and Treatment Strategy for Neuropsychiatric Disorders
オーガナイザー：御子柴克彦（東京大院・医・脳神経）
武田　雅俊（大阪大院・医・ポストゲノム疾患解析学講座）
座長：御子柴克彦（東京大院・医・脳神経）
佐藤　光源（東北福祉大）

SY27-01 小胞体ストレスと神経細胞死
ORP150, an ER-chaperone, suppresses neuronal cell death.
小川　智[1]
[1]金沢大院・医・神経分子

SY27-02 アルツハイマー病のワクチン療法
Vaccination therapy for Alzheimer's disease.
田平　武[1]、原　英夫[1]
[1]国立長寿医療センター研究所

SY27-03 疲労と疲労感の脳神経機構
Brain regions and neural circuits for fatigue and sensation of fatigue
渡辺　恭良[1]
[1]大阪市立大院・医・システム神経科学

SY27-04 ストレス一心因性精神障害の脳科学
Stress - neurobiology of psychogenic mental disorders
加藤　進昌[1]、笠井　清登[1]
[1]東京大院・医・精神医学

SY27-05 統合失調症の病態生理：特に候補遺伝子産物DISC1の大脳皮質構築にはたす役割から
Pathophysiology of schizophrenia; DISC1 and cerebral cortical development
澤　明[1]
[1]Johns Hopkins University School of Medicine Departments of Psychiatry and Neuroscience

SY27-06 双極性障害の分子基盤
Molecular basis of bipolar disorder
加藤　忠史[1]
[1]理研・脳科学センター・精神疾患動態

9:00～11:30 Room F
SY28-01～SY28-05 **薬剤による神経保護療法の現状と展望**
Recent advances and future aspects in the study of neuroprotective drugs
オーガナイザー：丸山和佳子（国立療養所中部病院長寿医療研究センター）
新田　淳美（名古屋大院・医・医学部附属病院薬剤部）

SY28-01 Mechanism behind Neuroprotection by Propargylamines
Wakako Maruyama[1]、Atsumi Nitta[2]、Masayo Nagai-Shamoto[1]、Makoto Naoi[3]
[1]Sect. Biochem., Dept. Exp. Gerontol., Natl. Center. Geriatr. & Gerontol、[2]Dept. Pharmac. & Pharmacol., Nagoya Univ. Grad. Sch. Med、[3]Dept Neurosci., Gifu Internat. Instit. Biotech.

SY28-02 疎水性ジペプチドLeu-Ileの神経保護作用
Hydrophobic dipeptide, Leu-Ile, protects against neuronal death by inducing BDNF and GDNF synthesis
新田 淳美[1]、鍋島 俊隆[1]、古川 昭栄[2]
[1]名古屋大学大学院・医療薬学・医学部附属病院・薬剤部、[2]岐阜薬科大学

SY28-03 Molecular mechanism underlying the neuroprotective and neurorescue activities of green tea polyphenol epigallocatechin-3-gallate
Silvia Mandel[1]、Moussa Youim[1]
[1]Res. Dept. Pharmacol. Faculty of Med, Technion

SY28-04 Intracellular mechanisms involved in the neuroprotective effects of IGF-1 and Gingko biloba in cultured hippocampal neurons
Zheng Wen-Hua[1]、Atsumi Nitta[1]、S. Bastianetto[1]、Y Han[1]、R Quirion[1]
[1]Douglas Hospital Research Centre, McGill University, Montreal

SY28-05 神経ペプチドPACAPによる古典的神経栄養因子の機能修飾
Neuropeptide PACAP: neuromodulatory effects on the actions of classical neurotrophins
新谷 紀人[1]、橋本 均[1]、馬場 明道[1]
[1]大阪大院・薬・神経薬理

9:00～11:30 Room I ISNシンポジウム

SY29-01～SY29-05 神経難病の神経化学的アプローチ
Neurochemical aspects and approaches for neurological diseases
オーガナイザー：遠山 正彌（大阪大院・医・ポストゲノム疾患解析学）
Peter Sonderegger (Department of Biochemistry, Zurich Univ)

SY29-01 プリオンの構造ダイナミクスに基づく創薬
Drug discovery for prion diseases based on the structural dynamics of prion protein
桑田 一夫[1]、西田 教行[2]、片峰 茂[2]
[1]岐阜大院・医・蛋白高次機能、[2]長崎大院・医歯薬・感染分子病態

SY29-02 Neurotrypsin, a synaptic serine protease involved in cognitive functions
Peter Sonderegger[1]
[1]Department of Biochemistry, Zurich Univ

SY29-03 小胞体ストレスとアルツハイマー病における神経細胞死
Molecular mechanism of cardiac hypertrophy induced by hypertension
Endoplasmic reticulum stress and neuronal death in Alzheimer disease.
今泉 和則[1]、遠山 正彌[2]
[1]奈良先端大・バイオ・細胞構造、[2]大阪大院・医・ポストゲノム疾患解析

SY29-04 アルツハイマー病βアミロイド線維形成・不安定化過程における分子間相互作用
Molecular interactions in the formation and destabilization of Alzheimer's β-amyloid fibrils in vitro
内木 宏延[1]
[1]福井大・医・分子病理

SY29-05 ABAD: a mitochondrial target of amyloid-beta peptide.
David Stern[1]
[1]Dean, Medical School, Medical College of Georgia

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9:00～11:30 Room J

SY30-01～SY30-06 糖鎖修飾と神経可塑性
Glycobiology and Plasticity of Nervous System

オーガナイザー：平林 義雄（理化学研究所脳総合科学研究センター）
加藤 啓子（大府立大院・農・獣医学）

SY30-01 *Essential roles of heparan sulfate proteoglycans in postsynaptic development*
Fumitoshi Irie[1]、Yu Yamaguchi[1]
[1]The Burnham Institute

SY30-02 グリコサミノグリカンによる神経回路形成の制御
Glycosaminoglycans regulate neural network formation
前田 信明[1]、秦 敏宏[1]、島崎由美子[1]、矢部 富雄[1]
[1]東京都神経研・分子発生

SY30-03 糖鎖異常による先天性筋ジストロフィー
Defective glycosylation in congenital muscular dystrophy
遠藤 玉夫[1]
[1]都老人研・糖蛋白質

SY30-04 ファブリー病の分子病理と酵素補充療法の開発
Molecular pathology of and development of enzyme replacement therapy for Fabry disease
桜庭 均[1]、千葉 靖典[2]、地神 芳文[2]、松澤 史子[3]、相川 聖一[3]、土居 洋文[3]
[1]東京都臨床医学総合研究所・臨床遺伝学、[2]産業技術総合研究所・糖鎖工学研究センター、[3]セレスターレキシコサイエンシーズ

SY30-05 Metabolic Labeling of Sialic Acid in Neurons
Kevin J. Yarema[1]
[1]The Johns Hopkins University, Baltimore, MD, USA

SY30-06 神経可塑性と空間学習におけるHNK-1糖鎖の関与
Involvement of the HNK-1 carbohydrate in synaptic plasticity and spatial learning
岡 昌吾[1]
[1]京都大院・薬・生体分子認識、CREST

14:40～17:40 Room A

SY31-01～SY31-04 脊髄損傷の神経修復
Spinal cord injury and neural repair

オーガナイザー：岡野 栄之（慶應義塾大・医・生理学教室）
西尾 健資（京都大院・医・認知行動脳科学）

SY31-01 成熟ラット脊髄切断モデルにおけるグリア環境の再構築と軸索再生
Reconstruction of periaxonal environment that supports axonal regeneration by CNS glial cells after transection of the spinal cord in adult rats
西尾 健資[1]
[1]京都大院・医・認知行動脳科学

SY31-02 脳脊髄液経由にニューロスフェアー、骨髄間質細胞を脊髄損傷部へ移植する新しい方法
A novel method for cell transplantation (neurosphere cells, bone marrow stromal cells) into damaged spinal cord via cerebrospinal fluid in rats
鈴木 義久[1]、張 弘富[1]、太田 正佳[1]、石川奈美子[1]、鈴木 茂彦[1]、出澤 真理[2]、井出 千束[2]
[1]京都大院・医・形成外科、[2]京都大院・医・機能微細形態学

SY31-03 P75受容体による中枢神経軸索再生阻害の分子機構
Axon growth inhibition signals from p75NTR
山下 俊英[1]
[1]千葉大院・医・神経生物

SY31-04 損傷脊髄の幹細胞治療
Stem cell therapy of injured spinal cord
岡野 栄之[1,2]
[1]慶應義塾大・医・生理学、[2]科学技術振興機構

14:40～17:40 Room C

SY32-01～SY32-05 高次脳機能発達障害の分子神経生物学的基盤
Molecular-neurobiological basis for the developmental defects of higher brain function

オーガナイザー：湯浅 茂樹（国立精神・神経センター神経研究所）
大隅 典子（東北大院・医・形態形成解析分野）

SY32-01 神経発達および精神機能における成長因子ミッドカインの役割
Roles of the growth factor midkine in neural development and mental functions
門松 健治[1]
[1]名古屋大院・医・生物化学

SY32-02 ウイルスによる行動異常：発症機序と高次脳発達障害モデルへの応用
Virus-induced neurobehavioral disorders: mechanisms and applications for developmental defect model
朝長 啓造[1]
[1]大阪大学・微研・ウイルス免疫

SY32-03 ニューロン新生低下と行動異常の関連
A potential link between impaired neurogenesis and behavior abnormalities
大隅 典子[1]、前川 素子[1]、高嶋 記子[2]、池上 史郎[2]、井ノ口 馨[2]
[1]東北大学院・医・創生応用医学研究センター、[2]三菱化学生命研

SY32-04 Fyn欠損マウスの行動ならびに精神薬理学的異常の神経機構
Behavioral and psychopharmacological abnormalities of Fyn-deficient mice and their clinical implications
服部功太郎[1,3]、八木 健[1,3]、湯浅 茂樹[2]
[1]大阪大院・生命機能・心生物学グループ、[2]国立精神神経センター神経研究所、[3]科学技術振興機構、クレスト

SY32-05 注意欠陥多動性障害（ADHD）の分子神経生物学的研究
Molecular and neurobiological study on attention deficit hyperactivity disorder (ADHD)
池田 和隆[1]、高松 幸雄[1]、山本 秀子[1]、猪子 香代[2]、曽良 一郎[3]
[1]都・精神研・分子精神、[2]都・精神研・児童思春期、[3]東北大院・医・精神神経生物

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14:40～17:40 Room D

SY33-01～SY33-05 脊椎動物中枢神経系形成のダイナミズム
Dynamism in vertebrate CNS development

オーガナイザー：仲村 春和（東北大院・生命科学研究科）
渡邉 裕二（東北大院・生命科学研究科）

SY33-01 発達段階依存的な大脳皮質ニューロンの移動様式
Cortical neurons at different developmental stages show distinct migratory behaviors
畠中由美子[1,2]、久永 真市[3]、村上富士夫[2,4]
[1]筑波大・基医・分子神経生物、[2]科学技術振興機構、[3]都立大・理、[4]大阪大・院・生命機能

SY33-02 大脳皮質の神経細胞移動におけるPax6遺伝子の役割
The role of Pax6 in tangential migration of cortical neurons
野村 真[1]、大隅 典子[1]
[1]東北大院・医・創生応用

SY33-03 骨髄系幹細胞制御におけるNotchシグナルの役割
The role of Notch signal in bone marrow-derived stem cells
勝部 憲一[1]、坂本 啓[1]、王 勝朝[1]、山口 朗[1]、梅澤 明弘[2]
[1]東京医歯大・院・口腔病理、[2]国立成育医療センター・生殖医療研究部

SY33-04 発生中の中枢神経系に発現する免疫グロブリンスーパーファミリーに属する新たな膜タンパク質
A novel cell surface protein of Ig superfamily member in developing CNS
渡邉 裕二[1]、豊田 礼子[1]、仲村 春和[1]
[1]東北大院・生命科学・脳構築

SY33-05 Slitrkファミリー膜貫通蛋白質の神経発生における役割
The role of Slitrk transmembrane protein family in neural development
有賀 純[1]、片山 圭一[1]、守村 直子[1]、佐藤 愛[1]
[1]理研・脳総研・比較神経発生

14:40～17:40 Room E

SY34-01～SY34-05 脳の社会適応化機構:環境・ホルモン情報と遺伝情報の統合と破綻
Mechanisms of Adaptive Socialization in the Brain: Integration and Disruption among Environmental, Hormonal and Gene Informations

オーガナイザー：筒井 和義（広島大学・総合科学部脳科学）
河田 光博（京都府立医大院・医・解剖学）

SY34-01 海馬におけるストレスホルモン応答に対する受容体の動態
Dynamism of stress relevant receptors in the hippocampus
河田 光博[1]
[1]京都府立医科大学・解剖学・生体構造科学

SY34-02 Functional genomics of hypothalamic homeostatic plasticity
David Murphy[1]
[1]Henry Wellcome Laboratories for Integrative Neuroscience and Endocrinology, Universityof Bristol

SY34-03 発生工学的手法を通してみた繁殖行動形成プロセスへのニューロステロイドホルモン環境の重要性
Analysis of crucial roles of the neurosteroidal environment in organization processes of reproductive behaviors by embryonic engineering
原田 信広[1]、Nicoletta Aste[1]、本田伸一郎[1]
[1]藤田保衛大・医・生化学

SY34-04 活動性を高める新規ニューロステロイドとストレス応答
A novel neurosteroid stimulating locomotor activity and acute stress response
筒井 和義[1,2]、松永 昌宏[1,2]、浮穴 和義[1,2]
[1]広島大・総合科学部脳科学・統合脳科学プロジェクト研究センター、[2]科学技術振興機構

SY34-05 報酬予期の神経表象と衝動的行為決定
Neural correlates of reward anticipation and impulsive choice.
松島 俊也[1]
[1]名古屋大院・生命農学

14:40～17:40 Room F

SY35-01～SY35-06 脳神経系のD-アミノ酸：機能と病態
D-Amino acids in the nervous systems: functions and pathophysiology

オーガナイザー：西川 徹（東京医歯大院・精神行動医科学分野）
白澤 卓二（東京都老人総合研究所）

SY35-01 視覚系におけるD-アスパラギン酸含有蛋白質の生成とその機構
Spontaneous D-aspartic acid formation in protein from elderly human lenses
藤井 紀子[1]
[1]京大・原子炉

SY35-02 アスパラギン酸残基異性化とアルツハイマー病神経変性
Isoaspartate Formation and Neurodegeneration in Alzheimer's Disease
白澤 卓二[1]
[1]東京都老人総合研究所・分子老化研究グループ

SY35-03 哺乳類体内におけるD-アスパラギン酸の役割
Biological function of D-aspartate in mammals
本間 浩[1]
[1]北里大学・薬学部

SY35-04 D-セリンと統合失調症
D-Serine and schizophrenia
西川 徹[1]、山本 直樹[1]、海野 麻未[1]、櫻井新一郎[1]、嶋津 奈[1]、谷口 豪[1]、石井 澄和[1]、岩間 久行[1]
[1]医歯大院・医歯総合・精神行動

SY35-05 脳における遊離型D-セリンの局在
Localization of free D-serine in the brain
仙波 禮治[1]
[1]愛知県コロニー・発達障害研究所

SY35-06 脳におけるD-アミノ酸酸化酵素の機能的・病態生理学的意義
Functional roles and pathophysiology of brain D-amino acid oxidase
福井 清[1]
[1]徳島大・酵素研・遺伝制御

14:40～17:40　Room I

SY36-01～SY36-05　デルタ２受容体の10年－最近の展開
10th anniversary of Delta2-Casting new light on its function

オーガナイザー：柚崎　通介（慶應義塾大・医・生理学）
三品　昌美（東京大院・医・分子神経生物学）

SY36-01　小脳回路形成と小脳学習の鍵分子としてのグルタミン酸受容体δ２
Glutamate receptor δ2 as a key molecule of cerebellar wiring and learning

三品　昌美[1]
[1]東京大院・医・分子神経生

SY36-02　グルタミン酸受容体デルタ２サブユニット欠損マウスにおける運動制御異常
Discoordination of motor control in a GluRδ2 knockout mouse

平野　丈夫[1,2]、吉田　盛史[1,2]、加藤　明[1,2]、大槻　元[1,2]
[1]京都大院・理・生物物理、[2]CREST, JST

SY36-03　成体小脳プルキニエ細胞特異的グルタミン酸受容体δ２欠損によりシナプス構造変化が引き起こされる
Synaptic mismatching and disconnection by inducible and Purkinje cell-specific ablation of glutamate receptor δ2 in mature cerebellum

竹内　倫徳[1]、宮崎　太輔[2]、渡辺　雅彦[2]、森　寿[1]、崎村　建司[3]、三品　昌美[1]
[1]東京大院・医・分子神経生物、[2]北海道大院・医・解剖発生、[3]新潟大・脳研・細胞神経生物

SY36-04　競合的小脳シナプス形成の分子基盤
Molecular mechanisms for competitive synaptic wiring in cerebellar Purkinje: GluRδ2 and P/Q-type calcium channel.

渡辺　雅彦[1]
[1]北海道大院・医・解剖発生

SY36-05　デルタ２グルタミン酸受容体の新しい活性化機構と機能
A novel activation mechanism and function of δ2 glutamate receptors

柚崎　通介[1]
[1]慶應大学・医・生理

一般演題（口演発表）
プログラム

	1st		2nd		3rd	
	Morning	Afternoon	Morning	Afternoon	Morning	Afternoon
神経疾患 I／Neurological Diseases I	Room B OB1-01~10					
神経疾患 II／Neurological Diseases II		Room B OB1-11~22				
再生、移植、神経細胞死／Regeneration, Transplantation and Neuronal Death			Room B OB2-01~11			
自律神経系、行動、睡眠、生体リズム／Autonomic Nervous System, Behavior, Sleep and Biological Rhythms				Room B OB2-12~23		
運動、視覚／Motor System, Visual System					Room B OB3-01~10	
学習、記憶、睡眠、生体リズム／ Learning and Memory, Sleep and Biological Rhythms						Room B OB3-11~23
視覚／Visual System	Room G OG1-01~04	Room G OG1-11~16		Room H OH2-11~22		
疾患モデル／Animal Models	Room G OG1-06~10					
老化、痴呆／Aging, Dementia		Room G OG1-11~21				
突起伸展、回路網形成／Axonal Outgrowth and Network Formation			Room G OG2-01~10			
受容体、輸送体／Receptors and Transporters				Room G OG2-11~16		
グリア、ミエリン、血液脳関門／Glia, Myelin and Blood-Brain Barrier				Room G OG2-17~22		
シナプス、シナプス可塑性／Synapse, Synaptic Plasticity					Room G OG3-01~10	
シナプス可塑性／Synaptic Plasticity						Room G OG3-11~19
情報伝達、伝達物質、受容体／Signal Transduction, Neurotransmitters and Receptors	Room H OH1-01~10					
栄養因子、サイトカイン／Trophic Factors and Cytokines		Room H OH1-11~16				
再生、移植／Regeneration, Transplantation		Room H OH1-17~22				
情報伝達、神経伝達物質／Signal Transduction and Neurotransmitters			Room H OH2-01~05			
軸索輸送、細胞運動、細胞骨格／Axonal Transport, Cell Motility and Cytoskeleton			Room H OH2-06~10			
イオンチャネル、興奮性膜／Ion Channels and Excitable Membranes					Room H OH3-01~07	
シナプス／Synapse						Room H OH3-08~12
パターン形成／Pattern Formation						Room H OH3-13~18
認知、コンピュータ科学／Cognition and Computer Science	Room J OJ1-01~10					
情報伝達、受容体、嗅覚、痛覚／Signal Transduction, Receptors, Olfaction and Pain		Room J OJ1-17~21				
イメージング、分子生物学的、細胞生物学的方法／Imaging, Molecular and Cellular Biological Techniques			Room J OJ2-01~10			
聴覚、嗅覚、味覚、化学感覚／Auditory System, Olfaction, Taste and Chemical Senses				Room J OJ2-11~16		
痛覚／Pain				Room J OJ2-17~22		
細胞分化／Cellular Differentiation					Room J OJ3-01~12	

◆◇◆ Oral Session ◆◇◆

9:00～10:15 Room B

OB1-01～OB1-05 神経疾患 I

Neurological Diseases I

座長：馬場 明道（大阪大院・薬・神経薬理）
功刀 浩（国立精神・神経センター・神経研究所）

OB1-01 中枢神経系におけるPACAPの分子薬理学的機能解析
Molecular and pharmacological analysis of PACAP in CNS functions
橋本 均[1]、新谷 紀人[1]、田中 一裕[1]、馬場 明道[1]
[1]大阪大院・薬・神経薬理

OB1-02 統合失調症脆弱性遺伝子ディスバインジンの関連解析と神経細胞における機能解析
Evidence of novel neuronal functions of dysbindin, a susceptibility gene for schizophrenia
橋本 亮太[1]、沼川 忠広[1]、矢ケ崎有希[1]、石本 哲也[1]、鈴木 竜世[1]、岩田 伸生[1]、尾崎 紀夫[1]、田口 隆久[1]、巽 雅彦[1]、上島 国利[1]、Richard Straub[1]、Daniel Weinberger[1]、功刀 浩[1]
[1]国立精神・神経センター・神経研究所・疾病研究第三部

OB1-03 Disrupted-In-Schizophrenia 1（DISC1）新規結合蛋白の機能解析
Novel zincfinger protein that interacts with Disrupted-In-Schizophrenia 1 (DISC1)
服部 剛志[1]、山口 淳[1]、井上 浄[1]、松崎 伸介[1]、本田 章子[1]、馬場 孝輔[1]、遠山 正彌[1]
[1]21世紀COEプログラム・大阪大院・医・ポストゲノム疾患解析学講座

OB1-04 DISC1/kinesin-IによるNUDEL/LIS1/14-3-3の輸送制御機構
DISC1 regulates the transport of NUDEL/LIS1/14-3-3 via Kinesin-I
田谷真一郎[1]、永井久美子[1]、篠田 友靖[1]、黒田 摂子[1]、貝淵 弘三[1]
[1]名古屋大院・医・神経情報薬理

OB1-05 統合失調症関連遺伝子産物DISC1の神経細胞における役割
Roles of Disrupted-in-Schizophrenia-1 (DISC1) in neuronal morphologies
永井久美子[1]、田谷真一郎[1]、篠田 友靖[1]、貝淵 弘三[1]
[1]名古屋大院・医・分子薬理学

10:15～11:30 Room B

OB1-06～OB1-10 神経疾患 I

Neurological Diseases I

座長：内田 さえ（東京都老人総合研究所）
山形 要人（東京都神経研・神経薬理）

OB1-06 ニューロン・グリア共培養系を用いた神経細胞の虚血耐性現象におけるアストロサイトの病態生理学的役割の解明
Reversed astrocytic glutamate transporter GLT-1 crucial to the development of neuronal ischemic tolerance in astrocyte/neuron co-cultures.
小杉 達郎[1]、河原 剛一[1]、田中 基樹[1]、山田 武志[1]、中島 崇行[1]
[1]北海道大院・情報科学・細胞情報

OB1-07 内側中隔核刺激は脳虚血によっておこるラット海馬での遅発性神経細胞死を防ぐ
Stimulating the medial septum can protect against ischemia-induced delayed neuronal death in rat hippocampus
堀田 晴美[1]、内田 さえ[1]
[1]東京都老人総合研究所・運動自律機能相関研究グループ

OB1-08 脳内セロトニン神経系は片頭痛病態（Spreading depression）に影響する
Brain serotonergic nervous system modulates spreading depression: A pathophysiology of migraine
崔 翼龍[1]、片岡 洋祐[1]、田村 泰久[1]、李 慶華[2]、野崎 聡[2]、水間 広[2]、
渡辺 恭良[2]、山田 久夫[1]
[1]関西医科大・解剖学第一講座、[2]大阪市立大院・医・システム神経科学

OB1-09 モルモット海馬スライスにおける，ビキュキュリン誘導てんかん波に対するシナプス可塑性の役割
The Role of Synaptic Plasticity in the Bicuculline-Induced Epileptic Discharges in Guinea-Pig Hippocampal Slices.
緒方 元気[1]、夏目季代久[1]
[1]九州工業大院・生命体工学研究科

OB1-10 カイニン酸誘発神経細胞脱落におけるプロスタグランジンの作用
The role of prostaglandin in neuronal loss induced by kainate
竹宮 孝子[1,2]、松村 潔[3]、前原 通代[4]、山形 要人[2]
[1]東京都神経研・神経薬理、[2]東京女子医大・第一生理、[3]京都大院・情報・生体情報処理、[4]東京女子医大・中央検査部

14:40～16:10 Room B

OB1-11～OB1-16 神経疾患 II
Neurological Diseases II

座長：川野 仁（東京都神経研・発生形態）
内山 安男（大阪大院・医・機能形態学）

OB1-11 先天的に多動性障害を呈するWigラットの解析
Analysis of spontaneously hyperactive Wig rats
長嶋 和郎[1]、石堂 正美[2]、澤 洋文[1]、森田 昌敏[2]、岡 修一[3]、二木 鋭雄[4]、
増尾 好則[5]
[1]北海道大院・医・分子細胞病理、[2]国立環境研・環境ホルモン/ダイオキシン研究領域プロジェクト、[3]産総研・生物機能工学研究部門、[4]産総研・ヒューマンストレスシグナル研究センター、[5]産総研・ヒューマンストレスシグナル研究センター

OB1-12 Fynチロシンキナーゼ欠損マウスにおける大脳新皮質形成異常の解析
Defective neocortical development in Fyn-tyrosine-kinase-deficient mice
湯浅 茂樹[1]、服部功太郎[2,3]、中平 英子[1]、八木 健[2,3]
[1]国立精神神経センター・神経研・微細構造、[2]大阪大院・生命機能研究科、[3]CREST（JST）

OB1-13 Complexin2遺伝子欠損マウスの母子分離ストレス脆弱性
Vulnerability of Complexin2 knockout mouse for maternal deprivation
山内 祥豪[1]、秦 麗紅[1]、谷 新医[1]、西原 真理[1]、澤田 健[1]、加藤 邦夫[1]、
井上 新平[1]
[1]高知大学・医・神経統御・神経精神病態医学

OB1-14 GM2ガングリオシド蓄積症モデルマウス網膜の神経突起再生能異常
Impaired neurite regeneration in the retina of a murine model of GM2 gangliosidosis
三五 一憲[1]、高野 雅彦[2]、安食 京子[1]、渡嘉敷晶子[1]、川野 仁[1]、堀江 秀典[3]、山中 正二[4]
[1]東京都神経研・発生形態、[2]北里大学・医・眼科、[3]早稲田大学・先端バイオ研究所、[4]横浜市立大学・医・病理学

OB1-15 カテプシンDノックアウトマウス、カテプシンBLダブルノックアウトマウスにおける、オートファジー関連蛋白MAP-LC3の挙動
Modification of LC3, a novel autophagosomal marker, in brain and peripheral tissues of mice deficient in cathepsin D and those doubly-deficient in cathepsins B and L
小池 正人[1]、柴田 昌宏[1]、和栗 聡[1]、木南 英紀[2]、Christoph Peters[3]、Kurt von Figura[4]、Paul Saftig[5]、八木沼洋行[6]、後藤 隆洋[7]、内山 安男[1]
[1]大阪大院・医・機能形態学、[2]順天堂大・医・生化学、[3]Inst. fur Molekulare Medizin und Zellforschung, Albert Ludwigs Univ. Freibrug, Freibrug, Germany、[4]Center for Biochemistry and Molecular Cell Biology, Goetingen Univ., Goetingen, Germany、[5]Dept of Biochemistry, Univ. Kiel, Kiel, Germany、[6]福島県立医大・医・解剖、[7]甲子園大・栄養

OB1-16 神経可塑性に関わるシアル酸付加の役割
Effect of sialylation on acquiring neural plasticity.
加藤 啓子[1]、岩森 正男[2]、平林 義雄[3]
[1]大阪府大・農・獣医・実験動物、[2]近畿大・理工・化学科、[3]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

16:10～17:40 Room B

OB1-17～OB1-22 神経疾患 II
Neurological Diseases II

座長：戸田 達史（大阪大院・医・ゲノム機能分野）
岡野 栄之（慶大・医・生理学）

OB1-17 Bax抑制ペプチド（BIP）はMachado-Joseph病のカスパーゼ非依存性細胞死を防ぐ
Bax-inhibitory peptide (BIP) prevents caspase-independent cell death in Machado-Joseph disease
横田 隆徳[1]、李 一[1]、松山 茂実[2]、水澤 英洋[1]
[1]東京医科歯科大学・医・神経内科、[2]The Department of Biochemistry, Medical College of Wisconsin, Milwaukee, U.S.A.

OB1-18 ハンチンチンにより誘導される小脳神経細胞特異的なHsp70は細胞死を抑制する
Neuron type-specific Hsp70 induction by huntingtin suppresses cell death in cerebellar neurons
田川 一彦[1]、星野 将隆[1]、奥田 智博[2]、村田 美穂[3]、金澤 一郎[3]、Wanker Erich E.[4]、岡澤 均[1,5]
[1]東京医科歯科大・難治研・神経病理、[2]東京都神経科学総合研究所、[3]国立精神・神経センター、[4]Max Delbrueck Center for Molecular Medicine、[5]PRESTO, JST

OB1-19 膜透過性阻害ペプチドQBP1を用いたポリグルタミン病に対する分子治療法の確立
Establishing a molecular therapy for the polyglutamine diseases using the cell permeable peptide inhibitor PTD-QBP1
ポピエル ヘレナ明子[1]、永井 義隆[1]、藤掛 伸宏[1]、戸田 達史[1]
[1]大阪大院・医・ゲノム機能

OB1-20 新規ALS原因遺伝子産物alsinによるSOD1誘導性神経細胞死抑制機構の解析
Alsin, the product of ALS2 gene, suppresses SOD1 mutant neurotoxicity via RhoGEF domain by interactiong with SOD1 mutants
金蔵 孝介[1]、橋本 祐一[1]、新倉 貴子[1]、相磯 貞和[2]、松岡 正明[1]、西本 征央[1]
[1]慶応義塾大院・医・薬理、[2]慶應大・医・薬理

OB1-21 神経系特異的新規ユビキチンリガーゼの機能的解析
Functional Analysis of Novel Ubiquitin Ligase, Potential Target Antigen of Paraneoplastic Neurologic Disorder
多田 敬典[1,2]、岡野ジェイムス洋尚[1,2]、松本 雅記[2,3]、中山 敬一[2,3]、鹿島 晴雄[4]、
岡野 栄之[1,2]
[1]慶大・医・生理学、[2]科技団・CREST、[3]九大・生医研・分子発現、[4]慶大・医・精神神経学

OB1-22 MuSK自己抗体により発症する重症筋無力症の分子病態についての解析
Autoantibodies against MuSK ectodomain inhibited multiple pathways of AChR clustering.
重本 和宏[1]、久保 幸穂[2]、太田 光熙[3]、太田 潔江[4]、丸山 直記[2]
[1]愛媛大学・医・衛生学、[2]都老人研・加齢臓器障害研究グループ、[3]神戸薬科大・臨床化学、
[4]国療・宇多野病院・臨床研究部

9:00～10:15 Room B

OB2-01～OB2-05 再生、移植、神経細胞死
Regeneration, Transplantation and Neuronal Death

座長：寺川 進（浜松医大・光量子医学研究センター）
吉田 純（名古屋大院・医・脳神経外科学）

OB2-01 カニクイサル胚性幹細胞を用いた選択的神経分化誘導と評価
Selective Neuronal Differentiation and Evaluation of Cynomolgus Embryonic Stem Cells
遠藤 乙音[1]、水野 正明[2]、梶田 泰一[1]、吉田 純[1]
[1]名古屋大院・医・細胞情報・脳神経外科学、[2]名古屋大院・医・細胞情報・遺伝子治療学

OB2-02 線条体に移植された神経上皮細胞の分化とシナプス再構築
Grafted neuroepithelial stem cells in the rat striatum differentiate to multiple neurons and receive synaptic inputs from the host area
籾山 俊彦[1]、内田 耕一[2]、峯 裕[2]、河瀬 斌[2]
[1]生理研・脳形態解析、[2]慶応大・医・脳神経外科

OB2-03 虚血サル海馬においては神経幹細胞は血管外膜に由来する
Vascular Adventitia Generates Neuronal Progenitors in the Monkey Hippocampus after Ischemia
山嶋 哲盛[1]、Anton Tonchev[1]
[1]金沢大院・医・脳機能制御

OB2-04 イノシトール7リン酸は細胞死を誘導する
Diphohoinositol pentakisphosphate (IP7) is a mediator as cell death
永田栄一郎[1]、Hongbo Luo[2]、Adolfo Saiardi[2]、鈴木 則宏[1]、Solomon Snyder[2]
[1]慶應義塾大・医・神経内科、[2]Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, MD, USA

OB2-05 急性および遅発性神経細胞死においてグルタミン酸により活性化されるカスパーゼ
Glutamate-induced caspase activation in acute and delayed neuronal death
山本 清二[1]、范 文英[1]、櫻井 孝司[1]、若園 佳彦[1]、寺川 進[1]
[1]浜松医大・光量子医学研究センター

10:15～11:45 Room B

OB2-06～OB2-11 再生、移植、神経細胞死
Regeneration, Transplantation and Neuronal Death

座長：久永 真市（都立大院・理・生物科学）
八木沼洋行（福島県立医大・医・第一解剖学）

OB2-06 Bax過剰発現はニワトリ胚における二つの異なったプログラムされた神経細胞死のあり方を示す
Bax overexpression reveals two distinct modes of programmed neuronal death in the chicken embryo
佐藤 昇[1]、佐久間千恵[1]、佐藤 康子[1]、八木沼洋行[1]
[1]福島県立医大・医・解剖学第一

OB2-07 NAIP発現誘導ドーパミン受容体リガンドの抗アポトーシス効果
Anti-apoptotic effect of the NAIP up-regulating dopamine receptor ligands
岡田 義則[1,2]、酒井 治美[1]、古曳 英理[3]、須賀恵津子[3]、田中 一則[1]、秦野 伸二[1,3]、大須賀 等[1,3]、池田 穣衛[1,3]
[1]東海大・総医研・分子神経、[2]東海大学・医・教育研究、[3]科技団・SORST

OB2-08 ニプラジロールは培養大脳皮質ニューロンにおいて一酸化窒素を遊離することによりグルタミン酸誘発アポトーシスを抑制する
Nipradilol inhibits glutamate-induced apoptosis in cultured cortical neurons via nitric oxide-dependent mechanism
田口 良太[1]、久米 利明[1]、香月 博志[1]、赤池 昭紀[1]
[1]京都大院・薬・薬品作用解析

OB2-09 神経細胞死におけるAATYKキナーゼのリン酸化状態の変化
Apoptosis-Associated Tyrosine Kinase (AATYK) undergoes phosphorylation during neuronal apoptosis.
友村美根子[1]、古市 貞一[1]
[1]理研・脳総研・分子神経形成研究チーム

OB2-10 ERストレスによる神経細胞死とCdk5活性化サブユニットp35のp25への限定分解
Calpain-dependent proteolysis of p35 Cdk5 activator to p25 in ER stress-induced neuronal cell death
斎藤 太郎[1]、今野 玄理[1]、浅田 明子[1]、久永 真市[1]
[1]都立大院・理・生物科学

OB2-11 ヒューマニン アンタゴニスト：二量体化作用欠失ヒューマニン変異体によるヒューマニン神経保護作用の阻害
Humanin antagonists
橋本 祐一[1]、新倉 貴子[1]、相磯 貞和[2]、寺下 謙三[1]、松岡 正明[1]、西本 征央[1]
[1]慶應大・医・薬理、[2]慶應大・医・解剖

14:40～16:10 Room B

OB2-12～OB2-17 自律神経系、行動、睡眠、生体リズム

Autonomic Nervous System, Behavior, Sleep and Biological Rhythms

座長：小林 茂夫（京都大院・情報・生体・認知情報学・生体情報処理）

金子 武嗣（京都大院・医・高次脳形態）

OB2-12 延髄縫線核に存在するsympathetic premotor neuron：発熱誘発や体温調節機能への関与

Identification of sympathetic premotor neurons in medullary raphe regions mediating fever and other thermoregulatory functions

中村 和弘[1]、松村 潔[2]、Thomas Huebschle[3]、武 勝昔[1]、Ruediger Gerstberger[3]、小林 茂夫[2]、金子 武嗣[1]

[1]京都大院・医・高次脳形態、[2]京都大院・情報・生体情報処理、[3]Inst Veterin-Physiol, Justus-Liebig-Univ, Germany

OB2-13 新生ラット摘出脳標本の辺縁系における自発バースト活動の時空間パターン及びその呼吸活動との関連

Spatio-temporal pattern of spontaneous burst activity in the limbic system and its relation to respiratory activity in the isolated brain preparation from newborn rat

鬼丸 洋[1]、本間 生夫[1]

[1]昭和大学・医・第二生理

OB2-14 延髄腹側サブスタンスP応答ニューロンによる悪心時の自律反応の制御

Substance P responsive neurons in the ventral medulla regulate nauseous autonomic reactions in rats.

氷見 直之[1]、古我 知成[2]、小橋 基[3]、辻岡 克彦[1]

[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、[3]岡山大院・医歯・口腔生理

OB2-15 ストレスによるマウス毛周期の成長期誘導遅延と、香料暴露による抑制

Odor restores hair cycle delay caused by immobilization stress in mice.

森山 未央[1]、尾仲 達史[2]、江浜 律子[1]、坂井 圭子[1]、土師信一郎[1]、岸本 治郎[1]

[1]株式会社資生堂・リサーチセンター、[2]自治医科大学・医・生理学

OB2-16 末梢の局所炎症はインターロイキン6を介して脳内プロスタグランジンE_2合成を促進する

Localized peripheral inflammation augments brain prostaglandin E_2 formation through interleukin-6 signaling

松村 潔[1]、Tammy Cartmell[2]、水島由美子[1]、細川 浩[1]、Stephen Poole[2]、小林 茂夫[1]

[1]京都大院・情報・知能情報・生体情報処理、[2]NIBSC, Hertfordshire, UK

OB2-17 ラットの視床下部室傍核におけるFos発現に及ぼす自発および強制走行の影響

Effects of spontaneous and forced running on induction of Fos expression in hypothalamic paraventricular nucleus of rats.

柳田 信也[1]、小林 良子[1]、古谷 累[1]、北 一郎[1]

[1]東京都立大院・理・身体運動科学

Sep 22 (Wed) /23 (Thu) Oral Session

16:10～17:40 Room B

OB2-18～OB2-23 自律神経系、行動、睡眠、生体リズム
Autonomic Nervous System, Behavior, Sleep and Biological Rhythms

座長：北 一郎（東京都立大院・理・身体運動科学）
香山 雪彦（福島県立医大・医・第二生理）

OB2-18 危険予期に伴う神経活動：fMRI研究
Neural Activity Associated With Risk Anticipation: An fMRI Study
福井 裕輝[1]、村井 俊哉[1]、花川 隆[2]、林 拓二[1]、福山 秀直[2]
[1]京都大院・医・脳病態生理学精神医学教室、[2]京都大院・医・高次脳機能センター

OB2-19 血圧の変化による覚醒反応に対する扁桃体中心核の役割
The role of the central nucleus of the amygdala in arousal response evoked by hypertension and hypotension in rats
小林 良子[1]、柳田 信也[1]、古谷 累[1]、北 一郎[1]
[1]東京都立大院・理・身体運動科学

OB2-20 2種のカルバコール誘導β振動に対するNMDA受容体活性の役割
The role of the activation of NMDA receptor in two types carbachol-induced βoscillation
新井 潤[1]、夏目季代久[1]
[1]九州工業大院・生命体工・脳情報

OB2-21 マウスの腎交感神経活動測定法
Method for Continuous measurement of renal sympathetic nerve activity in conscious mice
吉本 光佐[1]、三木 健寿[1]
[1]奈良女大院・人間文化・人間環境

OB2-22 レム睡眠発現ニューロンの活動を調節するGABA性入力について
Regulation of the firing of PS-on neurons by GABAergic inhibition
小山 純正[1]、高橋 和巳[1]、香山 雪彦[1]
[1]福島県立医大・医・第二生理

OB2-23 前頭前野における脳の状態に依存した周期的同期的活動
Brain-state-dependent synchronized oscillation in prefrontal cortex
坂田 秀三[1]、山森 哲雄[1]、櫻井 芳雄[2,3]
[1]基礎生物学研究所・種分化第一、[2]京都大院・文・心理学研究室、[3]CREST

9:00～10:15 Room B

OB3-01～OB3-05 運動、視覚
Motor System, Visual System

座長：藏田 潔（弘前大・医・第二生理学）
高田 昌彦（東京都神経研・統合生理）

OB3-01 立位想像と立位姿勢保持中の脳賦活
Brain activations during motor imagery of standing and assuming of standing postures
尾内 康臣[1]、菅野 敏彦[1]、吉川 悦次[2]、関根 吉統[1,2]、二ッ橋昌実[2]、小楠 智美[2]
[1]浜松医療センター・先端医療技術センター、[2]浜松ホトニクス

OB3-02 到達運動における座標変換を反映する運動前野腹側部の運動関連ニューロンの両側性活動
Bilateral activation of movement-related neurons in the ventral premotor cortex reflecting coordinate transformation in reaching
蔵田 潔[1]
[1]弘前大・医・第二生理

OB3-03 線条体局所神経回路における皮質入力の選択
Processing of cortical inputs in the Striatal local circuits
三浦 正巳[1]、鈴木 健雄[2,3]、増田 正雄[1]、西村 欣也[3]、青崎 敏彦[1]
[1]東京都老人総合研究所・神経回路動態、[2]都立墨東病院・麻酔、[3]順天堂大・医・麻酔

OB3-04 霊長類における皮質脊髄路損傷後の手指の巧緻運動の機能回復機構
Dexterous Finger Movements in Primate after Lesion of the Direct Corticomotoneuronal Connection
伊佐 正[1]、佐々木成人[2]、L-G Pettersson[3]、B Alstermark[4]、内藤 公郷[2]、吉村 和也[2]、関 和彦[1]、大木 紫[5]
[1]自然科学研究機構・生理学研究所・認知行動発達機構、[2]東京都神経研、[3]Inst Physiol Pharmacol, Dept Physiol Göteborg Univ, Göteborg, Sweden、[4]Dept Integr Med Biol sect Physiol, Umeå Univ, Umeå, Sweden、[5]杏林大学・生理学教室

OB3-05 大脳皮質MST野の両側破壊が眼球運動に及ぼす影響
Bilateral MST lesions severely impaired short-latency visual tracking eye movements
竹村 文[1]、井上 由香[1]、村田 弓[1]、河野 憲二[1,2]
[1]（独）産総研・脳神経情報、[2]京都大院・医・認知行動脳科学

10:15～11:30 Room B

OB3-06～OB3-10 運動、視覚
Motor System, Visual System

座長：河野 憲二（京都大院・医・認知行動脳科学）
伊佐 正（自然科学研究機構・生理学研究所）

OB3-06 網膜神経節細胞における周期的同期発火は視覚誘発性逃避行動に重要な役割を果たす
The oscillatory synchronization in retinal ganglion cells plays a key role in visually evoked escape behavior
雁木 美衣[1]、石金 浩史[1]、堀内 靖大[1]、本田 祥子[1]、立花 政夫[1]
[1]東京大院・人社・心理

OB3-07 MSTニューロンによる視覚刺激の動きの脳内表現
Neurons in the area MST represent visual movements in world-centered coordinates.
稲場 直子[1,2,3]、山根 茂[1,2]、河野 憲二[1,4]
[1]産総研・脳神経情報・システム脳科学、[2]筑波大院・医・生理、[3]日本学術振興会、[4]京都大院・医・認知行動脳科学

OB3-08 サッカード課題時の動機付けに関係した脚橋被蓋核のニューロン活動
Contribution of Pedunculopontine tegmental nucleus neurons to motivational state during visually guided saccade tasks
小林 康[1]、井上 由香[2]、相澤 寛[3]、伊佐 正[2]
[1]大阪大院・生命機能研究科、[2]生理学研究所・認知行動発達機構研究部門、[3]弘前大・医

OB3-09 眼球運動虫部皮質仮説によるsaccade適応の加速と消失現象の説明
Oculomotor vermis hypothesis applied to the acceleration and erasure of saccadic adaptation
藤田 昌彦[1]
[1]法政大学・工・経営工

OB3-10 下オリーブ小脳投射との関連における小脳皮質のアルドラーゼC発現パタンの機能的意義
Functional significance of aldolase C expression pattern in the cerebellar cortex in its relationship to the olivocerebellar projection
杉原 泉[1]、篠田 義一[1]
[1]東京医科歯科大学・大学院・システム神経生理学

14:40～16:10 Room B

OB3-11～OB3-16 学習、記憶、睡眠、生体リズム
Learning and Memory, Sleep and Biological Rhythms

座長：八尾 寛（東北大院・生命・脳機能解析）
城所 良明（群馬大・生体調節研究所）

OB3-11 ラットの感覚毛除去によるバレル野の睡眠時脳波ダイナミクスの変化
Changes of Sleep EEG Dynamics in Rat Barrel Cortex Associated with Long-term Sensory Deprivation
岩崎直穂子[1]、辛島 彰洋[1]、玉川 雄一[1]、中尾 光之[1]、片山 統裕[1]、山本 光璋[1]
[1]東北大院・情報科学・バイオモデリング論分野

OB3-12 BMAL1の過剰発現は、視交叉上核ニューロンに見られるサーカディアン・カルシウム濃度リズムを抑制する
Over-expression of BMAL1 suppressed circadian cytosolic Ca^{2+} rhythms in cultured suprachiasmatic nucleus neurons
池田 真行[1,2]、杉山 崇[2]、吉岡 亨[2]、池田 正明[3]
[1]（財）大阪バイオサイエンス研究所・分子行動生物学部門、[2]早稲田大学・先端バイオ研究所、[3]埼玉医科大学・ゲノム医学研究センター

OB3-13 RORレスポンスエレメント結合因子による*Bmal1*リズム性発現の制御
Modulation of the circadian change of *Bmal1* promoter activity by ROR response element binding factors
池田 正明[1,2]、熊谷 恵[1,2]、徐 海源[1,2]、楊 芳[1,2]、林 隆司[1,2]、野村 正彦[2]
[1]埼玉医大・ゲノム医学研究センター、[2]埼玉医大・生理

OB3-14 時計遺伝子period発現部位のシナプス伝達阻害は長期記憶の呼び出しを阻害する
Disruption of synaptic transmission through *period*-expressing cells blocks retrieval of long-term memory in *Drosophila*
坂井 貴臣[1]、北本 年弘[2]、城所 良明[1]
[1]群馬大院・医・遺伝発達行動学、[2]The University of Iowa College of Medicine, Iowa City, USA

OB3-15 絵の再認記憶に関するfMRIを用いた研究
Neural correlates of recognition memory for pictures - an fMRI study-
飯高 哲也[1,3]、松本 敦[1]、野川 純平[1]、定藤 規弘[2,3]
[1]名古屋大院・環境・心理、[2]自然科学研究機構・生理学研究所、[3]JST/RISTEX

OB3-16 空間学習マウスにおける海馬苔状線維シナプス新生の形態計測解析
Morphometric analysis of hippocampal mossy fiber synaptogenesis in spatially learning mice
八尾 寛[1,2]、和地 恵[1,2]、奈良 聡[1,2]、鎌田 真希[2]、石塚 徹[1,2]、仲井 邦彦[3]、佐藤 洋[3]
[1]東北大院・生命・脳機能解析、[2]CREST, 科学技術振興機構、[3]東北大院・医・環境保健医学

16:10～17:55 Room B

OB3-17～OB3-23 学習、記憶、睡眠、生体リズム
Learning and Memory, Sleep and Biological Rhythms
座長：櫻井 芳雄（京都大院・文・心理学）
西条 寿夫（富山医科薬科大・医・システム情動科学）

OB3-17 サル連合記憶に対する下部側頭葉細分野損傷効果
Lesion effects of inferotemporal subdivisions on associative memory in monkeys
柳沼 重弥[1]、大沢 康隆[1]、山口 清子[1]
[1]東京都神経研・認知行動

OB3-18 サブミリ秒のニューロン間相互作用を検出するリアルタイム式スパイク・ソーティング
Real-time sorting for sub-millisecond neuronal interactions *in vivo*
高橋 晋[1,2]、櫻井 芳雄[1,3]
[1]京都大院・文・心理、[2]日本学術振興会、[3]CREST, 科学技術振興機構

OB3-19 刺激提示時間のワーキングメモリーにおけるサル前頭連合野ニューロンの相関活動
Neuronal correlation in the monkey prefrontal cortex in working memory for stimulus duration
櫻井 芳雄[1,2]、高橋 晋[1]
[1]京都大院・文・心理学、[2]科学技術振興機構・CREST

OB3-20 新規場所符号化におけるラット海馬神経活動に対する上乳頭体核の関係
Relationships between the hippocampus and the supramammillary nucleus for spatial encoding of a novel place in rats
関野 祐子[1,2]、伊藤 真[2,3]、銅谷 賢二[2,3]、白尾 智明[1,2]
[1]群馬大学院・医・高次細胞機能、[2]戦略基礎、[3]科学技術振興機構、OIST

OB3-21 非空間的記憶過程における海馬CA3領野内NMDA受容体の役割：瞬目反射条件付け課題を用いて
Hippocampal CA3 NMDA receptors are crucial for adaptive timing of trace eyblink conditioned response
岸本 泰司[1,2]、中沢 一俊[2,3]、利根川 進[3]、桐野 豊[1,4]、狩野 方伸[1]
[1]金沢大院・医・シナプス発達・機能学、[2]NIMH, NIH, U.S.A、[3]Center for Learning and Memory, MIT, U.S.A、[4]東大院・薬

OB3-22 Rat hippocampal neuronal activity during a delayed stimulus-response association task on a mobile robot.
Samantha Dayawansa[1]、Tsuneyuki Kobayashi[1,2]、Katsumi Umeno[1,2]、Etsuro Hori[1,2]、Toru Tazumi[1,2]、Taketoshi Ono[1,2]、Hisao Nishijo[1,2]
[1]System Emotion Sci, Fac Medicine, Toyama Med & Pharmaceu Univ, Toyama.、[2]CREST, JST Tokyo.

OB3-23 摂食によって増加するグルコース濃度は学習記憶に必須
Glucose increase in CSF by food intake is necessary to learning and memory
大村 裕[1]、粟生 修司[2]、福永 浩司[3]、高野 志伸[4]、石橋 賢[4]、佐々木和男[4]
[1]九州大院・医・統合生理、[2]九州工業大学・生命体工学、[3]東北大学・薬・薬理、[4]富山大学・工・生体情報

9:00～10:00 Room G
OG1-01～OG1-04 視覚
Visual System

座長：大澤 五住（大阪大院・生命機能・視覚神経科学）
程 康（理化学研究所・脳科学総合研究センター）

OG1-01 サルCIP（caudal intraparietal）野ニューロンの三次元曲面図形に対する選択的反応
Three-dimensional curvature tuning in area CIP (caudal intraparietal) of macaque monkey
勝山 成美[1]、長沼 朋佳[1]、酒田 英夫[2]、泰羅 雅登[1]
[1]日本大院・医・応用システム神経科学、[2]聖徳栄養短期大学

OG1-02 内因性光学計測法によるラット視覚野両眼、単眼領域の可視化および組織化学的検証
Histochemical confirmation of binocular and monocular zones identified by intrinsic optical imaging in rat visual cortex
大橋 一徳[1,2]、田中 繁[1,2]
[1]九州工業大学・生命体工学研究科、[2]理化学研究所・脳科学総合研究センター・視覚神経回路モデル研究チーム

OG1-03 水平・垂直線条に反応する第一次視覚野領域の更なる機能的分割
Functional Segregation of the Cortical Region Representing Cardinal Contours
王 鋼[1]、黒岩 大[1]
[1]鹿児島大・工・生体工学科

OG1-04 Molecular organization of the ferret visual thalamus
Hiroshi Kawasaki[1,2]、Justin C. Crowley[1]、Frederick J. Livesey[3]、Lawrence C. Katz[1]
[1]HHMI & Duke Univ. Med. Cntr., Durham, USA、[2]The Univ. of Tokyo, Tokyo, Japan、[3]Wellcome/CR UK institute & Univ. of Cambridge, Cambridge, UK

10:00～11:15 Room G
OG1-06～OG1-10 疾患モデル
Animal Models

座長：貫名 信行（理化学研究所・脳科学総合研究センター）
中別府雄作（九州大・生体防御医学研究所・脳機能制御）

OG1-06 αシヌクレイン・トランスジェニックマウスの作出と解析
Generation and analysis of transgenic mice that express human α-synuclein in dopamine neurons
石井 愛子[1]、若松 正樹[1]、鵜飼友里子[1]、坂神 純子[1]、岩田 晋吾[1]、小野美恵子[1]、中村 厚[1]、小林 和人[2]、岩坪 威[3]、吉本 真[1]
[1]大正製薬（株）・医薬研究所、[2]福島県立医大・生体情報伝達研・生体機能研究、[3]東京大院・薬・臨床薬学

OG1-07 パーキンソン病の病態の一部を示すαシヌクレイン・トランスジェニックマウスの作出
Generation of transgenic mice that show a part of pathological features of Parkinson's disease
吉本 真[1]、石井 愛子[1]、鵜飼友里子[1]、坂神 純子[1]、岩田 晋吾[1]、小野美恵子[1]、小林 和人[2]、岩坪 威[3]、若松 正樹[1]
[1]大正製薬（株）・医薬研究所、[2]福島県立医大・生体情報伝達研・生体機能研究、[3]東京大院・薬・臨床薬学

OG1-08 MPTP処置C57BL/6マウスの黒質線条体ドパミン神経変性に伴う運動機能異常
Nigrostriatal dopaminergic degeneration causes motor dysfunction as detected by the tail suspension test in MPTP-treated C57BL/6 mice
森 厚詞[1,2]、大橋 聡[1]、中井 正三[1]、光本 泰秀[1]、森泉 哲次[2]
[1]大塚製薬株式会社・探索第二研究所、[2]信州大・医・人体構造学講座

OG1-09 Involvement of Neuromelanin in Selective Vulnerability of Dopamine Neurons in Parkinson disease
Makoto Naoi[1]、Wakako Maruyama[2]、Masayo Nagai-Shamoto[2]
[1]Dept Neurosci, Gifu Internat. Inst. Biotech.、[2]Sect. Biochem., Dept. Exp. Gerontol., Natl. Center Geriatr. & Gerontol.

OG1-10 マンガン誘発ドパミン神経毒性とパーキンの保護効果におけるキノン体生成の関与
Role of quinone formation in protective effects of parkin against manganese-induced dopaminergic cytotoxicity
浅沼 幹人[1]、宮崎 育子[1]、小川 紀雄[1]
[1]岡山大院・医歯・神経情報学

14:40～15:55 Room G

OG1-11～OG1-15 老化、痴呆
Aging, Dementia

座長：芝崎 太（都臨床研・細胞生理）
加藤 武（横浜市大・総合理学・分子認識部門）

OG1-11 脊髄損傷後Semaphorin3A阻害剤投与による損傷軸索の再生と機能回復
Axonal Regeneration and Functional Recovery after Administration of Strong and Selective Semaphorin3A Inhibitor into the Injured Spinal Cord
岩波 明生[1,2]、金子慎二郎[1,2]、中村 雅也[2]、岡野ジェームズ洋尚[1]、岸野 晶祥[3]、菊池 薫[3]、木村 徹[3]、熊谷 和夫[3]、五嶋 良郎[4]、戸山 芳昭[2]、岡野 栄之[1]
[1]慶大・医・生理学・整形外科、[2]慶大・医・整形外科、[3]住友製薬・研究本部、[4]横浜市大・医・薬理学

OG1-12 神経突起伸展及びシナプス形成を機序とするwithanolide類の空間記憶障害改善作用
Ameliorative effects of withanolide derivatives on spatial memory impairment by neurite outgrowth and synaptic construction
久保山友晴[1,2]、東田 千尋[1]、小松かつ子[1,2]
[1]富山医薬大・和漢薬研・薬効解析センター、[2]富山医薬大・21世紀COEプログラム

OG1-13 新規ペプチド化合物CsJの虚血性脳障害治療薬への応用
Potent neuroprotective effect of CsJ, a novel cyclosporin A-analogue in ischemic brain damage
芝崎 太[1]、坂田 和彦[1]、内田 和代[1]、中田 裕久[2]、工藤 佳久[3]、内野 博之[4]
[1]都臨床研・細胞生理、[2]藤沢製薬、[3]東薬大・生命科学・生体高次機能、[4]東京医大・八王子医療センター・麻酔科

OG1-14 Clozapine but no haloperidol reverses the effect of MK-801 on neuropeptide-processing enzymes in rat brain regions
Mohammad Arif[2]、Toshiyuki Chikuma[2]、Takeshi Kato[1]
[1]Laboratory of Natural Information Science, Graduate School of Integrated Science, Yokohama City University、[2]Department of Hygenic Chemistry, Showa Pharmaceutical University

OG1-15 βアミロイドによるcalsyntenin familyの選択的発現制御とその神経細胞死への役割
Distinct expression of calsyntenin family in β-treated neurons and its role in neuronal death
内田 洋子[1]、五味不二也[1]
[1]都老人総合研究所・老年病ゲノム

15:55～17:25 Room G

OG1-16～OG1-21 老化、痴呆
Aging, Dementia

座長：武田 雅俊（大阪大院・医・精神医学）
井原 康夫（東京大院・医・基礎神経医学）

OG1-16 βAPPのγ切断の細胞内局在部位の検討
Subcellular localization of γ-cleavage of βAPP.
福森 亮雄[1]、大河内正康[1]、姜 経緯[1]、木村 亮[1]、谷井 久[2]、工藤 喬[1]、田上 真次[1]、武田 雅俊[1]
[1]大阪大院・医・精神医学、[2]三重大・医・精神科

OG1-17 血液脳関門を介したAβ1-40排出輸送特性の解析
Characteristics of Aβ1-40 efflux transport mechanism at the blood-brain barrier
伊藤 慎悟[1]、森 しのぶ[1]、堀 里子[1,2,3]、大槻 純男[1,2,3]、寺崎 哲也[1,2,3]
[1]東北大院・薬・薬物送達、[2]東北大・未来研、[3]SORST

OG1-18 アルツハイマー病脳でのCa^{2+}/カルモデュリン依存性蛋白質燐酸化酵素IIによるタウの燐酸化反応
Phosphorylation of tau by CaM kinase II in Alzheimer's disease brain
山本 秀幸[1]、村山美由紀[2]、高島 明彦[2]
[1]熊本大院・医薬・細胞情報薬理、[2]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

OG1-19 タウオパチーの線虫モデル
C. elegans model of tauopathy
丁 震[1]、宮坂 知宏[1]、安藤 恵子[2]、三谷 昌平[2]、井原 康夫[1]
[1]東京大院・医・脳神経病理、[2]東京女子医大・第二生理学

OG1-20 Immature Nicastrin Stabilizes APH-1 independent of PEN-2 and Presenilin
Keiro Shirotani[1]、Dieter Edbauer[1]、Stefan Prokop[1]、Harald Steiner[1]、Christian Haass[1]
[1]Laboratory for Alzheimer's and Parkinson's disease, Department of Biochemistry, Adolf-Butenandt-Institute, Ludwig-Maximilians-University

OG1-21 赤ワイン関連ポリフェノールのアルツハイマー病βアミロイド線維に対する分解作用
Potent anti-amyloidogenic and fibril-destabilizing effects of red wine-related polyphenols in vitro
小野賢二郎[1]、吉池 裕二[2]、高島 明彦[2]、長谷川一浩[3]、内木 宏延[3]、山田 正仁[1]
[1]金沢大院・医・脳老化神経病態学、[2]理化学研究所・脳科学総合研究センター、[3]福井大学・医・第2病理学

Sep 22 (Wed) Oral Session

9:00～10:15 Room G

OG2-01～OG2-05 突起伸展、回路網形成
Axonal Outgrowth and Network Formation

座長：山本 亘彦（大阪大院・生命機能・脳神経工学）
吉原 良浩（理化学研究所・脳科学総合研究センター）

OG2-01 新規細胞間シグナルシステムCD47-SHPS-1系の神経系における機能
A novel intercellular communication system, the CD47-SHPS-1 system, in neuronal network
大西 浩史[1]、宮下 元明[1]、金子 由夏[1]、林 朗子[1]、富澤 恭子[1]、小林 久江[1]、岡澤 秀樹[1]、的崎 尚[1]
[1]群馬大学・生調研・生理活性物質センター

OG2-02 嗅覚神経系発達過程におけるホメオボックス型転写因子Arxの機能解析
Arx homeobox gene is essential for development of mouse olfactory system
吉原 誠一[1]、大道 佳世[2]、柳澤 昌子[2]、北村 邦夫[2]、吉原 良浩[1]
[1]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、[2]三菱化学生命科学研究所

OG2-03 神経網膜におけるWnt-LEF-1シグナルによる神経突起の伸長の制御
Regulation of neurite extension of neural retina by Wnt-LEF-1 signalling pathway
大内 靖夫[1]、新井 賢一[1]、渡辺すみ子[1]
[1]東大院・医・分子細胞生物学

OG2-04 GSK-3βとCRMP-2による神経極性の制御機構
GSK-3β regulates phosphorylation of CRMP-2 and neuronal polarity
吉村 武[1]、河野 洋治[1]、貝淵 弘三[1]
[1]名古屋大院・医・神経情報薬理学

OG2-05 大脳皮質の神経回路形成におけるT－カドヘリンの発現
T-cadherin expression in cortical circuit formation
服部 祐紀[1]、乾 佳代[1]、小林久美子[1]、小林 裕明[1]、武内 恒成[2]、乗岡 茂巳[1]、山本 亘彦[1]
[1]大阪大院・生命機能、[2]名古屋大院・理・生命理学

10:15～11:30 Room G

OG2-06～OG2-10 突起伸展、回路網形成
Axonal Outgrowth and Network Formation

座長：桜井 正樹（帝京大・医・生理学）
吉岡 亨（早稲田大学・先端バイオ研究所）

OG2-06 突起伸展機能に見られる軸索の可塑性
Axonal plasticity in neurite extention
堀江 秀典[1]、小柳 清光[2]、中嶋 聡美[1]、河上江美子[2]、佐藤真奈美[1]、門屋 利彦[3]、吉岡 亨[1]、Wolfram Tetzlaff[4]
[1]早稲田大学・先端バイオ研究所、[2]東京都神経科学総合研究所・神経病理、[3]キリンビール株式会社・医薬カンパニー生産技術研究所、[4]ICORD (Int. Collaboration on Repair Discoveries), University of British Columbia

OG2-07 Fyn欠損マウスに見られる水頭症の解析
Severe hydrocephalus in *fyn*$^{-/-}$ mice
後藤 純[1]、手塚 徹[1]、中澤 敬信[1]、山本 雅[1]
[1]東大・医科研・癌細胞シグナル

OG2-08 皮質脊髄路シナプス除去過程における臨界期の *in vitro* モデル
An *in vitro* model of critical period for corticospinal synapse elimination during development
大野 孝恵[1]、桜井 正樹[1]
[1]帝京大・医・生理学

OG2-09 小脳前核細胞群の核の起源は片側菱脳唇由来か、それとも両側菱脳唇由来か
Unilateral and bilateral origins of precerebellar neurons as revealed by exo utero gene transfer.
川内 大輔[1,3]、斎藤哲一郎[2]、村上富士夫[1]
[1]大阪大院・生命機能・脳発生生理、[2]京都大・再生医科学・発生分化、[3]SORST, 日本科学技術振興事業団

OG2-10 発達期皮質脊髄路シナプス退行の時間経過
Time course of corticospinal synapse regression during development
上山 勉[1]、吉岡 昇[1]、桜井 正樹[1]
[1]帝京大学・医・生理学

14:40～16:10 Room G

OG2-11～OG2-16 受容体、輸送体
Receptors and Transporters

座長：福田 敦夫（浜松医大・生理学第一）
川上 潔（自治医大・分子病態治療研究センター）

OG2-11 摂食調節分子メラニン凝集ホルモン受容体1のインターナリゼーションの解析
Characterization of rat melanin-concentrating hormone receptor 1 internalization
斎藤祐見子[1]、手塚 満恵[1]、丸山 敬[1]
[1]埼玉医大・薬理

OG2-12 AMPA受容体サブユニットタンパクの選択的会合
Preferred assembly of AMPA receptor subunit
鈴木 岳之[1]、James Brorson[2]
[1]共立薬科大学・基礎生物、[2]Department of Neurology, University of Chicago, IL, USA

OG2-13 Gタンパク質共役型受容体C06H5.7のアゴニストの同定
Identification of agonists for a putative G-protein-coupled receptor, C06H5.7
矢上 達郎[1]、青木 令奈[1]、片山 梨絵[1]、宮前 丈明[1]、中村 史雄[1]、梶原 康宏[2]、山本 直毅[2]、五嶋 良郎[1]
[1]横浜市大・医・薬理学、[2]横浜市大・理・機能科学

OG2-14 ナトリウムポンプα2サブユニットの周産期呼吸中枢神経系における役割
Na, K-ATPase α2 subunit is indispensable for functional maturation of respiratory neural network
池田 啓子[1]、鬼丸 洋[2]、川上 潔[1]
[1]自治医大・分子病態治療研究センター・細胞生物、[2]昭和大学・医・第二生理、[3]自治医大・分子病態治療研究センター・細胞生物

OG2-15 電気生理学的手法を用いたK^+-Cl^-共輸送体機能の新規評価法
A novel method to assess the function of a K^+-Cl^- cotransporter by using electrophysiological technique
井上 浩一[1]、山田 順子[2]、上野 伸哉[3]、福田 敦夫[1,2]
[1]浜松医大・医・一生理、[2]静岡大学・院・電子科研、[3]弘前大・医・脳研

OG2-16 マウスGLUT1 mRNAの脳内発現に対するGLUT1遺伝子欠損の影響
Effect of GLUT1 deficient in developmental expression of mouse GLUT1 mRNA
大槻 純男[1,2,3]、吉川 多鶴[1]、堀 里子[1,2,3]、寺崎 哲也[1,2,3]
[1]東北大院・薬・薬物送達、[2]東北大・未来研、[3]SORST・科技団

16:10～17:40 Room G

OG2-17～OG2-22 グリア、ミエリン、血液脳関門
Glia, Myelin and Blood-Brain Barrier
座長：古市 貞一（理化学研究所・脳科学総合研究センター）
寺崎 哲也（東北大院・薬・薬物送達）

OG2-17 BMP受容体シグナリングと脳血管機能
Astrocytic BMP Receptor IA signal maintains the cerebral blood vessel
山田 真久[1]、結城 宗浩[1]、岸田 治夫[1]、佐藤 友美[2]、古市 貞一[2]、三品 裕司[3]
[1]理研・脳科学・山田研究ユニット、[2]理研・脳科学・分子神経形成研究チーム、[3]NIH・NIEHS・分子発生生物学研究室

OG2-18 中枢ミエリンパラノードループ膜に特異的に発現する新しい膜貫通型糖タンパク
A novel, noncompact myelin-pecific, transmembrane glycoprotein in CNS
吉川 文生[1]、長倉 祐子[1]、遠山稿二郎[2]、端川 勉[3]、森田 規之[1]、佐藤 明[1]、古市 貞一[1]
[1]理化学研究所・脳センター・分子神経形成、[2]岩手医大・医・第二解剖、[3]理化学研究所・脳センター・神経構築技術開発

OG2-19 老化に伴うミエリン塩基性蛋白の発現
The expression of Myelin Basic Protein 21.5KDa isoform with aging
小見山貴継[1]、仲尾 保志[1]、戸山 芳昭[1]、山本 雅浩[2]、福武 昌人[2]、河喜多卓也[3]、清和 千佳[4]、阿相 皓晃[4]
[1]慶應義塾大学・整形外科学教室、[2]ツムラ漢方薬理研究所、[3]カネボウ漢方ヘルスケア研究所、[4]東京都老人総合研究所

OG2-20 脳内クレアチン合成酵素（guanidinoacetate N-methyltransferase）及びクレアチンキナーゼの免疫組織化学的解析
Immunohistochemical analysis of creatine biosynthetic enzyme, guanidinoacetate N-methyltransferase (GAMT), and creatine kinases (CKs) in the mouse brain
立川 正憲[1]、深谷 昌弘[2]、寺崎 哲也[1,3,4]、大槻 純男[1,3,4]、渡辺 雅彦[2]
[1]東北大院・薬・薬物送達、[2]北大院・医・解剖発生、[3]東北大・未来研、[4]SORST・科技団

OG2-21 脳、網膜、末梢組織におけるマウスABCG2の局在解析
Localization of ABCG2 in mouse brain, retina and peripheral tissues
淺島 朋子[1,2,3]、立川 正憲[1]、堀 里子[1,2,3]、大槻 純男[1,2,3]、渡辺 雅彦[4]、寺崎 哲也[1,2,3]
[1]東北大院・薬・薬物送達学、[2]東北大・未来研、[3]SORST・科学技術振興機構、[4]北大院・医

OG2-22 ラット脈絡叢におけるsterol輸送担体のLXRを介した発現制御
LXR-mediated regulation of sterol transporters in rat choroid plexus
藤吉 正哉[1]、堀 里子[1,2,3]、須田 太郎[1]、松田 明大[1]、大槻 純男[1,2,3]、寺崎 哲也[1,2,3]
[1]東北大院・薬・薬物送達、[2]東北大・未来研、[3]SORST・科技団

Sep 23 (Thu) Oral Session

9:00～10:30 Room G

OG3-01～OG3-06 シナプス、シナプス可塑性
Synapse, Synaptic Plasticity

座長：高橋 智幸（東京大院・医・神経生理）
重本 隆一（自然科学研究機構・生理学研究所）

OG3-01 タンパク質のリン酸化は、神経末端でのシナプス小胞の回収を起こす
Protein phosphorylation induces retrieval of synaptic vesicles at nerve terminals of Drosophila
黒見 坦[1]、本多 敦子[1]、城所 良明[2]
[1]群馬大・医行動生理、[2]群馬大学・生調研

OG3-02 細胞接着分子テレンセファリンの樹状突起選択的局在機構の解明
Polarized dendritic targeting of a cell adhesion molecule telencephalin
三津井五智子[1]、斉藤美知子[1]、林 研[1]、吉原 良浩[1]
[1]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

OG3-03 中枢神経系シナプス前終末におけるGTP結合タンパク質依存的小胞エンドサイトーシス
GTP binding protein-dependent vesicle endocytosis at a fast central synapse
山下 貴之[1]、髭 俊秀[1]、高橋 智幸[1]
[1]東京大院・医・神経生理

OG3-04 内因性カンナビノイドによる線条体抑制性シナプス伝達の逆行性調節
Endocannabinoid-mediated retrograde suppression of GABAergic inhibitory transmission in the striatum
鳴島 円[1]、狩野 方伸[1]
[1]金沢大院・医・シナプス発達・機能学

OG3-05 Differential control of synaptic versus ectopic release of synaptic vesicles
Ko Matsui[1]、Craig E. Jahr[1]
[1]Vollum Institute, Oregon Health and Science University, Oregon, USA

OG3-06 小脳運動学習による平行線維シナプスのAMPA受容体の減少
Reduction of AMPA receptor content in parallel fiber synapses by cerebellar motor learning
馬杉一時田美和子[1,2,3]、中館 和彦[1,4]、首藤 文洋[5]、王 文[1]、Elek Molnar[6]、永雄 総一[5]、重本 隆一[1,2]
[1]自然科学研究機構・生理学研究所・脳形態解析、[2]科学技術振興機構・戦略的創造研究推進事業、[3]日本学術振興会、[4]獨協医科大学・解剖学、[5]理研BSI・運動学習制御、[6]MRC, Univ. of Bristol, UK.

10:30～11:30 Room G

OG3-07～OG3-10 シナプス、シナプス可塑性
Synapse, Synaptic Plasticity

座長：小松由紀夫（名古屋大・環境医学研究所・視覚神経科学）
村瀬 一之（福井大・工・知能システム）

OG3-07 チロシンキナーゼ欠損型TrkBによるグリア細胞の形態制御
A truncated TrkB receptor, T1, regulates glial cell morphology via Rho GDI1
大平 耕司[1,2]、熊ノ郷晴子[1]、佐原 資謹[1]、本間 光一[3]、平井 啓久[2]、中村 俊[1]、林 基治[2]
[1]精神神経セ・神経研・診断、[2]京大・霊長研・分子生理、[3]東大・薬

OG3-08 LTDに伴う海馬錐体細胞スパインの縮小
Shrinkage of Dendritic Spines Associated with Induction of Long-Term Depression
本間 光一[1]、Qiang Zhou[2]、Mu-ming Poo[2]
[1]帝京大学・薬・病態生化学、[2]Department of Molecular and Cell Biology, University of California, Berkeley, USA

OG3-09 脱分極で誘発されるラット視覚野抑制性伝達の長期増強、長期抑圧は異なるサブタイプの電位依存性Caチャネルに依存する
Depolarization-induced LTD and LTP at inhibitory synapses depend on the activation of distinct voltage-gated Ca channel subtypes in rat visual cortex.
黒谷 亨[1]、小松由紀夫[1]
[1]名古屋大学・環境医学研究所・視覚神経科学

OG3-10 CaMKII-transgenic mouseにおける長期増強の光計測による時空間解析
Enhancement of optical signals recorded from CA1 of CaMKII-transgenic mouse during long term potentiation.
小島比呂志[1]、佐藤 正康[2]、長宗 伸朋[2]、塚田 稔[2]
[1]玉川大学・学術研究所、[2]玉川大学・工・学習・記憶システム工学科

14:40～16:55 Room G

OG3-11～OG3-19 シナプス可塑性
Synaptic Plasticity

座長：白尾 智明（群馬大院・医・高次細胞機能）
井ノ口 馨（三菱化学生命科学研究所）

OG3-11 PKGはVesl-1S-EGFP融合蛋白の樹状突起スパインへの取り込みを制御する
Protein kinase G regulates entry of Vesl-1S-EGFP fusion protein into dendritic spines
岡田 大助[1]、小沢 史子[1]、井ノ口 馨[1]
[1]三菱化学生命科学研究所・井ノ口グループ

OG3-12 樹状突起スパイン形成のAMPA受容体とNMDA受容体活性による二方向性制御
AMPA and NMDA receptor activities bidirectionally regulate postsynaptic actin organization in dendritic spine formation during development
高橋 秀人[1]、水井 利幸[1]、関野 祐子[1]、山崎 博幸[1]、白尾 智明[1]
[1]群馬大院・医・高次細胞機能

OG3-13 NMDA受容体NR2Bサブユニットのリン酸化残基（Tyr-1472）改変マウスの解析
NMDAR2B tyrosine-phosphorylation modulates synaptic plasticity and memory formation in amygdala
中澤 敬信[1]、駒井 章治[2]、城山 優治[2]、渡部 文子[2]、深谷 昌弘[3]、手塚 徹[1]、渡辺 雅彦[3]、真鍋 俊也[2]、山本 雅[1]
[1]東京大学医科学研究所・癌細胞シグナル研究分野、[2]東京大学医科学研究所・神経ネットワーク分野、[3]北海道大学大学院・医学系研究科・解剖発生学分野

OG3-14 シナプス可塑性発現時におけるRabaptin-5/Neurocresinのスパインにおける動態の可視化
Dynamics of rabaptin-5/ neurocrescin are involved in LTD expression in cultured hippocampal neurons.
清末 和之[1]、中山貴美子[1]、田口 隆久[1]
[1]産総研・セルエンジニアリング研究部門・ニューロニクス

OG3-15 発達期皮質脊髄路シナプスの再分布－膜電位感受性色素を用いた光学的測定
Developmental redistribution of corticospinal synapses: An optical recording study with fast voltage-sensitive dyes
前田 仁士[1]、大野 孝恵[1]、桜井 正樹[1]
[1]帝京大・医・生理

OG3-16 Frequency-Specific Spike Timing-Dependent Modifications of Excitatory Synapses on GABAergic Multipolar Interneurons
Jiangteng Lu[1]、Jiangping Zhao[1]、Chengyu Li[1]、Chienping Wu[1]、Shumin Duan[1]、Muming Poo[1,2]
[1]Institute of Neuroscience, Chinese Academy of Sciences, Shanghai, China、[2]Division of Neurobiology, Department of Molecular and Cell Biology, University of California, Berkeley, CA

OG3-17 ラット脊髄後角におけるサイレントなシナプス前終末の賦活による長期増強
Long-term potentiation by activation of silent presynaptic terminals in the rat spinal dorsal horn
池田 弘[1]、村瀬 一之[1]
[1]福井大・工・知能システム

OG3-18 凍結割断レプリカ標識法による海馬歯状回LTP誘導後のAMPA型グルタミン酸受容体の定量的解析
Quantitative analysis of AMPA receptors after LTP induction in the dentate gyrus *in vivo*
深澤 有吾[1]、斉藤 喜人[2]、井ノ口 馨[2]、Elek Molnar[3]、重本 隆一[1]
[1]自然科学研究機構・生理学研究所・脳形態解析部門、[2]三菱化学生命科学研究所、[3]Univ. of Bristol, Bristol, UK

OG3-19 late-LTPを誘発したラットCA1領域における遺伝子プロファイリング
Gene expression profile of late LTP-induced rat hippocampal CA1
谷口真理子[1]、吉岡 昇[2]、瀬々 潤[3]、水野 智之[2]、倉橋 浩樹[4]、桜井 正樹[2]、戸田 達史[1]
[1]大阪大院・医・ゲノム機能、[2]帝京大・医・生理、[3]東京大院・新領域創生、[4]藤田保健衛生大学・医・分子遺伝

9:00～10:15 Room H

OH1-01～OH1-05 情報伝達、伝達物質、受容体
Signal Transduction, Neurotransmitters and Receptors
座長：西川 徹（東京医科歯科大院・医歯学総合・精神行動医科学）
津田 正明（富山医薬大・薬・分子神経生物）

OH1-01 ラット大脳新皮質においてD-セリンによって発現が誘導される遺伝子 dsr-2 の研究
Cloning of a D-serine-regulated transcript *dsr-2* from the rat cortex
谷口 豪[1]、山本 直樹[1]、土田 英人[2]、海野 麻未[1]、嶋津 奈[1]、櫻井新一郎[1]、西川 徹[1]
[1]東京医科歯科大院・医歯学・精神行動医科学分野、[2]京都府立医大院・医・精神機能病態学

OH1-02 D-セリン制御分子 *dsm-1* の発現クローニング
Expression cloning of a D-serine modulator gene *dsm-1*
嶋津 奈[1]、山本 直樹[1]、海野 麻未[1]、櫻井新一郎[1]、西川 徹[1]
[1]東京医科歯科大院・医・精神行動医科学

OH1-03 大脳・海馬特異的RhoGEF, TECHによるコアクチベーターMALの核移行とSRF依存性転写活性化
SRF-mediated transcription through nuclear translocation of MAL coactivator by TECH, a neuronal RhoGEF
田渕 明子[1,2]、M Estevez[2]、J Henderson[2]、R Marx[2]、塩田 惇[1]、津田 正明[1]、J Baraban[2]
[1]富山医薬大・薬・分子神経生物、[2]Dept Neurosci, Johns Hopkins Univ, Sch Med, Baltimore, MD, USA

OH1-04 NGFによるGTP cyclohydrolase Iの誘導にはRas-MEK経路が必要である
Ras-MEK pathway is required for NGF-induced expression of the GTP cyclohydrolase I gene
伊藤 岳人[1]、鈴木 崇弘[1]、一瀬 宏[1]
[1]東工大院・生命理工

OH1-05 NMDA投与後にFyn欠損マウスの扁桃体で発現量が変化する遺伝子のcDNAアレイ解析
cDNA array analysis of gene expression profiles in the amygdaloid subdivisions of Fyn- deficient mice following NMDA administration
甲斐 信行[1,3]、八木 健[2]、湯浅 茂樹[1]
[1]国立精神・神経センター 微細構造研究部、[2]大阪大院・生命機能研究科・時空生物学講座 心生物学グループ、[3]福島医大・生体機能

10:15～11:30 Room H

OH1-06～OH1-10 情報伝達、伝達物質、受容体
Signal Transduction, Neurotransmitters and Receptors

座長：田口 隆久（産業技術総合研究所）
狩野 方伸（金沢大院・医・シナプス発達・機能学）

OH1-06 代謝型グルタミン酸レセプターは初代培養大脳皮質神経回路網における自発的カルシウム発火頻度を調節する
Glutamate regulates the frequency of spontaneous synchronized Ca^{2+} spikes through group II metabotropic glutamate receptor in cultured mouse cortical networks
安本 史恵[1,3]、根岸 隆之[1,3]、石井 寿幸[1]、久和 茂[1]、黒田洋一郎[2,3]、吉川 泰弘[1,3]
[1]東京大院・農学生命科学・実験動物、[2]東京都神経科学総合研究所、[3]戦略的基礎研究推進事業（CREST）

OH1-07 線条体における自発 Ca^{2+} 濃度上昇への大脳皮質運動野の影響
The spontaneous Ca^{2+} elevation in the striatum is affected by motor cortex
小山内 実[1]、山田 尚宏[1]、大星 文人[1]、八木 哲也[1]
[1]大阪大院・工・電子

OH1-08 海馬ニューロンにおけるホスホリパーゼC活性の脱分極による促進
Enhancement of receptor-mediated phospholipase C activation by the preceding depolarization in hippocampal neurons
橋本谷祐輝[1]、少作 隆子[1]、福留 優子[1]、狩野 方伸[1]
[1]金沢大院・医・シナプス発達・機能学

OH1-09 小脳プルキンエ細胞での平行線維入力誘発性イノシトール1,4,5-三リン酸産生における代謝型グルタミン酸受容体とAMPA受容体のクロストーク
Cross talk between metabotropic glutamate receptor and AMPA receptor in parallel fiber-induced inositol 1,4,5-trisphosphate production in cerebellar Purkinje cells
大久保洋平[1]、柿澤 昌[1]、廣瀬 謙造[1]、飯野 正光[1]
[1]東京大院・医・細胞分子薬理

OH1-10 シナプス増強による神経活動時空間パターンの調整
Modulation of spatiotemporal pattern in neuronal activities by synaptic enhancement.
工藤 卓[1,2]、田口 隆久[3]
[1]産業技術総合研究所・セルエンジニアリングRI・ニューロニクスRG、[2]科学技術振興機構戦略的創造研究推進事業PRESTO・相互作用と賢さ領域、[3]産業技術総合研究所・セルエンジニアリングRI

14:40～16:10 Room H

OH1-11～OH1-16 栄養因子、サイトカイン
Trophic Factors and Cytokines
座長：松岡 一郎（北海道大・創成科学研究機構・大学院・薬学研究科）
古川 昭栄（岐阜薬科大・薬・分子生物学）

OH1-11 NecdinとTrkAおよびp75神経栄養因子受容体との相互作用
Necdin interacts with TrkA and p75 neurotrophin receptors
桑子賢一郎[1]、吉川 和明[1]
[1]大阪大学・蛋白研・機能制御

OH1-12 エンドセリンによるexon特異的なアストロサイトのBDNF遺伝子転写促進
Endothelin Increases the Expressions of Exon III- and IV-Containing BDNF Transcripts in Cultured Astrocytes
小山 豊[1]、馬場 明道[2]、松田 敏夫[1]
[1]大阪大院・薬・複合薬物動態、[2]大阪大院・薬・神経薬理

OH1-13 ヒトBDNFのSNPsの分子機能解析
SNPs in human BDNF gene; Decreased trophic effects of BDNF SNPs variants to CNS neurons
小清水久嗣[1]、清末 和之[1]、鈴木 辰吾[1,2]、原 とも子[1]、Bai Lu[3]、小島 正己[1,2]
[1]産総研・セルエンジニアリング・セルダイナミクス、[2]SORST、[3]NICHD, NIH, MD, USA

OH1-14 脳由来神経栄養因子（BDNF）によりラフトへ移行するレセプター型チロシンキナーゼTrkB
Brain-derived neurotrophic factor induced recruitment of receptor tyrosine kinase TrkB into lipid rafts in primary neurons
鈴木 辰吾[1,2]、沼川 忠広[3]、小清水久嗣[1]、原 とも子[1]、小島 正己[1,2]
[1]AIST・セルエンジニアリング・セルダイナミクス、[2]JST・SORST、[3]国立精神・神経センター・神経研究所

OH1-15 大脳皮質神経前駆細胞の位置取りに対する脳由来神経栄養因子の影響
BDNF influences the decision of the laminar fate of cortical progenitors of mice
福光 秀文[1]、大塚 正成[1]、村井 里菜[1]、古川 昭栄[1]
[1]岐阜薬科大学・分子生物学教室

OH1-16 中枢神経における新規構造「フラクトン」を構築するIV型コラーゲン
Type IV collagen of the novel structure "fractone" in mouse central nervous system
米澤 朋子[1]、遊木 陽子[1]、赤木 雪恵[1]、内藤 一郎[2]、平野 修一[1]、大塚 愛二[2]、佐渡 義一[3]、二宮 義文[1]
[1]岡山大院・医歯学・分子医化学、[2]岡山大院・医歯学・人体構成学、[3]重井医学研

16:10～17:40 Room H

OH1-17～OH1-22 再生、移植

Regeneration, Transplantation

座長：井出 千束（京都大院・医・生体構造医学機能微細形態学）
篠原 一之（長崎大院・医歯薬・神経機能）

OH1-17 骨髄間質細胞からの神経細胞誘導法とパーキンソンモデルラットへの移植応用
Induction of neuronal cells from bone-marrow stromal cells and application for Parkinson's disease model rats
出澤 真理[1]、管野 洋[2]、鈴木 義久[3]、張 弘富[3]、井出 千束[1]
[1]京都大院・医・生体構造医学講座機能微細形態学、[2]横浜市大・医・脳神経外科、[3]京都大院・医・形成外科学

OH1-18 神経幹細胞の自己複製能に対する時計遺伝子の周期的調節機構
The periodic regulation of the neural stem cell proliferation by clock-related genes
守屋 孝洋[1]、堀江 信貴[1]、平石 敬三[1]、三留 雅人[2]、篠原 一之[1]
[1]長崎大院・医歯薬・神経機能、[2]北海道大院・歯・口腔機能

OH1-19 転写因子RX/raxの発現によるマウスES細胞の網膜亜集団への分化
Retinal fate specification of mouse embryonic stem cells by ectopic expression of Rx/rax, a homeobox gene
渡辺すみ子[1]、田畑 陽子[1]、大内 靖夫[1]、神谷 温之[2]、真鍋 俊也[3]、新井 賢一[4]
[1]東京大・医科研・染色体制御、[2]北海道大学院・院・分子解剖、[3]東京大・医科研・神経ネットワーク、[4]東京都臨床研

OH1-20 網膜前駆細胞の新規表面抗原、SSEA-1の同定
Identification of SSEA-1 as a novel surface antigen for retinal progenitor cells
高祖 秀登[1]、新井 賢一[1]、渡辺すみ子[1]
[1]東大・医科研・染色体制御

OH1-21 ラット線条体に移植した神経幹細胞、アストロサイト、PC12細胞のMR追跡
MR tracking of neural stem cells, astrocytes and PC 12 cell transplanted into the rat striatum
遠山 育夫[1]、高田 達之[2]、鳥居 隆三[2]、加藤 雅也[3]、近藤 靖[4]、犬伏 俊郎[1]
[1]滋賀医科大学・分子神経科学研究センター、[2]滋賀医科大学・動物生命科学研究センター、[3]石原産業（株）・中央研究所、[4]田辺製薬（株）・創薬研究所

OH1-22 CD34陽性細胞移植による脳梗塞の血管再生治療の基礎的検討
Administration of CD34+ cells post-stroke enhances angiogenesis and neurogenesis in a murine model
松山 知弘[1]、田口 明彦[2]、西村 裕之[1]、成冨 博章[2]、芳川 浩男[1]
[1]兵庫医大・医・内科、[2]国立循環器病センター・内・脳血管

9:00～10:15 Room H

OH2-01～OH2-05 情報伝達、神経伝達物質

Signal Transduction and Neurotransmitters

座長：福永 浩司（東北大院・薬・薬理学）
福田 光則（理化学研究所・福田独立主幹研究ユニット）

OH2-01 海馬神経細胞におけるCaM kinase kinase活性の検討
Characterization of the activity of CaM kinase kinase in the hippocampal neurons.
笠原 二郎[1]、福永 浩司[1]
[1]東北大院・薬・薬理

OH2-02 CAD 細胞の突起伸展における CaM kinase II の役割
Role of CaMKII on neurite outgrowth of CAD cells
有永 和代[1]、山内 卓[1]
[1]徳大院・薬・生化学

OH2-03 PC12細胞からの有芯顆粒開口放出におけるシナプトタグミンIXの役割
Synaptotagmin IX, a major Ca^{2+} sensor for dense-core vesicle exocytosis in PC12 cells
福田 光則[1]
[1]理研・福田独立主幹研究ユニット

OH2-04 キンギョMb1型双極細胞におけるCa^{2+}マイクロドメインと開口放出部位
Ca^{2+} microdomains and fusion sites of synaptic vesicles in goldfish retinal bipolar cells
緑川 光春[1]、浅香 知光[1]、立花 政夫[1]
[1]東京大・人社・心理学

OH2-05 GABA刺激による情報伝達はsrc protein tyrosine kinaseを介して、細胞のredox活性とリンクしている
GABA signaling couples to cellular redox activity by mediation of Src
丸山 悦子[1,2]、小川 健司[1]、遠藤 昌吾[3]、辻本 雅文[1]、次田 皓[2]
[1]理研・細胞生化学、[2]プロテオミクス研究所、[3]遠藤研究ユニット

10:15～11:30 Room H

OH2-06～OH2-10 軸索輸送、細胞運動、細胞骨格
Axonal Transport, Cell Motility and Cytoskeleton

座長：貝淵 弘三（名古屋大院・医・神経情報薬理）
高坂 新一（国立精神・神経センター神経研究所）

OH2-06 CRMP-2によるSra-1/WAVE1複合体の神経軸策成長円錐への輸送
CRMP-2 is involved in kinesin I-dependent transport of Sra-1/WAVE1 complex to growth cone of axon
河野 洋治[1]、吉村 武[1]、貝淵 弘三[1]
[1]名古屋大院・医・細胞情報薬理

OH2-07 膜蛋白として発現する微小管結合蛋白MAP1Bとシナプス形成
Expression of some microtubule-associated protein 1B as a transmembrane protein during synaptogenesis in vitro.
北村 主税[1,2]、白井 京子[1]、田代 朋子[1,2]
[1]青山学院大学・理工・化学・生命科学科、[2]CREST-JST

OH2-08 キネシンが運ぶRNA-タンパク複合体
Kinesin transports RNA: Isolation and characterization of the RNA-transporting complex
金井 克光[1]、堂前 直[2]、広川 信隆[1]
[1]東京大院・医・細胞生物、[2]理研

OH2-09 NUDELのリン酸化・脱リン酸化の制御機構の解析
The analysis of regulation mechanism of NUDEL phosphorylation/dephosphorylation.
豊岡 和人[1]、Anthony Wynshaw-Boris[2]、広常 真治[1]
[1]大阪市大院・医・細胞機能制御学、[2]School of Medicine, UCSD

OH2-10 ニューロフィラメントゲル形成に対する脱リン酸化の影響ーリアルタイム観察と生物物理的解析
Effect of dephosphorylation on neurofilament gel formation - real-time observation and biophysical analysis
佐野 浩樹[1,2]、小野 れい[1]、中林 和也[1]、倉知 正[3]、田代 朋子[1,2]
[1]青山学院大・理工・化学生命科学、[2]科学技術振興事業団、[3]群馬大・医・分子病態

14:40～16:10 Room H

OH2-11～OH2-16 視覚
Visual System

座長：細谷 俊彦（理化学研究所・脳科学総合研究センター）
政井 一郎（理化学研究所・政井独立主幹研究ユニット）

OH2-11 哺乳動物網膜内における受容体型タンパク質チロシンホスファターゼζ/βの発現と視覚機能への関与
Expression and function of receptor-type protein tyrosine phosphataseζ/β in mammalian retina
高雄 元晴[1]、野田 昌晴[1,2]
[1]自然科学研究機構・基生研・感覚情報、[2]CREST

OH2-12 ゼブラフィッシュ眼球におけるオプシンキナーゼの発現量の比較
Expression levels of opsin kinases in the zebrafish eye
和田 恭高[1]、杉山 純一[1]、岡野 俊行[1,2]、松川 淑恵[3]、河村 悟[3]、深田 吉孝[1]
[1]東大・院理・生化、[2]JST・さきがけ、[3]阪大・院生命機能

OH2-13 視細胞外節における円盤膜形成に異常を示すゼブラフィッシュ突然変異体 *twilight*
twilight, a zebrafish mutant showing defects in the outer segment formation of photoreceptors
西脇 優子[1]、小森 敦子[1]、相良 洋[2]、鈴木えみ子[2]、岡本 仁[3,4]、政井 一郎[1]
[1]理研・政井独立主幹研究ユニット、[2]東大・医科研・分子構造解析、[3]理研・脳センター、[4]科技団・戦略

OH2-14 網膜AIIアマクリン細胞の活動電位の特性と電位依存性Na^+チャネルの局在
Properties of spikes and localization of voltage-gated Na^+ channels in retinal AII amacrine cells
田丸 文信[1]、中平 健祐[1]、渡辺 修一[1]
[1]埼玉医大・生理

OH2-15 カエル網膜における周期的発火生成機構
Neural mechanisms of light-evoked oscillations in the frog retina
山田 義之[1]、荒井 格[1]、立花 政夫[1]
[1]東京大学・人社・心理

OH2-16 視覚入力の相関に対する網膜の適応
Retinal Adaptation to Space-Time Correlations
細谷 俊彦[1,2]、Markus Meister[2]
[1]理研・脳センター・臨界期・細谷ユニット、[2]Harvard U., Cambridge, USA

Sep 22 (Wed) /23 (Thu) Oral Session

16:10～17:40 Room H

OH2-17～OH2-22 視覚
Visual System

座長：三上 章允（京都大・霊長類研究所・行動発現分野）
柿木 隆介（自然科学研究機構・生理学研究所）

OH2-17 ヒト初期視覚野におけるコントラスト順応：事象関連fMRI研究
Contrast adaptation in human early visual cortex: an event related fMRI study
程 康[1]、Justin L. Gardner[1]、孫 沛[1]、R. Allen Waggoner[1]、上野 賢一[1]、
田中 啓治[1]
[1]理化学研究所・脳科学研究所

OH2-18 ヒト視覚腹側路におけるプライミング効果の時間的動態
Temporal dynamics of neural adaptation effect in the human visual ventral stream
野口 泰基[1]、乾 幸二[1]、柿木 隆介[1]
[1]生理学研究所 統合生理研究系

OH2-19 ヒト一次視覚野における時間周波数優位性領域：高解像度fMRI研究
Temporal frequency dominance domains in human primary visual cortex as revealed by high resolution fMRI study
孫 沛[1]、上野 賢一[1]、R. Allen Waggoner[1]、田中 啓治[1]、程 康[1]
[1]理化学研究所・脳科学総合研究センター

OH2-20 干渉刺激によって相違が生じる２つのタイプの遅延期間中の神経活動
Differential Effect by Distractors on Delay-Period Activity in Monkey Inferotemporal Cortex during Pair Association Task
竹田 真己[1]、納家 勇治[1]、宮下 保司[1]
[1]東京大院・医・統合生理

OH2-21 マカクザルの臭周囲皮質および下側頭葉皮質における報酬関連活動
Effect of reward contingency on discharges of cells in macaque inferior temporal areas
最上 嗣生[1]、田中 啓治[1]
[1]理化学研究所・BSI・認知機能表現

OH2-22 他者の運動観測に関わる事象関連電位
An event-related potential associated with human action observation.
福島 宏器[1]、開 一夫[1]
[1]東京大院・総合文化・広域システム科学

9:00～10:45 Room H

OH3-01～OH3-07 イオンチャネル、興奮性膜
Ion Channels and Excitable Membranes

座長：小澤 瀞司（群馬大院・医・神経生理）
岡 良隆（東京大院・理・生物科学）

OH3-01 ヤムシ横紋筋リアノジン受容体は細胞内カルシウム放出チャネルとして機能していない
The ryanodine receptor in striated muscle of chaetognaths does not function as an intracellular Ca^{2+}-release channel
井上 勲[1]、Quentin Bone[2]、筒井 泉雄[3]
[1]徳島大・酵素研、[2]MBA Laboratory, Plymouth, UK、[3]一橋大

OH3-02 脳血管内皮不死化細胞（t-BBEC 117）におけるプリン受容体活性化による過分極応答の新規メカニズム
Novel mechanism underlying marked hyperpolarization by activation of purinoceptor in t-BBEC 117
山崎 大樹[1]、青山 峰芳[2]、大矢 進[1]、村木 克彦[1]、浅井 清文[2]、今泉 祐治[1]
[1]名古屋市大院・薬・細胞分子薬効解析、[2]名古屋市大院・医・分子神経生物

OH3-03 脊髄後角深層細胞におけるATP P2X受容体の機能意義について
Functional role of ATP P2X receptors in deep dorsal horn neurons of the spinal cord
中塚 映政[1]、塩川 浩輝[2]、藤田 亜美[1]、古江 秀昌[2]、吉村 恵[2]、熊本 栄一[1]
[1]佐大・医・生体構造機能学、[2]九州大学院・医学研究院・統合生理学

OH3-04 ヒト上皮型Na^+チャネルδサブユニットのpH感受性
Protons activate the δ-subunit of the epithelial Na^+ channel in humans
山村 寿男[1]、鵜川 眞也[1]、植田 高史[1]、長尾 正崇[2]、島田 昌一[1]
[1]名古屋市大院・医・分子形態、[2]名古屋市大院・医・法医科学

OH3-05 終神経GnRHニューロンにおける電位依存性Ca^{2+}チャネルの発現と修飾
Voltage-gated Ca^{2+} channels of Terminal Nerve (TN) Gonadotropin-Releasing Hormone (GnRH) Neurons and its modulation by GnRH.
羽田 幸祐[1]、岡 良隆[1]
[1]東京大院・理・生物科学

OH3-06 GFAPプロモーターによってGluR2を発現するトランスジェニックマウスの作製
Transgenic mice that express GluR2 by glial cells specific promoter
都筑 馨介[1]、石内 勝吾[2]、小澤 瀞司[1]
[1]群馬大院・医・神経生理、[2]群馬大院・医・脳脊髄病態外科

OH3-07 Spatiotemporal specificity in modification of dendritic integration by correlated activity
Ning-Long Xu[1]、Chang-Quan Ye[1]、Xian Zhang[1]、Chien-Ping Wu[1]、Shumin Duan[1]、Mu-Ming Poo[1,2]
[1]Institute of Neuroscience, CAS, Shanghai, China、[2]Division of Neurobiology, Department of Molecular and Cell Biology, University of California, Berkeley

14:40～15:55 Room H

OH3-08～OH3-12 シナプス
Synapse

座長：立花 政夫（東京大院・人文社会・心理学）
真下 節（大阪大院・医・機能調節医学）

OH3-08 標的細胞の大きさに応じて適切な大きさのシナプスを形成する仕組み
Experience-driven regulation of synaptic size in *Drosophila* Neuromuscular Junctions
森本一谷藤高子[1]、中山 浩明[1]、風間 北斗[1]、能瀬 聡直[1]
[1]東京大院・理・物理学

OH3-09 ラット小脳プルキンエ細胞平行線維シナプスにおける構成性エンドサイトーシス阻害によるAMPA受容体表面発現の増強
Up-regulation of AMPA-receptor surface-expression by blocking of constitutive endocytosis in parallel fiber synapse of rat cerebellar Purkinje cell.
山口 和彦[1]、立川 哲也[1,2]、小野寺加代子[1,2]
[1]理研・脳センター・記憶学習、[2]CREST/JST

OH3-10 マウス網膜外網状層におけるグルタミン酸取り込みによる信号調節
Signal modulation by glutamate uptake in the mouse outer retina
長谷川 淳[1]、立花 政夫[1]
[1]東京大・人社・心理学

OH3-11 神経伝達物質放出における、プレシナプスactive zone蛋白質CAST-RIM-Bassoon複合体の生理的意義の解析
Physical and Functional Interaction of the Active Zone Proteins, CAST, RIM1, and Bassoon, in Neurotransmitter Release
大塚 稔久[1]、力津絵津子[1]、井上 英二[1]、俵田 真紀[1]、井上真理枝[1]、持田 澄子[2]、高井 義美[3]
[1]カン研究所、[2]東京医科大学・生理学教室、[3]大阪大院・医・分子生理化学教室

OH3-12 脊髄後角第2層における持続性GABA, A受容体介在性抑制性電流
The tonic inhibition mediated by γ Aminobutyric AcidA receptor in substantia gelatinosa neurons
高橋亜矢子[1]、内田 一郎[1]、真下 節[1]、山中 博樹[2]、徳永 敦[2]、野口 光一[2]
[1]大阪大院・医・生体機能調節講座（麻酔科）、[2]兵庫医科大学・医・第二解剖講座

15:55～17:25 Room H

OH3-13～OH3-18 パターン形成
Pattern Formation

座長：前田 信明（東京都神経研・分子発生）
田辺 康人（三菱化学生命科学研究所）

OH3-13 マウス脳におけるヘパラン硫酸硫酸転位酵素群の発現時期および発現部位の解析
Developmental and regional expression of heparan sulfate sulfotransferases in the mouse brain
矢部 富雄[1]、秦 敏宏[1]、何 王玉[1]、前田 信明[1]
[1]（財）東京都神経研・分子発生

OH3-14 大脳皮質構築を調節するCajal-Retzius細胞の発生・発達機構の解析
Generation of Cajal-Retzius Cells from the Caudal-Medial Wall of Telencephalic Vesicles
田辺 康人[1]
[1]三菱化学生命研・神経構築

OH3-15 大脳皮質投射ニューロンの放射状移動に必須なFGF18-ETS転写因子群の新規フィードバックシグナル
A novel feedback signal through FGF18-ETS transcription factors is critical for the radial migration of cerebral cortical neurons
長谷川 潤[1]、芦垣しづ子[1]、高松 昌子[1]、大林 典彦[2]、高田 慎治[2]、田辺 康人[1]
[1]三菱化学生命科学研究所、[2]岡崎国立共同研究機構・統合バイオサイエンスセンター

OH3-16 大脳皮質層形成における神経細胞の分離機構解明に向けた*in silico*実験の試み
Attempt of computational approach toward understanding the neural cell segregation during the cortical layer formation
前田 卓哉[1]、味岡 逸樹[1]、仲嶋 一範[1,2]
[1]慶應大・医・解剖、[2]慈恵医大・DNA研

OH3-17 小脳の縦縞状区画化は小脳プルキンエ細胞の誕生する日によって規定される
Mediolateral compartmentalization of the cerebellum is determined on the "birth date" of Purkinje cells
橋本 光広[1]
[1]理研 脳総研 発生神経生物

OH3-18 脊椎動物における形態形成と神経回路形成のKaedeとケージ蛍光色素をもちいた研究
Large-scale morphogenesis and neural network formation in a vertebrate, analyzed by using a photo-convertible coral fluorescent protein
八田 公平[1]、辻井 仁美[1]
[1]理研・CDB

9:00～10:15 Room J

OJ1-01～OJ1-05 **認知、コンピュータ科学**
Cognition and Computer Science

座長：宮下 保司（東京大院・医・統合生理）
尾上 浩隆（東京都医学研究機構・東京都神経科学総合研究所）

OJ1-01 空間的及び非空間的な注意のシフトと関連した後頭頂皮質における脳活動の解離
Dissociable activity in posterior parietal cortex associated with spatial and nonspatial attentional shifts
浅利 知輝[1]、小西 清貴[1]、地村 弘二[1]、宮下 保司[1]
[1]東京大院・医・統合生理

OJ1-02 カウンティングにおける運動前野の役割
Role of the premotor cortex in counting of large numbers
神作 憲司[1,2]、Benjamin Carver[2]、Ari Johnson[2]、Mark Hallett[2]
[1]自然科学研究機構・生理学研究所・心理生理学研究部門、[2]NINDS, Bethesda, USA

OJ1-03 前頭前野ニューロンにおける行動境界表現
Representation of action boundaries in the prefrontal cortex of the macaque monkey
藤井 直敬[1,2]、Ann Graybiel[2]
[1]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、[2]MIBR and BCS, MIT, Boston MA, USA

OJ1-04 多次元視覚探索課題遂行中のV4野におけるボトムアップとトップダウン信号の相互作用
Interaction between bottom-up and top-down signals in area V4 during a multi-dimensional visual search task
小川 正[1]、小松 英彦[1]
[1]岡崎国立共同研究機構・生理学研究所・感覚認知情報

OJ1-05 記憶負荷に伴う前頭前野一下側頭葉の活性化;学習の構えを習得したサルを用いたPET実験
Prefrontal-inferior temporal activation in the effort for the memory load; PET study of macaque monkeys with learning-set.
尾上 浩隆[1]、横山ちひろ[2]、山本 茂幸[3]、尾上 嘉代[3]、塚田 秀夫[4]、渡辺 恭良[3]
[1]（財）東京都医学研究機構・東京都神経科学総合研究所・心理学部門、[2]京都府立医科大学・精神医学教室、[3]大阪市大院・医・システム神経科学、[4]浜松ホトニクス（株）・中央研究所

Sep 21 (Tue) Oral Session

10:15～11:30 Room J

OJ1-06～OJ1-10 認知、コンピュータ科学 Cognition and Computer Science

座長：八木 哲也（大阪大院・工・電子工学）
外池 光雄（産業技術総合研究所）

OJ1-06 内側前頭前野のドパミン放出が視覚性探索課題遂行に関わる
Medial prefrontal dopamine release during a visual search task
澤本 伸克[1,2]、Gary Hotton[2]、Nicola Pavese[2]、Paola Piccini[2]、David J Brooks[2]
[1]京大・医・脳センター、[2]Hammersmith Hospital, London, UK

OJ1-07 母語及び第二言語の獲得過程において特異的に変動するブローカ野の課題選択的な活動
Task-selective activation in Broca's area is differentially modulated during first and second language acquisition
辰野 嘉則[1,2]、酒井 邦嘉[1,2]
[1]東京大院・総合文化、[2]CREST・JST

OJ1-08 単語親密度の視覚誘発脳磁図反応に対する影響の検討
The Effect of Word Familiarity on Visually Evoked Magnetic Fields
原田 暢善[1]、岩木 直[1]、中川 誠司[1]、山口 雅彦[1]、外池 光雄[1]
[1]産業技術総合研究所・関西センター・人間福祉医工学研究部門

OJ1-09 ラット海馬体ニューロンの順列および文脈への相関性
Neural correlates to sequence and context in the rat hippocampal formation
田渕 英一[1,2]、古沢 明美[1,2]、堀 悦郎[1,2]、梅野 克身[1,2]、小野 武年[3]、西条 寿夫[1,2]
[1]富山医薬大・医・システム情動科学、[2]創造科学推進事業、[3]富山医薬大・医・分子統合情動神経科学

OJ1-10 一次視覚野単純型細胞の方位選択性を模擬するVLSIシステム
An orientation selective VLSI system emulating the simple cell response of the primary visual cortex
下ノ村和弘[1]、谷口 直哉[2]、八木 哲也[1,2]
[1]科学技術振興機構、[2]大阪大院・工・電子工学

14:40～16:10 Room J

OJ1-11～OJ1-16 視覚 Visual System

座長：藤田 一郎（大阪大院・生命機能・認知脳科学）
小松 英彦（生理学研究所・感覚認知情報研究部門）

OJ1-11 両眼非対応要素を用いた単眼性奥行き知覚に関する研究
Monocular features define stereoscopic depth of binocularly unpaired component
牧野 悌也[1]、矢野 雅文[1]
[1]東北大学・電気通信研究所・実世界コンピューティング

OJ1-12 奥行き弁別における大脳皮質MT野の課題依存性
Task specific effects of microstimulation in area MT during depth discrimination
宇賀 貴紀[1,2]、Gregory C. DeAngelis[2]
[1]順天堂大学・医・生理学第一、[2]Washington University School of Medicine

OJ1-13 サル視覚皮質V4野における相対視差選択性
Selectivity for relative disparity in V4
梅田 和昌[1]、田辺 誠司[2]、藤田 一郎[2]
[1]大阪大院・基礎工・認知脳科学、[2]大阪大院・生命機能・認知脳科学

OJ1-14 サルV4野における、ランダムドットステレオグラムとソリッド図形ステレオグラムに対する両眼視差チューニングの相違
Disparity tuning of area V4 neurons differs when tested with dynamic random-dot stereograms or solid-figure stereograms
土井 隆弘[1]、田辺 誠司[1]、梅田 和昌[2]、藤田 一郎[1]
[1]大阪大院・生命機能・認知脳科学、[2]大阪大院・基礎工・認知脳科学

OJ1-15 色カテゴリー課題と色弁別課題中のTE野細胞の色選択性
Area TE neurons have invariant color selectivity for both categorization and discrimination tasks
鯉田 孝和[1]、小松 英彦[1,2]
[1]生理学研究所・感覚認知情報研究部門、[2]総合研究大学院大学

OJ1-16 周辺運動錯視と固視微動
A peripheral motion illusion and fixational eye movements
村上 郁也[1]、北岡 明佳[2]、蘆田 宏[3,4]
[1]NTTコミュニケーション科学基礎研究所・人間情報研究部、[2]立命館大学文学部心理学科、
[3]京都大院・文学研究科、[4]Department of Psychology, Royal Holloway, University of London, Egham, UK

16:10～17:25 Room J
OJ1-17～OJ1-21 **情報伝達、受容体、嗅覚、痛覚**
Signal Transduction, Receptors, Olfaction and Pain
座長：齋藤 尚亮（神戸大学・バイオシグナル研究センター）
大瀧 丈二（神奈川大・理・生物科学）

OJ1-17 VGLUT3/PPTB産生皮質インターニューロンによる化学特異的神経回路
Chemically Specific Circuit Composed of VGLUT3- and PPTB-Producing Interneurons in Rat Neocortex
日置 寛之[1]、藤山 文乃[1]、中村 和弘[1]、松田 和郎[1]、金子 武嗣[1]
[1]京都大院・医・高次脳形態

OJ1-18 DGKサブタイプα、β、γの生後発達における変化
Differential developmental expression of three DGK subtypes, α, β and γ.
足立 直子[1]、大保 三穂[1]、谷口 泰造[1]、餅田 泰人[1]、竹中 利佳[1]、白井 康仁[1]、
齋藤 尚亮[1]
[1]神戸大学・バイオシグナル研究センター

OJ1-19 エンドセリンによる神経前駆細胞のDNA合成促進：プロテインキナーゼCとインテグリンの関与
Endothelin-stimulated DNA synthesis in neural progenitor cells: Involvement of protein kinase C and integrin
浅野 富子[1]、森下 理香[1]、篠原 春夫[2]、上田 浩[3]、加藤 兼房[1]
[1]愛知県コロニー・研・神経制御、[2]東海学園大・人間健康学、[3]岐阜大・工・生命工学

OJ1-20 嗅神経細胞におけるBcl-6の発現
Bcl-6 expression in olfactory sensory neurons
大瀧 丈二[1]
[1]神奈川大・理・生物科学

OJ1-21 ラット脊髄後角内痛覚神経回路網に対する細胞外ATPによる修飾作用
Effects of extracellular ATP on the neuronal network function of the rat spinal cord dorsal horn
青山 龍馬[1]、岡田 泰昌[2]、福田健太郎[1]、吉田 英彰[1]、中村 雅也[1]、千葉 一裕[1]、
戸山 芳昭[1]
[1]慶應義塾大学・医・整形外科学教室、[2]慶應義塾大学・月が瀬リハビリテーションセンター内科

9:00～10:15 Room J

OJ2-01～OJ2-05 イメージング、分子生物学的、細胞生物学的方法
Imaging, Molecular and Cellular Biological Techniques

座長：壇 一平太（食品総合研究所）
小田 一郎（株式会社島津製作所 基盤技術研究所）

OJ2-01 二重干渉問題と脳活動に関するヒトPET研究
Neural mechanism of dual task: A human PET study
望月 秀紀[1,2]、田代 学[2]、岡村 信行[2]、伊藤 正敏[3]、谷内 一彦[2]
[1]国立精神神経センター・神経研・モデル動物開発部、[2]東北大学院・医・薬理、[3]東北大・サイクロトロンRIセンター

OJ2-02 開口放出における分泌顆粒のdocking/fusion部位 ―全反射顕微鏡（TIRF）による解析―
Different fusion sites between 1st- and 2nd-phase insulin release from rat pancreatic β cells analyzed by TIRF
今泉 美佳[1]、西脇知世乃[1]、菊田 敏輝[1]、長井進太郎[1]、中道 洋子[1]、永松 信哉[1]
[1]杏林大・医・生化学

OJ2-03 NIRSとfMRIによるリンゴの皮むきに伴う脳活性解析
Cortical activation during apple peeling as studied by NIRS and fMRI
岡本 雅子[1]、檀 はるか[1]、清水 公治[2]、武尾 和浩[2]、網田 孝司[2]、小田 一郎[2]、
小西 郁夫[2]、五十部誠一郎[1]、鈴木 建夫[1]、神山かおる[1]、檀 一平太[1]
[1]食品総合研究所・製造工学研、[2]島津製作所

OJ2-04 10-20法を介した確率的頭表－脳表3次元対応化
Probabilistic anatomical cranio-cerebral correlation via the international 10-20 system
檀 一平太[1]、岡本 雅子[1]、檀 はるか[1]、坂本 晋子[1]、武尾 和浩[2]、河野 悟[2]、
小田 一郎[2]、五十部誠一郎[1]、鈴木 建夫[1]、神山かおる[1]、清水 公治[2]
[1]食品総合研究所・製造工学研、[2]島津製作所

OJ2-05 脳虚血モデルにおける移植神経幹細胞の *in vivo* bioluminescence imaging
Imaging of Stem Cell Recruitment to Ischemic Infarcts in a Murine Model
石井 賢[1,3]、Dong-Eog Kim[3]、Dawid Schellingerhout[3]、Ralph Weissleder[3]、
中村 雅也[1]、岡野 栄之[2]、戸山 芳昭[1]
[1]慶應大学・整形外科、[2]慶應大学・生理、[3]CMIR, MGH/Harvard Medical School, Boston, USA

10:15～11:30 Room J

OJ2-06～OJ2-10 イメージング、分子生物学的、細胞生物学的方法 Imaging, Molecular and Cellular Biological Techniques

座長：木村 文隆（大阪大院・医・神経生理）
金村 米博（産業技術総合研究所）

OJ2-06 Imaging regenerative peripheral axons in live mice by fibered confocal fluorescence microscopy

Benjamin Abrat[1]、Igor Charvet[2]
[1]Mauna Kea Technologies、[2]Faculte de Medecine, UNIGE, Departement de Morphologie

OJ2-07 局所エレクトロポーレーションによる脳限定領域へのsiRNAの導入 RNAi-induced gene silencing by local electroporation (RISLE): Method to knockdown a target protein at the defined brain region in vivo

茜谷 行雄[1]、Bin Jiang[1]、津本 忠治[1]
[1]大阪大院・医・神経生理

OJ2-08 白金黒電極を用いた電気化学ポンプによる神経伝達物質の高速微小投与 Rapid microadministration of neurotransmitter toward neuron by electrochemical pump using blakened platinum electrode

吉見 靖男[1]、篠田 賢一[1]、中川 大輔[1]、後藤 孝博[1]
[1]芝浦工大・工・化学工学

OJ2-09 SELDI-TOFMSを用いた薬剤安全性評価 Safety Assessment of Neuronal Drugs using Protein Expression Profiles by SELDI-TOFMS

山崎 智彦[1,2]、小林 哲[1]、森 英樹[1]、三宅 淳[1]、金村 米博[1]
[1]産総研・セルエンジニアリング研究部門、[2]新エネルギー・産業技術総合開発機構・産業技術フェロー

OJ2-10 マウス小脳発達のトランスクリプトーム・データベース Cerebellar development transcriptome database

古市 貞一[1]、佐藤 明[1]、定方 哲史[1]、松木 亨[1]、吉川 文生[1]、森田 規之[1]、佐藤 友美[1]、白石一山口陽子[1]、菅 恵美子[1]、関根友紀子[1]、北村 愛子[1]、柴田 泰行[1]
[1]理研・脳センター・分子神経形成

14:40～16:10 Room J

OJ2-11～OJ2-16 聴覚、嗅覚、味覚、化学感覚／Auditory System, Olfaction, Taste and Chemical Senses

座長：澁木 克栄（新潟大・脳研・システム脳生理）
島田 昌一（名古屋市立大院・医・分子形態）

OJ2-11 モルモット一次聴覚野における神経活動の伝播 Propagation of Electrically Evoked Activity in Guinea Pig Primary Auditory Cortex

宋 文杰[1]、川口 英夫[2]、十時慎一郎[1]、井上 雄二[1]、桂 卓成[2]、前田 新一[1]、稲垣 眞司[1]
[1]大阪大院・工・電子、[2]（株）日立製作所 基礎研究所

OJ2-12 線虫*C. elegans*の化学走性の可塑性におけるインシュリン様シグナル伝達経路の機能 The insulin-like signaling pathway regulates the plasticity of salt chemotaxis in *C. elegans*

富岡 征大[1]、國友 博文[1]、飯野 雄一[1]
[1]東京大学・遺伝子実験施設

OJ2-13 線虫の嗅覚可塑性のメカニズム：Ras-MAPK経路によるグルタミン酸受容体の局在の制御
The Ras-MAPK pathway controls neural circuit-dependent odor adaptation by regulating localization of glutamate receptors in *C. elegans*
広津 崇亮[1,2]、飯野 雄一[2]
[1]京大・院・生命科学、[2]東大・遺伝子実験施設

OJ2-14 塩分摂取行動の制御は脳弓下器官のNaxチャンネルが担う
Nax channel in the subfornical organ is essential for the control of salt-intake behavior
檜山 武史[1,5]、渡辺 英治[2,4]、岡戸 晴生[3]、野田 昌晴[1,5]
[1]基礎生物学研究所・感覚情報処理研究部門、[2]基礎生物学研究所・形質転換生物研究施設、[3]東京都神経科学総合研究所・分子神経生理研究部門、[4]PRESTO、[5]CREST・JST

OJ2-15 βカリオフィレンが月経周期に伴う抑うつ気分に及ぼす影響
Effects of β -caryophyllene on the depressive mood during the Menstrual Cycle
諸伏 雅代[1]、西谷 正太[1]、篠原 一之[1]
[1]長崎大院・医歯薬・医療科学・神経機能学

OJ2-16 Gustatory responses in the hippocampal CA1 area in freely behaving rats
Son A Ho[1,2]、E Tabuchi[1,2]、K Umeno[1,2]、E Hori[1,2]、T Ono[1]、H Nishijo[1,2]
[1]System Emotional Neuroscience, Toyama Medical and Pharmaceutical University、[2]CREST, JST, Tokyo, Japan.

16:10～17:40 Room J

OJ2-17～OJ2-22 痛覚 Pain

座長：野口 光一（兵庫医科大・第二解剖学）
杉本 幸彦（京都大院・薬・生体情報制御）

OJ2-17 脊髄損傷後痛覚過敏に対するアデノシンの効果
Effect of adenosine on hyperalgesia after spinal cord injury
森野 忠夫[1]、尾形 直則[1]、堀内 秀樹[1]、濱本雄一郎[1]、山本 晴康[1]
[1]愛媛大学・整形外科

OJ2-18 脊髄痛覚伝達におけるニューロメジンUの解析
The effect of Neuromedin U on excitatory synaptic transmission in the rat spinal cord
森山麻衣子[1,2,3]、古江 秀昌[1]、寺西 仁志[2]、加納 龍彦[3]、児島 将康[2]、吉村 恵[1]
[1]九州大院・医学研究院・統合生理学分野、[2]久留米大学 分子生命科学研究所 遺伝情報研究部門、[3]久留米大学・医・麻酔学教室

OJ2-19 ラット脊髄後角膠様質におけるドーパミンの痛覚抑制作用
Anti-nociceptive action of dopamine in substantia gelatinosa neurons of the rat spinal cord.
玉江 昭裕[1]、中塚 映政[2]、古賀 浩平[1]、加藤 剛[1]、古江 秀昌[1]、吉村 恵[1]
[1]九州大院・医学研究院・統合生理学、[2]佐賀大学・医・生体構造機能学講座・神経生理学分野

OJ2-20 実験的胃潰瘍に見られる痛覚過敏における神経成長因子の役割
Roles of nerve growth factor in gastric hyperalgesia induced by experimental ulcer in the rat
尾崎 紀之[1]、伊藤千香子[1]、篠田 雅路[1]、杉浦 康夫[1]
[1]名古屋大院・医・機能形態

OJ2-21 求心路遮断痛患者における大脳運動野刺激による反対側視床の活性化
Activation of contralateral thalamus in deafferentation pain (DP) patients after successful motor cortex stimulation (MCS)
齋藤 洋一[1]、貴島 晴彦[1]、大崎 康宏[2]、西村 洋[2]、加藤 天美[1]、橋川 一雄[3]、畑澤 順[2]、吉峰 俊樹[1]
[1]大阪大院・神経機能制御外科学、[2]大阪大院・生体情報医学講座（トレーサ情報解析学）、[3]京都大学学医学研究科附属高次脳機能総合研究センター

OJ2-22 H_1およびH_2遺伝子欠損マウスにおけるニューロパチックペインの発現機構
Mechanisms of neuropathic pain in histamine H_1 and H_2 receptors gene knockout mice
谷内 一彦[1]、J. I. Mobarakeh[1,2,3]、加藤 元久[1]、櫻田 忍[4]、H. Akbari[5]、M. H. Heidari[3]
[1]東北大院・医・細胞薬理、[2]Department of Physiology and Pharmacology, Institute Pasteur of Iran, Iran、[3]Department of Anatomy and Cell Molecular Biology, Research Center of Shaheed Beheshti, Iran、[4]東北薬大・機能形態、[5]Department of Pharmaceutices, Faculty of Pharmacy, Tehran University of Medical Science, Iran

14:40～16:10 Room J

OJ3-01～OJ3-06 細胞分化
Cellular Differentiation

座長：田賀 哲也（熊本大・発生医学研究センター・転写制御分野）
堀尾 嘉幸（札幌医大・医・薬理）

OJ3-01 ヒストン脱アセチル化酵素Sir2αの神経幹細胞増殖・分化における関与
Effects of histone deacetylase Sir2α on proliferation and differentiation of neural stem cells.
久原 真[1,2]、松本 博之[2]、堀尾 嘉幸[1]
[1]札幌医大・医・薬理、[2]札幌医大・医・神経内科

OJ3-02 ヒストン脱アセチル化阻害剤による神経幹細胞のニューロン分化誘導
HDAC inhibitor-mediated neuronal differentiation of adult neural progenitors
中島 欽一[1]、Jenny Hsieh[2]、桑原 知子[2]、田賀 哲也[1]、Fred Gage[2]
[1]熊大・発医研・転写制御、[2]The Salk Institute

OJ3-03 胎生期脳におけるAINTの役割
Role of AINT in the developing brain
落合 和[1,2]、滝沢 琢己[3]、中島 欽一[2]、田賀 哲也[2]
[1]熊本大学・医・細胞情報薬理、[2]熊本大学・発生研・転写制御、[3]群馬大学・医・小児科

OJ3-04 哺乳類の神経発生におけるRNA結合蛋白質Musashiの機能解析
Roles of Musashi Family in mammalian nervous development
芝田 晋介[1,3]、榊原 伸一[2]、今井 貴雄[1,3]、岡野ジェイムス洋尚[1,3]、岡野 栄之[1,3]
[1]慶應大・医・生理学、[2]獨協医大・解剖、[3]科技団・戦略

OJ3-05 エピジェネティクス制御による中枢神経幹細胞のアストロサイト分化制御機構
Developmental stage dependent regulation of epigenetic modification of a premature astrocytic gene promoter
波平 昌一[1]、中島 欽一[1]、田賀 哲也[1]
[1]熊本大・発医研・転写制御

OJ3-06 Dlx1 and Dlx2 homeobox genes determine competent state of retinal progenitor cells
Koji Shibasaki[1]、Lin Gan[1]
[1]Center for Aging and Dev. Biol., University of Rochester School of Medicine, Rochester, NY, U.S.A.

16:10～17:40 Room J

OJ3-07～OJ3-12 細胞分化
Cellular Differentiation

座長：仲嶋　一範（慶大・医・解剖学）
山森　哲雄（基礎生物学研究所・種分化機構第一研究部門）

OJ3-07 成体ラット側頭葉におけるニューロン新生
Neurogenesis beneath the temporal neocortex in the adult rat brain
竹村　典子[1,2]
[1]東京大院・医・神経生物、[2]東京大院・医・神経ネットワーク

OJ3-08 bFGFによる大脳皮質神経上皮細胞から大脳基底核原基神経上皮細胞への運命転換
Ventralizing effect of bFGF on cortical neuroepithelial cells
椙松昌彦[1]、鹿川　哲史[1]、福田　信治[1]、井上　俊洋[1]、田賀　哲也[1]
[1]熊本大学・発生医学研究センター・転写制御分野

OJ3-09 大脳皮質の発生において脳室帯で特異的に発現する分泌型あるいは細胞表面分子のスクリーニング
Screening of the ventricular zone-specific secretory or cell surface molecules in the developing cerebral cortex
味岡　逸樹[1]、前田　卓哉[1]、仲嶋　一範[1,2]
[1]慶應・医・解剖、[2]慈恵医大・DNA研・分子神経

OJ3-10 分化途上のニューロンに発現している新規転写因子 ZFH-5 は，内在性アンチセンスRNAにより負の制御を受けている
Novel transcription factor ZFH-5 expressed in the differentiating neurons is negatively regulated by the natural antisense RNA
小峰由里子[1]、中村　健司[2]、勝木　元也[1]、山森　哲雄[1]
[1]基礎生物学研究所、[2]三菱化学生命科学研究所

OJ3-11 ゼブラフィッシュにおける、Engrailed-1 発現細胞の脊髄神経回路中での機能
Engrailed-1 expression marks a primitive class of inhibitory spinal interneuron
東島　眞一[1,2]
[1]自然科学研究機構・岡崎統合バイオサイエンスセンター、[2]Dept of Neurobiology, SUNY Stony Brook, NY, USA

OJ3-12 ER81は齧歯類と霊長類の大脳皮質第５層の投射神経細胞の一部に発現している
ER81 is expressed in a subpopulation of layer 5 projection neurons in rodent and primate cerebral cortices
米島　宏幸[1]、山崎　信介[1]、Courtney C J Voelker[2]、Zoltán Molnár[2]、藤田　一郎[1]、山本　亘彦[1]
[1]大阪大院・生命機能・認知脳科学、[2]Dept. of Human Anatomy and Genetics, Univ. of Oxford, Oxford, UK

一般演題（ポスター発表）プログラム

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内臓感覚／Viscerosensory System		P2-180	
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◆◇◆ Poster Session ◆◇◆

神経伝達物質、修飾物質
Neurotransmitters and Modulators

P1-001 新規ペプチドINSL7/relaxin3のラット脳内発現
The expression of INSL7/relaxin3 in the rat brain
田中 雅樹[1]、飯島 典生[1]、宮本 容正[1]、小澤 一史[2]
[1]京都府立医大院・生体機能形態科学、[2]京都府立医大院・生体構造科学

P1-002 マーモセット脳内ChATニューロンの分布：免疫組織学的解析
Distribution of cholinergic neurons in the common marmoset brain
岩佐 峰雄[1]、唐沢 延幸[1]、竹内 輝美[1]、山田 敬喜[2]、片山 一道[3]、森 琢磨[3]、
清水 慶子[3]、林 基治[3]
[1]星城大学・リハビリテーション学部、[2]藤田保健衛生大学・衛生学部、[3]京都大学・霊長類研究所

P1-003 ラット眼組織のコリン神経支配：新規アセチルコリン合成酵素pChATによる再評価
Re-evaluation of cholinergic innervation of rat ocular tissues using choline acetyltransferase of a peripheral type as a marker
安原 治[1]、相見 良成[1]、松尾 明典[1]、Jean-Pierre Bellier[1]、Mohamed Elnasharty[1]、
遠山 育夫[1]、木村 宏[1]
[1]滋賀医科大・分子神経研

P1-004 2つのタイプのコリンアセチル基転移酵素の細胞内動態の差異
Differences in intracellular translocation between two types of choline acetyltransferase of the rat
松尾 明典[1]、Jean Pierre Bellier[1]、久野 正[1]、相見 良成[1]、安原 治[1]、遠山 育夫[1]、
齋藤 尚亮[1,2]、木村 宏[1]
[1]滋賀医大・分子神経科学研究センター、[2]神戸大・バイオシグナル研究センター

P1-005 培養神経細胞におけるアデノシンA3受容体のN型Caチャネルに対する効果
Effects of adenosine A3 receptor on N-type Ca^{2+} channels in cultured rat striatal neurons
井草 幸夫[1]
[1]埼玉県立大学 生理

P1-006 同族モノアミントランスポーターによる細胞外モノアミンの調節
Regulation of extra-cellular monoamine by cognate monoamine transporter
小林 秀昭[1]、沈 昊偉[2]、萩野 洋子[2]、仲 真樹[1]、近江 香予[1]、池田 和隆[2]、
沼地陽太郎[1]、曽良 一郎[1,2]
[1]東北大院・医・精神神経生物、[2]（財）東京都医学研究機構・東京都精神医学総合研究所

P1-007 ヒトチロシン水酸化酵素1型のN端の除去は本酵素の細胞内安定性を増加させる
Deletion of N-terminus of human tyrosine hydroxylase type 1 dramatically enhances the stability in mammalian cells
中島 昭[1]、金子 葉子[1]、森 啓至[1]、永津 俊治[2]、太田 明[1]
[1]藤田保健衛生大学・医学部・第1生理、[2]藤田保健衛生大学・総合医科学研究所・難病治療共同研究部門

P1-008 セロトニンは5-HT1A受容体を介してオレキシン神経細胞を過分極させる
Serotonergic regulation of the orexin/hypocretin neurons through the 5-HT1A receptor
山中 章弘[1,2,3]、村木 暢[1]、辻野なつ子[1]、Thomas Kilduff[3]、後藤 勝年[1]、櫻井 武[1,2]
[1]筑波大学・基礎医学系・薬理学、[2]ERATO柳沢オーファン受容体プロジェクト、[3]SRI International

P1-009 セロトニン遊離薬は5-HT$_{1D}$受容体を介して脊髄単シナプス反射を抑制する
Endogenously released 5-HT depress the spinal monosynaptic reflex via 5-HT$_{1D}$ receptors
本多 基子[1]、今井田佳子[1]、田辺 光男[1]、小野 秀樹[1]
[1]名古屋市大院・薬・中枢神経機能薬理学

P1-010 ラット黒質線条体系ドーパミンニューロン破壊後縫線核線条体系セロトニンニューロンにおけるドーパミン代謝の免疫組織学的検討
Dopamine is present in raphe-striatal serotonergic neurons of rats with nigro-striatal dopaminergic denervation
前田 哲也[1]、長田 乾[1]、吉田 泰二[2]、神成 一哉[3]
[1]秋田県立脳血管研究センター神経内科学研究部、[2]秋田県立脳血管研究センター病理学研究部、[3]弘前大学医学部第三内科

P1-011 AD/HDモデル：高血圧自然発症ラット（SHR）に対するmilnacipranの作用
An effect of milnacipran on norepinephrine content in spontaneously hypertensive rat: AD/HD model rat.
石松 秀[1]、木谷 有里[1]、桑波田 卓[1]、赤須 崇[1]
[1]久留米大学・医・第2生理

P1-012 AD/HDモデル：高血圧自然発症ラット（SHR）の青斑核ニューロンに対するMethylphenidateの作用
Effect of Methylphenidate on Locus Coeruleus Neurons in Spontaneously Hypertensive Rat: an Animal Model of AD/HD.
木谷 有里[1]、石松 秀[1]、桑波田 卓[1]、赤須 崇[1]
[1]久留米大・医・第2生理

P1-013 脱分極刺激誘発ドパミン遊離に対する組織プラスミノーゲンアクチベーター－プラスミン経路の関与
Tissue plasminogen activator-plasmin system modulates depolarization-evoked dopamine release
永井 拓[1]、橋本 和樹[1]、亀井 浩行[1]、田熊 一浩[1]、鍋島 俊隆[2]、山田 清文[1]
[1]金沢大院・自然科学・病院薬学、[2]名古屋大院・医・医療薬学、附属病院薬剤部

P1-014 イオンチャネル型ガンマアミノ酪酸受容体を活性化する芳香物質のマウス行動への影響
Behavioral effects in mice of the fragrant compounds which activate ionotropic GABA receptors
青島 均[1]、葛城 将文[1]、梅津 豊司[2]
[1]山口大 理 自然情報、[2]独立法人国立環境研究所

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P1-015 PACAP欠損マウスにおけるエタノール誘発体温低下および睡眠作用の減弱
Reduced hypothermic and hypnotic responses to ethanol in PACAP-deficient mice
田中 一裕[1]、橋本 均[1]、新谷 紀人[1]、山本亜希子[1]、馬場 明道[1]
[1]大阪大院・薬・神経薬理学

P1-016 不応期終了後かつGABA抑制開始以前に働く皮質フィードバック興奮抑制
An intermediate type of feedback regulation of cortical excitability that is not attributable to synaptic inhibition or refractoriness
村田 宮彦[1]、加藤 伸郎[1]
[1]京都大院・医・認知行動脳科学

P1-017 三叉神経運動核におけるGABAならびにグリシン免疫陽性終末の発生学的検索
GABA and/or glycine immunoreactive synapses in the trigeminal motor nucleus of rats during postnatal development
林 弘之[1]、都築 英子[1]、河田 亮[1]、東 一善[1]、高橋 浩二[1]、高橋 理[1]
[1]神奈川歯科大学・歯・人体構造学・組織学分野

P1-018 ラット大脳皮質ニューロンにおけるGABA$_A$受容体を介するトニック抑制
Tonic GABA$_A$ receptor-mediated inhibition in rat neocortex
山田 順子[1]、山本 純偉[2]、上野 伸哉[3]、古川 智範[2]、福田 敦夫[1,2]
[1]静岡大院・電子科研・生体情報処理、[2]浜松医大・医・生理学第一、
[3]弘前大・脳研・機能回復

P1-019 サル前頭前皮質においてGABA$_A$受容体はコラム活動を形成する
GABA$_A$ receptors shape columnar activity in the monkey prefrontal cortex
平田 快洋[1,2]、澤口 俊之[1,2]
[1]北海道大院・医・高次脳機能学、[2]科学技術振興事業機構戦略的基礎研究

P1-020 Realtime RT-PCR法によるラット脳内各部位におけるGABABR1, R2 subunitの定量的mRNA解析
Quantitative mRNA analysis of GABABR1 and R2 subunits in various regions of the rat brain by real-time RT-PCR
菊山 裕貴[1]、豊田 裕敬[1]、中川 俊正[2]、神原 清人[3]、水野 貴史[1]、吉田 祥[1]、
森本 一茂[4]、川野 涼[1]、太田 宗寛[1]、堤 淳[1]、渡辺 正仁[3]、松村 人志[5]、
米田 博[1]
[1]大阪医科大学 神経精神医学教室、[2]大阪医科大学 病態検査学教室、[3]大阪医科大学 第二解剖学教室、[4]大阪医科大学 中央検査部、[5]東亜大学 医療工学部

P1-021 脳幹聴性中継核におけるGABA, glycineニューロンの不均質分布
Regional expression of GABA, glycine and related neuronal markers in rat auditory brainstem
佐久間 覚[1]、宝谷 剛志[1]、加瀬 政彦[1]、堤 俊之[1]、杉本 哲夫[1]
[1]関西医大・脳構築学

P1-022 小脳発生期におけるGABA伝達機構
GABAergic signaling in the developing cerebellum
高山 千利[1]、井上 芳郎[1]
[1]北海道大・医・分子解剖学

P1-023 培養小脳組織からのGABA放出パターンの観察
The observation of spatiotemporal GABA release patterns in cerebella culture
上松 正和[1,5]、柳川右千夫[2,4]、小幡 邦彦[3]、吉田 祥子[1]
[1]豊橋技術科学大学・物質工学系、[2]群馬大院・医・遺伝発達行動学、[3]理研・脳センター、[4]科学技術振興機構・CREST & SORST、[5]豊橋技術科学大学・未来技術流動センター

P1-024 仙髄におけるグリシンとGABAのシナプス小胞への充填速度の差異による共放出の出現
Distinct re-filling profiles of inhibitory neurotransmitters into presynaptic terminals projecting to spinal neurons
窪田 寿彦[1]、桂林秀太郎[2]、赤池 紀生[3]、福田 敦夫[1]
[1]浜松医科大学 医 生理学第一講座、[2]Dept. Mol. & Gene., Baylor Coll. Med., Houston, USA、[3]熊本保健科学大学

P1-025 細胞性粘菌の発達におけるグルタメイトシグナリングの役割
The role of glutamate signaling in development of *Dictyostelium discoideum*.
眞田 法子[1]、谷浦 秀夫[1]、米田 幸雄[1]
[1]金沢大・院・自然科学・薬物

P1-026 放射線による中枢神経障害発症におけるグルタミン酸の役割
Role of glutamate on radiation-induced functional damage of the central nervous system in rats
山本 浩一[1]、野原 恭子[1]、安藤 興一[2]、大和谷 厚[1]
[1]大阪大院・医・保健・生体情報、[2]放射線医学総合研究所

P1-027 非定型抗精神病薬クロザピンのラット内側前頭前野細胞外グルタミン酸レベルに対する効果:脳内微小透析法による検討
Effects of clozapine on extracellular glutamate in rat medial prefrontal cortex
黒木 俊秀[1]、梶畑 俊雄[1]、中原 辰雄[1]、神庭 重信[1]
[1]九州大院・医・精神病態医学

P1-028 低亜鉛食で飼育したマウスの海馬苔状線維の異常興奮
Enhanced Excitability of Hippocampal Mossy Fibers under Dietary Zinc Deficiency
武田 厚司[1]、山田 浩平[1]、南 彰[1]、長野 哲雄[2]、奥 直人[1]
[1]静岡県立大学・薬・医薬生命化学、[2]東京大学・薬・薬品代謝化学

P1-029 ラット海馬におけるエストロゲン受容体分布の解析
Localization of Estrogen Receptor α in the Rat Hippocampus
向井 秀夫[1,2]、村上 元[1,2]、北條 泰嗣[1,2]、小南 思郎[4]、William Janssen[4]、John Morrison[4]、木本 哲也[1,2]、川戸 佳[1,2]
[1]東京大院・総合文化・広域科学、[2]科学技術振興機構戦略的基礎研究、[3]広島大学総合科学部、[4]Fishberg Center of Neurobiology of Aging, Mount Sinai School of Medicine

P1-030 転写因子から見た脳におけるエストロゲンーニコチン相関
Estrogen and nicotine interactions in terms of transcription factors in the brain
笹井 友介[1]、新郷 明子[1]、鬼頭 昭三[1]
[1]兵庫大・健康科学部・脳科学実験室

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P1-031 海馬におけるエストロゲンによるERとIGF-1のmRNA発現に対するカイニン酸とニコチンの作用
Kainic acid and nicotine have reverse effects on estrogen-induced ER and IGF-1 mRNA expressions in the hippocampus.
桂　昌裕[1]、新郷　明子[1]、鬼頭　昭三[1]
[1]兵庫大・健康科学部・脳科学実験室

P1-032 NMDA全身投与後のマウス海馬におけるAP1とTex261の発現調節
Regulation of AP1 and Tex261 gene expression in mouse hippocampus after intraperitoneal injection of NMDA.
飯島　壮悟[1]、谷浦　秀夫[1]、米田　幸雄[1]
[1]金沢大院・自然科学・薬物学

P1-033 エレクトロポレーション法により同定した新皮質ニューロンにおけるGABA$_A$レセプターを介するシグナルの特性
Properties of GABA$_A$ receptor-mediated signaling in neocortical neurons with identified birth period by *in utero* electroporation of EGFP.
古川　智範[1]、山田　順子[2]、井上　浩一[1]、天野　賢[1]、福田　敦夫[1]
[1]浜松医科大学生理学第一講座、[2]静岡大学大学院電子科学研究科

P1-034 クロマフィン細胞におけるPKCアイソフォームによる開口分泌制御
Regulation of exocytotic events by protein kinase C isoform in single adrenal chromaffin cells
笹川　展幸[1]、村山　典恵[1]、熊倉鴻之助[1]
[1]上智大学・生命科学研究所・神経化学

P1-035 培養神経細胞における細胞活動と神経栄養因子
Effect of neurotrophins on neural acitivities in dissociated neurons
鳥光　慶一[1,2]、古川由里子[1,2]、河西奈保子[1]、韓　春錫[1]、藤本　一朗[3]
[1]NTT 物性科学基礎研究所、[2]SORST/JST、[3]東京大学医科学研究所、NTT-IMSUT

P1-036 PKCによる分泌顆粒供給の時空的調節
Tempo-spatial regulation of the vesicle dynamics by PKC in adrenal chromaffin cells.
熊倉鴻之助[1]、笹川　展幸[1]、村山　典恵[1]
[1]上智大・生命研

受容体、輸送体
Receptors and Transporters

P1-037 皮質線条体終末および視床線条体終末におけるシナプス前性AMPAレセプター
Presynaptic Localization of AMPA-type Glutamate Receptor in Corticostriatal and Thalamostriatal Axon Terminals
藤山　文乃[1]、倉本恵理子[1]、岡本　敬子[1]、日置　寛之[1]、古田　貴寛[1]、周　里鋼[1]、野村　儀[2]、金子　武嗣[1]
[1]京都大院・医・高次脳形態、[2]京都大学 医学部保険学科 理学療法学専攻

P1-038 小脳バーグマングリアにおけるAMPA型グルタミン酸受容体の局在
Concentrated localization of AMPA-type glutamate receptors in Bergmann glial plasmalemma facing synaptic cleft at Purkinje cell synapses
深谷 昌弘[1]、渡辺 雅彦[1]
[1]北海道大院・医・解剖発生

P1-039 NMDA受容体のインターナライゼーション
Internalization of NMDA receptors
中道 範隆[1]、及川 弘崇[1]、神戸 悠輝[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物

P1-040 NMDA受容体GluRε/NR2サブユニットはGluRζ1/NR1サブユニットのポストシナプスへの局在化とタンパク安定化に必須である
NMDA receptor GluRε/NR2 subunits are essential for postsynaptic localization and protein stability of GluRζ1/NR1 subunit
阿部 学[1]、深谷 昌弘[2]、三品 昌美[3]、渡辺 雅彦[2]、崎村 建司[1]
[1]新潟大・脳研・細胞神経生物、[2]北海道大院・医・解剖発生、[3]東京大院・医・分子神経生物

P1-041 培養神経細胞の成熟に伴うNMDA感受性の変化
Altered sensitivity to NMDA during maturation in cultured rat brain neurons
神戸 悠輝[1]、中道 範隆[1]、及川 弘崇[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物学

P1-042 マウス成熟脳海馬由来神経系前駆細胞のNMDAシグナル応答性
NMDA signaling to adult neurogenesis in murine hippocampus
渡邉 真未[1]、米山 雅紀[1]、玉置 啓祐[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物

P1-043 成人脳に高発現するGluR4サブユニットの新規選択的スプライシングアイソフォーム
GluR4c, an alternative splicing isoform of GluR4 is abundantly expressed in adult human brain
河原 行郎[1]、伊藤 杏子[1]、孫 慧[1]、伊藤 雅之[2]、金澤 一郎[1,2]、郭 伸[1]
[1]東京大院・医・神経内科、[2]国立精神神経センター

P1-044 リガンド結合による代謝型グルタミン酸受容体の二量体構造変化
Ligand-induced rearrangement of the dimeric conformation of metabotropic glutamate receptor1α
立山 充博[1,2]、阿部 秀樹[1,2]、中田 裕康[3]、齋藤 修[3]、久保 義弘[1,2]
[1]生理研・分子生理・神経機能素子、[2]CREST、[3]神経研・生体機能分子

P1-045 小脳プルキンエ細胞においてグルタミン酸受容体GluRδ2とPSD足場蛋白質Shankは直接結合している
Direct interaction of GluRδ2 with Shank scaffold proteins in cerebellar Purkinje cells
植村 健[1]、森 寿[1]、三品 昌美[1]
[1]東大院・医・分子神経生物学、SORST/JST

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P1-046 C末領域によるδ2グルタミン酸受容体の細胞内局在の調節
C-terminal domain of δ2 glutamate receptor controls its intracellular localization
松田 信爾[1]、松田 恵子[1]、柚崎 通介[1]
[1]慶應大・医・生理

P1-047 ゼブラフィッシュグルタミン酸受容体δ2は小脳プルキンエ細胞様の結合様式を持つ神経細胞に特異的に発現している
Zebrafish glutamate receptor δ2 is expressed in selective neurons with wiring patterns similar to cerebellar Purkinje cells
三上 義礼[1]、吉田 知之[1]、松田 尚人[1]、三品 昌美[1]
[1]東京大院・医・分子神経生物学

P1-048 ラット線条体におけるgroup III 代謝型グルタミン酸受容体陽性の軸索終末とコリン作動性ニューロンの樹状突起との関係
Association of group III metabotropic glutamate receptor-equipped axon terminals with the dendrites of cholinergic neurons in rat striatum
倉本恵梨子[1]、藤山 文乃[1]、重本 隆一[2]、金子 武嗣[1]
[1]京都大院・医・高次脳形態学、[2]生理学研究所・脳形態解析部門

P1-049 ラットfreeze-lesionモデル動物における$GABA_A$及びグリシン受容体の発現変化
Expressional changes in the $GABA_A$ and glycine receptor subunit mRNAs in the rat freeze-lesion model of cortical malformations.
岡部 明仁[1]、清水一岡部千草[1]、W Kilb[2]、小林 可奈[1]、川島 裕也[1]、高山 洋平[1]、HJ Luhmann[2]、福田 敦夫[1]
[1]浜松医大・医・一生理、[2]Inst of Physiol and Pathophysiol, Johannes Gutenberg University Mainz, German

P1-050 ラット大脳皮質アストロサイトにおける$GABA_B$受容体の機能発現
Functional expression of metabotropic $GABA_B$ receptor in rat cerebrocortical astrocytes
藤田 卓也[1]、和田美由紀[1]、岡 美智子[1]
[1]京都薬科大・薬剤学

P1-051 ドパミン受容体作動薬のHomer1a遺伝子発現に対する影響
Dopamine-D1 and D2 receptor agonists differently modulate gene expression of Homer 1a.
山田 英孝[1]、中原 辰雄[1]、黒木 俊秀[1]、橋本喜次郎[1]、堤 哲之[1]、平野 誠[1]
[1]肥前精神医療センター・臨床研究部・生化学研究室

P1-052 ラット脳膜標品における5-HT刺激性[^{35}S]GTPγS結合に対する5-HT受容体拮抗薬の影響
Effects of several 5-HT receptor antagonists on 5-HT-stimulated [^{35}S]GTPγS binding in rat brain membranes
小田垣雄二[1]、豊嶋 良一[1]、山内 俊雄[1]
[1]埼玉医大・精神

P1-053 アデノシン処理による脊髄培養アストロサイトの興奮性アミノ酸処理機能の変化
Change of Glutamate transporter and glutamine sunthetase in rat cultured astrocytes by adenosine exposure: Role for neuroprotection via EAA scavenging
尾形 直則[1]、森野 忠夫[1]、堀内 秀樹[1]、濱本雄一郎[1]、山本 晴康[1]
[1]愛媛大・医・整形外科

P1-054 ヒトノルエピネフリントランスポータースプライスバリアントの機能発現調節
Regulated expression of human norepinephrine transporter splice variants
北山 滋雄[1]、十川 千春[1]、十川 紀夫[1]、熊谷 圭[2]、森田 克也[2]、土肥 敏博[2]、小野寺憲治[1]
[1]岡山大院・医歯総合・歯科薬理、[2]広島大院・医歯薬総合・歯科薬理

P1-055 細胞外グルコースによる骨芽細胞GABAトランスポーター活性の調節
Regulation by extracellular glucose of GABA transporter activity in rat calvarial osteoblasts.
藤森さゆ美[1]、大澤 壮登[1]、檜井 栄一[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物学

P1-056 小胞性モノアミントランスポーター完全欠損マウスではドーパミントランスポーター活性が低下している
Inhibitory regulation of plasma dopamine transporter activity by complete knockout of vesicular monoamine transporter-2 (VMAT-2)
山本 秀子[1]、亀ヶ谷悦子[1]、萩野 洋子[1]、池田 和隆[1]、山本 敏文[1,2]、岸田真紀子[1]、沼知陽太郎[3]、竹島多賀夫[4]、George Uhl[5]、曽良 一郎[1,3]
[1]都精神研・分子精神医学、[2]横浜市立大・院・総理、[3]東北大学・院・医、[4]鳥取大学・医、[5]Mol. Neurobiol. Branch, NIDA-IRP, NIH, Baltimore, USA

P1-057 高親和性コリントランスポーターのシナプス小胞への局在：免疫電顕による検討
Ultrastructural localization of high-affinity choline transporter on synaptic vesicles.
中田 和子[1]、奥田 隆志[2]、三澤日出巳[1]
[1]東京都神経研・神経学、[2]東京大院・医・神経生化

P1-058 ラット脳における中性アミノ酸トランスポーターASCT1の糖鎖修飾の脳内部位差
Regional differences on glycosylation of neutral amino acid transporter ASCT1 in rat brain
山本 敏文[1]、宅間 仁志[1]、竹内紗貴子[1]、古屋 茂樹[2]、平林 義雄[2]、渡辺 雅彦[5]、池田 和隆[3]、額田 敏秀[4]、山本 秀子[3]
[1]横浜市大院・総理・分子認識、[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グループ、[3]東京都精神医学総合研究所、分子精神医学部門、[4]東京都精神医学総合研究所、脳細胞情報伝達部門、[5]北海道大学医学研究科解剖発生

P1-059 輸送系asc中性アミノ酸トランスポーターAsc-1のラット大脳皮質および小脳皮質における局在解析
Neuronal localization of a system asc neutral amino acid transporter (Asc-1) in the rat cerebral and cerebellar cortices.
徳永 元秀[1]、松尾 洋孝[1]、中田 隆博[2]、武田湖太郎[1]、石嶺 久子[1]、小林 靖[2]、金井 好克[3]、遠藤 仁[3]、福田 潤[1]
[1]防衛医大・生理学第一、[2]防衛医大・解剖学第二、[3]杏林大・医・薬理学

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P1-060 3種類のNBCアイソフォーム別のマウス脳内局在
Developmental expression pattern of sodium bicarbonate cotransporter NBC in mouse brain
藤本 一朗[1]、Giuseppina Priori[1]、関 常司[2]、鳥光 慶一[3]、御子柴克彦[1]
[1]東大・医科研・神経情報シグナルNTT-IMSUT、[2]東大・医学部・内科、[3]日本電信電話株式会社・NTT物性基礎研

P1-061 SH-SY5Y細胞によるクエン酸アルミニウム取り込みに対するシステムXc⁻の関与
Contribution of System Xc⁻ to Al Citrate Uptake by SH-SY5Y Cells
長井 克仁[1]、長澤 一樹[1]、赤木 淳哉[1]、小間 峰登[1]、下濱 俊[2]、藤本 貞毅[1]
[1]京都薬科大学・衛生化学、[2]京都大学・医学部・神経内科学

P1-062 ラットアストロサイトにおけるカルニチントランスポーターOCTN2の機能的発現
Functional expression of the organic cation/carnitine transporter OCTN2 in rat astrocytes
稲津 正人[1]、宮下 協二[1]、前原 勝之[1]、武田 弘志[1]、松宮 輝彦[1]
[1]東京医大・薬理

P1-063 海馬CA1領域におけるシナプス前性TrkB受容体の役割
Roles of presynaptic TrkB receptors in the hippocampal CA1 region
熊澤 紀子[1]、加藤 永子[2]、竹内 倫徳[2]、三品 昌美[2]、真鍋 俊也[1]
[1]東大・医科研・神経ネットワーク分野、[2]東大・院医・分子神経生物、SORST/JST

P1-064 海馬CA3領域のTrkB受容体欠損C57BL/6マウス
C57BL/6 mice lacking TrkB neurotrophin receptor in hippocampal CA3 region
加藤 永子[1]、竹内 倫徳[1]、松代はるか[1]、森 寿[1]、深谷 昌弘[2]、渡辺 雅彦[2]、
明石 馨[3]、崎村 建司[3]、三品 昌美[1]
[1]東京大院・医・分子神経生物学、[2]北大院・医・機能形態学、[3]新潟大・脳研・細胞神経生物学

P1-065 海馬神経細胞における神経活動依存性TrkB FL mRNA安定化のsynergisticなシグナル制御
Synergistic regulation of activity-dependent stabilization of trkB FL mRNA in cultured hippocampal neuron
大河内謙太郎[1]、堂薗 礼子[1]、大江 総一[1]、小島 正巳[2]、米田 悦啓[1]
[1]大阪大学大学院・生命機能研究科・細胞ネットワーク講座・細胞内分子移動学、[2]産業技術総合研究所・セルエンジニアリング研究部門・セルダイナミクス研究グループ

P1-066 神経幹細胞の増殖に対するロイコトリエンB_4の作用
Effect of leukotriene B_4 on neural stem cell proliferaiton
工藤 千穂[1]、和田孝一郎[1]、中島 淳[2]、山本 晋[3]、上崎 善規[1]
[1]大阪大院・歯・薬理、[2]横浜市大・医・三内、[3]小野薬品・創薬研

P1-067 各種静脈麻酔薬のシグマ1受容体に対する作用
Effects of several intravenous anesthetics on sigma-1 receptors
山田麻起子[1]、中尾 慎一[1]、高森 康晴[2]、小田 紀子[2]、片岡 洋祐[2]、山田 久夫[2]、
新宮 興[1]
[1]関西医科大学大学院・麻酔科、[2]関西医科大学 システム細胞科学

P1-068 コルチコステロイドレセプターとインポーチンの生細胞内におけるダイナミクス
Dynamics of corticosteroid receptors and importins in living neural cells and non-neural cells
西　真弓[1]、宇宿　智裕[1]、河田　光博[1]
[1]京都府立医科大学大学院・生体構造科学

P1-069 ラット胎児脳内特定部位からの神経系前駆細胞の単離
Neural progenitor cells isolated from discrete brain regions of embryonic rats
米山　雅紀[1]、玉置　啓祐[1]、米田　幸雄[1]
[1]金沢大院・自然科学・薬物

P1-070 神経栄養活性を持つアデニンに対する結合サイトの性状解析
Characterization of binding sites for adenine, a neurotrophic factor of cultured Purkinje cells
渡辺　俊[1,2]、池北　雅彦[1]、中田　裕康[2]
[1]東京理科大院・理工・応用生物、[2]東京都神経科学総合研究所・生体機能分子

P1-071 生体試料による原子間力顕微鏡AFMを用いたIP3レセプターの表面構造解析
AFM image of inositol 1,4,5-trisphosphate receptor (IP3R) tetramer structure in its native environment
須原和歌子[1]、後藤東一郎[2]、小林　未明[1,2]、鳥光　慶一[2]、御子柴克彦[1]、藤本　一朗[1]
[1]東大・医科研・神経情報シグナルNTT-IMSUT、[2]日本電信電話株式会社・NTT物性基礎研

P1-072 ニューロペプチドY Y1 受容体 mRNA のげっ歯類脳内分布およびメラノコルチン４受容体との共発現
Neuropeptide Y Y1 receptor mRNA in the rodent brain: Distribution and coexpression with the melanocortin-4 receptor
岸　敏郎[1]
[1]島根大・医・解剖学講座神経形態学

P1-073 NPY-Y5受容体ノックアウトマウスでの摂食行動の調節
Feeding behavior of NPY-Y5 knock out mice
山口　剛[1]、大谷　桂[1]、長友　孝文[1]、三富　明夫[1]、浦山　勝裕[1]、樋口　宗史[1]
[1]新潟大院・医歯・分子細胞医学・薬理学

P1-074 海馬、視床下部領域におけるミネラルコルチコイド受容体とグルココルチコイド受容体の共存について
Colocalization pattern of mineralocorticoid receptor (MR) and glucocorticoid receptor (GR) in the rat hippocampus and paraventricular nucleus
韓　芳[1]、小澤　一史[1]、松田　賢一[1]、河田　光博[1]
[1]京都府立医科大学・生体構造科学

P1-075 エストロゲン受容体プロモーター制御下でGFPを発現するトランスジェニックラット
Transgenic rats expressing green fluorescent protein under the control of estrogen receptor α promoter
濱田　知宏[1]、和田一木山裕子[1]、佐久間康夫[1]
[1]日本医大・医・生理学第一

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P1-076 カイニン酸傷害海馬におけるエストロゲン受容体の発現
ER expression in the kainic acid-injured hippocampus
木村 泰大[1]、新郷 明子[1]、鬼頭 昭三[1]
[1]兵庫大・健康科学部・脳科学実験室

P1-077 インターロイキン1βによるラットアストロサイトにおけるアクアポリン4の誘導
Interleukine-1β induces aquaporin 4 expression in rat astrocytes
祖父江和哉[1]、山本 直樹[2]、伊藤 弘晃[1]、森島 徹朗[1]、青山 峰芳[2]、梅西 文範[3]、
勝屋 弘忠[1]、浅井 清文[2]
[1]名古屋市立大学大学院・医・危機管理医学、[2]名古屋市立大学大学院・医・分子神経生物学、
[3]Division of Renal Diseases and Hypertension, Department of Medicine, Univ of Colorado Health Sciences Center, Denver, USA

P1-078 A_1アデノシン受容体と$P2Y_2$受容体のオリゴマー形成
Hetero-oligomerization of A_1 Adenosine receptors and $P2Y_2$ purinergic receptors
津賀 浩史[1]、吉岡 和晃[1]、神谷 敏夫[1]、中田 裕康[1]
[1]東京都神経科学総合研究所生体機能分子研究部門

P1-079 パーキンソン病原因遺伝子parkinによるATP受容体の機能制御
Regulation of P2X recepters by parkin
佐藤あゆみ[1]、真子 好正[1]、西川 香里[2]、青木公美子[2]、和田恵津子[2]、青木 俊介[2]、
和田 圭司[2]、野田 百美[1]
[1]九州大院・薬・病態生理、[2]国立精神神経センター

P1-080 ウサギ迷走神経吸息促進反射は孤束核P2X受容体を介する
P2X receptor mediates vagal inspiration-promoting reflex in the rabbit
高野 一夫[1]、繁冨 英治[2]、加藤 総夫[2]
[1]慈恵医大・薬理学、[2]慈恵医大・神経生理

P1-081 ニコチン適用に伴う培養アストロサイトの細胞内遊離Ca^{2+}濃度変化
Changes in intracellular free Ca^{2+} by nicotine in cultured astrocytes
及川 弘崇[1]、中道 範隆[1]、神戸 悠輝[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物学

P1-082 シンタキシン5はゴルジ装置の構造に影響する
Syntaxin 5 affects organization of the Golgi apparatus
服部 浩史[1]
[1]杏林大・医・細胞生理

P1-083 addicsin/GRAP3-18結合タンパク質Aip-1の同定
Identification of Aip-1 as an addicsin/GTRAP3-18 associated protein
秋月さおり[1]、宅森 将人[1,2]、池本 光志[1,2]
[1]産業技術総合研究所・年齢軸生命工学研究センター、[2]東邦大院・理学研究科

P1-084 3,4-Dihydroxybenzaldehyde, 線虫C06H5.7に対する新規アゴニスト
3,4-Dihydroxybenzaldehyde, a novel agonist for *Caenorhabditis elegans* C06H5.7
青木 令奈[1]、矢上 達郎[1]、片山 梨絵[1]、宮前 丈明[1]、中村 史雄[1]、梶原 康宏[2]、
山本 直毅[2]、五嶋 良郎[1]
[1]横浜市立大院・医・分子薬理神経生物学、[2]横浜市立大院・総合理学研究科

P1-085 膜内微小領域（ラフト）に存在するATPaseの解析
Identification of raft-localized ATPases of synaptic membranes
前川 昌平[1]、芳仲 幸治[1]、田口 勝敏[1]、熊ノ郷晴子[2]、中村 俊[2]
[1]神戸大学大学院・自然科学・生命機構科学、[2]国立精神・神経センター、神経研

情報伝達、変換、修飾
Signal Transduction and Modulation

P1-086 ラット脳由来細胞膜ラフト領域を構成する 4-5F6 抗原の同定と機能解析
Identification and characterization of 4-5F6 antigen as the neural tissue-enriched synaptic protein in the lipid raft of rat brain
田口 勝敏[1]、熊ノ郷晴子[2]、中村 俊[2]、宮田 清司[3]、前川 昌平[1]
[1]神戸大院・自然科学・生命機構科学専攻、[2]国立精神・神経センター神経研究所、診断研究部 、[3]京都工芸繊維大学、繊維学部、応用生物学科

P1-087 PtprzによるGit1チロシンリン酸化レベルの制御
Identification of tyrosine phosphorylation sites of Git1, a substrate for Ptprz
深田 斉秀[1,2]、藤川 顕寛[1,2]、野田 昌晴[1,2]
[1]自然科学研究機構 基礎生物学研究所 感覚情報処理、[2]戦略的創造研究推進事業 科学技術振興機構

P1-088 Mカリウムチャンネルのムスカリン抑制に必要なAKAP79/150の最小機能部位の同定
MAPPING ESSENCIAL DOMAIN OF AKAP79/150 FOR MUSCARINIC SUPPRESSION OF THE M-CURRENT
星 直人[1]、東田 陽博[1]、John Scott[2]
[1]金沢大院・医・脳細胞遺伝子、[2]Howard Hughes Medical Institute and Vollum Institute, Oregon Health Sciences University, Portland, USA

P1-089 培養神経細胞におけるビタミンDとグルタメイトのクロストーク
Crosstalk between vitamin D and glutamate signaling in primary cultured rat cortical neurons.
伊藤 実[1]、倉本 展行[1]、杉山 千絵[1]、谷浦 秀夫[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物学

P1-090 ラット脳弓下器官ニューロンにおけるノルアドレナリンの作用
Action of noradrenaline on rat subforcal organ (SFO) neurons in slice preparation
本田 栄子[1]、小野堅太郎[1]、稲永 清敏[1]
[1]九州歯科大・生理

P1-091 二蛍光同時イメージングによる細胞内マグネシウム貯蔵庫の解明
Investigation of Intracellular Magnesium Stores by Simultaneous Fluorescent Imagings in PC12 Cells
久保田 健[1]、新藤 豊[1]、小松 広和[2]、小川 宏人[3]、北村美一郎[1]、鈴木 孝治[2,4]、岡 浩太郎[1]
[1]慶大院・理工・基礎理工、[2]慶大・理工・応化、[3]埼玉医大・生物、[4]JST-CREST

Sep 21 (Tue) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

P1-092 3-phosphoinositide-dependent protein kinase 1 (PDK1) の偽基質によるPKCの機能調節
Pseudosubstrate of 3-phosphoinositide-dependent protein kinase 1 (PDK1) regulates PKC functions
関　貴弘[1]、松林　弘明[1]、天野　託[1]、斎藤　尚亮[2]、酒井　規雄[1]
[1]広島大院・医歯薬・神経精神薬理、[2]神戸大・バイオシグナル研・分子薬理

P1-093 転写調節因子CREBに対する、新規相互作用タンパク質の同定と機能解析
Identification and analysis of new interacting proteins with transcription factor CREB
関　信亮[1]、高橋　清文[1]、亀田　明美[1]、江副　浩和[1]、細田　広司[1]、岩本　拓[1]、舛重　正一[1]、喜田　聡[1]
[1]東京農業大学・動物分子生物学研究室

P1-094 生体内におけるFRETを利用したαCaMKII活性化のモニタリング
Visualization and monitoring of αCaMKII activation *in vivo* using FRET
加藤　健一[1]、岩本　拓[1]、舛重　正一[1]、喜田　聡[1]
[1]東京農業大学 バイオサイエンス

P1-095 不活性型Ca^{2+}/カルモジュリン依存性プロテインキナーゼIIαサブユニット遺伝子改変マウスの作成と解析
Transgenic mice carrying inactivated α subunit of Ca^{2+}/calmodulin-dependent protein kinase II
山肩　葉子[1,2,3]、井本　敬二[1,2]、戸塚　昌子[1]、八木　健[1]、小幡　邦彦[1]、柳川右千夫[1,2,3]
[1]生理研、[2]総研大、[3]CREST

P1-096 CaM キナーゼ II と MAP キナーゼ系を介する mPer1 の転写調節機構
Transcriptional regulation of mPer1 promoter through CaM kinase II and MAP kinase pathways
福永　浩司[1]、野村　和美[2]、竹内　有輔[1]
[1]東北大院・薬・薬理学、[2]熊本大・医・細胞情報薬理

P1-097 カムキナーゼIIによるタウのアルツハイマー病のリン酸化サイトのリン酸化
Phosphorylation of Tau to sites found in Alzheimer's disease brain is catalyzed by CaMKII
一瀬　竜也[1]、吉村　好之[1]、山内　卓[1]
[1]徳島大院・薬・生化学

P1-098 神経伝達物質放出におけるカルモジュリン阻害剤の影響
THE EFFECT OF OPHIOBOLIN A, A NOVEL CALMODULIN ANTAGONIST, ON NEUROTRANSMITTER RELEASE FROM PC12 AND RAT CEREBELLAR GRANULE CELLS
安藤　公祐[1]、工藤　佳久[1]、高橋　正身[2,3]
[1]東京薬科大院・生命科学・生体高次機能学、[2]北里大・医学部・代謝蛋白学、[3]CREST（科学技術振興機構）

P1-099 ステロール調節配列結合タンパク質によるヒト脳アシル-CoA水解酵素遺伝子の発現調節
Sterol regulatory element-binding protein-2 modulates human brain acyl-CoA hydrolase gene transcription
高木 充弘[1]、吉田 智洋[1]、須賀 哲弥[1]、野水 基義[1]、山田 純司[1]
[1]東京薬大・薬・病態生化学

P1-100 カルシウムシグナルによるミトコンドリア形態変化の制御
Regulation of mitochondrial dynamics and morphology by calcium signaling
松下 正之[1]、陸 雲飛[2]、冨澤 一仁[1]、李 順愛[3]、韓 小建[1]、竹居 孝二[3]、松井 秀樹[1,3]
[1]岡山大院・医・細胞生理、[2]Preventure Program, JST, Japan、日本科学技術事業団、[3]岡山大院・医・生化学

P1-101 カルパインによるNMDAレセプターの断片化と機能阻害
Calpain-Mediated Cleavage and Down-Regulation of NMDA Receptors
呉 海燕[1]、富沢 一仁[1]、E Yuen[2]、陸 雲飛[3]、松下 正之[1]、Z Yan[2]、松井 秀樹[1]
[1]岡山大学大学院・医歯学総合研究科・生体制御・細胞生理、[2]State Univ. of New York、[3]JST, Okayama

P1-102 NYAN1: the tyrosine kinase substrate that connects the NMDA receptor to Akt activation.
横山 一剛[1]、山本 雅[1]
[1]東京大学・医科学研究所・癌細胞シグナル

P1-103 PI (4,5) P_2マイクロドメインと開口放出部位
PI (4,5) P_2 microdomains and the sites of exocytosis
青柳 共太[1]、菅谷津貴子[1]、梅田 真郷[2]、山本 清二[3]、寺川 進[3]、高橋 正身[1,4]
[1]北里大・医・代謝蛋白、[2]京大・化学研、[3]浜松医大・光量子、[4]CREST JST

P1-104 リカレントネットワーク情報伝達のガンマ波による調整
Gamma oscillations enhance temporal precision in CA3 recurrent network
藤澤 茂義[1]、松木 則夫[1]、池谷 裕二[1]
[1]東京大院・薬・薬品作用

P1-105 ショウジョウバエ脳のリン酸化タンパク質の同定
Identification of a 32 kDa phosphoprotein in Drosophila brain
井上 宏子[1]
[1]早稲田大学・理工学部

P1-106 カテプシンZの分子生物学的解析
Molecular analyses for cathepsin Z
水野 龍義[1,2]、米田 託成[1,2]、遠山正彌[1,2]
[1]大阪大院・医・ポストゲノム疾患解析（第二解剖）、[2]21世紀COE program

Sep 21 (Tue) Poster Session

Room P

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an even number 15:00～16:00

P1-107 mRNA結合蛋白であるHuDは、PC12細胞においてCARM1によってメチル化されており、NGFによる神経様細胞分化時に脱メチル化される
HuD, a mRNA binding protein, is methylated by CARM1 in PC12cells and it is demethylated during neuronal differentiation by NGF.
藤原 達司[1]、森 泰丈[1]、宮田 信吾[1]、池中 建介[1]、遠山 正彌[1]
[1]大阪大院・医・ポストゲノム疾患解析学講座

P1-108 マウスの発生過程における、protein arginine N-methyltransferase3 (PRMT3) の局在、及び相互作用するタンパク質の探索の局在
A localization of protein arginine N-methyltransferase 3 (PRMT3) and its interacting proteins in the mouse development.
池中 建介[1,2]、森 泰丈[1,2]、宮田 信吾[1,2]、藤原 達司[1,2]、遠山 正彌[1,2]
[1]大阪大院・医・ポストゲノム疾患解析学、[2]21世紀COE program

P1-109 ギセリン遺伝子発現に至る情報伝達経路の解析
Signalling pathways for gicerin gene experession
小浜 恵子[1]、三木 直正[1]、平 英一[1]
[1]大阪大院・医・情報薬理

P1-110 ストレス刺激に対するニコチンの作用：*in vitro*実験
Effects of nicotine on stress stimulation: *in vitro* experiments
新郷 明子[1]、鬼頭 昭三[1]
[1]兵庫大・健康科学部・脳科研

P1-111 ヒポカルシンによるmixed lineage kinase (MLK) 3活性の制御
Regulation of mixed lineage kinase (MLK) 3 activity by hippocalcin
小林 正明[1]、谷 真利子[1]、高松 研[1]
[1]東邦大・医・第2生理

P1-112 顎下腺導管系における交感神経アドレナリン受容体および細胞内情報伝達系分子
Sympathetic adrenoreceptors and intracellular signal transduction molecules in the duct system of the submandibular gland
森田 泰博[1]、湯浅 繁一[1]
[1]香川県立保健医療大学

P1-113 ラット脳内におけるイオタ型ジアシルグリセロールキナーゼの遺伝子クローニング
Cloning and characterization of diacylglycerol kinase ι splice variants in rat brain
後藤 薫[1]、伊藤 吏[1]、八月朔日泰和[1]、斎野一齋藤幸子[1]、近藤 尚武[2]
[1]山形大・医・組織細胞生物、[2]東北大院・医・細胞組織

P1-114 ラット脳内におけるベータ型ジアシルグリセロールキナーゼの局在について
Localization of diacylglycerol kinase β in the brain
八月朔日泰和[1]、齋藤 尚亮[2]、近藤 尚武[3]、後藤 薫[1]
[1]山形大・医・組織細胞生物学、[2]神戸大学・バイオシグナル研究センター、[3]東北大院・医・細胞組織

P1-115 マウス脳におけるPurα結合蛋白の性質
Characterization and partial purification of novel Purα binding proteins in mouse brain
曽　玲暉[1]、郭　哲輝[1]、三木　直正[1]
[1]大阪大院・医・第一薬理

P1-116 新規グアニンヌクレオチド交換因子RhoGEF10の性状解析
Characterization of Rho guanine-nucleotide exchange factor implicated in peripheral nervous disease
松本　浩[1]、大岡　嘉治[2]、稲垣　忍[2]
[1]大阪大院・歯・第一口外、[2]大阪大院・医・保健

P1-117 マウス線条体におけるFoxp1遺伝子の発現
Expression of Foxp1 in the mouse striatum
森川　吉博[1]、田村　志宣[1]、久岡　朋子[1]、仙波恵美子[1]
[1]和歌山医大・医・第二解剖

P1-118 *In vitro* 虚血性神経細胞死に対するprotein kinase A活性化の関与
Protein kinase A contributes to the irreversible depolarization following *in vitro* ischemia in rat hippocampal CA1 neurons.
田中永一郎[1]、東　英穂[1]
[1]久留米大学・医・生理学第1講座

P1-119 細胞外ATPを介する表皮－知覚神経連関
Skin-to-sensory neurons interaction via ATP
小泉　修一[1]、藤下加代子[2]、井上かおり[3]、津田　誠[2]、井上　和秀[2,4]
[1]国立衛研・薬理、[2]国立衛研・代謝生化学、[3]資生堂リサーチセンター、[4]九州大院・薬・分子制御

P1-120 *Xenopus* oocyte follicular cellのアデノシンで発生するK^+電流応答に対するインスリンの作用
Facilitatory effect of insulin on adenosine-induced K^+ current response in the follicular cells of *Xenopus* oocyte
渡辺　修二[1]、木村　眞吾[1]、川崎　敏[1]、藤田　玲子[2]、平野　浩子[2]、松本光比古[3]、佐々木和彦[1]
[1]岩手医大・医・第一生理、[2]岩手医大・教養・化学、[3]弘前大・医・保健

P1-121 FGFシグナルによる神経突起形成におけるRnd1の機能解析
Rnd1 directly binds to FRS2β and provides control mechanism for FGF-induced neurite outgrowth
原田　周[1]、加藤　裕教[1]、根岸　学[1]
[1]京都大院・生命・生体システム

P1-122 G protein-coupled receptor kinase 4 (GRK4) による$GABA_B$受容体脱感作の作用機序
Desensitization by GRK4 of the $GABA_B$ receptor-mediated signaling.
上園　保仁[1]、金出　政人[1,2]、松本　正順[2]、北條美能留[2]、貝原　宗重[1]、澄川　耕二[2]、谷山紘太郎[1]
[1]長崎大院・医歯薬総合・病態解析制御・医薬理、[2]長崎大院・医歯薬総合・病態解析制御・麻酔科学

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軸索輸送、細胞運動、細胞骨格
Axonal Transport, Cell Motility and Cytoskeleton

P1-123 レセプター型チロシンキナーゼ様分子RYKの発生期大脳における機能解析
Receptor tyrosine kinase-related protein RYK regulates cell migration during cortical development
神鳥 和代[1]、田仲 真弓[1]、高坂 新一[1]
[1]国立精神神経センター・神経研・代謝

P1-124 大脳皮質GABA作動性ニューロンの吻尾軸方向への移動
Tangential migration of cortical GABAergic neurons along rostrocaudal axis
田中 大介[1]、柳川右千夫[2]、小幡 邦彦[3]、村上富士夫[1]
[1]大阪大院・生命機能・脳神経工学、[2]群馬大院・医、[3]理研・BSI

P1-125 Rac活性化因子であるP-Rex1の神経系発生過程での発現、及び細胞移動に於ける役割
P-Rex1, a Rac specific activator - its expression during brain development and involvement in cell migration
吉澤 匡人[1,2]、川内 健史[1,2]、曽根 雅紀[1]、鍋島 陽一[1]、星野 幹雄[1,2]
[1]京都大院・医・腫瘍生物、[2]科学技術振興機構 さきがけ

P1-126 神経特異的K^+-Cl^-輸送体KCC2の異所性発現は発達中の大脳皮質の神経移動に影響しない
Ectopic expression of neuron-specific K^+-Cl^- cotransporter, KCC2, does not affect neuronal migration in developing cerebral cortex
天野 賢[1]、井上 浩一[1]、山田 順子[2]、古川 智範[1]、福田 敦夫[1]
[1]浜松医科大学・医・生理第一、[2]静岡大・院・電子科研

P1-127 海馬神経細胞におけるドレブリンAとPSD95の局在
Immunocytochemical analysis of Drebrin A and PSD-95 in cultured hippocampal neurons
花村 健次[1]、水井 利幸[1]、白尾 智明[1]
[1]群馬大院・医・高次細胞機能

P1-128 ドレブリンA過剰発現によるFアクチンとPSD-95の樹状突起フィロポディアへの集積
Drebrin A accumulates F-actin and PSD-95 into dendritic filopodia of immature hippocampal neurons
水井 利幸[1]、高橋 秀人[1]、白尾 智明[1]
[1]群馬大院・医・高次細胞機能学

P1-129 ドレブリン結合タンパク質DBP1の樹状突起スパインでの局在はc末端領域の配列に依存する
Localization of drebrin-binding protein 1 in dendritic spine depends on its C-terminal region
山崎 博幸[1]、水井 利幸[1]、白尾 智明[1]
[1]群馬大院・医・高次細胞機能

P1-130 キネクチンの膜結合部位の同定
Identification of membrane binding domain of kinectin
豊島 至[1]、菅原 正伯[1]、大川 聡[1]
[1]秋田大・医・第1内科

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an even number 15:00～16:00

P1-131 キネシンによるCRMP-2/チューブリン複合体の軸索輸送機構の解析
Tubulin and CRMP-2 complex is transported via conventional kinesin
木村 俊秀[1]、有村奈利子[1]、貝淵 弘三[1]
[1]名古屋大院・医・神経情報薬理

P1-132 神経細胞内のポストゴルジの膜小器官は微小管によって軸索の方向をしる
Microtubules provide directional cues for polarized axonal transport through interaction with kinesin motor head
中田 隆夫[1]、廣川 信隆[1]
[1]東大院 医 細胞生物解剖

P1-133 正常型プリオン蛋白質の細胞内輸送機構の解析
Anterograde and Retrograde Intracellular Trafficking of Fluorescent Cellular Prion Protein
渡邊 光太[1,2]、八谷 如美[1,2]、山田真紀子[1,2]、逆瀬川裕二[1,2]、金子 清俊[1,2]
[1]国立精神神経センター 神経研究所 疾病研究第7部、[2]（独）科学技術振興機構

P1-134 進化的に保存されているセリン/スレオニンキナーゼであるUNC51は、神経の発生および軸索輸送においてTrio、Racとともに協調的にはたらいている。
The Conserved Ser/Thr Kinase UNC51 Functions with Trio and Rac to Regulate Neural Development and Axonal Transport in *Drosophila*.
望月 洋明[1]、戸田 浩史[1]、鈴木えみ子[2]、浜 千尋[3]、友田 利文[4]、古久保一徳永克男[1]
[1]筑波大院・生命環境・構造生物、[2]国立遺伝学研究所・構造遺伝学研究センター、[3]理化学研究所・発生再生科学総合研究センター、[4]シティオブホープ・ベックマン研究所

P1-135 微小管切断蛋白、カタニンの軸索伸長特異的な役割
Axon-elongation specific role of microtubule-severing protein, katanin, in cultured neurons
林 謙介[1,2]、平井 里香[2]、西郷 薫[3]、程 久美子[3]
[1]上智大・生命研、[2]群馬大・生体調節研、[3]東大院・理・生化

P1-136 平面内細胞極性関連遺伝子群の中枢神経系における発現解析
The planar cell polarity (PCP) -related genes are expressed in the CNS.
木田 泰之[1]、小椋 利彦[1]
[1]東北大・加齢研・神経機能

P1-137 MAPsリン酸化による微小管-ニューロフィラメント間の動的相互作用の調節：*in vitro*輸送系を用いた解析
Regulation of neurofilament motility along microtubules by phosphorylated and dephosphorylated MAPs
倉知 正[1]、Jean-Francois Leterrier[2]、小宮 義璋[1]、田代 朋子[3]
[1]群馬大院・医・分子細胞機能学、[2]UMR 6558 CNRS, LBSC, Univ. Poitiers, Poitiers, France、[3]青山学院大・理工・化学生命科学

グリア、ミエリン
Glia and Myelin

P1-138 脱随モデルマウスにおける脳内遺伝子変化の解析
DNA microarray analysis of gene expression during demyelination
馬 堅妹[1]、田中 謙二[1]、山田 元[1]、松本 路生[1]、竹林 浩秀[1]、池中 一浴[1]
[1]自然科学研究機構・生理研・分子神経生理

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P1-139 カプリゾンによる脱髄の分子メカニズムについて
FcRγ/Fyn participate in the mouse brain during cuprizone-induced demyelination and remyelination
清和 千佳[1]、田中嘉代子[2]、中原 仁[3]、阿相 皓晃[1]
[1]東京都老人総合研究所・グリア細胞研究グループ、[2]埼玉医科大学・中央研究施設、[3]慶応義塾大学・医学部

P1-140 ラジアルグリア細胞とアストロサイト、脳室上衣細胞はalpha-actinin-1を発現する
Radial glial cells, astrocyte and ependymal cells express alpha-actinin-1
小川 泰弘[1]、竹林 浩秀[1]、Mathiew Rasband[2]、岩崎 靖乃[1]、Jim Trimmer[3]、池中 一裕[1]
[1]自然科学研究機構 生理学研究所 分子神経生理研究部門、[2]Connecticut Health Center Univ. connecticut. USA、[3]California Univ, Davis. USA

P1-141 中枢神経軸索ランビエ絞輪における脳特異的リンクプロテイン1、Bral1/Hapln2の機能解析
Extracellular matricies at the node of Ranvier in the central nervous system.
別宮 洋子[1]、二宮 善文[1]、大橋 俊孝[1]
[1]岡山大院・医歯・分子医化学

P1-142 培養ミクログリアに対するリポ蛋白分画の効果：HDLによる食作用の阻害
Effect of lipoproteins on rat cultured microglia: Inhibition of phagocytosis by high-density lipoprotein fraction
中村 洋一[1]、浅井 絵理[1]、本吉 晃子[1]、河南有希子[1]、森山 光章[1]
[1]大阪府立大院・農学生命・獣医・統合生理

P1-143 $P2X_7$受容体活性化ミクログリアの神経保護作用
Neuroprotective effect of TNF released from $P2X_7$ receptor-activated microglia
鈴木 智久[1]、松原 明代[1]、高坂 新一[2]、井上 和秀[3]、秀 和泉[1]、仲田 義啓[1]
[1]広島大院・医歯薬総合・薬効解析科学、[2]国立精神・神経セ・代謝、[3]国立医薬品食品研・代謝生化学

P1-144 Iba1-EGFPトランスジェニックマウスの作製；ミクログリアの可視化技術の開発
Generation of Iba1-EGFP transgenic mouse; Visualization of microglia
平澤 孝枝[1]、大澤 圭子[1]、今井 嘉紀[1,2]、内野 茂夫[1]、高坂 新一[1]
[1]国立精神神経セ・神経研・代謝、[2]愛媛大学・医・分子細胞生命科学

P1-145 ラット中枢神経系におけるオリゴデンドロサイト前駆細胞の発達
Development of oligodendroglial progenitor cells in the central nervous system of rats
鶴尾 吉宏[1]、上山 敬司[1]、伊藤 隆雄[1]、仲井 沙織[1]、阿相 皓晃[2]
[1]和歌山医大・医・第一講座、[2]東京都老人総合研究所・グリア細胞研究グループ

P1-146 オリゴデンドロサイト分化における$p21^{Cip1/WAF1}$の役割
Roles for $p21^{Cip1/WAF1}$ during oligodendrocyte differentiation
谷内 孝次[1]、田中 啓之[1]、遠山 正彌[1]
[1]大阪大院・医・ポストゲノム疾患解析

P1-147 ガレクチン1はアストロサイトの分化、および脳由来神経栄養因子産生を誘導する
Galectin-1 induces astrocyte differentiation, which leads to production of brain-derived neurotrophic factor
佐々木 翼[1]、平林 淳[2]、萬谷 博[1]、笠井 献一[3]、遠藤 玉夫[1]
[1]都老人研・糖蛋白質研究、[2]産総研・糖鎖工学研究センター ・糖鎖構造解析チーム、[3]帝京大・薬・生物化学

P1-148 ラジアルグリア/アストロサイト細胞系譜で特異的に発現するシスタチオニンβシンターゼの神経疾患への関与
Predominant expression of cystathionine β-synthase (CBS) in the radial glia/astrocyte cell lineage and its relationship to neurological disorders
榎戸 靖[1]、岩沢 和[1]、鈴木 恵里[1]、行方 和彦[1]、木村 英雄[1]
[1]国立精神・神経センター、神経研究所、遺伝子工学研究部

P1-149 培養大脳皮質アストロサイトにおけるcAMPおよびcGMPによる星状化をセロフェンド酸は促進する
Serofendic acid promotes stellation induced by cAMP and cGMP analogs of cultured cortical astrocyte
久米 利明[1]、伊藤 亮[1]、田口 良太[1]、香月 博志[1]、赤池 昭紀[1]
[1]京都大院・薬・薬品作用解析

P1-150 小脳アストロサイトにおける甲状腺ホルモン依存性GLASTの発現調節と水酸化PCBによる影響
Effects of hydroxy-polychlorinated biphenyls (OH-PCBs) on thyroid-hormone-dependent expression of GLAST, Bergmann glial specific glutamate transporter, in cerebellar astrocytes
木村－黒田純子[1,3]、永田 功[1,3]、黒田洋一郎[2,3]
[1]東京都神経科学総合研究所・脳構造研究部門、[2]東京都神経科学総合研究所、[3]科学技術振興機構、CREST

P1-151 エストロゲンによるアストロサイトのグルタミン酸取り込み阻害の作用メカニズム
The mechanisms underlying the inhibitory effect of estrogens on the L-glu uptake by astrocytes
佐藤 薫[1]、松木 則夫[2]、大野 泰雄[1]、中澤 憲一[1]
[1]国立衛研・薬理、[2]東京大院・薬・薬品作用

P1-152 培養アストロサイトにおけるグルタミントランスポーターの発現
Expression of glutamine transporter in cultured astrocytes
小椋 正人[1]、高野 桂[1]、中道 範隆[1]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物学

P1-153 ミクログリアとアストロサイトに対するポリアミン毒性
Polyamine toxicity in cultured rat brain microglia and astrocytes
高野 桂[1]、小椋 正人[1]、中村 洋一[2]、米田 幸雄[1]
[1]金沢大院・自然科学・薬物、[2]大阪府立大院・農学生命科学・統合生理

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P1-154 髄鞘形成におけるSema4Dの役割の検討
Sema 4D deficiency up-regulates myelination in mouse brain
谷口 佳孝[1,2]、松山 知弘[3]、芳川 浩男[3]、古郷 幹彦[2]、古山 達雄[4]、稲垣 忍[4]
[1]関西労災病院・口腔外科、[2]大阪大院 歯 口腔外科1、[3]兵庫医大 総合内科 神経・SCU科、[4]大阪大院 医 保健 神経生物

P1-155 ニワトリ視神経でのnetrin-1、Sema3Aおよびそのレセプターの発現パターンの解析
The expression pattern of netrin-1, Sema3A and their receptors in the chick optic nerve
渡辺 啓介[1,2]、池中 一裕[1,2]、小野 勝彦[1,2]
[1]総研大・生命科学、[2]生理研・分子神経生理

P1-156 PLPトランスジェニックマウスでの中枢神経伝導速度と不応期の検討
In vivo analysis of the conduction velocity of the central neural pathways in the PLP transgenic mice
田中 久貴[1]、池中 一裕[1]、伊佐 正[2]
[1]自然科学研究機構 生理学研究所 分子神経生理研究部門、[2]自然科学研究機構 生理学研究所 認知行動発達機構研究部門

P1-157 未分化アストロサイト、分化アストロサイトにおけるSNAREタンパク質の発現
The expression of SNARE proteins in undifferentiated or differentiated astrocytes
志賀 葉月[1]、伊藤 悦朗[1,2]
[1]北大・院理・生物科学、[2]北大・創成

P1-158 ミエリン由来軸索伸展阻害因子のlipid raftを介するシグナル伝達機構
Binding of soluble myelin-associated glycoprotein to specific gangliosides induces the association of p75 NTR to lipid rafts and signal transduction
藤谷 昌司[1,2]、川合 寛道[2]、山下 俊英[1]
[1]千葉大院・医・神経生物、[2]滋賀医大・医・神経内科

P1-159 ヒト神経系におけるContactin associated protein (Caspr) 4の分子間相互作用の解析
Distribution and molecular interaction of contactin associated protein (Caspr) 4 in human nervous system.
二川 俊隆[1]、大磯 茂[1]、武田 泰生[1]、山田 勝士[1]
[1]鹿児島大院・医歯学総合・薬物動態制御学分野

P1-160 トランスジェニックマウスを用いたL－セリン合成酵素Phgdh遺伝子の中枢神経系における発現制御機構の解析
Phgdh gene promoter directs transgene expression to neuroepithelial stem cells and radial glia *in vivo*
古屋 茂樹[1]、木下 雅美[1,2]、篠田 陽子[1]、境 和久[1]、吉田 一之[1,3]、三苫 純也[4]、端川 勉[1]、渡辺 雅彦[5]、町田 武生[2]、平林 義雄[1]
[1]理化学研究所・脳科学総合研究センター、[2]埼玉大学理学部生体制御学科、[3]宇都宮大学・ベンチャービジネスラボラトリー、[4]The Burnham Inst., La Jolla, USA、[5]北海道大学医学研究科 解剖発生

P1-161 大脳皮質－線条体切片培養系におけるATPγSによるMCP-1発現
ATPγS induces MCP-1 expression in organotypic cortico-striatal slice cultures
片山 貴博[1]、伊藤 美聖[1]、山崎 裕子[1]、神谷 明裕[1]、金子 周司[1]、佐藤 公道[1,2]、南 雅文[1]
[1]京都大院・薬・生体機能解析、[2]奈良先端科学技術大学院大学

P1-162 運動神経損傷によりミクログリアで誘導されるannexin IIIの発現
Annexin III expression is induced in microglia in response to motor nerve injury
小西 博之[1]、濤川 一彦[1]、木山 博資[1]
[1]大阪市大院・医・機能細胞形態学

P1-163 ショウジョウバエ成虫脳における神経細胞死と傷害に依存的なグリア細胞の増殖
Glial cell proliferation induced by the programmed cell death and neural injury in the *Drosophila* adult brain
加藤健太郎[1]、粟崎 健[1,2]、伊藤 啓[1,3]
[1]東大・分生研・高次構造、[2]さきがけ、科学技術振興機構、[3]バイオインフォマティクス、科学技術振興機構

P1-164 ノックアウトマウスを用いた損傷脳組織におけるアストロサイトの機能解析
Functional analysis of astocytes in lesion site using knockout mouse
池島一片岡宏子[1]、沈 勁松[2]、名竹 洋子[1]、斎藤 三郎[1]、湯浅 茂樹[3]
[1]慈恵医大・DNA研・分子免疫、[2]慈恵医大・DNA研・遺伝子治療、[3]国立精神神経センター・神経研究所・微細構造

P1-165 培養ラット大脳皮質ミクログリアのカチオンチャネルに対するGLP-1の作用
Effects of GLP-1 on cation channels in cultured microglias from the rat's cortex
岡 淳一郎[1]、谷光かほり[1]、伊藤 論史[1]、岩井 孝志[1]
[1]東京理科大・薬・薬理

P1-166 培養アストロサイトにおけるグルタミン酸受容体誘導性カルシウム振動に対するPKCの役割
PKC modulation of glutamate-induced astrocyte calcium oscillation
森田 光洋[1]、中根 晃[1]、工藤 佳久[1]
[1]東京薬科大・生命科学・生体高次

P1-167 薬物依存の研究（第396報）：Methamphetamine誘発astrocyte活性化におけるNMDA受容体およびPKCの関与
Studies on drug dependence (Rept. 396) : Involvement of NMDA receptor associated with PKC activation in methamphetamine-activated astrocyte
宮竹真由美[1]、成田 年[1]、鈴木 勉[1]
[1]星薬大・薬品毒性

P1-168 ヒトおよびラットのGMFBとGMFGの組織分布および年齢別血清濃度測定
Tissue distribution and age-related changes of human and rat GMFB and GMFG
青山 峰芳[1]、稲垣 雅昭[1,2]、祖父江和哉[2]、山本 直樹[1]、森島 徹朗[1,2]、森山 昭彦[3]、勝屋 弘忠[2]、浅井 清文[1]
[1]名市大院・医・分子神経生物学、[2]名市大院・医・危機管理医学、[3]名古屋市立大学大学院システム自然科学研究科

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P1-169 大脳皮質凍結損傷後のGMFBの発現誘導
Expression of GMFB after cortical cryogenic injury
堀田 直和[1]、青山 峰芳[1]、稲垣 雅昭[1,2]、祖父江和哉[2]、石原 正司[1]、多田 豊曠[3]、浅井 清文[1]
[1]名市大院・医・分子神経生物学、[2]名市大院・医・危機管理医学、[3]名市大・看護学部

P1-170 硫酸化グルクロン酸含有糖鎖に対する種々のグルクロニダーゼの基質特異性：HNK-1糖鎖構造決定のツールとして
Specificities of various glucuronidases to the oligosaccharides containing sulfated glucuronic acids
北村 邦男[1]、藤牧 香代[1]、熊谷 恵[1]、野村 正彦[1]
[1]埼玉医大・生理学

P1-171 In vitroでオリゴデンドロサイトとアストロサイトを認識するモノクローナル抗体12F7
The monoclonal antibody, 12F7, recognizes antigens in both oligodendrocytes and astrocytes *in vitro*
吉村 和法[1,2]、亀谷富由樹[3]、山室 裕[4]、藤牧 香代[1]、田中嘉代子[5]、桜井 洋子[2]、穐田 真澄[5]、野村 正彦[1]、阿相 皓晃[2]
[1]埼玉医科大学・医・生理学、[2]東京都老人総合研究所・グリア細胞研究グループ、[3]東京都精神医学総合研究所・分子神経生物学、[4]日本大学・生物資源学部・動物資源科学科、[5]埼玉医科大学・中央研究施設・形態部門

P1-172 チャネル局在化に対するシュワン細胞微絨毛の役割
The role of Schwann cell microvilli on the axonal organization
星 登美子[1,2]、山口 宜秀[2]、馬場 広子[2]
[1]東京薬科大学・生命科学・生体高次機能、[2]東京薬科大学・薬・機能形態

P1-173 ウズラの網膜と視蓋の発達過程におけるQH1陽性細胞の分布
The distribution of QH1-positive cells in the developing quail retina and optic tectum
安田ゐう子[1]、藤田 晢也[1]
[1]（財）ルイ・パストゥール医学研究センター・基礎

P1-174 パラノーダルジャンクション形成不全に伴う脊髄構成タンパク質の変化
Changes of protein composition in spinal cord by the defect of paranodal junction
鈴木 彩佳[1]、山口 宜秀[1]、馬場 広子[1]
[1]東京薬科大・薬・機能形態

P1-175 コネキシンチャネルを介したカルシウム流入によるミクログリアの活性化
Connexin hemichannels regulate microglial activation
杉山 崇[1]、池田 真行[1,2]、吉岡 亨[1]
[1]早稲田大・先端バイオ研、[2]大阪バイオサイエンス・分子行動生物学部門

P1-176 軸索－グリア間相互作用の異常に伴う神経細胞の遺伝子発現変化
Changes of neuronal gene expression by disruption of axo-glial communication
梶ヶ谷仁志[1]、馬場 広子[1]
[1]東京薬科大・薬・機能形態

P1-177 断眠によるリポカリン型プロスタグランジンD合成酵素のオリゴデンドログリアでの遺伝子発現の増加
Upregulation of lipocalin-type prostaglandin D synthase in oligodendrocytes during sleep deptivation
江口 直美[1]、坂田 三恵[1]、星川有美子[1]、松本 重子[1]、永田奈々恵[1]、裏出 良博[1]、早石 修[1]
[1]財団法人 大阪バイオサイエンス研究所

P1-178 シャーファー側枝入力によってラット海馬CA1アストログリア細胞に誘起される膜電流の解析
Membrane currents induced by Schaffer collateral inputs in rat hippocampal CA1 astrocytes
鈴木 崇之[1]、真仁田 聡[1]、工藤 佳久[1]、宮川 博義[1]
[1]東京薬科大学・生体高次機能学

P1-179 Myelinogenesis of the optic nerve in Neonatal diabetic rat model
Hamdino M Attia[1]、H Hida[1]、H Shimizu[1]、H Nishino[1]
[1]Dept. of Neuro-physiol. & Brain Sci., Nagoya City Univ. Grad. Sch. Med. Sci., Nagoya

P1-180 マイクログリアは神経外胚葉形質を持つ細胞に転換しうる
Microglia transform into cells with neuroectodermal phenotypes
横山 彰子[1]、今井 嘉紀[1]、田中 潤也[1]
[1]愛媛大学・医・形態系専攻

血液脳関門
Blood-Brain Barrier

P1-181 Tie2-GFPトランスジェニックマウスを用いた高純度脳毛細血管内皮細胞単離
Fluorescence activated purification of brain capillary endothelial cells from Tie2-EGFP transgenic mouse
寺崎 哲也[1,2,3]、大倉 直人[1]、堀 里子[1,2,3]、大槻 純男[1,2,3]
[1]東北大院・薬・薬物送達、[2]東北大・未来研、[3]SORST・科技団

P1-182 血液脳関門を介した24S-hydroxycholesterolの排出機構解析
Efflux Transport of 24S-hydroxycholesterol across the blood brain barrier
松田 明大[1]、手塚 和宏[1]、堀 里子[1,2,3]、大槻 純男[1,2,3]、寺崎 哲也[1,2,3]
[1]東北大院・薬・薬物送達、[2]東北大・未来研、[3]SORST

P1-183 ラットにおける冬眠特異的蛋白質の脳内輸送
Transport of hibernation-specific protein from blood into CSF in rats
大西 裕子[1]、安武かおり[1]、近藤 宣昭[1]
[1]三菱化学・生命科学研究所・冬眠制御

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運動
Motor System

P1-184 障害物、傾斜トレッドミル上二足歩行運動課題の実行により賦活される脳領域
Exploration of brain activity during bipedal walking on perturbed and inclined treadmill belt: PET study
森　大志[1,2]、中溝　克巳[3]、塚田　秀夫[4]、森　茂美[2]
[1]山口大学・農・獣医生理、[2]生理学研究所・生体システム研究部門、[3]近畿大学・医・生理、[4]浜松ホトニクス（株）中央研究所

P1-185 運動遂行想像課題中の中心前回脳活動
Brain activity during motor execution and imagery in the human precenral gyrus
花川　隆[1,2]、Hallett Mark[2]
[1]京都大学院・医・高次脳機能センター、[2]NIH, Bethesda, USA

P1-186 マーモセット大脳皮質運動関連領野の電気刺激による同定とその入力の解析
Intracortical microstimulation mapping and input organization of the primary motor and premotor cortical areas of the marmoset
徳野　博信[1]、知見　聡美[1,2]
[1]東京都神経科学総合研究所・脳構造、[2]学振・科技特

P1-187 サル一次運動野の神経活動から上肢運動を再現する
Direct estimation of arm movements from neuronal activities in the primary motor area of monkeys
広瀬　秀顕[1,2]、小池　康晴[3]、大場　健嗣[1]、筧　慎治[1]、飯島　敏夫[1]
[1]東北大学院・生命科学研究科、[2]科学技術振興機構、[3]東京工業大学・精密工学研究所

P1-188 神経変性疾患における協調運動障害の特徴
Characteristics of limb coordination in degenerative neurological diseases
松尾　善美[1]、淺井　義之[2,3]、野村　泰伸[2]、佐藤　俊輔[2]、米田　稔彦[4]、井上　悟[1]、水庫　功[5]、阿部　和夫[6]
[1]大阪大学医学部附属病院・リハビリテーション部、[2]大阪大学大学院基礎工学研究科機能創成専攻生物工学講座、[3]Fandazione I.S.I. Neuroheuristic Group、[4]神戸大学大学院医学系研究科保健学専攻、[5]三菱電機エンジニアリング（株）、[6]大阪大学大学院医学系研究科神経機能医学講座神経内科学

P1-189 課題達成のためのインピーダンス制御
Impedance control for task optimization
大須理英子[1]、森重　健一[2]、上村　直樹[3]、岩崎　裕志[3]、宮本　弘之[2]、和田　安弘[3]、川人　光男[1]
[1]国際電気通信基礎技術研究所・脳情報研究所・認知神経科学研究室、[2]九州工業大学生命体工学研究科、[3]長岡科学技術大学

P1-190 感覚運動連関に対する注意の影響 －経皮的磁気刺激法による検討－
Effect of spatial attention on human sensorimotor integration studied by trancranial magnetic stimulation
Mamdouh Ali Kotb[1]、美馬　達哉[1]、植木　美乃[1]、Begum Tahamina[1]、福山　秀直[1]、長峯　隆[1]
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P1-191 ピンセットを用いたサル摂餌運動における眼球-頭-手の協調運動の解析
Eye-head-hand coordination in food taking movements using a forceps in monkeys
佐々木成人[1]、本郷　利憲[1]、平井　直樹[2]、内藤　公郷[1]、Ekaterina Petropavlovskaya[1]
[1]東京都神経科学総合研究所、[2]杏林大学、生理

P1-192 片足立位時の姿勢維持における足首関節の制御機構：
The role of ankle joint in the control of postural stability during upright standing on one foot
江　依法[1]、長崎　幸雄[2]、松浦　陽[1]、古田　善伯[3]、松岡　敏男[2]、木村　英紀[1]
[1]独立法人理化学研究所、生物制御システム研究チーム、[2]岐阜大学 医学部 スポーツ医・科学講座、[3]岐阜大学 教育学部 保健体育学講座

P1-193 被殻から一次運動野への多シナプス性入力様式-狂犬病ウイルスの逆行性経シナプス輸送を用いた解析
Somatotopic arrangement of multisynaptic inputs from putamen to primary motor cortex as visualized by retrograde transneuronal transport of rabies virus
高田　昌彦[1]、宮地　重弘[1]、陸　暁峰[2]、今西美知子[1]、伊東　由美[1]、南部　篤[3]
[1]東京都神経研・統合生理、[2]順天堂大学・医・第一生理、[3]生理学研究所・システム生理

P1-194 ラット大脳基底核直接路における体部位再現
Somatotopical organization of the cortico-striato-entopeduncular pathway of rats
知見　聡美[1,2]、徳野　博信[1]
[1]東京都神経研・脳構造、[2]学振・科技特

P1-195 除脳ウサギの歩行運動誘発野刺激により発現する歩行運動の特徴
Locomotor patterns evoked by stimulation of supraspinal locomotion-inducing sites in decerebrate rabbits
松山　清治[1]、小林　卓[1]、青木　藩[1]
[1]札幌医大・医・第二生理

P1-196 歩行観察による運動前野背側部の脳賦活 ～近赤外線光イメージング研究～
Dorsal Premotor Cortex Activation during Observation of Human Gait: A fNIRS Study
小野　剛[1]、宮井　一郎[1]、鈴木　三央[1]、小田　一郎[2]、小西　郁夫[2]、久保田　競[1]
[1]ボバース記念病院・神経リハビリテーション研究部、[2]島津製作所・基盤技術研究所

P1-197 共同運動の発現と制御に対する小脳切除の影響
Influences of cerebellar hemispherectomy on generation and control of compound movements
玄番　央恵[1]、中尾　和子[1]、松崎　竜一[1]
[1]関西医大・第二生理

P1-198 手首運動制御における小脳プルキンエ細胞活動の座標系
Coordinate frames of Purkinje cell activities for motor control
松井　航[1]、角田　吉昭[2]、飯島　敏夫[1]、Donna Hoffman[3]、Peter Strick[3]、
筧　慎治[1,2]
[1]東北大院・生命科学・脳情報処理、[2]科学技術振興機構、[3]Center for the Neural Basis of Cognition, University of Pittsburgh School of Medicine, USA

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P1-199 サル前頭前野から一次運動野への多シナプス性入力-狂犬病ウイルスの逆行性経シナプス輸送による可視化
Multisynaptic inputs from prefrontal cortex to primary motor cortex as visualized by retrograde transneuronal transport of rabies virus
宮地 重弘[1]、陸 暁峰[1,2]、今西美知子[1]、伊東 由美[1]、南部 篤[3]、高田 昌彦[1]
[1]東京都神経研・統合生理、[2]順天堂大学・医・第一生理、[3]生理学研究所・生体システム

P1-200 ラット2足歩行モデルと正常ラットのトレッドミル歩行における筋活動に関する研究
Differences of EMG activities of trunk and hindlimb muscles of bipedal-walking rat model (BRM) and normal rat during treadmill locomotion.
平野 真嗣[1]、漆川 美穂[1]、森 大志[1]、和田 直己[1]、森 茂美[2]
[1]山口大学・農・獣医生理、[2]生理研

P1-201 二足歩行能力を運動学習により獲得した成ラット脊髄運動細胞の形態学的考察
Morphological changes in spinal motorneurons after the acquisition of bipedal walking in adult rats
漆川 美穂[1]、宮田 浩文[2]、森 大志[1]、和田 直己[1]、森 茂美[3]
[1]山口大学・農・獣医生理、[2]山口大学・農・生物機能、[3]生理学研究所

P1-202 ネコのLevel, Upslope, Downslope トレッドミル歩行における体軸の運動学的、最長筋の筋電図学的研究
EMG activity of the longissimus muscle and kinematics of the spinal column during level, upslope and downslope treadmill walking in cats
和田 直己[1]、鈴木 郷[1]、神田 健郎[2]
[1]山口大学・農・獣医生理、[2]東京都老人総合研究所

P1-203 二足歩行能力を運動学習により獲得した成ラット筋骨格系の形態学的考察
Morphological changes in skeleto-muscular system after acquisition of bipedal walking in adult rats
鈴木 郷[1]、岩元わかな[1]、森 大志[1]、和田 直己[1]、森 茂美[2]
[1]山口大学・農・獣医生理、[2]生理学研究所

P1-204 groupIとII感覚入力の最長筋運動ニューロンにおける統合制御に関する研究
Integration of group I and II muscle afferents from various parts of the body on motoneurons innervating m.longissimus lumborum (Long).
加藤 規寛[1]、和田 直己[1]、神田 健郎[2]
[1]山口大学・農・獣医生理、[2]東京都老人総合研究所

P1-205 運動機能と脊髄運動ニューロン
Functionally essential neuronal population of spinal motor neurons
掛川 晃[1]、横内久美子[1]、森泉 哲次[1]
[1]信州大・医・人体構造

P1-206 筋緊張制御の脊髄内介在細胞神経機構
Functional organization of spinal interneuronal systems involved in the control of postural muscle tone
高草木 薫[1]、柏柳 誠[1]
[1]旭川医大・医・第二生理

P1-207 豊かな環境は線条体6水酸化ドーパミン注入後の運動機能障害を弱める
Environmental enrichment attenuates motor dysfunction following intrastriatal 6-hydroxydopamine (6-OHDA) lesion
浦川 将[1]、飛田 秀樹[1]、Cha-Gyun Jung[1]、西野 仁雄[1]
[1]名古屋市立大学院・医・脳神経生理

P1-208 大脳皮質脊髄投射ニューロンより発する線条体及び視床への軸索支配を解析する
Reconstruction of axon collaterals arising from single corticospinal neurons in the striatum and thalamus
古田 貴寛[1]、松田 和郎[1]、金子 武嗣[1]
[1]京都大院・医・高次脳形態学

P1-209 パーキンソン病における淡蒼球内代謝型グルタミン酸受容体の動作様式
Downregulation of Metabotrophic Glutamate Receptor 1α in Globus Pallidus of Parkinsonian Monkeys
橘 吉寿[1]、金田 勝幸[2,3,4]、南部 篤[1,3]、畑中 伸彦[1]、今西美知子[2]、喜多 均[4]、重本 隆一[3,5]、高田 昌彦[2,3]
[1]生理研・生体システム、[2]東京都神経研・統合生理、[3]CREST、[4]Dept. Anat. & NeuroBiol., Univ. of Tennessee, Memphis, TN, USA、[5]生理研・脳形態解析

P1-210 視床下核高頻度刺激が、パーキンソン病サルの淡蒼球ニューロンの活動に及ぼす影響
Pallidal activity during high frequency stimulation of the subthalamic nucleus in parkinsonian monkeys
南部 篤[1]、橘 吉寿[1]、金田 勝幸[2]、喜多 均[3]、高田 昌彦[2]
[1]生理研・生体システム、[2]東京都神経研・統合生理、[3]Dept of Anat and Neurobiol, Col of Med, Univ of Tennessee, Memphis, USA

P1-211 視床下核ニューロンは選択的な神経路を介してドーパミン依存性行動を協調する
Subthalamic neurons coordinate dopamine-mediated behavior through differential neural pathways
小林 和人[1]、八十島安伸[1]、小山 純正[2]、香山 雪彦[2]
[1]福島医大・医・生体機能、[2]福島医大・医・第二生理

P1-212 腹側被蓋野におけるドーパミン神経細胞の投射経路
Axonal arborization of ventral tegmental area dopaminergic neurons: single-cell study
松田 和郎[1]、古田 貴寛[1]、金子 武嗣[1]
[1]京都大院・医・高次脳形態

P1-213 GAD67-GFPノックインマウスを用いた上丘中間層GABA作動性ニューロンの電気生理学的及び形態学的特性
Morphological and electrophysiological properties of GABAergic neurons in the superior colliculus (SC) of GAD67-GFP knock-in mice
伊佐かおる[1]、Thongchai Sooksawate[1,2]、Mary Behan[3]、小幡 邦彦[4]、柳川右千夫[5,6]、伊佐 正[1]
[1]生理研・発達生理・認知行動発達、[2]Fac of Pharmac Sci, Chulalongkorn Univ, Thailand、[3]Dep of Comp Biosci, Univ of Wisconsin, USA、[4]理研・脳センター、[5]群馬大・院・遺伝発達行動学、[6]科学技術振興機構・CREST & SORST

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P1-214 Cholinergic Responses of Neurons in the Intermediate Layer of Rat Superior Colliculus

Thongchai Sooksawate[1]、Tadashi Isa[2]
[1]Dep. Physiol, Fac. Pharmaceutical Sc., Chulalongkorn University, Bangkok, Thailand、[2]Dep. Dev. Physiol, Natl. Inst. Physiol. Sc., Japan

P1-215 ラット黒質網様部の刺激は顎筋、舌筋および顔面筋の活動を誘発する
Stimulation of the rat substantia nigra pars reticulata induces jaw, tongue and facial muscle activity

天野仁一朗[1]、村田　貴俊[1]、河岸　重則[1]、吉野　賢一[1]
[1]九州歯科大・口腔科学

P1-216 閉口筋運動ニューロンと興奮性シナプス結合を形成する網様体ニューロンの同定
Reticular neurons located caudal to the trigeminal motor nucleus form excitatory synaptic connections with jaw-closing motoneurons.

高松　純一[1]、井上　富雄[2]、時田 賢一[2]、新谷　明幸[1]、古屋　良一[1]、川和　忠治[1]
[1]昭和大学・歯学・補綴学、[2]昭和大学口腔生理学教室

P1-217 顎運動に関わる多シナプス性神経回路の同定
Organization of multisynaptic pathways innervating the jaw-closing and jaw-opening muscles in the rat: a transneuronal tracing study using rabies virus.

畑中　伸彦[1]、宮地　重弘[2]、橘　吉寿[1]、井上　智[3]、南部　篤[1]、高田　昌彦[2]
[1]自然科学研究機構・生理学研究所・生体システム研究部門、[2]東京都神経研 統合生理、[3]国立感染研

P1-218 ラット延髄腹側呼吸性ニューロン群および上部頸髄吸息ニューロンへの大脳性支配
Cerebral control of the medullary ventral respiratory neuron group and upper cervical inspiratory neurons in the rat

藤戸　裕[1]、Ying Cao[1]、青木　藩[1]
[1]札幌医大・医・第二生理

P1-219 マウスの呼吸パターン形成におけるヒスタミンH1受容体の役割
Contribution of histamine H1 receptors to the breathing pattern formation in mice

岩瀬みち子[1]、宮本　謙一[1]、本間　生夫[1]
[1]昭和大・医・第二生理

P1-220 正常ラットの哺乳量の測定
Measurement of sucking amounts in developing rats

福島菜奈恵[1]、横内久美子[1]、森泉　哲次[1]
[1]信州大・医・人体構造

P1-221 舌下神経と哺乳量
Hypoglossal nerve resection and sucking capability

横内久美子[1]、藤田　研也[1]、福島菜奈恵[1]、森泉　哲次[1]
[1]信州大・医・人体構造

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P1-222 舌下神経の生存・成長への影響
Differential effects of hypoglossal nerve resections on the survival of developing rats
福山 哲広[1]、小池 健一[1]、横内久美子[2]、森泉 哲次[2]
[1]信州大・医・小児科、[2]信州大・医・人体構造

P1-223 ネコ運動皮質のGABAニューロンによる舌運動制御
Cortical control of GABA neurons during cat tongue movements
平場 久雄[1]
[1]日本大学・歯学部・生理学教室

P1-224 脳出血モデルラットにおけるトレッドミル訓練の大脳皮質運動野の厚さに対する影響
The effect of treadmill running on motor cortex thickness after intracerebral hemorrhage in rats
加藤 尚子[1,2]、増田 匡[2]、飛田 秀樹[2]、石田 和人[1,2]、猪田 邦雄[1]、西野 仁雄[2]
[1]名古屋大院・医・リハビリテーション、[2]名古屋市立大・院・医・脳神経生理

P1-225 内包出血モデルラットにおける運動機能障害の原因としての軸索障害
Axonal damage causes motor dysfunction in internal capsule hemorrhage model rats
増田 匡[1]、飛田 秀樹[1]、熊崎 路子[1]、櫻井 輝美[1]、西野 仁雄[1]
[1]名古屋市立大学大学院・医学研究科・脳神経生理学

P1-226 ラットの空中立ち直り反射における神経回路：破壊実験
Lesion studies on neuronal substrates for rat air righting reflex
竹田 敏幸[1]、朝生 淳一[1]、山口 峻司[1]
[1]山形大院・理工・生体センシング機能工

P1-227 動的な系列運動生成の神経機序：高磁場MRIを用いて
Neural substrates for predicting a dynamic abstract motor sequence: a high-field fMRI study
平田 智秋[1]、堀江 亮太[1]、Allen Waggoner[1]、上野 賢一[1]、程 康[1]、田中 啓治[1]、谷 淳[1]
[1]理化学研究所 脳科学研究センター

P1-228 動作分析装置を用いた指の単一関節の最小移動距離
Minimal voluntary movement of a single finger joint analyzed by a motion analysis system
小林 宣泰[1]、八島美登里[2]、岡田 真優[3]
[1]金沢大・医・保・作業療法学専攻、[2]金沢社会保険病院、[3]穴水ヘルスケアサポートセンター

P1-229 視覚性単純反応時間課題中の背側運動前野に対する経頭蓋磁気刺激の影響
Effects of Transcranial Magnetic Stimulation to human dorsal premotor cortex during simple visual reaction time task
大谷めぐみ[1]、橋本 敏宏[1]、松村 道一[1]、内藤 栄一[1]
[1]京都大院・人間環境学・共生人間学

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P1-230 サル背側視床における遅延期間活動はサッカードのタイミングを予測する
Delay period activity in the central thalamus of monkeys predicts saccade timing
田中 真樹[1]
[1]北大院・医・認知行動

P1-231 GluR δ２欠損マウスにおける下オリーブ核破壊の反射性眼球運動への効果
Lesion of inferior olivary nuclei reduces abnormal dynamics of reflexive eye movements in a GluRδ2 knockout mouse
吉田 盛史[1,3]、加藤 明[1]、姫嶋由布子[1]、大槻 元[1,3]、船曳 和雄[2,3]、平野 丈夫[1,3]
[1]京都大院・理・生物物理、[2]京都大院・医・先端領域融合医学研究機構、[3]戦略的創造研究推進事業 科学技術振興機構

P1-232 小脳片葉領域は輻輳眼球運動に関与する
The cerebellar floccular region is involved in vergence eye movements
津布久 崇[1]、赤尾 鉄平[1]、McCrea Robert[2]、Kurkin Sergei[1]、福島 順子[1]、福島 菊郎[1]
[1]北海道大院・医・統合生理学・認知行動、[2]シカゴ大・医・生理学

P1-233 小脳半球皮質の微小刺激により誘発される眼球運動
Effects of microstimulation of monkey cerebellar hemispheric lobule VI/VII on eye movements.
北澤 宏理[1]、大木 雅文[1,2]、平松 敬人[3]、北村 泰子[4]、山田 仁三[4]、永雄 総一[1]
[1]理研BSI・運動学習制御、[2]東京大学医学部耳鼻咽喉科、[3]自治医科大学医学部脳神経外科、[4]東京医科大学第2解剖学教室

P1-234 輻輳運動のフィードバック制御及びプログラム制御に関するGABA作用
GABAergic contribution to feedback-controlled and programmed convergence eye movements
戸田 春男[1]、板東 武彦[1]
[1]新潟大院・医・統合生理

P1-235 前頭眼野におけるバーストニューロンの分類とその役割の検討
Classification of the bursting neurons and their functional roles in the frontal eye fields (FEF).
片井 聡[1,2]、猿渡 正則[1]、井上 雅仁[1]、三上 章允[1]
[1]京都大霊長類研究所・行動発現分野、[2]信州大・第三内科

P1-236 前頭眼野後部領域の滑動性眼球運動ニューロンにおける奥行き方向刺激に対する視覚応答について
Visual responses of pursuit neurons in the caudal frontal eye fields (FEF) to motion-in-depth stimuli
赤尾 鉄平[1]
[1]北海道大院・医・認知行動学

P1-237 過去の経験に基づく追跡眼球運動のオンラインゲイン制御
Online gain control for smooth pursuit based on the past experiences
田端 宏充[1,2]、三浦健一郎[1]、河野 憲二[1]
[1]京都大院・医・認知行動脳科学、[2]日本学術振興会

P1-238 ΔFosBのみ発現する *fosB*$^{d/d}$マウスとFosBとΔFosBともに欠損する *fosB*-nullマウスの行動解析
Behavioral analysis of *fosB*$^{d/d}$ mice which express only ΔFosB and *fosB*-null mice lacking both FosB and ΔFosB.
大西　克典[1]、大西一本田陽子[1]、山崎　勝久[1]、山口　浩雄[1]、梶谷　康介[1]、冨永　洋平[1]、作見　邦彦[1]、中別府雄作[1]
[1]九州大学・生医研・脳機能

P1-239 mGluR1bレスキューマウスの作製および解析
Generation and analysis of mGluR1b-rescue mice
大谷　善昭[1]、橋本　浩一[2]、深谷　昌弘[3]、中尾　和貴[4]、新石　健二[1]、渡辺　雅彦[3]、狩野　方伸[2]、饗場　篤[1]
[1]神戸大院・医・細胞生物学、[2]金沢大院・医・シナプス発達・機能学、[3]北大院・医・解剖発生学、[4]理研・発生再生総合研究セ

P1-240 GAD67遺伝子欠損マウス胎仔における頭部と舌の自発運動と神経発射
Development of spontaneous movement of the head and tongue in the mouse embryos lacking glutamate decarboxylase 67
常川　直子[1]、荒田　晶子[2]、小幡　邦彦[1]
[1]理研・脳センター・神経回路メカニズム、[2]理研・脳センター・記憶学習

P1-241 線虫の前進運動持続時間の調節に関与する部分神経回路の同定
Neurons Regulating the Duration of Forward Locomotion in *Caenorhabditis elegans*
若林　篤光[1]、長田　貴史[1]、佐々木聖夏[1]、新貝　鉚蔵[1]
[1]岩手大・工・福祉

P1-242 ゼブラフィッシュの逃避運動を発現する後脳の分節間相同ニューロン
Functional relationships of segmentally homologous hindbrain neurons in controlling escape behaviors of zebrafish
小橋　常彦[1]、小田　洋一[1]
[1]大阪大院・生命機能・脳神経工学

P1-243 2誘引物質の同時提示がC. エレガンスの化学走性に及ぼす効果
Effect of simultaneous presentation of two attractants on chemotactic response of *C. elegans*
及川　智宏[1]、松浦　哲也[2]、若林　篤光[2]、新貝鉚蔵[1,2]
[1]岩手大院・工・情報、[2]岩手大・工・福祉システム

P1-244 C.エレガンスの野生種とGABA変異種における加齢による移動運動の変化
Changes in phenotypes of locomotion with aging in wild-type and GABAergic mutants of *C. elegans*
林　琳[1]、星　勝徳[2]、小栗栖太郎[2]、若林　篤光[2]、青島　均[3]、新貝　鉚蔵[2]
[1]岩手大院・工・電子情報、[2]岩手大・工・福祉システム、[3]山口大・理

P1-245 アメフラシの食物嗜好性行動におけるLEタイプニューロンの機能
Function of the LE type neuron in Aplysia food preference behavior
衣川亜衣子[1]、長濱　辰文[2]
[1]神戸大院・自然科学・生命科学、[2]東邦大・薬・物理

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P1-246 パルス型弱発電魚の発電周波数は感覚入力により誘発・制御される
Sensory inputs modulate the frequency of electric organ discharges (EOD) in pulse-type weakly electric fish, Mormyrid.
菅原 美子[1]、大西 啓允[2]、配島 聖治[2]、桑名 俊一[1]、菅原 隆[3]
[1]帝京大・医・生理学、[2]芝浦工大・工学部・電子工学科、[3]埼玉医科大・生物

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P1-247 アテンションがバイオロジカルモーション知覚に与える影響：事象関連電位計測による研究
Attentional effect to the biological motion perception: An event-related potentials study
平井 真洋[1]、千住 淳[1]、福島 宏器[1]、開 一夫[1]
[1]東京大院・総合文化・広域科学

P1-248 追跡眼球運動中における視覚刺激の動きの知覚
Accuracy of object motion perception during smooth pursuit
三浦健一郎[1]、和田 佳郎[2]、田端 宏充[1,3]、瀧 正勝[1]、河野 憲二[1]
[1]京都大院・医・認知行動脳科学、[2]県立奈良医大、[3]日本学術振興会

P1-249 回転・並進運動による3次元構造に関する脳活動のfMRI解析
fMRI Analysis of Brain Activity on Three-Dimensional Structure from Rotational and Translational Motion
山本 哲也[1]、高橋 成子[2]、花川 隆[3]、浦山 慎一[3]、福山 秀直[3]、江島 義道[4]
[1]京都大学大学院人間・環境学研究科、[2]京都市立芸術大学、[3]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、[4]京都工芸繊維大学

P1-250 多義的運動視知覚刺激を用いた局所運動情報の能動的統合プロセスの解明
Active integration of local motion signals revealed by coherency among multiple multistable motion stimuli
井戸 喜英[1]、花沢 明俊[1]
[1]九工大・院生命体・脳情報

P1-251 方位マップは暗室飼育条件下でも形成されるのか?
Are orientation maps formed even under the dark rearing condition?
谷 利樹[1]、今村 一之[1]、Ribot Jerome[1]、田中 繁[1]
[1]理化学研究所・脳科学総合研究センター・視覚神経回路モデル研究チーム

P1-252 両眼受容野解析による面の傾き感受性
3-D surface orientation sensitivity in area 18 of the cat.
眞田 尚久[1]、大澤 五住[2]
[1]大阪大院・基礎工・生物工、[2]大阪大院・生命機能

P1-253 ネコ18野における複雑型細胞受容野構成の解析
Spatial pooling of subunits in complex receptive fields in the cat area 18
木村 塁[1]、岸本 知之[2]、五住 大澤[2]
[1]大阪大院、基礎工、[2]大阪大院、生命機能

P1-254 弁別訓練によるサル一次視覚野の方位表現への影響
Effect of discrimination training on orientation representation in V1 of a monkey.
脇田 真清[1]
[1]京都大・霊長研・行動神経

P1-255 ネコ視覚野における文脈依存的反応修飾のコントラスト依存性
Contrast-dependency of contextual modulation in the cat visual cortex
定金 理[1]、尾関 宏文[1]、内藤 智之[1]、赤崎 孝文[1]、佐藤 宏道[1]
[1]大阪大学・健康体育部

P1-256 一次視覚野と外側膝状体における受容野外刺激からの文脈依存的修飾効果の比較
Comparison of contextual modulation by the receptive field surround between V1 and LGN in the cat
内藤 智之[1]、定金 理[1]、赤崎 孝文[1]、佐藤 宏道[1]
[1]大阪大学健康体育部

P1-257 マカクザルMT野における図形選択的神経細胞活動
Shape selective activities recorded in the area MT of macaque monkeys
半田 高史[1,2]、片井 聡[1]、久能 玲子[1]、海野 俊平[1]、井上 雅仁[1]、三上 章允[1]
[1]京都大学・霊長類研究所・行動発現分野、[2]日本福祉大院・人間情報

P1-258 ヒト視覚皮質における色空間比較処理のfMRI解析
FMRI analysis of color contrast processing in the human visual cortex
近藤 あき[1]、中越明日香[2]、山本 洋紀[1]、田中 忠蔵[2]、梅田 雅宏[2]、江島 義道[3]
[1]京都大学・院・人間・環境学、[2]明治鍼灸大学、[3]京都工芸繊維大学

P1-259 サル下側頭皮質における色処理の局在
Specialization for color processing in the inferior temporal cortex of the monkey
安田 正治[1,2]、鯉田 孝和[2]、小松 英彦[2]
[1]総研大、[2]生理学研究所・感覚認知情報

P1-260 大脳皮質一次視覚野における色表現
Color selectivity and functional connectivity of macaque V1 neurons: simultaneous recording from adjacent cells
村上 浩文[1]、森本 広志[2]、田村 弘[1]、藤田 一郎[1]
[1]大阪大院・生命機能・認知脳科学、[2]大阪大院・基礎工・認知脳科学

P1-261 色の弁別反応時間における左右大脳半球の機能差
Hemispheric dominance in color discrimination using reaction time task
佐々木 仁[1]、西尾 啓[1]、森本 晶子[1]、青木 亮三[1]、福田 淳[1]
[1]大阪大院・医・情報生理

P1-262 色誘導が透明視条件下の色恒常性に果たす役割
Color induction contributes to color constancy in the perception of color transparency
青木 麗[1]、花沢 明俊[1]
[1]九工大・院生命体・脳情報

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P1-263 遮蔽物体の脳内表象に関するfMRI研究
The representation of occluded object: an fMRI Study
番　浩志[1]、花川　隆[2]、浦山　慎一[2]、福山　秀直[2]、中越明日香[3]、山本　洋紀[1]、田中　忠蔵[3]、梅田　雅宏[3]、江島　義道[4]
[1]京都大学大学院人間・環境学研究科、[2]京都大学大学院医学研究科高次脳機能センター、[3]明治鍼灸大学、[4]京都工芸繊維大学

P1-264 神経活動を抑制した視覚野における眼優位可塑性に競合メカニズムは関与するか?
Dose competitive mechanism contribute to the ocular dominance plasticity in the pharmachologically inhibited visual cortex?
春田　雅之[1]、畠　義郎[2]
[1]鳥取大 生命 神経生物学、[2]鳥取大院 医 生体高次機能

P1-265 両眼視差からの奥行き知覚に対する視野闘争の影響
Effect of binocular rivalry on the perception of depth from binocular disparity
沖石　梓[1]、花沢　明俊[1]
[1]九工大・院生命体・脳情報

P1-266 初期視覚野細胞における奥行き方向への運動選択性
Looming selectivity of cells in the early visual cortex
西本　伸志[1]、大澤　五住[2]
[1]大阪大院・基礎工・生物工学、[2]大阪大院・生命機能

P1-267 TE野神経細胞の2つの発火状態と状態遷移の細胞間同期
Synchronous transition between two activity states of neurons in area TE
内田　豪[1]、福田　光洋[1]、谷藤　学[1]
[1]理研・BSI

P1-268 初期視覚野のスパイク神経細胞モデル:単純型細胞の刺激線分長とコントラスト依存性
Spiking neural network model of the primary visual cortex: contrast and stimulus length dependence of neuronal responses
宮下　真信[1]、田中　繁[1]
[1]理研・BSI・視覚神経回路モデル

P1-269 光学的記録を行った一次視覚野におけるGABAニューロンのトポロジー解析
Topological analysis of GABAergic neurons in optically-recorded primary visual cortex
福田　孝一[1]、Ralf Aw Galuske [2]、Wolf Singer[2]、小坂　俊夫[1]
[1]九州大院・医・神経形態学、[2]Max Planck Institute for Brain Research

P1-270 サルTE野における内因性光信号イメージングと膜電位イメージングにより計測した神経活動の空間パターンの直接比較
Comparison of Functional Maps Revealed by *in vivo* Voltage-sensitive Dye Imaging and Optical Intrinsic Signal Imaging in Monkey IT Cortex
本間　良太[1]、谷藤　学[1]
[1]理化学研究所・脳科学総合研究センター

P1-271 サル視床枕核における内在性線維連絡
Intra-pulvinar long distance intrinsic connections
井村 幸介[1]
[1]理化学研究所・脳科学総合研究センター・脳皮質機能構造研究チーム

P1-272 向網膜投射の標的細胞
Retinal target cells of the isthmo-optic projection
内山 博之[1]、青木 謙二[1]、米澤 真司[1]、有村 ふく[1]、大野 裕史[1]
[1]鹿児島大・工・情報工学

P1-273 網膜神経細胞のグルタミン酸応答－膜電位感受性色素による記録
Glutamate responses of the retinal neurons recording with voltage sensitive dye
霜田 幸雄[1]
[1]東京女子医大・総研

P1-274 ロイコトリエンA_4水解酵素の視覚系および他の感覚神経系における分布の検討
Distribution of leukotriene A_4 hydrolase in the visual and other sensory systems
千葉 陽一[1]、島田 厚良[1]、佐藤 衛[1]、齋藤 優子[1]、河村 則子[1]、花井 敦子[1]、
細川 昌則[1]、清水 孝雄[2]
[1]愛知県心身障害者コロニー・発達障害研・病理、[2]東京大院・医・細胞情報

P1-275 トリアムシノロンの酸化ストレス負荷した網膜色素上皮への影響
The Effect of Triamcinolone Acetonide to Retinal Pigment Epithelial Cells by Oxidative Stress
松田 理[1,2,3]、五味 文[2]、大島 佑介[2]、田野 保雄[2]、遠山 正彌[1,3]
[1]大阪大院・医・ポストゲノム疾患解析学、[2]大阪大院・医・眼科学、[3]21世紀COEプログラム

聴覚、前庭感覚
Auditory and Vestibular System

P1-276 マウス蝸牛神経背側核におけるReelinの発現
Expression of reelin in the dorsal cochlear nucleus of the mouse
高岡 由佳[1]
[1]神戸大院・医・脳科学・神経発生

P1-277 ほ乳類の蝸牛神経核において、ムスカリン性受容体を活性化することでgranule cellsとGolgi cellsは過分極応答を示す
Activation of muscarinic receptor activation induces hyperpolarization in granule cells and Golgi cells of the mammalian cochlear nucleus
入江 智彦[1]、福井 巌[1]、大森 治紀[1]
[1]京都大院 医 神経生物学

P1-278 音刺激によって誘発されるラット脳幹のc-Fos 発現の分布について
c-Fos expression in rat brainstem evoked by aoustic stimuli
小崎 寛子[1]
[1]国立印刷局東京病院耳鼻咽喉科

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P1-279 ラット内側前庭神経核のグルタミン酸作動性、GABA作動性ニューロンの発火特性
Firing properties of glutamatergic and GABAergic neurons of the rat medial vestibular nucleus
齋藤 康彦[1]、高澤 知規[1]、小澤 瀞司[1]
[1]群馬大学院・医・神経生理

P1-280 下丘単一細胞または細胞集団による両耳間位相差表現の文脈依存性
Context Dependency on the Interaural-Phase-Difference Representation by Single Neurons and by Neural Populations in the Inferior Colliculus
古川 茂人[1]、牧 勝弘[1]、柏野 牧夫[1]、力丸 裕[2]
[1]NTT-CS研・人間部・人覚G、[2]同志社大・工・知識工

P1-281 上オリーブ核スライス培養におけるOlivocochlear Efferent線維の成長
OLIVOCOCHLEAR EFFERENT FIBERS IN CULTURE OF SLICES OF SUPERIOR OLIVE COMPLEX
福田 潤[1]、石嶺 久子[1]、武田湖太郎[1]、徳永 元秀[1]、松尾 洋孝[1]
[1]防衛医大・第一生理

P1-282 ネコ外転神経核へ直接投射する卵形嚢一次求心性神経と有毛細胞のシナプス結合
Innervation patterns of afferents in the utricular macula projecting directly to the abducens nucleus in the cat
今川美登里[1]、吉濱 勲[2]、佐々木光美[1]、佐藤 斎[1]、内野 善生[1]
[1]東京医大・第二生理、[2]東京医大・電顕室

P1-283 コイ科魚類の隆起前核を介した下行性聴覚神経路
Descending auditory pathways through the nucleus anterior tuberis in cyprinids
山本 直之[1]、伊藤 博信[1]
[1]日本医科大学・第二解剖

P1-284 大脳皮質介在ニューロンにおける興奮性の長期亢進
Long-Term Enhancement of Excitability in a Type of Neuron in the Cerebral Cortex
窪田 道典[1]、細川 浩[1]、堀川 順生[2]
[1]東京医科歯科大学・難治疾患研究所・情報医学研究部門、[2]豊橋技術科学大学・知識情報工学系

P1-285 経頭蓋的イメージングで捉えたマウス聴覚野機能マップの可塑性
Trans-cranial imaging of tonotopic map plasticity in the mouse auditory cortex
高橋 邦行[1]、工藤 雅治[1]、渋木 克栄[1]
[1]新潟大・脳研・システム脳生理

P1-286 ラットの二音弁別における聴覚野の役割
Roles of the auditory cortex in sound pitch discrimination in rats
小野健太郎[1]、工藤 雅治[1]、渋木 克栄[1]
[1]新潟大・脳研・システム脳生理

P1-287 一次および高次聴覚皮質における無音と雑音区間を含む純音に対する応答の光学的計測
Optical imaging of responses to gap- and noise-inserted tones in primary and belt fields of guinea pig auditory cortex
堀川 順生[1]、宮本 章弘[1]、窪田 道典[2]
[1]豊橋技科大・知識情報、[2]東京医歯大・難研

P1-288 モルモット一次聴覚皮質の聴覚誘導課題に対する神経応答
Neuronal Response to auditory-induction tasks in the primary auditory cortex of guinea pigs
高浦 加奈[1]、岡崎俊太郎[1]、武田湖太郎[1]、渋谷 和磨[2]、塚田 稔[2]、富田 豊[1]
[1]慶應義塾大学院・理工学研究科・基礎理工学、[2]玉川大学工学部

P1-289 スナネズミのコミュニケーション音声に関連した聴覚野の神経活動
Neural activities in gerbil auditory cortex responding to sounds related to communication
土居 節[1]、力丸 裕[1]
[1]同志社大院・工・知識・知覚認知機構研究室

P1-290 近赤外レーザーによる砂ネズミ聴覚野の神経活動の可逆的不活性化
Reversible inactivation of neural activities in gerbil auditory cortex by near-infrared laser.
川崎 敏生[1]、力丸 裕[1]
[1]同志社大院・工・知識・知覚認知機構研究室

P1-291 ジュウシマツHVC核の神経接続様式と自己歌への聴覚応答様式
Input output connections and neural activities in the HVC of Bengalese finches
奥村 哲[1]、岡ノ谷一夫[2,3,4]、谷 淳[1]
[1]理化学研究所・BSI・動的認知行動研究チーム、[2]千葉大学・文・認知行動行動科学、[3]さきがけ研究21、[4]理化学研究所・BSI・生物言語研究チーム

P1-292 ショウジョウバエ全聴覚感覚細胞の包括的な同定と分類
Comprehensive classification of Drosophila auditory sensory neurons
上川内あづさ[1,2]、伊藤 啓[1,2]
[1]東京大学・分子細胞生物学研究所・高次構造、[2]科学技術振興機構、バイオインフォマティクス

嗅覚、味覚、化学感覚
Olfaction, Taste and Chemical Senses

P1-293 ラット嗅球背側部の糸球における２種類の匂い分子の混合に対する応答
Responses of glomeruli to the mixture of two odorants in the rat dorsal OB
松本 英之[1]、森 憲作[1]
[1]東京大院・医・細胞分子生理学

P1-294 マウス嗅覚受容細胞におけるキラルなニオイ分子間の識別
Discrimination between chiral pairs in mouse olfactory receptor neurons
廣野 順三[1]、浜名 洋[1,2]、来住美和子[1]、江村 誠[3]、松田 洋幸[3]、所 一彦[3]、佐藤 孝明[1]
[1]産総研・セルエンジニアリング、[2]日本学術振興会、[3]高砂香料工業総合研究所

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P1-295 マウス副嗅球における鋤鼻ニューロンの受容パターン
Connection pattern of vomeronasal receptor afferents to an accessory olfactory bulb projection neuron
横須賀 誠[1]、佐原 資謹[2]、小牟田 縁[2]、中村 俊[2]、Richard Costanzo[3]、市川 眞澄[4]
[1]聖マリアンナ医大・医・解剖学、[2]国立精神神経センター、[3]Dept Physiol, Virginia Commonwealth Univ, Richmond, VA, USA.、[4]東京都神経学総合研究所

P1-296 ゼブラフィッシュにおける二つの異なる嗅覚神経回路の可視化
Two distinct neural pathways in the zebrafish olfactory system visualized by transgenesis with cell type-specific promoters
佐藤 友紀[1]、宮坂 信彦[1]、吉原 良浩[1]
[1]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

P1-297 梨状皮質におけるニオイ応答の電気生理学的メカニズム
Electrophysiological bases of odor-induced neural responses in the piriform cortex
石川 享宏[1]、佐藤 孝明[2]、筧 慎治[1]、飯島 敏夫[1]
[1]東北大院・生命科学・脳情報処理、[2]産業技術総合研究所 セルエンジニアリング研究部門

P1-298 モルモットの梨状葉皮質第IIaとIIb層の単一神経細胞の軸索投射に関する定量的解析
Quantitative analysis of axon collaterals of single neurons in layers IIa and IIb of the piriform cortex of the guinea pig
Junli Yang[1]、Qurash Afraz Ul[1]、陳 少雲[1]、石川 陽一[1]、角田 幸子[1]、岸 清[1]
[1]東邦大学・医・解剖学第一講座

P1-299 線虫*C.elegans*の嗅覚応答を調節するG_o-G_qシグナル経路の解析
The G_o and G_q signaling pathways modulate olfactory responses in *C.elegans*
松木 正尋[1]、國友 博文[1]、飯野 雄一[1]
[1]東京大・遺伝子実験施設

P1-300 鋤鼻系組織におけるprotein kinase Cの分布
Distribution of protein kinase C (PKC) in vomeronasal system
阿久津仁美[1]、山本 欣郎[1]、谷口 和之[1]
[1]岩手大学・農・獣医解剖

P1-301 ショウジョウバエ幼虫・成虫脳における*ems*遺伝子の解析
Expression and functions of the cephalic homeobox gene *empty spiracles* in the olfactory and gustatory centers of the *Drosophila* Brain
丸山 泰[1]、戸谷 洋子[1]、古久保一徳永克男[1]
[1]筑波大院・生命環境・構造生物

P1-302 ショウジョウバエ甘味受容機構におけるGsの関与
Drosophila sweet taste perception is mediated by Gs
上野 耕平[1]、古波津 創[2]、磯野 邦夫[2]、城所 良明[3]
[1]群馬大院・医・遺伝発達行動学、[2]東北大院・情科・情報生物、[3]群馬大学・生体調節研究所

P1-303 T1r3 KO, gustducin KO, Trpm5 KOマウス鼓索神経におけるグルマリンの甘味抑制
Gurmarin inhibition of sweet taste responses of the chorda tympani nerve in T1r3 KO, gustducin KO and Trpm5 KO mice
安松 啓子[1]、重村 憲徳[1]、吉田 竜介[1]
[1]九州大院・歯・口腔機能解析

P1-304 マウスdpa遺伝子座はグルマリン感受性甘味受容機構に関与している
The dpa locus may influence gurmarin sensitive receptor component in mice
重村 憲徳[1]、安松 啓子[1]、吉田 竜介[1]、二ノ宮裕三[1]
[1]九州大院・歯・口腔機能解析

P1-305 甘味物質に応答するマウス味細胞におけるgustducinとT1r3の発現
Expression of α-gustducin and T1r3 in taste receptor cells responding to sweet substances in mice
吉田 竜介[1]、實松 敬介[1]、重村 憲徳[1]、安松 啓子[1]、二ノ宮裕三[1]
[1]九大院・歯・口腔機能解析

P1-306 味覚認知と脳内モノアミン神経系との関連
Relationship between taste perception and the brain monoamine nervous system in rats.
山本千珠子[1]、山田 朋子[2]、乾 賢[1]、武田 弘志[2]、山本 隆[1]
[1]大阪大学・院人間科学・行動生理、[2]東京医科大学 薬理学講座

P1-307 覚醒サル大脳皮質味覚関連領野の味刺激に対する応答
Gustatory Neural Responses in Fronto-Opercular and Adjacent Cortices in Monkeys
平田 真一[1]、中村 民生[1]、井福 裕俊[1]、小川 尚[1]
[1]熊本大・医・知覚生理学

P1-308 線虫*C. elegans*における水溶性物質への化学走性に関する可塑性異常変異体の解析
Analysis of mutants abnormal in the plasticity of salt chemotaxis in *C. elegans*
池田 大祐[1]、國友 博文[1]、飯野 雄一[1]
[1]東京大学・遺伝子実験施設

P1-309 成体マウス嗅球において新規に同定された顆粒細胞の解析
A Novel Type of Granule Cells in the Olfactory Bulb of Adult Mouse
成塚 裕美[1]、森 憲作[1]、山口 正洋[1]
[1]東京大院・医・細胞分子生理

P1-310 ナメクジの中枢セロトニン神経系の組織学的および薬理学的解析
Histological and pharmacological analysis of the central serotonergic system in the slug *Limax valentianus*
白畑 孝明[1]、渡辺 恵[1]、桐野 豊[1]
[1]東京大院・薬・神経生物物理

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痛覚
Pain

P1-311 一次知覚ニューロンにおけるP38の活性化とperipheral sensitization
Activation of p38 MAPK in primary afferent neurons by noxious stimulation and its involvement in peripheral sensitization
水島 敏行[1]、小畑 浩一[1]、野口 光一[1]
[1]兵庫医大第2解剖学教室

P1-312 侵害受容性ニューロンに対する免疫抑制剤FK506の直接作用
Direct action of immunosuppressant FK506 on nociceptive sensory neurons
片野坂公明[1]、水村 和枝[1]、矢島 弘毅[1]、仙波恵美子[2]
[1]名古屋大・環境医学研究所、[2]和歌山県立医科大・第二解剖

P1-313 足蹠部と腰部における神経因性炎症の違い
Neurogenic inflammation in the plantar and hip skin areas
小山 なつ[1]、岩下 成人[1]、横田 敏勝[1]
[1]滋賀医科大学

P1-314 末梢グルタミン酸受容体に対するケタミン皮下注射の効果
Effects of ketamine on peripheral glutamate receptors
岩下 成人[1]、小山 なつ[1]
[1]滋賀医大・大学院・生理1

P1-315 炎症性痛覚過敏における脊髄内グリア型グルタミン酸トランスポーターGLT-1の役割
Involvement of spinal glial glutamate transporter GLT-1 in inflammatory hyperalgesia in rats
中川 貴之[1]、南 雅文[1]、金子 周司[1]、佐藤 公道[1,2]
[1]京都大院・薬・生体機能解析、[2]奈良先端科技大

P1-316 末梢神経損傷による接着因子L1-CAMの発現変化と疼痛発現への関与
Expression of L1-CAM in rat dorsal root ganglia and spinal cord following nerve injury
山中 博樹[1]、野口 光一[1]
[1]兵庫医科大学・解剖学第二講座

P1-317 後根神経節発生過程におけるTRPチャンネルの発現の変化について
Differential temporal expression of temperature-activated transient receptor potential channels in developing dorsal root ganglia
田村 志宣[1]、森川 吉博[1]、仙波恵美子[1]
[1]和歌山医大・医・第二解剖

P1-318 CCIモデルのDRGにおけるERKとp38の活性化
Differential activation of MAPK in injured and uninjured DRG neurons after CCI
小畑 浩一[1]、野口 光一[1]
[1]兵庫医大・第2解剖

P1-319 ラット脊髄後角膠様質神経細胞における興奮性・抑制性感覚入力と形態との相関
Correlation between morphology and sensory input of excitatory and inhibitory transmission in substantia gelatinosa neurons of adult rat spinal cord *in vitro*
八坂 敏一[1]、加藤 剛[1]、園畑 素樹[1]、玉江 昭裕[1]、古江 秀昌[1]、吉村 恵[1]
[1]九大院・医・統合生理

P1-320 ニワトリ腰髄後角におけるCyclooxygenase-2とGABAの共存細胞について
Cyclooxygenase-2- and GABA-immunoreactivities are often colocalized in cells in the lumbar dorsal horn of the chicken
川手 豊子[1]、坂本 宏史[2]、青木 航洋[3]、山田 明香[3]、熱海佐保子[1]
[1]山梨大院・医工・解剖2、[2]健康科学大・理学療法、[3]山梨大院・医・整形外科

P1-321 老齢ラット脊髄侵害受容ニューロン活動の慢性炎症による変調
Effect of chronic inflammation on dorsal horn nociceptive neurons in aged rats
北川 純一[1]、島 麻子[1]、小山 なつ[2]、岩田 幸一[1]
[1]日本大学・歯・生理、[2]滋賀医大・生理

P1-322 マウス脊髄後角深層における抑制性神経伝達に対するサブスタンスPの作用
The effects of substance P on phasic and tonic inhibitory action in deep dorsal horn neurons of mice spinal cord.
安宅 豊史[1,2]、Jianguo Gu[2]、Xing Hong[2]、馬場 洋[1]
[1]新潟大・医・麻酔、[2]Dep. of Oral Surgery, College of Dentistry, University of Florida, Gainesville, Florida

P1-323 顎顔面領域における炎症関連遺伝子の同定 －cDNAマイクロアレイによる解析－
Analysis of gene expression in rat Trigeminal ganglion neurons following maxillofacial inflammation using cDNA microarray
松橋 瞳[1]、国分 暁子[2]、姫野 勝仁[2]、田中 丈也[2]、増田 裕次[1]、岩田 幸一[3]、金銅 英二[1,2]
[1]松本歯科大学・総合歯科医学研究所・顎口腔機能制御学部門、[2]松本歯科大学大学院・顎口腔機能制御学講座、[3]日本大学・歯学部・生理学講座

P1-324 実験的モノニューロパシーラット（ベネットモデル）の末梢及び中枢神経系の電子顕微鏡解析
Ultrastructural analysis on the peripheral and the central nervous system of an experimental mononeuropathy rat (Bennett's model)
本多たかし[1]、池田 寛子[1]
[1]福島県立医大・医・解剖学第二

P1-325 ラット坐骨神経切断によって帯状回に出現するc-fos様蛋白産生細胞
C-Fos cells in the cingulate gyrus after cutting the sciatic nerve of rats
山田 仁三[1]、北村 泰子[1]
[1]東京医大・解剖学第二

P1-326 ラット前帯状回への痛覚上行路：狂犬病ウィルスを用いた逆行性越シナプストレーシング
Ascending pain pathways to the anterior cingulate cortex in rats: retrograde transneuronal tracing with rabies virus
岩田 幸一[1]、宮地 重弘[2]、今西美智子[2]、坪井 美行[1]、寺本 浩平[1]、高田 昌彦[2]
[1]日本大学・歯・生理学、[2]東京都立神経科学総合研究所

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P1-327 帯状回ニューロンは機械的侵害刺激の強さを反応の長さによりコードする
Duration of nociceptive responses encodes stimulus intensity in the medial prefrontal cortex
富田美穂子[1]、張　日輝[2]、片山　洋子[1]、川上　順子[1]
[1]東京女子医大・医・第一生理、[2]瀋陽体育学院・運動生理

P1-328 ホルマリン誘発侵害刺激に対する中枢神経系の反応の性差 －pCREB発現を神経活動の指標として－
Sex difference in the response of the CNS to formalin-induced nociceptive stimuli as revealed by the expression of phosphorylated cAMP response element binding protein (pCREB)
萩原　裕子[1]、舩橋　利也[1]、貴邑冨久子[1]
[1]横浜市大院・医・神経内分泌学

P1-329 反対側の脊髄後角へ投射する青斑核ニューロンの脊髄下行路
A possible spinal pathway of contralaterally projecting coeruleospinal neurons in the rat
鶴岡　正吉[1]、前田　昌子[1]、長澤　郁子[1]、井上　富雄[1]
[1]昭和大学・歯・口腔生理学

P1-330 慢性膵炎における求心性神経線維の膵内分枝増生
Intrapancreatic neuronal branching of afferent fibers in chronic pancreatitis
高御堂祥一郎[1]、片岡　洋祐[1]、崔　翼龍[1]、棚野　晃秀[1]、山田　久夫[1]
[1]関西医科大学 システム細胞科学

P1-331 ヒト三叉神経節感覚神経におけるカプサイシンレセプター（TRPV1）とアミロライド感受性陽イオンチャンネル（ASIC）の共存
Coexpression of vanilloid receptor subtype-1 and acid-sensing ion channel genes in the human trigeminal ganglion neurons
鵜川　眞也[1]、植田　高史[1]、山村　寿男[1]、長尾　正崇[2]、島田　昌一[1]
[1]名古屋市立大院・医・分子形態、[2]名古屋市立大院・医・法医科学

P1-332 ブラジキニンによる細胞内カルシウム上昇反応のプロスタグランジンEP3受容体を介した増強
Prostaglandin EP3 receptor-mediated augmentation of bradykinin-induced increase in intracellular calcium
小崎　康子[1]、神部　福司[2]、妹尾　久雄[2]、水村　和枝[1]
[1]名古屋大学・環医研・神経性調節、[2]名古屋大学・環医研・内分泌・代謝分野

P1-333 プロスタグランジンE_2によるTRPV1活性調節機構
Modulation of TRPV1 activity by prostaglandin E_2
森山　朋子[1]、東　智広[1]、冨樫　和也[1]、瀬木　恵里[2]、飯田　陶子[1]、富永　知子[1]、
杉本　幸彦[3]、成宮　周[2]、富永　真琴[1]
[1]三重大院・医・分子細胞生理、[2]京大院・医・神、[3]京大院・薬・生体情報制御

P1-334 プロスタグランジンI_2によるTRPV1活性調節機構
Modulation of TRPV1 activity by prostaglandin I_2
東　智広[1]、森山　朋子[1]、冨樫　和也[1]、瀬木　恵里[2]、飯田　陶子[1]、富永　知子[1]、
杉本　幸彦[3]、成宮　周[2]、富永　真琴[1]
[1]三重大院・医・分子細胞生理、[2]京都大院・医・神経細胞薬理、[3]京都大院・薬・生体情報制御

P1-335 抗リン酸化TRPV1抗体の機能解析
Functional analysis of anti-phosphorylated TRPV1 antibody
村山奈美枝[1]、沼崎　満子[1]、富永　真琴[1]
[1]三重大院・医・分子細胞生理

P1-336 顎関節炎により誘発される顔面皮膚を支配する大型三叉神経節ニューロンの興奮性の変化
Change in the excitability of large-diameter TRG neurons innervating intact cutaneous area after TMJ inflammation
武田　守[1]、谷本　武[1]、池田　水脈[1]、那須　優則[2]、角井　淳[1]、島　幸夫[3]、
太田　英彦[3]、松本　茂二[1]
[1]日本歯大・歯・生理、[2]日本歯大・歯・共同研セ、[3]杏林大・保健・生化

P1-337 後根神経節細胞は刺激頻度に応じて活性化する
Frequency-dependent activation of small, medium and large sizes of dorsal root ganglion neurons in vitro
古賀　浩平[1]、古江　秀昌[1]、吉村　恵[1]
[1]九州大院・医・統合生理

P1-338 伸張性収縮後にin vitroラット神経ー筋標本から記録した筋C線維受容器の反応
Responses of muscle C-fiber sensory receptors recorded from rat muscle-nerve preparations in vitro after eccentric contraction
田口　徹[1]、佐藤　純[1]、水村　和枝[1]
[1]名古屋大

P1-339 ラット脊髄膠様質神経興奮のバニロイド受容体とプリン受容体を介した温度による抑制
Temperature-dependent inhibition of C fiber-evoked excitation via vaniloid and purinergic receptors in the substantia gelatinosa of rat spinal cord slices revealed by optical imaging
楠堂　圭[1]、林　貴之[1]、池田　弘[1]、村瀬　一之[1]
[1]福井大学・工・知能システム

P1-340 In vivo パッチクランプ法を用いたラット脊髄痛覚伝達抑制機序の解析
In vivo patch-clamp analysis of interaction of excitatory and inhibitory synaptic inputs in the substantia gelatinosa of the rat spinal dorsal horn
古江　秀昌[1]、加藤　剛[1]、八坂　敏一[1]、水口　洋子[1]、吉村　恵[1]
[1]九州大院・医・統合生理

P1-341 吻側延髄腹内側部刺激によるラット脊髄膠様質細胞における抑制性シナプス入力の変化
Stimulation of the rostral ventromedial medulla facilitates inhibitory neurotransmission in the substantia gelatinosa of the rat spinal cord -In vivo patch-clamp analysis-
加藤　剛[1]、古江　秀昌[1]、八坂　敏一[1]、阿部　恵子[1]、吉村　恵[1]
[1]九州大学院、医、統合生理

Sep 21 (Tue) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

P1-342 カイニン酸類似キノコ毒、アクロメリン酸の急性および遅延性アロディニア誘導効果
Acute and late effects on induction of allodynia by acromelic acid, a mushroom poison related structurally to kainic acid
松村 伸治[1]、南 敏明[2]、西澤 幹雄[1]、笹栗 靖之[3]、伊藤 誠二[1]
[1]関西医大・医化学、[2]大阪医大・麻酔、[3]産業医大・第二病理

P1-343 下歯槽神経損傷によるアロディニア誘発ラットの三叉神経脊髄路核尾側亜核浅層領域での興奮性アミノ酸量の変動と一酸化窒素の関わりについて
Involvement of NO in increase of exitatory amino acids level in the trigeminal nucleus caudalis of the rats with nerve injury-induced allodynia
米原 典史[1]、藤田 豊大[1]、金 幼虹[1]、吉村 昌和[2]、上崎 善規[1]
[1]大阪大院・歯・薬理、[2]丸石製薬株式会社中央研究所

P1-344 脊髄内P2Xプリン受容体による長期持続性アロディニア
Long-lasting tactile allodynia mediated by spinal P2X-purinoceptors in rats
張 楠楠[1]、中川 貴之[1]、福井 真人[1]、南 雅文[1]、金子 周司[1]、佐藤 公道[1,2]
[1]京都大院・薬・生体機能解析、[2]奈良先端科技大

P1-345 神経因性疼痛モデルラットに発現するアロディニアに対する抗うつ薬髄腔内投与の効果
Effects of intrathecal injection of antidepressants on allodynia in the rat neuropathic pain model
池田 哲也[1]、武田龍一郎[2]、安部 博史[2]、石田 康[2]、西森 利數[1]
[1]宮崎大学・医・生物、[2]宮崎大学・医・精神医学

P1-346 光コヒーレンス断層法による慢性疼痛に伴うマウス大脳皮質の可塑的変化の測定
Optical Coherence Tomography revealed in vivo the Rise of Cortical Plasticity of Adult Mice in response to Peripheral Neuropathic Pain
大井 康浩[1]
[1]大阪大院・歯・顎口腔病因病態制御学

P1-347 神経因性疼痛モデル動物に対するミルナシプランの疼痛軽減効果
Effect of systemic milnacipran administration in rats with neuropathic pain.
武田龍一郎[1]、安部 博[1]、橋口 浩志[1]、池田 哲也[2]、石田 康[1]、西森 利數[2]
[1]宮崎大学・医学部・精神医学講座、[2]宮崎大学・医学部・生物学教室

P1-348 神経因性疼痛へのラマトロバンの鎮痛効果
Analgesic effect of ramatroban on neuropathic pain
徐 麗[1]、馬渕 圭生[1]、松村 伸治[1]、伊藤 誠二[1]
[1]関西医大・医化学

P1-349 LPA受容体を介した神経因性疼痛の分子機構
The molecular mechanisms of neuropathic pain through LPA receptor
藤田 亮介[1]、井上 誠[1]、Md Harunor Rashid[1]、松本みさき[1]、植田 弘師[1]
[1]長崎大院・医歯薬・分子薬理

P1-350 平滑筋におけるホルマリン痛と静磁場の効果
Effects of static magnetic field for muscular contraction by pain
千葉 惇[1]、生塩 研一[1]、稲瀬 正彦[1]
[1]近畿大・医・生理学1

Sep 22 (Wed) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

イオンチャネル、興奮性膜
Ion Channels and Excitable Membranes

P2-001 マウス腹側歯状回のmossy cellの律動性バースト発火はGABA$_B$受容体による持続性の抑制を受けている
GABA$_B$ receptor-mediated inhibition of bursting ventral mossy cells in the developing mouse dentate gyrus
神野 尚三[1]、石塚 智[2]、小坂 俊夫[1]
[1]九州大院・医・神経形態、[2]九州工大院・生命体・脳情報

P2-002 篩状神経節ニューロンのTTX-R Na^+チャネル電流に対するProtein kinase C活性の影響
Effect of PKC activity on the TTX-R Na^+ current in the nodose ganglion neurons
池田 水脈[1]、吉田 清輝[1]、角井 淳[1]、中野友花子[1]、松本 茂二[1]
[1]日本歯大・歯・生理

P2-003 *tottering*ミュータントマウス海馬で発生するコリン作動性リズム
Cholinergic oscillation in hippocampus of *tottering* Ca^{2+} channel mutant mice.
下野 健[1]、三木 崇史[2]、清中 茂樹[2]、森 泰生[2]
[1]アルファメッドサイエンス株式会社、[2]京都大院・工学研究科・合成／生物化学専攻 分子生物化学分野

P2-004 大脳皮質神経細胞の時間変動電流入力に対するスパイク発火時刻の予測
Predicting spike timings of current-injected neurons
坪 泰宏[1]、金子 武嗣[2]、篠本 滋[3]
[1]玉川大学・学術研究所、[2]京都大院・医、[3]京都大院・理

P2-005 治療的電撃けいれん（ECT）はHomer1a発現を介して新皮質錐体細胞を過分極させる
Electroconvulsion (ECT) hyperpolarizes neocortex pyramidal cells by inducingHomer1a/Vesl1S expression
阪上 優[1]、山本 兼司[2]、杉浦 重樹[3]、井ノ口 馨[4]、林 拓二[1]、加藤 伸郎[2]
[1]京都大院・医・精神医学、[2]京都大院・医・認知行動、[3]奈良医大、[4]三菱化学

P2-006 グリア細胞におけるNa_xチャンネルの局在と機能
Localization and function of Na_x sodium channel in glial cells
渡辺 英治[1]、檜山 武史[2,5]、児玉 隆治[3]、岡戸 晴生[6]、野田 昌晴[2,5]
[1]基生研・形質転換、[2]基生研・感覚情報、[3]基生研・発生生物、[4]さきがけ研究21、科学技術振興機構、[5]戦略的基礎研究推進事業、科学技術振興機構、[6]分子神経生理、東京都神経研

P2-007 ラット脳弓下器官ニューロンにおけるTEA感受性と低感受性I_{AS}
TEA sensitive and less-sensitive I_{AS} in rat subfornical organ neurons
小野堅太郎[1]、豊野 孝[2]、本田 栄子[1]、稲永 清敏[1]
[1]九歯大・生理、[2]九歯大・口腔解剖2

P2-008 GABAA受容体阻害による神経細胞死における電位依存性カルシウムチャネルの関与
VDCC and neuronal cell death induced by GABAA receptor inhibition
河西奈保子[1]、韓 春錫[1]、島田 明佳[1]、鳥光 慶一[1,2]
[1]日本電信電話（株）・NTT物性基礎研、[2]SORST

Sep 22 (Wed) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

P2-009 坐骨神経標本の極興奮に対するチャネル阻害剤の効果
Effects of channel blockers on the polar excitations in frog sciatic nerves
丹羽麻由香[1]、木暮 信一[1]
[1]創価大院・工・生物工

P2-010 バルビツレートのラット黒質ニューロンATP感受性Kチャネルに対する抑制作用
Inhibitory effects of barbiturates on ATP-sensitive K channels in rat substantia nigra
安藤 富男[1]、大塚 立夫[1]、石和 大[1]、紙谷 義孝[1]、山田 芳嗣[1]
[1]横浜市大院・医・生体制御・麻酔科学

P2-011 バースト発火型ニューロンのネットワークにおける同期的活動 --- シナプス部位と神経伝達物質の影響
The effects of the synaptic location on the synchronized activity in neural networks of fast rhythmic bursting neurons
竹川 高志[1]、青柳富誌生[1]、深井 朋樹[2]
[1]京都大院・情報、[2]玉川大・工

P2-012 Homer1a/Vesl1SはL型チャンネルを通るスパイク誘発性カルシウム流入を増強する
Homer1a/Vesl1S enhances spike-induced Ca^{2+} influx through L-type channels
山本 兼司[1,2]、阪上 優[2]、下濱 俊[1]、杉浦 重樹[3]、井ノ口 馨[4]、加藤 伸郎[2]
[1]京都大院・医・神経内科、[2]京都大院・医・認知行動、[3]奈良医大・組替DNA研究施設、[4]三菱化学生命科学研究所

P2-013 過興奮性神経細胞膨張からの回復における容積感受性Cl^-チャネルの関与
Volume-sensitive Cl^- channels are involved in recovery from excitotoxic neuronal swelling
井上 華[1,2]、岡田 泰伸[1,2]
[1]自然科学研究機構・生理学研究所・機能協関、[2]科学技術振興機構

P2-014 青斑核単離ニューロンにおけるオレキシン-A及び-Bの脱分極作用には非選択性カチオン及びカリウムコンダクタンスが関与する
Depolarizing effects of orexin-A and -B on isolated locus coeruleus neurons are mediated through nonselective cationic and K^+ conductances
村井 恵良[1]、赤池 忠[1]
[1]北海道大院・歯・口腔機能

P2-015 脱分極で発生する外向き電流応答の5-HTによる増強効果と低分子量G蛋白RhoとRho-kinaseの関与
Involvement of Rho and Rho-kinase in the augmenting effect of 5-HT on the depolarization-induced outward current response
川崎 敏[1]、木村 眞吾[1]、藤田 玲子[2]、渡辺 修二[1]、佐々木和彦[1]
[1]岩手医大・医・第一生理、[2]岩手医大・教養・化学

P2-016 StargazinファミリーはAMPA型受容体のサブユニットか?
Is Stargazin Involved in the AMPA Receptor Channel Complex?
山崎 真弥[1,4]、少作 隆子[2]、深谷 昌弘[3]、狩野 方伸[2]、渡辺 雅彦[3]、崎村 建司[1,4]
[1]新潟大・脳・細胞神経生物、[2]金沢大院・医・シナプス発達機能学、[3]北海道大院・医・生体構造解析、[4]SORST・JST

P2-017 海馬CA1ニューロンにおけるAMPAおよびQuisqualic acid投与で発生する電流応答の性質
Characteristics of AMPA- and Quisqualic acid-induced current responses in the CA1 pyramidal neuron of rat hippocampal slice
木村 眞吾[1]、川崎 敏[1]、藤田 玲子[2]、渡辺 修二[1]、佐々木和彦[1]
[1]岩手医大・医・第一生理、[2]岩手医大・教養・化学

P2-018 Baculovirus 発現系を用いた ATP 受容体 $P2X_2$ 蛋白の精製
Purification of recombinant protein of ATP receptor $P2X_2$ using baculovirus expression system.
久保 義弘[1]、山本 友美[1]、三尾 和弘[2]、佐藤 主税[2]
[1]生理学研究所・神経機能素子、[2]産業技術総合研究所・脳神経情報・構造生理

P2-019 P2Y1受容体の活性化を介した海馬抑制性介在ニューロンの興奮
Activation of P2Y1 receptors directly excites inhibitory interneurons in the hippocampus
川村 将仁[1]、加藤 総夫[2]
[1]慈恵医大・薬理学講座第1、[2]慈恵医大・神経生理

P2-020 ラット青斑核ニューロン興奮性に及ぼすセボフルレンの作用の分子標的
Sevoflurane affects multiple membrane conductance in the locus coeruleus of the rat
安井 豊[1,2]、正木 英二[2]、加藤 総夫[1]
[1]慈恵医大・神経生理、[2]慈恵医大・麻酔科学

P2-021 N型電位依存性カルシウムチャネル欠損マウスにおける孤束核シナプス伝達のGタンパク質共役型受容体による制御
Altered mechanism of transmitter release regulation by G-protein-coupled receptors in the autonomic sensory synapse in mice lacking N-type calcium channels.
繁冨 英治[1]、山崎 弘二[1]、西田 基宏[2]、森 泰生[3]、加藤 総夫[1]
[1]慈恵医大・神経生理、[2]九州大院・薬・薬物中毒学、[3]京都大院・工・合成・生物化学・分子生物化学

P2-022 生後発達に伴う前シナプス活動電位変化のメカニズム
Mechanisms underlying developmental shortening of presynaptic action potential at the calyx of Held
中村 行宏[1]、高橋 智幸[1]
[1]東京大院・医・神経生理

P2-023 ラット膀胱に投射する感覚神経におけるCMR1の発現
Expression of CMR1 in the rat sensory neurons innervating the urinary bladder
和中 明生[1]、松吉ひろ子[1]、辰巳 晃子[1]、眞部 孝幸[1]
[1]奈良県立医科大学 第二解剖

Sep 22 (Wed) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

P2-024 マイクログリアのpHホメオスターシス：細胞アシドーシスにおける膜電位依存性プロトンチャネルの役割
pH homeostasis of microglia: The voltage-gated proton channel serves as a pH regulator upon cell acidosis
久野みゆき[1]、森畑　宏一[2]、川脇　順子[3]、酒井　　啓[1]、澤田　　誠[4]、蔦田　強司[2]、森　　啓之[1]
[1]大阪市立大院・医・分子細胞生理学、[2]大阪市立大院・医・神経内科、[3]大阪市立大院・医・中央研究室、[4]藤田保健衛生大・難病治療共同研究部門

P2-025 海馬CA1領域における細胞外電場に対する応答特性へのhの寄与は小さい
Hyperpolarization-activated currents play a minor role in unique distribution of DC field-induced responses in CA1 region of rat hippocampal slice
秋山　博紀[1]、鈴木　崇之[1]、岡野　祥子[1]、工藤　佳久[1]、宮川　博義[1]、井上　雅司[1]
[1]東薬大院・生命科学・生体高次機能学

P2-026 IRBITによるリン酸化依存的なイノシトール－1,4,5－三リン酸受容体の制御
IRBIT regulates inositol 1,4,5-trisphosphate receptor in a phosphorylation-dependent manner
安東　英明[1]、水谷　顕洋[2]、松浦　　徹[3]、御子柴克彦[1,2,3]
[1]理研・脳研・発生神経生物、[2]科技団、[3]東京大院・医科研・脳神経

P2-027 ラット後根神経節細胞の過分極活性化陽イオン電流に対する細胞内ATP及びプロテインキナーゼAの作用
Effect of intracellular ATP and PKA on the hyperpolarization-activated cation current in rat dorsal root ganglion neurons
駒切　　洋[1]、北村　直樹[1]
[1]北大・獣・生理

P2-028 スパイク同期現象の安定化メカニズム －FS細胞、LTS細胞に関して－
Stability of Synchronous Solutions in Weakly Coupled Neuron Networks
野村　真樹[1,2]、青柳富誌生[1,2]
[1]京都大院・情報学・複雑系科学専攻、[2]科学技術振興機構

P2-029 樹脂酸による電位依存性K^+チャネル活性化とその機構
The activation of voltage-gated K^+ channels induced by rosin acid
坂本　多穂[1]、大矢　　進[1]、村木　克彦[1]、今泉　祐治[1]
[1]名古屋市立大院・薬・細胞分子薬効解析

P2-030 テトロドトキシン非感受性ナトリウムチャネル，Nav1.9，において観察される一過性電流増大（キンドリング）の解析
Cellular mechanism regulating the upregulation of Nav1.9, a TTX-resistant Na channel, in mouse dorsal root ganglion neurons
柿村　順一[1]、松冨　智哉[1]、鄭　　泰星[1]、中本　千泉[1]、仲田　義啓[2]、緒方　宣邦[1]
[1]広島大院・医歯薬学総合研究科・神経生理学、[2]広島大院・医歯薬学総合研究科・薬理学

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P2-031 マウス後根神経節ニューロンの活動電位発生におけるTTX －感受性およびTTX－ 非感受性ナトリウムチャネルの機能的役割
Functional roles of TTX-sensitive and TTX-resistant sodium channels in action potential generation of the mouse dorsal root ganglion neurons.
中本 千泉[1]、鄭 泰星[1]、松冨 智哉[1]、柿村 順一[1]、仲田 義啓[2]、緒方 宣邦[1]
[1]広島大院・医歯薬学総合研究科・神経生理学、[2]広島大院・医歯薬学総合研究科・薬理学

P2-032 PSD-95とGKAPによるNMDA受容体チャンネル活性のサブタイプ特異的調節
Subtype-specific modulation of the channel activity of the NMDA receptor by PSD-95 and GKAP
山田 康枝[1]、乾 誠[1]
[1]山口大・医・病態薬理

P2-033 N型カルシウムチャネルを強制発現させたBHK N101細胞の性質について
Characteristics of N-type calcium channels stably expressed in BHK N101cells
和田 哲幸[1]、川口 明範[1]、阿部 順一[1]、今西 孝至[1]、吉田 繁[2]、森 泰生[3]、
井本 敬二[4]、市田 成志[1]
[1]近畿大・薬・生物化学、[2]近大・理工・生命科学、[3]京都大院・工学専攻、[4]自然科学研究機構・生理学研究所

P2-034 Aチャネルによる EPSPの分流抑制の緩和とその神経間相互作用への影響
Reduction of EPSP Shunting by A-channels and its Effect on Neural Interaction
青西 亨[1,3]、宮川 博義[2]、井上 雅司[2]、岡田 真人[3,4]
[1]東工大院・総合理工学・知能システム、[2]東京薬科大学、[3]理化学研究所、[4]科学技術振興機構

P2-035 未分化及び分化したNG108-15細胞における高濃度KClによって惹起されたCa流入の性質について
Characteristics of calcium influx elicited by high-KCl in undifferentiated and differentiated NG108-15 cells
今西 孝至[1]、市田 成志[1]、松島加代子[1]、川口 明範[1]、和田 哲幸[1]、吉田 繁[2]
[1]近畿大・薬・生物化学、[2]近畿大・理工・生命科学

P2-036 過分極で活性化される陽イオンチャネルにおける第一膜貫通領域の構造解析
Helical Structure of S1 in the Hyperpolarization-Activated Cation Channel
石井 孝広[1]、大森 治紀[1]
[1]京都大院・医・生理

シナプス
Synapse

P2-037 機械的に強く接着したシナプス前部構造と後部構造の速い形態変化
Morphological Changes of Tightly Associated Presynaptic Varicosities and Postsynaptic Spines
梅田 達也[1]、海老原達彦[2]、岡部 繁男[1,3]
[1]東京医科歯科大院・医歯学総合・細胞生物学、[2]産業技術総合研究所・神経情報研究部門、
[3]戦略的基礎研究推進事業・科学技術振興機構

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P2-038 カルモジュリンキナーゼIIによるLRP13のPSD-95, SAP97結合の制御
LRP13 is a PSD target protein for PSD-95 and SAP97, regulated by the Ca2+/calmodulin-dependent protein kinase II.
鈴木 龍雄[1]、田 慶宝[1]、宮沢 昌子[1]、岡野 照[1]、山内 卓[3]、吉村 好之[3]、中山 耕造[2]、阪上 洋行[4]、近藤 尚武[4]、遠藤 省吾[5]
[1]信州大・院・医・神経可塑性学、[2]信州大・医・解剖、[3]徳島大・薬・生化学、[4]東北大・院・医・細胞生物学、[5]理研

P2-039 幼若ラット小脳プルキンエ細胞単一シナプスにおけるAMPA受容体数および密度の推定
The Number and Density of AMPA Receptors in Single Synapses in Immature Rat Cerebellum
田中 淳一[1]、松崎 政紀[2]、足澤 悦子[1,3]、籾山 明子[1]、Molnar Elek[5]、河西 春郎[2,3,4]、重本 隆一[1,3,4]
[1]生理学研究所・脳形態解析、[2]生理学研究所・生体膜、[3]総合研究大院、[4]戦略的創造研究推進事業、[5]Univ. Bristol, UK

P2-040 MAGUKファミリータンパク質p55（ラット）のシナプスにおける役割
Characterization of rat p55, a MAGUK family member, in the synapse
張 静萍[1]、田 慶宝[1]、岡野 照[1]、阪上 洋行[2]、近藤 尚武[2]、鈴木 龍雄[1]
[1]信州大・院・医・神経可塑性学、[2]東北大・院・医・細胞生物学

P2-041 シナプス膜とlipid raftに局在する新規nNOS結合タンパク質NIDD
NIDD, a novel DHHC-containing protein, targets neuronal nitric oxide synthase (nNOS) to the synaptic membrane through a PDZ-dependent interaction and regulates nNOS activity
齋藤 文典[1]、田 慶宝[1]、岡野 照[1]、阪上 洋行[2]、近藤 尚武[2]、鈴木 龍雄[1]
[1]信州大・院・医・神経可塑性学、[2]東北大・院・医・細胞生物学

P2-042 神経系, 造血細胞特異的BAALC 1-6-8 proteinのシナプス局在と性質
BAALC 1-6-8 protein targets to postsynaptic lipid raft by its N-terminal myristoylation and palmitoylation, and interacts specifically with α subunit of Ca2+/calmodulin-dependent protein kinase II.
王 新[1]、田 慶[1]、岡野 照[1]、阪上 洋行[2]、近藤 尚武[2]、鈴木 龍雄[1]
[1]信州大・院・医・神経可塑性学、[2]東北大・院・医・細胞生物学

P2-043 シナプトタグミンIのC2Bドメインの速い神経伝達における役割
Roles of the C2B domain of Synaptotagmin I for fast synaptic transmission in *Drosophila* embryos
田村 拓也[1]、Noreen E. Reist[2]、城所 良明[1]
[1]群馬大学・医・行動生理、[2]Col St Univ

P2-044 シナプス小胞リサイクリングにおけるチュブリンの役割
Roles of Tubulin in synaptic vesicle recycling
本多 敦子[1]、黒見 坦[1]、城所 良明[1]
[1]群馬大院・医・遺伝発達行動学

P2-045 VGLUT2含有興奮性終末入力のある皮質棘突起の抑制性支配
GABAergic input association with the VGLUT2-expressed terminal at the spine head of cortical neurons
窪田 芳之[1]、畑田小百合[1]、根東 覚[1]、川口 泰雄[1]
[1]自然科学研究機構・生理研・大脳神経回路論

P2-046 ベンゾジアゼピン類によるGABA遊離の増強
Benzodiazepines facilitate spontaneous GABA release at mouse hippocampal CA1 pyramidal neurons
石橋 仁[1]、梶原三紗子[1]、小野原伸也[1]、鍋倉 淳一[2]
[1]九州大院・医・分子機能生理、[2]岡崎国立共同研究機構生理学研究所

P2-047 シナプス前後ニューロンのカルシウムイメージングが明らかにしたコオロギ気流感覚系における刺激方向抽出メカニズム
Calcium imaging of pre- and postsynaptic neurons reveals cellular basis for directional tuning properties in cricket cercal sensory system
小川 宏人[1]、岡 浩太郎[2]、John P. Miller[3]
[1]埼玉医大・生物、[2]慶大・理工・生命情報、[3]Center for Computational Biology, Montana State Univ.

P2-048 海馬CA3におけるシナプスの形態計測
Morphometrical analysis of synaptic boutons in the rat hippocampal CA3.
松田 正司[1]、小林 靖[2]、石塚 典生[3]
[1]愛媛大・医・解剖学第一、[2]防衛医科大・解剖学第二、[3]東京都神経科学総合研究所・脳構造研究部門

P2-049 β-neurexin はゼブラフィッシュ嗅神経系の軸索終末への VAMP2-EGFP の集積を抑制する
β-Neurexin suppressed VAMP2-EGFP accumulation in axon terminals of zebrafish olfactory sensory neurons
増田 賢嗣[1]、吉田 知之[1]、松田 尚人[1]、三品 昌美[1]
[1]東大院・医・分子神経生物

P2-050 小脳プルキンエ細胞AMPA受容体構成性エンドサイトーシスにおけるPKCならびにp38 MAPKの関与
Involvement of PKC and p38 MAPK in constitutive trafficking of AMPA receptor in cerebellar purkinje cell.
立川 哲也[1,2,3]、工藤 佳久[2]、山口 和彦[1,3]
[1]理研・脳センター・記憶学習、[2]東薬大・生命科学・生体高次機能、[3]CREST/JST

P2-051 ミオシンVとシンタキシンのカルシウム依存性結合の解析
Characteristics of the Ca^{2+}-dependent binding between myosin-V and syntaxin-1A
渡部 通寿[1]、牛木 辰男[2]、石川 良樹[3]、笹川 展幸[4]、熊倉鴻之助[4]、五十嵐道弘[1]
[1]新潟大院・医歯学総合研究科・分子細胞機能学（医学部生化学第二）、[2]新潟大院・医歯学・顕微解剖学、[3]群馬大・医・薬理学、[4]上智大・生命科学研

P2-052 前頭皮質における線条体投射錐体細胞の皮質内結合選択性
Recurrent excitatory connections between corticostriatal pyramidal cells
森島美絵子[1]、川口 泰雄[1]
[1]総研大・生命科学・生理学研究所・大脳神経回路論

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P2-053 視床において同期的神経活動を誘発するシナプス結合様式
Thalamic synchronizer: revealed by direct measurement of divergent synaptic connections
井上　剛[1,2]、井本　敬二[1,2]
[1]生理学研究所、[2]総合研究大院・生命科学研究科

P2-054 ラット脊髄後角におけるエンドモルフィンの痛覚情報伝達制御の作用機序
Cellular mechanisms for the modulation by endomorphins of nociceptive transmission in the rat spinal dorsal horn
藤田　亜美[1]、古賀亜希子[1]、中塚　映政[1]、熊本　栄一[1]
[1]佐大・医・生体構造機能学

P2-055 海馬テタヌス後脱分極と同期的オシレーションに関与する介在細胞サブタイプの同定
Network mechanism of GABA-dependent post-tetanic depolarization and seizure-like afterdischarge in the rat hippocampus
塚元　葉子[1]、礒村　宜和[1]、今西美知子[1]、高田　昌彦[1]
[1]東京都神経研・統合生理

P2-056 Neuropsinによるスパインの形態変化
Neuropsin/KLK8-induced spine dynamics in hippocampal neurons
石川　保幸[1]、伊藤　瑞幾[1]、塩坂　貞夫[1]
[1]奈良先端科学技術大院・細胞構造学

P2-057 遺伝子ターゲッティング法を用いたシナプトフローリンによる開口放出の測定
Cell type specific measurement of exocytosis by conditionally targeted expression of synapto-pHluorin
荒木　力太[1,2]、石塚　徹[1,2]、柳川右千夫[3]、阪上　洋行[4]、小橋　雄一[1,2]、明石　馨[5,6]、平林　敬浩[7]、宮崎　純一[8]、崎村　建司[5,6]、八尾　寛[1,2]
[1]東北大院・生命・脳機能解析、[2]CREST・科学技術振興機構、[3]群馬大院・医・脳神経発達統御学講座・遺伝発達行動学、[4]東北大院・細胞生物学講座・細胞組織学、[5]新潟大・脳研究所・細胞神経生物学、[6]SORST・科学技術振興機構、[7]自然科学研究機構・生理学研究所・高次神経機構研究部門、[8]大阪大院・医・幹細胞制御

P2-058 カルシウムチャネルα1Aサブユニット欠損マウスのプルキンエ細胞体における興奮性および抑制性入力
Excitatory and inhibitory innervation on to Purkinje cell soma in calcium channel α1A subunit deficient mice
宮崎　太輔[1]、Hee-Sup Shin[2]、渡辺　雅彦[1]
[1]北海道大院・医・解剖発生、[2]National Creative Research Initiative Center for Calcium and Learning, Korea Institute of Science and Technology

P2-059 マウス視床VB核の興奮性シナプス伝達に対するkainate 受容体の制御機構
Distinct regulation of kainate receptors in the corticothalamic and primary sensory (leminiscal) synapses in the ventrobasal thalamus
宮田麻理子[1,2]、井本　敬二[1,2]
[1]生理学研究所・生体情報研究系、[2]The Graduate University for Advanced Studies

P2-060 VAMPファミリー含有小胞の解析
Molecular characterization of VAMP-2, 3 and 4-containing intracellular vesicles
西宮聖一朗[1]、板倉　誠[1]、小寺　義男[2]、前田　忠計[2]、高橋　正身[1]
[1]北里大学・医療衛生・代謝蛋白、[2]北里大学・理・生物動力学

P2-061 synaptic vesicleとactive zoneに結合している新規serine/threonine kinase SAD-1の機能解析
SAD-1 is Associated with Synaptic Vesicles and the Active Zone and Implicated in Ca^{2+}-dependent Exocytosis
井上　英二[1]、力津絵津子[1]、俵田　真紀[1]、井上真理絵[1]、大塚　稔久[1]
[1]株式会社カン研究所

P2-062 ガン抑制遺伝子APCはPSD-95のクラスタリングを介してシナプス伝達に関与する
Tumor suppressor protein APC affects synaptic signal transduction via clustering of PSD-95
大熊　真人[1]、下村　敦司[2]、向後　晶子[2]、野村　隆士[2]、千田　隆夫[2]、宮地　栄一[1]
[1]藤田保健衛生大・医・生理II、[2]藤田保健衛生大・医・解剖I

P2-063 成熟マウス小脳におけるシナプス後膜AMPA受容体の活動に依存したシナプス前終末の機能維持
Postsynaptic AMPA receptor activity maintains presynaptic function in the adult cerebellum
柿澤　昌[1,2]、宮崎　太輔[3]、飯野　正光[1]、渡辺　雅彦[3]、狩野　方伸[2]
[1]東京大院・医・細胞分子薬理、[2]金沢大院・医・シナプス発達機能学、[3]北海道大院・医・解剖発生学

P2-064 神経細胞におけるRING-H2タイプユビキチンリガーゼ、hVPS18の機能解析
Characterization of the RING-H2 type ubiquitin ligase, hVPS18, in the rat cortical neurons
與五沢里美[1]、赤澤　智宏[1]、中村　泰子[1]、高坂　新一[1]
[1]国立精神神経センター・神経研・代謝

P2-065 ラット背外側中隔核における単シナプス性抑制性シナプス後電流の5-HT1B受容体によるシナプス前抑制
Presynaptic inhibition of monosynaptic IPSCs mediated by 5-hydroxytryptamine 1B receptors in rat dorsolateral septal nucleus
蓮尾　博[1]、松岡　稔昌[2]、浅海　安雄[1,2]、赤須　崇[1]
[1]久留米大・医・生理学2、[2]久留米大・医・神経精神科

P2-066 メンブレントラフィックに関わる線虫Sec1/Munc-18ファミリー遺伝子 *Ce-vps45*の機能解析
Functional analysis of the *C. elegans* Sec1/Munc-18 family gene *Ce-vps45* involved in membrane traffic
安藤　恵子[1]、町山　悦子[1]、野口　幸子[1]、三谷　昌平[1]
[1]東女医大・医・第2生理

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P2-067 特徴周波数で異なるニワトリ大細胞核の情報処理機構
Tonotopic gradients of membrane and synaptic properties for neurons of the chicken nucleus magnocellularis
福井　巌[1]、大森　治紀[1]
[1]京都大院・医・神経生物

P2-068 発達マウス脳におけるプレセベリンファミリーの発現と成熟マウス小脳におけるプレセレベリン3の分子局在
Expression of precerebellin family in the developing mouse brain and subcellular localization of precerebellin-3 in the adult cerebellum
三浦会里子[1]、渡辺　雅彦[1]
[1]北大院・医・解剖発生

P2-069 Active Zone 蛋白質 CASTのマウス網膜における局在
The Active Zone Protein CAST is a Component of the Conventional and Ribbon Synapses in Mouse Retina
俵田　真紀[1]、井上　英二[1]、力津絵津子[1]、井上真理枝[1]、大塚　稔久[1]
[1]カン研究所

P2-070 ミダゾラムは大脳皮質第V層の微小抑制性シナプス後電流の頻度をα7ニコチン性アセチルコリン受容体を介して増加させるがII層の錐体細胞では増加させない
Midazolam increases Miniature IPSCs frequency in layer V but not in layer II neocortical pyramidal neurons via α7 nicotinic ACh receptor
山本　純偉[1]、山田　順子[2]、上野　伸哉[3]、福田　敦夫[1,2]
[1]浜松医科大学・生理学第一講座、[2]静岡大院・電子科学研究科、[3]弘前大学脳研リハビリテーション医学

P2-071 ニューレグリン受容体ErbBは脂質マイクロドメイン（ラフト）に極在する
ErbB Neuregulin Receptors are Localized to Lipid Rafts
尾崎美和子[1]、大須賀　壮[2]、大島恵理子[2]、平林　義雄[2]
[1]理研・脳センター・記憶学習、[2]理研・脳センター・平林研究ユニット

P2-072 ラット背外側中隔核（DLSN）ニューロンにおけるdopamineの抑制性シナプス伝達に対する効果
Effects of dopamine on the inhibitory synaptic transmission in neurons of the rat dorsolateral septal nucleus (DLSN)
浅海　安雄[1,2]、蓮尾　博[1]、赤須　崇[1]
[1]久留米大・医・生理学2、[2]久留米大・医・精神神経

P2-073 カゼインキナーゼ2阻害剤によるシナーフィン局在変化の抑制
Inhibition of the dynamic movement of synaphin/complexin by a casein kinase II inhibitor
車多　厚志[1]、西須　英雄[1]、小谷　昌司[2]、阿部　輝雄[1]
[1]新潟大・脳研・細胞神経生物学、[2]新潟大・理・生物

P2-074 ラットスライスを用いた一次聴覚野と周辺領野の機能結合の解析
Properties of inter-areal circuits between primary auditory cortex and the surrounding areas in rat cerebral slices
菱田　竜一[1]、北浦　弘樹[1]、鎌谷　大樹[1]、工藤　雅治[1]、澁木　克栄[1]
[1]新潟大・脳研・システム脳生理

P2-075 新生ラット辺縁帯におけるシナプス伝達の光学的・化学的解析
Optical and chemical analyses of synaptic transmission in neonatal rat marginal zone (MZ)
千　太哲[1]、中村　直人[2]、天野　賢[1]、古川　智範[1]、山田　順子[3]、W. Kilb[4]、中原大一郎[2]、H.J. Luhmann[4]、福田　敦夫[1,3]
[1]浜松大・医・生理第一、[2]浜松大・医・心理学、[3]静岡大・院・電子科研、[4]Inst Physiol Pathophysiol, J Gutenberg Univ Mainz, Mainz, Germary

P2-076 Regulation of excitatory synaptic transmission by a neuronal ubiquitin ligase
Takeshi Otsuka[1]、Michael_D. Ehlers[1]
[1]Department of Neurobiology, Duke University Medical Center, NC, USA

シナプス可塑性
Synaptic Plasticity

P2-077 ラット後背側及び腹側海馬－前頭前野路のpaired-pulse facilitation特性の比較
Difference in dynamical property to paired-pulse facilitation of two LTP-inducible hippocampal-prefrontal cortex pathways of rats
倉持　真人[1,2]、伊崎　義憲[3]、大富美智子[2]、瀧田　正寿[1]
[1]産総所・人間福祉医工学・ニューロバイオニクスG、[2]東邦大・理・生物分子、[3]聖マリアンナ医科大・生理

P2-078 マウス視覚野興奮性ニューロンの樹状突起におけるセリンプロテアーゼを介した経験依存性形態変化
Experience-dependent pruning of dendritic spines in mouse visual cortex by tissue-type plasminogen activator
俣賀　宣子[1]、水口　陽子[1]、ヘンシュ貴雄[1]
[1]理化学研究所・脳総研・神経回路発達

P2-079 刺激特異的に発現するタンパクHomer1aの生理的役割
Activity-inducible protein Homer1a promotes redistribution of postsynaptic protein Homer1c in cultured rat hippocampal neurons
井上由理子[1]、有働　洋[1]、杉山　博之[1]
[1]九州大院・システム生命科学

P2-080 ニジマス視蓋網膜視蓋間シナプスにおける長期増強
Long-term potentiation in retinotectal synapses in the optic tectum of rainbow trout
木下　雅恵[1]、小林　卓[1]、細川　敏幸[2]、浦野　明央[1]、伊藤　悦朗[1,3]
[1]北海道大院・理・生物科学、[2]北海道大・高等教育センター、[3]北海道大・創成機構

P2-081 シナプス関連蛋白ArcとCaMKIIの相互作用の可視化
Interaction between Arc and CaMKII monitored by fluorescence resonance energy transfer
奥野　浩行[1,2]、Shoaib Chowdhury[2]、Paul Worley[2]、尾藤　晴彦[1,3]
[1]東京大院・医・神経生化学、[2]Dept. of Neurosci., Johns Hopkins Univ. Sch. of Med., Baltimore, USA、[3]科学技術振興機構 SORST

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an even number 15:00～16:00

P2-082 *vesl-1* mRNA polyadenylationは細胞外刺激により制御される
Extracellular stimuli regulate polyadenylation of *vesl-1* mRNAs that encode synaptic scaffold proteins
新堀　洋介[1,2]、井ノ口　馨[1,2]
[1]三菱化学生命科学研究所、[2]横浜国大・環境情報

P2-083 小脳GABA伝達のシナプス前抑制を仲介するAMPA型グルタミン酸受容体の性質
Characterization of AMPA receptors that mediate presynaptic inhibition of cerebellar GABAergic transmission
佐竹伸一郎[1]、井本　敬二[1]
[1]生理研・生体情報

P2-084 17beta-estradiolによる海馬CA1錐体神経細胞の形態変化
Morphological changes of hippocampal CA1 pyramidal cells caused by 17beta-estradiol
釣木澤朋和[1,2]、川戸　佳[1,2]
[1]東京大院・広域科学、[2]CREST

P2-085 海馬分散培養の繰り返しAキナーゼ活性化後の長期シナプス新生：シナプスの機能性
Activity-dependent synaptogenesis in cultured hippocampal neurons by repetitive PKA activation.
山本　恵[1]、冨永一吉野恵子[1,2]、小倉　明彦[1,2]
[1]大阪大院・理・生物科学専攻・神経可塑性生理学研究グループ、[2]大阪大院・生命機能・生命機能学専攻・神経可塑性生理学研究グループ

P2-086 サル1次視覚野と下側頭葉皮質TE野間で差次的発現を示す遺伝子の同定
Identification of genes differentially expressed between V1 and area TE of the macaque monkey
松岡　洋祐[1]、濱田　俊[1]、田村　弘[1]、米島　宏幸[1]、八木　健[1]、藤田　一郎[1]
[1]大阪大院・生命機能・心生物学グループ、認知脳科学研究室

P2-088 大脳皮質の神経細胞にみられるStargazin familyの異なったフォスファターゼの制御
Different phosphatases regulate stargazin family in cortical neurons.
稲村実穂子[1]、板倉　誠[1]、岡本　浩嗣[1]、外　須美夫[1]、高橋　正身[1,2]
[1]北里大学・医、[2]CREST

P2-089 LTDの繰り返し誘発による長期シナプス廃止
Loss of synapses provoked by repeated inductions of LTD in cultured hippocampal slice
上窪　裕二[1]、篠田　陽[1]、冨永一吉野恵子[1]、小倉　明彦[1]
[1]大阪大院・生命機能・神経可塑性生理学

P2-090 大脳新皮質孤立培養神経細胞における興奮性シナプス結合の活動依存的な成熟
Activity-dependent maturation of excitatory synaptic connections in solitary neuron cultures of neocortex
高田　直樹[1]、柳川右千夫[2]、小幡　邦彦[3]、小松由紀夫[1]
[1]名古屋大・環境医学研究所、[2]群馬大院・医学系研究科、[3]理研・脳科学総合研究センター

P2-091 長期シナプス新生とグルタミン酸によるLTP誘発との相関性
Long-lasting synaptogenesis after repetitive exposures to glutamate depends on the induction of LTP
冨永一吉野恵子[1]、谷口　直子[1]、小倉　明彦[1]
[1]大阪大院・生命機能研究科

P2-092 視機性眼球運動の長期適応後に起こるマウス小脳片葉のシナプス密度減少
Reduction of synapse density after long-term adaptation of horizontal optkinetic response in the mouse flocculus
中舘　和彦[1,2,3]、首藤　文洋[3,4]、永雄　総一[3,4]、重本　隆一[2,3]
[1]獨協医大・解剖学（組織）、[2]自然科学研究機構・生理学研究所・脳形態解析、[3]科学技術振興機構戦略的創造研究推進事業、[4]理研BSI・運動学習制御

P2-093 神経ステロイドDHEAS慢性投与によるラット海馬CA1長期増強の誘導促進
Chronic exposure of DHEAS primes LTP induction in rat hippocampal CA1
陳　　玲[1]、戴　　小牛[2]、曽我部正博[1,2,3]
[1]JST国際共同研究「細胞力覚プロジェクト」、[2]名大・医・生理2、[3]岡崎生理研・分子生理

P2-094 成熟ネコ視覚野における逆説眼優位シフトと通常シフトの同時誘導
Noradrenergic activation restores ocular dominance plasticity in adult cats irrespective of normal or paradoxical shift
今村　一之[1]、田中　　繁[1]
[1]理研・BSI・視覚神経回路モデル研究チーム

P2-095 発達期視覚野における視覚入力によるERK 1/2活性の調節
Regulation of ERK 1/2 activation by visual input in the developing visual cortex
高村　明孝[1]、一坂　吏志[2]、林　　千尋[2]、牧　　廣利[2]、畠　　義郎[1]
[1]鳥取大・院・医・生体高次機能、[2]鳥取大・医・生命・神経生物

P2-096 CRE転写調節因子ICER欠失の扁桃体キンドリングに及ぼす効果
Effects of a lack of CRE-mediated transcription factor ICER on amygdala kindling
児島　伸彦[1]、池田　敏男[2]、糸原　重美[2]、遠藤　昌吾[1]
[1]理研・脳センター・神経回路メカニズム、[2]理研・脳センター・行動遺伝

P2-097 M1ムスカリン性アセチルコリン受容体を介した海馬シナプス可塑性の調節
Cholinergic regulation of hippocampal synaptic plasticity via M1 muscarinic receptors
篠江　　徹[1]、松井　　稔[1]、真鍋　俊也[1]
[1]東京大・医科・基礎医科学部門・神経ネットワーク分野

P2-098 海馬長期増強におけるニューロプシンの役割
Role of neuropsin in hippocampal long-term potentiation in vivo
田村　英紀[1]、塩坂　貞夫[1]
[1]奈良先端科学技術大院・バイオサイエンス・細胞構造

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P2-099 17β-estradiolは海馬シナプス伝達効率を調節する
Modulation of excitatory neurotransmission by 17β-estradiol in the hippocampus
城間 晋作[1]、井戸 喜英[1]、米谷快男児[1]
[1]九州工大・情報工・生命情報

P2-100 海馬CA3野ヘテロシナプティックLTP誘導の膜特性依存性
Bistable membrane properties as a determinant of heterosynaptic CA3 LTP induction
柄本 昌子[1]、松木 則夫[1]、池谷 裕二[1]
[1]東京大院・薬・薬品作用

P2-101 Presynaptic nicotinic acetylcholine receptors regulate transmitter release in adult songbird motor control nucleus
張田 由希[1]、Hessler Neal[1]
[1]理研・脳科学総合研究センター・発声行動機構チーム

P2-102 最初期遺伝子Arc mRNA樹状突起輸送機構の解析
Identification of a Dendritic Targeting Element in the 3'UTR of Arc mRNAs
小林 裕明[1]、山本 祥也[2]、丸尾 知彦[2]、村上富士夫[1,3]
[1]大阪大院・生命機能・脳神経工学、[2]大阪大院・基礎工学・生物工学、[3]科学技術振興事業団

P2-103 PKA活性化による長期可塑性シナプス新生におけるBDNFの関与
Involvement of BDNF in the long-lasting synaptogenesis after repetitive activation of PKA
谷口 直子[1]、冨永一吉野恵子[1]、小倉 明彦[1]
[1]大阪大院・生命機能研究科・神経可塑性生理学研究室

P2-104 リン酸化酵素CK2はNMDA受容体依存性の海馬長期増強に必要である
The protein kinase CK2 is required for the induction of NMDA receptor-dependent long-term potentiation in rat hippocampus
木村 梨絵[1]、山田 麻紀[1]、西山 信好[1]、松木 則夫[1]
[1]東京大院・薬・薬品作用

P2-105 主嗅球における長期増強のPI-3キナーゼ抑制作用
Inhibition of phosphoinositide 3-kinase impairs LTP in the main olfactory bulb
張 敬姫[1]、奥谷 文乃[1]、黄 光哲[1]、椛 秀人[1,2]
[1]高知大・医・統合生理学、[2]生理学研究所

P2-106 副嗅球におけるLTPのノルアドレナリンによる促進：細胞機構
Noradrenaline enhancement of LTP in the accessory olfactory bulb: a cellular mechanism
黄 光哲[1]、椛 秀人[1,2]
[1]高知大・医・統合生理学、[2]生理学研究所

P2-107 幼弱海馬における異シナプス性長期抑圧メカニズム
Mechanism of heterosynaptic LTD in neonatal hippocampus
安田 浩樹[1,2]、津本 忠治[1,2]
[1]大阪大院・医・神経生理、[2]戦略的創造研究推進事業

P2-108 多点電極アレイ記録法を用いたマウス海馬スライスのLTPの長時間解析
Long-term analysis of LTP in the mouse hippocampal slice using multielectrode array recording
別所 親房[1]、星野 基[1]、榎本 孝幸[1]
[1]京産大・理・物理

P2-109 成熟した海馬神経培養細胞における神経回路再編成のメカニズムの解析
Re-organization mechanisms of grown-up neuronal circuit in dissociated hippocampal pyramidal neurons
中山貴美子[1]、清末 和之[1]、田口 隆久[1]
[1]産総研・セルエンジニアリング・ニューロニクス

P2-110 Enhanced mossy fiber plasticity in mice with increased kainate receptor function
Koichi Ito[1]、Geoffrey T. Swanson[1]
[1]Department of Pharmacology and Toxicology, University of Texas Medical Branch, Texas, USA

P2-111 Direct Focal Detection of Axonal Terminal-Branch Variability in *Drosophila* Learning Mutants: The Role of cAMP in Transmission Precision, Output levels, Activity-Dependent Plasticity, and Ca^{2+} Regulation
Atsushi Ueda[1]、Chun-fang Wu[1]
[1]Department of Biological Sciences, University of Iowa, Iowa, USA

P2-112 Effects of Capsaicin on Neocortical Long-term Depression in Awake and Freely Moving Rat
Alireza Komaki[1,2]、Hossein Esteky[1,3]、Fereshteh Motamedi[1,2]
[1]Departement of Physiology, School of Medicine, Shaheed Beheshti University of Medical Science, Tehran, Iran、[2]Neuroscience Research Center, Shaheed Beheshti University of Medical Science, Tehran, Iran、[3]IPM School of Cognitive Sciences, Niavaran, Tehran, Iran

パターン形成
Pattern Formation

P2-113 ネトリン1欠損マウス脊髄ではオリゴデンドロサイト前駆細胞の出現部位が腹側方向に変異する
Ventral shift of the site of oligodendrocyte origin in the netrin-1 deficient mouse spinal cord
小野 勝彦[1]、渡辺 啓介[1,2]、竹林 浩秀[1]、池中 一裕[1,2]
[1]生理研・分子神経生理、[2]総研大

P2-114 DNAマイクロアレイを用いたマウス網膜における領域特異的発現分子の探索
Large-scale screening of topographic molecules in the embryonic mouse retina using oligo DNA microarrays.
中村 隆弘[1,2]、新谷 隆史[1,2]、野田 昌晴[1,2]
[1]大学共同利用機関法人・自然科学研究機構・基礎生物学研究所・感覚情報処理研究部門、
[2]戦略的創造研究推進事業・科学技術振興機構

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P2-115 子宮内エレクトロポレーション法によるマウス海馬原基における神経細胞の発生・移動の解析
Neuronal generation and migration in the mouse hippocampal primordium as revealed by in utero electroporation
中平 英子[1]、湯浅 茂樹[1]
[1]国立精神神経センター・神経研究所・微細構造研究部

P2-116 子宮内エレクトロポレーションを用いた放射状グリアの選択的破壊による皮質形成機構の研究
Selective ablation of radial glia through the expression of suicide gene by in utero electroporation
長谷川明子[1]、池島 宏子[2]、福村 怜子[1]、湯浅 茂樹[1]
[1]国立精神神経センター・神経研究所・微細構造、[2]慈恵医大・DNA医学研究所・分子免疫学研究部門

P2-117 マウス大脳皮質視覚野における抑制性神経細胞の誕生時期依存的配置機構
Birth-date dependent alignment of GABAergic interneurons in the developing mouse visual cortex
四津 真人[1]、田畑 秀典[1]、仲嶋 一範[1,2]
[1]慶應大・医・解剖、[2]慈恵医大・DNA研・分子神経生物学

P2-118 マウスCcd1アイソフォームの同定と神経系での発現解析
Identification and neural expression of mouse Ccd1 (Coiled-coil-DIX1).
桝 和子[1]、塩見 健輔[1]、兼元みずき[1]、吉田さちね[1]、相馬 克徳[1]、桝 正幸[1]
[1]筑波大院・人間総合科学・分子神経

P2-119 リーラーマウスにおける嗅内野海馬投射の経路異常
Abnormal Formation of Entorhinohippocampal Projection of Reeler Mutant Mouse
村岡 大輔[1]、寺島 俊雄[1]
[1]神戸大院・医・脳科学講座・神経発生学分野

P2-120 新生齧歯類の延髄半分割スライス標本における呼吸ニューロンの律動的バーストの発現
The rhythmic bursts of the respiratory activity neurons in medullary half-slice preparations of neonatal rodents
小林 卓[1]、藤戸 裕[1]、松山 清治[1]、青木 藩[1]
[1]札幌医大・医・第二生理

P2-121 成獣マウス脳における *SulfFP1* mRNAの発現解析
Expression analysis of *SulfFP1* mRNA in the adult mouse brain
吉田さちね[1]、桝 和子[1]、長嶺 聖史[1]、大戸 達之[1]、小池 誠一[1]、桝 正幸[1]
[1]筑波大院・人間総合科学・感性認知脳科学

P2-122 リーラマウスの皮質視蓋路の生後発達の解析
Postnatal development of corticotectal projection of the reeler mouse
馬場 孝輔[1]、榊原 俊介[1]、薛 富義[1]、村岡 大輔[1]、寺島 俊雄[1]
[1]神戸大学・医・脳科学講座・神経発生学

P2-123 ゼブラフィッシュ *SulfataseFP* 遺伝子の機能解析
Functional analysis of zebrafish *SulfFP* genes during neural development
塩見 健輔[1]、内田 博[1]、桝 和子[1]、桝 正幸[1]
[1]筑波大院・人間総合科学・分子神経

P2-124 前脳形成におけるzinc finger遺伝子 *fez*および *fezl*の役割
A role of the zinc finger genes *fez* and *fezl* in the forebrain formation
平田　務[1]、中澤　祐人[1]、須田　容子[2]、中尾　和貴[3]、日比　正彦[1]
[1]理研・発生再生研・体軸形成、[2]理研・発生再生研・ボディプラン、[3]理研・発生再生研・変異マウス

P2-125 ラット胎生期視床の前後部において発現の異なる分子の解析
Differential gene expression analysis of anterior and posterior thalamus in rat embryo
山田　早織[1]、山口　宜秀[1]、馬場　広子[1]
[1]東京薬科大・薬・機能形態

P2-126 ラット胎生期前脳のプロテオミクス解析
Proteomic analysis of rat embryonic forebrain
山口　宜秀[1]、山田　早織[1]、馬場　広子[1]
[1]東京薬科大・薬・機能形態

P2-127 ボンベシン様ペプチド受容体の脳の形成における役割の解析
Role of bombesin-like peptide receptors on developmental chick brain
岩渕　舞子[1]、田中　光一[1]、浜崎　浩子[1]
[1]東京医科歯科大・医歯学総合研究科・認知システム学

P2-128 マウス神経発生の高精度デジタルアトラスプロジェクト
A project on superfine digital atlas of the development of the mouse nervous system
原　嘉信[1]
[1]神経発生研究所

P2-129 小脳区域化関連遺伝子のin situ Hybridizationスクリーニング
In situ hybridization screen for genes involved in mouse cerebellar compartmentalization
森田　規之[1]、古市　貞一[1]
[1]理研・脳科学・分子神経形成

P2-130 メダカ脳の神経路形成:脊椎動物の神経路形成の共通性
Axonogenesis in the medaka embryonic brain
石川　裕二[1]、山本　直之[2]、吉本　正美[2]、伊藤　博信[2]、保田　隆子[1]、丸山　耕一[1]、景　崇洋[1]
[1]放射線医学総合研究所、[2]日本医科大・第2解剖

P2-131 リーラーマウスにおける梨状葉皮質の層構築異常
Layer disorganization in reeler piriform cortex.
宮下　英将[1]、小川　正晴[1]
[1]理研・脳研・細胞培養技術開発

Sep 22 (Wed) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

細胞分化
Cellular Differentiation

P2-132 bFGF存在下の神経幹細胞におけるnecdinの発現上昇
Upregulation of necdin expression in bFGF-treated neural stem cells *in vitro*
西村伊三男[1]、田島　亜紀[1]、吉川　和明[1]
[1]大阪大・蛋白研・機能制御

P2-133 ES細胞から神経幹細胞および神経細胞の分化誘導
Simple procedure for production of neural stem cells and neurons from embryonic stem cells
中山　孝[1]、百木　智子[1]、山口　和彦[2]、鈴木　豊[3]、近藤　靖[3]、祭　友昭[4,5]、井上　順雄[1,5]
[1]横浜市大・医・生化学、[2]理研・脳科学・記憶学習機構研究チーム、[3]田辺製薬・先端医学研究所、[4]慈恵医大・整形、[5]都立保健科学大・保健科学部

P2-134 ラット幼弱脳および成体脳におけるNeurogenesin-1の発現分布
The distribution of Neurogenesin-1 in the developing and adult rat brain
山下　寛奈[1]、三河須美子[1]、王　涼[1]、佐藤　康二[1]
[1]浜松医大・医・解剖1

P2-135 海馬歯状回におけるGABAergic systemによる成体ニューロン新生の促進
Excitatory GABAergic inputs promoting adult neurogenesis in dentate gyrus
戸塚　祐介[1]、福田　諭[1]、吉田　美香[1]、宮本　有正[1]、久恒　辰博[1]
[1]東京大院・新領域・先端生命

P2-136 ニューロンにおけるショウジョウバエMAGEの発現解析
Expression of *Drosophila* MAGE in brain neurons
清水　尚子[1]、西村伊三男[1]、吉川　和明[1]
[1]大阪大・蛋白質研究所・機能制御部門

P2-137 Delta細胞内ドメインは神経分化に関するシグナルを伝える
Intracellular domain of delta provides signaling for neurogenesis
平栃　正博[1,2]、林　健太郎[1]、倉持　有[2]、今村　健志[3]、中山　耕造[1]
[1]信州大院・医・加齢適応医科学・分子腫瘍学、[2]キッセイ薬品工業（株）、[3]癌研究会

P2-138 前脳のOlig2陽性細胞はGABA作動性ニューロンやオリゴデンドロサイト前駆細胞に分化する
Olig2 positive cells differentiate into GABAergeic neurons and/or Oligodendrocyte progenitors in the forebrain
古性　美記[1,2]、竹林　浩秀[2]、政平　訓貴[2,3]、小野　勝彦[2]、池中　一裕[2]
[1]総研大・生命科学・生理化学、[2]自然科学研究機構・生理研・分子神経生理、[3]高知大・医・脳神経外科

P2-139 脳虚血後の霊長類海馬における新生ニューロンの増加
Enhanced Neurogenesis in the Dentate Gyrus of Non-Human Primates after Focal Ischemia
纐纈　大輔[1]、古市　泰久[2]、前田　雅志[2]、松岡　信也[2]、宮本　有正[1]、久恒　辰博[1]
[1]東京大院・新領域・先端生命、[2]藤沢薬品工業株式会社・薬理研究所

P2-140 In vitroにおける小脳プルキンエ細胞樹状突起形成過程の経時観察
Time-lapse observation of dendrite formation of cerebellar Purkinje cells in vitro
田中 正彦[1]、柳川右千夫[2,3]、小幡 邦彦[4]、丸野内 棣[1]
[1]藤田保健衛生大・総医研・応用細胞学、[2]群馬大院・医・遺伝発達行動学、[3]科学技術振興機構・CREST & SORST、[4]理研・脳センター

P2-141 EmxとDlx発現細胞の細胞系譜解析
In utero fate mapping of Emx- and Dlx-expressing cells in the mouse forebrain
鹿川 哲史[1]、荒木 喜美[2]、田賀 哲也[1]
[1]熊本大・発生研・転写制御、[2]熊本大・発生研・臓器形成

P2-142 発生期ニワトリ網膜の双極細胞とアマクリン細胞で発現している新規bHLH型転写抑制因子NeuroABの解析
A novel basic helix-loop-helix (bHLH) transcription repressor, NeuroAB, expressed in bipolar and amacrine cells in the developing chick retina
大河原 剛[1,2]、新谷 隆史[1,2]、三枝 智香[1,2]、湯浅一河田純一[1,2]、高橋 正和[1,2]、野田 昌晴[1,2]
[1]大学共同利用機関法人・自然科学研究機構・基礎生物学研究所・感覚情報処理研究部門、[2]CREST, JST

P2-143 生後海馬由来培養スライスにおける新生細胞の形質
Phenotypes of postnatally generated cells in hippocampal slice culture
鎌田 真希[1]、李 仁勇[1]、橋本 美香[1,2]、角田 雅昭[1,2]、岡田 広司[3]、小柳 義夫[4]、石塚 徹[1,2]、八尾 寛[1,2]
[1]CREST・科学技術振興機構、[2]東北大院・生命・脳機能解析、[3]東北大院・医・微生物、[4]京都大・ウイルス研・感染病態

P2-144 CreER™/loxPシステムを用いたOlig2発現細胞の細胞系譜解析
A long-term lineage analysis of Olig2-expressing cells by CreER™/loxP system
政平 訓貴[1,2]、丁 雷[1]、竹林 浩秀[1]、小野 勝彦[1]、清水 惠司[2]、池中 一裕[1]
[1]岡崎国立共同研究機構・生理学研究所・分子神経生理、[2]高知大・医・脳神経機能統御学教室

P2-145 Notchシグナルによる未分化神経幹細胞から神経幹細胞への分化制御
Primitive neural stem cells from the mammalian epiblast differentiate to definitive neural stem cells under the control of Notch signaling
等 誠司[1,2]、Raewyn Seaber[2]、Derek Van Der Kooy[2]、池中 一裕[1]
[1]生理研・分子神経生理、[2]Dept. Med. Genet. Microbiol., Univ. Toronto, Toronto, Canada

P2-146 生後初期海馬歯状回に存在する神経前駆細胞の性質とその細胞運命
Nature, migration and fate of hilar proliferating cells in the postnatal rat dentate gyrus
難波 隆志[1,2]、並木 秀男[2]、石 龍徳[1]
[1]順天堂大・医・解剖学、[2]早稲田大・院・理工・生命理工

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P2-147 Gab1を介したEGFシグナルによるOlig2陽性神経系前駆細胞の選択的な増殖
Gab1 mediates EGF signal inducing selective proliferation of Olig2-expressing neural progenitor cells in the developing spinal cord
早川 佳芳[1,3]、西田 圭吾[2]、平野 俊夫[2]、島崎 琢也[1,3]、岡野 栄之[1,3]
[1]慶應大・医・生理、[2]大阪大院・医・腫瘍病理、[3]科技団・戦略

P2-148 神経分化におけるL3/Lhx8の役割
Search for roles of the L3/Lhx8 on retinoic acid-induced neural differentiation in neuro2A by in vitro approaches using the siRNA
眞部 孝幸[1]、辰巳 晃子[1]、松吉ひろこ[1]、井上 政秀[1]、和中 明生[1]
[1]奈良医大・医・第二解剖

P2-149 海馬神経新生へのPax6の関与
Pax6 haploinsufficiency impairs hippocampal neurogenesis
前川 素子[1,2]、湯浅 茂樹[2]、大隅 典子[1]
[1]東北大院・医・形態形成解析、[2]国立精神神経センター・神経研究所・微細構造研究部

P2-150 コンドロイチナーゼABCを用いたin vitroにおけるヒト神経幹細胞/前駆細胞のコリン作動性ニューロンへの分化誘導法の開発
In vitro differentiation of cholinergic neurons from human neural stem cell/progenitor cell (hNSPC) using chondroitinase ABC
児玉 恵理[1]、山崎 智彦[1]、山本 篤世[1]、森 英樹[1]、三宅 淳[1]、金村 米博[1]
[1]産総研・セルエンジニアリング

P2-151 後脳と脊髄でみられるOlig3系譜細胞の腹側方向への移動
Dorsal-to-ventral migration of Olig3 lineage cells in the fetal mouse hindbrain and spinal cord
丁 雷[1]、竹林 浩秀[1]、田中 謙二[1]、小野 勝彦[1]、千坂 修[2]、池中 一裕[1]
[1]自然科学研究機構・生理学研究所・分子神経生理、[2]京都大院・生命科学研究科・統合生命科学専攻

P2-152 マウス大脳皮質および線条体のチロシン水酸化酵素mRNA発現細胞
Cortical and striatal expression of tyrosine hydroxylase mRNA in neonatal and adult mice
齋野一齋藤幸子[1,4]、小林 和人[2]、岡野 栄之[3]、後藤 薫[1]、Harriet Baker[4]
[1]山形大・医・組織細胞生物、[2]福島県立医大・生体機能、[3]慶応大・医・生理、[4]Burke Med. Res. Inst., New York, USA

P2-153 発達期のラット脳におけるATBF1とZFH4の発現の解析
Expression analysis of ATBF1 and ZFH4, the zinc-finger and homeodomain family proteins, in developing rat brains
石井 陽子[1]、野上 重治[1]、川口 誠[1]、尾矢 剛志[1]、笹原 正清[1]
[1]富山医科薬科大・医学部・病理学第二講座

P2-154 大脳皮質原基の非脳室面分裂細胞は、脳室面で生まれ、細胞周期進行中に脳室面離脱を決める。
Non-surface-dividing cortical progenitor cells: Their decision to leave the surface during cell cycle progression
宮田 卓樹[1,2,3]、齋藤加奈子[1,3]、小川 正晴[2]
[1]名大院・医・細胞生物、[2]理研・脳セ・細胞培養、[3]CREST・JST

P2-155 ショウジョウバエの神経特異的C2HC型Znフィンガー遺伝子 *dnzf* 変異体の解析
Mutation analysis of the *Drosophila* neural C2HC-type Zn finger gene *dnzf*
大迫 俊二[1]、橋爪 卓[1,2]、山田 隆文[1]
[1]東京都神経研・基盤技術、[2]東邦大院・理

P2-156 ホヤ中枢神経系特異的遺伝子プロモーターの体系的機能解析
Systematic analysis of central nervous system-specific promoters in ascidian tadpoles
日下部岳広[1]、吉田 麗子[1]、堀江 健生[1]、櫻井 大介[1]、川上 功[1]、津田 基之[1]
[1]兵庫県立大院・生命理学

P2-157 幼弱マウス小脳から調製したEGF応答性増殖細胞の解析
Characterization of the proliferating cells in early postnatal mouse cerebellum
岡野ー内田孝幸[1]、小宮 義璋[1]、石崎 泰樹[1]
[1]群馬大院・医・分子細胞機能

P2-158 プルキンエ細胞樹状突起の方向決定における顆粒細胞神経繊維束の役割：三次元的な小脳培養系での解析
Role of aligned neurite bundles of granule cells in determination of Purkinje cell dendrite orientation in 3-dimensional (3D) cerebellar cultures
永田 功[1]、木村ー黒田純子[1]
[1]東京都医学研究機構・都神経研・脳構造

P2-159 神経幹細胞の周辺環境を構築しているコンドロイチン硫酸プロテオグリカンの同定と機能
Chondroitin sulfate proteoglycans in the milieu of neural stem cell
伊田みちる[1]、周尾 卓也[1]、時田 義人[1]、中西 圭子[1]、松井ふみ子[1]、青野 幸子[1]、大平 敦彦[1]
[1]愛知県心身障害者コロニー・発達障害研究所・周生期学部

P2-160 神経興奮によるラット大脳皮質細胞の増殖と分化への影響について
The effect of neural activity on cell proliferation and differentiation in the cerebral cortex of adult rats
田村 泰久[1]、片岡 洋祐[1]、崔 翼龍[1]、小田ー望月紀子[1]、山田 久夫[1]
[1]関西医科大学・システム細胞科学

P2-161 成体マウス海馬歯状回におけるカイニン酸投与後のネスチン陽性細胞の動態
Dynamics of nestin-positive cells in the adult dentate gyrus after kainate administration
福田 諭[1]、戸塚 祐介[1]、宮本 有正[1]、久恒 辰博[1]
[1]東京大院・新領域・先端生命・細胞応答化学

P2-162 ダブルトランスジェニックマウスを用いた凍結脳損傷後におけるOlig2発現細胞の動態
The lineage tracing of Olig2 expressing cells in the adult mice brain after cryo-injury using tamoxifen inducible Cre/loxP system
辰巳 晃子[1]、竹林 浩秀[2]、眞部 孝幸[1]、池中 一浩[2]、和中 明生[1]
[1]奈良医大・医・解剖学、[2]自然科学研究機構・生理学研究所・分子神経生理研究部門

Neuro 2004

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P2-163 マウス中枢神経系発生過程におけるシスタチンCの発現と機能解析
The expression and functional studies of Cystatin C in the developing mouse central nervous system
成瀬 雅衣[1,2]、長谷川明子[2]、等 誠司[2]、池中 一裕[2]
[1]総合研究大学院大学・生理学研究所・分子神経生理、[2]自然科学共同研究機構・生理学研究所・分子神経生理

P2-164 G1/S 期細胞抑制剤による神経への分化誘導
Neuronal differentiation is promoted by treatment with G1/S cell cycle blockers to neurospheres
Hye-Jung Kim[1]、飛田 秀樹[1]、鄭 且均[1]、西野 仁雄[1]
[1]・医・脳神経生理

P2-165 新規神経特異的遺伝子ファミリー, BRINPの機能解析
Functional Characterization of a Novel Neuron-Specific Protein Family, BRINP
松岡 一郎[1,2]、森 卓哉[2]、小林三和子[1]
[1]北海道大・創成科学研究機構、[2]北海道大院・薬学研究科

P2-166 生後ラット延髄および知覚神経節におけるGAP-43の分布変化
Distribution of GAP-43 in the medulla oblongata and sensory ganglion of rat during postnatal development
村田 祐造[1]、李 明子[1]、増子 貞彦[1]
[1]佐賀大・医・生体構造機能学講座

P2-167 妊娠ラット母体に投与した植物性エストロゲン様物質Genisteinの青斑カテコールアミン神経核に及ぼす影響
Influence of prenatal exposure of Genistein, a phytoestrogen, on the catecholaminergic neurons in the locus coeruleus in the rat
井岡 真基[1]、片岡 洋佑[1]、山田 久夫[1]
[1]関西医科大学・システム細胞科学（解剖学第一）

P2-168 FGF2による神経幹細胞分化制御機構の解析
FGF2 signaling regulates proliferation and maintenance of multipotentiality of neural stem cells via the glycogen synthase kinase3β pathway.
清水 健史[1]、鹿川 哲史[1]、田賀 哲也[1]
[1]熊本大・発生研・転写制御

P2-169 神経特異的RNA結合蛋白質Huの生化学的解析
Biochemical study of neuronal RNA binding protein Hu
矢野 真人[1,2,3]、岡野ジェイムズ洋尚[1,3]、岡野 栄之[1,3]
[1]慶應大・医・生理学、[2]大阪大院・医・機能形態、[3]CREST・JST

P2-170 哺乳類新皮質において層特異的発現をする遺伝子の種間比較解析
A comparative analysis of layer-specific genes in mammalian neocortex
渡我部昭哉[1]、大澤 園子[1]、小松 勇介[1]、端川 勉[2]、山森 哲雄[1]
[1]基生研・種分化第一、[2]理研・脳センター・神経構築

P2-171 虚血誘導性神経新生に対するプロスタグランジンE2の効果
Effects of prostaglandin E2 on ischemia-induced neurogenesis
内田 克哉[1]、井樋 慶一[1]
[1]東北大院・情報・情報生物

P2-172 N-myc downregulated gene 1 (NDRG1) は出生後脳において分化段階の神経系細胞に発現する
N-myc downregulated gene 1 (NDRG1) is expressed in developing neural cells in the postnatal brain
鈴木 諭[1]、中島健太郎[1]、佐藤 卓士[1]、岩城 徹[1]、小野 眞弓[2]、桑野 信彦[2]
[1]九州大院・医・神経病理、[2]九州大院・医・生化学

P2-173 Notch-RBP-J シグナルは大脳皮質層形成を調節する
Notch-RBP-J signaling regulates cortical laminar fates.
谷垣 健二[1]、山本 典生[2]、辻 正幸[2]、本庶 佑[2]
[1]滋賀県立成人病センター 研究所、[2]京都大院・医・分子生物学教室

体性感覚
Somatosensory System

P2-174 ニホンザル第二体性感覚野手指再現領域は2つの領域に分けられる
Functional subdivisions of the hand representation in the Japanese monkey second somatosensory cortex
田岡 三希[1,2]、戸田 孝史[1,3]
[1]東邦大・医・第一生理、[2]東京医科歯科大・院・認知神経生物、[3]大阪大院・歯・口腔生理

P2-175 ヒスタミン誘発掻痒関連反応と一次感覚神経における細胞内カルシウム濃度に対するインターロイキン-2の影響
Effects of interleukin-2 on histamine-induced itch-associated responses and intracellular Ca^{2+} concentration in primary sensory neurons in mice
安東 嗣修[1]、榎本 愛[1]、今野 光洋[1]、野島 浩史[1]、倉石 泰[1]
[1]富山医薬大・薬・薬品作用

P2-176 fMRIを用いたヒト2次体性感覚野における皮膚局所擦過刺激の表象研究
Representation of The Secondary Somatosensory Cortex in local rubbing stimulation: an fMRI study
中越明日香[1]、福永 雅喜[2]、梅田 雅宏[2]、青木伊知男[2]、森 勇樹[1]、樋口 敏宏[1]、田中 忠蔵[1]
[1]明治鍼灸大学・脳神経外科、[2]明治鍼灸大学・医療情報学

P2-177 ラットの1本のヒゲを用いた刺激方向弁別課題における視床核VPmとPOmの機能の違い
Differential roles of the thalamic VPm and POm nuclei in a directional discrimination task with a single whisker of the rat.
鳴海 毅亮[1]、小山 知子[1]、高島 一郎[2]、覚 慎治[1]、飯島 敏夫[1]
[1]東北大院・生命科学研究科・脳情報処理、[2]独立法人産業技術総合研究所・脳神経情報研究部門

Sep 22 (Wed) Poster Session

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an even number 15:00～16:00

P2-178 メントールおよび冷刺激によるメダカの麻酔反応と異常遊泳行動
Anesthetic response and abnormal behavior to menthol and cold stimulation in rice fish
笠井 聖仙[1]
[1]鹿児島大学・理・生命化学科

P2-179 硬骨魚類コイの三叉神経感覚核の出力
Efferent connections of the sensory trigeminal nucleus in the carp
Hao-Gang Xue[1]、山本 直之[1]、楊 春英[1]、伊藤 博信[1]
[1]日本医科大学・解剖学第二教室

内臓感覚
Viscerosensory System

P2-180 スズキ型魚類ティラピアの糸球体前核交連部の線維連絡
Fiber connections of the nucleus preglomerulosus pars commissuralis in a cichlid fish, *Tilapia*.
吉本 正美[1]、山本 直之[1]、伊藤 博信[1]
[1]日本医科大学・解剖学第2

疾患モデル
Animal Models

P2-181 脳損傷後の機能回復に伴う運動皮質でのGAP-43発現
GAP-43 mRNA is increased in the motor cortex of the recovered brain-injured monkey
村田 弓[1,2]、肥後 範行[1]、大石 高生[3]、山下 晶子[4]、松田 圭司[1]、林 基治[3]
[1]産総研・脳神経情報、[2]筑波大院・人間総合科学、[3]京都大・霊長研・器官調節、[4]日大・医・応用システム神経科学

P2-182 フクチン欠損キメラマウスにおける大脳皮質層構築異常
Ectopic corticospinal and corticothalamic neurons in the cerebral necortex of *fukutin*-deficient chimeric mice
山本 達朗[1]、武田 聖[2]、美崎佳寿代[1]、村岡 大輔[1]、戸田 達史[3]、寺島 俊雄[1]
[1]神戸大院・医・脳科学・神経発生、[2]大塚GEN研究所、[3]大阪大院・医・ポストゲノム疾患解析学・ゲノム機能

P2-183 脊髄損傷モデルにおける損傷程度と客観的運動障害評価法の有効性の比較
Differential evaluations of rat hind-limb function after spinal cord injury
濱本雄一郎[1]、尾形 直則[1]、森野 忠夫[1]、堀内 秀樹[1]、山本 晴康[1]
[1]愛媛大・整形外科

P2-184 白血病阻害因子の脳内投与によるドパミン・シグナルおよび潜在制止の異常
Central Administration of Leukemia Inhibitory Factor Disrupts Dopaminergic Signaling and Latent Inhibition
渡部雄一郎[1,2]、武井 延之[1]、柿田 明美[3]、染矢 俊幸[2]、那波 宏之[1]
[1]新潟大・脳研・分子神経生物、[2]新潟大院・医歯・精神医学、[3]新潟大・脳研・脳疾患標本解析

P2-185 線条体における上皮成長因子はドパミン代謝と機能に関与する
Striatal Epidermal Growth Factor/ErbB1 Signal Alters Dopamine Metabolism and Functions
水野　誠[1]、鈴木　雅子[1]、江田　岳誉[1]、金　善和[1]、那波　宏之[1]
[1]新潟大・脳研究所・分子神経生物

P2-186 リーラーヘテロマウス海馬におけるNMDA受容体の検討
NMDA receptors are increased in the hippocampus of heterozygous Reeler mice.
伊早坂智子[1]、服部功太郎[1,2]、八木　健[1,2]
[1]大阪大院・生命機能研究科、[2]科学技術振興機構

P2-187 *Als2*ノックアウトマウスの作出と解析
Generation and characterization of the *Als2* knockout mice
秦野　伸二[1,2]、角田　茂[3]、須藤カツ子[3]、大友　麻子[1]、國田　竜太[2]、水村　光[2]、鈴木　恭子[1]、岩倉洋一郎[3]、池田　穣衛[1,2]
[1]東海大・総医研・分子神経、[2]発展・科技、[3]東大・医科研

P2-188 脳に特異的に発現するプロテオグリカン NGC のノックダウンマウスにおける行動異常
Mice with down-regulated expression of neuroglycan C, a CNS-specific transmembrane proteoglycan, show abnormal behaviors
青野　幸子[1]、黒田　喜幸[1]、時田　義人[1]、周尾　卓也[1]、松井ふみ子[1]、伊田みちる[1]、中西圭子[1]、平野佳奈子[1]、小野　教夫[2]、孫田　信一[2]、伊川　正人[3]、岡部　勝[3]、大平　敦彦[1]
[1]愛知県コロニー・研・周生期、[2]愛知県コロニー・研・遺伝、[3]大阪大学遺伝情報実験センター・遺伝子機能解析分野

P2-189 糖尿病モデル動物・GKラットの膵島における開口放出関連遺伝子発現の変化
Alterations in the expression of genes involved in exo- and endo-cytosis in the pancreatic islets of the diabetic GK rats
八田真以子[1]、永松　信哉[2]、田代　朋子[1]
[1]青山学院大・理工・化学生命科学、[2]杏林大・医・第2生化学

P2-190 Cre/loxPシステムを用いたα-synucleinトランスジェニックマウスの作製・解析
Generation and analysis of α-synuclein transgenic mice using a Cre/loxP system
安田　徹[1]、多田　昇弘[2]、小林　和人[3]、喜納　裕美[1]、望月　秀樹[1]、水野　美邦[1]
[1]順天堂大・医・脳神経内科、[2]順天堂大・医・アトピー疾患研究センター、[3]福島県立医大・生体情報伝達研

P2-191 共焦点蛍光顕微鏡を用いたラット胎児期腸管におけるPGP9.5陽性神経系構築過程の免疫組織学的検討
The development of PGP9.5-immunopositive neural network structure using the confocal laser scanning microscopy in the embryonic rat intestine.
棚野　晃秀[1,2]、山田　久夫[1]、高御堂祥一郎[1]、片岡　洋祐[1]、渡辺　淳[1]、濱田　吉則[2]、上山　泰男[2]
[1]関西医科大学・システム細胞科学、[2]関西医科大学・外科学

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an even number 15:00～16:00

P2-192 サリドマイド、バルプロ酸を用いた自閉症モデルラットにおけるセロトニン神経系の異常
thalidomide- or valproic acid- induced autism model rats show serotonergic abnormalities
成田奈緒子[1]、成田　正明[1]
[1]筑波大・基礎医学系・解剖学

P2-193 自閉症モデル動物における甲状腺ホルモンの関与
Animal model of autism: possible implication of thyroid hormone
定松　美幸[1]、金井　裕彦[1]、黒川　清[2]、加藤　進昌[3]
[1]滋賀医大・精神、[2]滋賀医大・解剖、[3]東京大院・医・神経精神科

P2-194 ラット線条体へのガンマナイフ照射法についての検討
An experimental targeting protocol for gamma knife irradiation on rat brain.
神山　暢夫[1]、片山　洋子[1]、富田美穂子[1]、徳丸　治[1]、林　基弘[2]、川上　順子[1]
[1]東京女子医大・医・第一生理学、[2]東京女子医大・医・脳神経外科学

発育、発達障害
Developmental Disorders

P2-195 発達期のサル大脳新皮質におけるneurograninの遺伝子発現変化
Development of neurogranin mRNA in the monkey cerebral neocortex
肥後　範行[1]、大石　高生[1,2]、山下　晶子[3]、村田　弓[4]、林　基治[2]
[1]産総研・脳神経情報、[2]京都大・霊長研・器官調節、[3]日大・医・応用システム神経科学、[4]筑波大院・人間総合科学

P2-196 二次元電気泳動による新生仔期セロトニン枯渇モデルラットの脳におけるプロテオム解析
Proteomic analysis in rat brain with neonatal serotonin depletion using two-dimensional electrophoresis
李　慶華[1]、野崎　聡[1]、水間　広[1]、渡辺　恭良[1]
[1]市大院・医・システム神経科学

P2-197 放射線により神経細胞の遊走障害とL1 moleculeの関連
Relationship between neuronal migration and expression of L1 molecule in the brain following prenatal exposure to irradiation
孫　学智[1]、張　鋭[2]、崔　春[2]、高橋千太郎[1]、久保田善久[1]、澤田　和彦[2]、福井　義浩[2]
[1]放医研・比較環境グループ、[2]徳島大・医・発生発達医学講座・機能解剖学

P2-198 転写抑制蛋白RP58は皮質層形成と皮質－視床経路形成に必要である。
Requirement of a POZ/zinc finger transcriptional repressor, RP58, for cortical lamination and pathway formation
岡戸　晴生[1]、川野　仁[2]、松田潤一郎[3]、寺島　俊雄[4]、葛西　正孝[5]
[1]東京都神経科学総合研究所・分子神経生理、[2]東京都神経科学総合研究所・発生形態、[3]国立感染症研究所・獣医科学、[4]神戸大院・解剖、[5]国立感染症研究所・免疫

P2-199 妊娠末期にあるラットの母体および胎児視床下部室傍核におけるFos発現に対する慢性ストレスの影響
Effects of repeated stress on Fos expression in the maternal and fetal rat hypothalamic paraventricular nucleus in late pregnancy
戸部　郁代[1]、石田　佳幸[1]、田中　増郎[1]、遠藤　久[1]、富士岡　隆[1]、中村　彰治[1]
[1]山口大・医・高次神経科学講座

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P2-200 オリジナルオリゴDNAマイクロアレイを用いたマウス小脳における周産期期甲状腺機能低下症の影響
Effect of perinatal hypothyroidism on gene expression in the developing mouse cerebellum studied with an original oligoDNA microarray
高橋 理貴[1,3]、今村 誠[1,3]、近藤 恭光[2]、田代 英夫[2]、田代 朋子[1,3]
[1]青山学院大・理工・化学生命科学、[2]理化学研究所・田代分子計測工学研究室、[3]行政独立法人 科学技術振興事業

P2-201 周産期甲状腺機能攪乱による注意欠陥多動性障害様行動の惹起
Attention-deficit and hyperactive neurobehavioural characteristics by perinatal hypothyroidism in rats
根岸 隆之[1,5,6]、須崎 真悟[2]、高橋 理貴[1,6]、今村 誠[1,6]、川崎 勝義[3,6]、石井 寿幸[2]、田代 朋子[1,6]、黒田洋一郎[4,6]、吉川 泰弘[2,6]
[1]青山学院大学・理工・化学生命科学科、[2]東京大院・農学生命科学研究科、[3]星薬科大学・心理、[4]東京都立神経研、[5]日本学術振興会・特別研究員、[6]CREST・JST

P2-202 胎児期および哺乳期における仔ラットへのビスフェノールA曝露の影響 －機序について－
Perinatal exposure to BPA affects ER expression in rat hippocampus
許 暁彬[1,2]、劉 影[1]、牛島 廣治[2]、加藤 進昌[1]
[1]東京大院・医・精神神経科、[2]東京大院・国際保健学発達医科学

P2-203 ビスフェノールAの胎生期曝露はラット脳機能の性分化を障害する
Fetal exposure to bisphenol A impairs sexual differentiation of brain functions in rats
藤本 哲也[1]、久保 和彦[2]、粟生 修司[1]
[1]九州工大院・生命体工学・脳情報、[2]九州大院・医学研究院・耳鼻咽喉科学

変性疾患
Neurodegenerative Diseases (Parkinson's Disease and others)

P2-204 線条体へのMPP+注入後の黒質における酸化的ストレスとミクログリア活性化
Oxidative stress and microglial activation in the substantia nigra following intrastriatal injection of MPP+
三輪 英人[1]、久保 友美[1]、森田 修平[1]、中西 一郎[1]、近藤 智善[1]
[1]和歌山県立医大・神経内科

P2-205 MPTPによるマウス黒質ドパミンニューロンの酸化障害と酸化プリンヌクレオシド三リン酸分解酵素MTH1による神経保護作用
MPTP-induced oxidative damage in dopaminergic neurons of mouse substantia nigra and neuro-protection by MTH1 encoding an oxidized purine nucleoside triphosphatase
山口 浩雄[1]、古市 正人[1]、大野みずき[1]、作見 邦彦[1]、中別府雄作[1]
[1]九州大・生医研・脳機能

P2-206 ^{1}H-MRSによるMPTP誘発パーキンソン病モデルマウスのin vivo解析
^{1}H-MRS identify changes in energy metabolism in the striatum of MPTP-treated C57BL/6 mice
中井 正三[1]、古賀けい子[1]、石川 誠[1]、森 厚詞[1]、大橋 聡[1]、光本 泰秀[1]
[1]大塚製薬（株）・探索第二研究所

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P2-207 6-Hydroxydopamine による Bip/Grp78 の発現誘導における p38 MAP kinase の関与
Induction of Bip/Grp78 by 6-hydroxydopamine (6-OHDA) is regulated by p38 MAPK activation in human neuroblastoma SH-SY5Y cells
山室 晶子[1]、桑田るみこ[1]、岸野 高志[1]、吉岡 靖啓[1]、荻田喜代一[2]、前田 定秋[1]
[1]摂南大・薬・薬物治療学、[2]摂南大・薬・薬理学

P2-208 MPP+で障害された培養ドーパミンニューロン終末の再生について
Recovery of dopaminergic neurons damaged by 1-methyl-4-phenylpyridinium in vitro
西 克典[1]
[1]東京都神経研・神経学

P2-209 小胞体関連分解 (ERAD) 関連分子ヒトHRD1によるPael-Rの分解
Degradation of Pael-R by human HRD1 involved in ER-associated Degradation (ERAD)
大村 友博[1]、金子 雅幸[1]、村橋 果林[1]、野村 靖幸[1]
[1]北海道大院・薬・薬理学

P2-210 ドパミン，レボドパはpolyubiquitinationを促進する －パーキンソン病におけるレビー小体形成機序
Dopamine and levodopa promote polyubiquitination in dopaminergic cells: possible mechanisms in Lewy body formation in Parkinson disease
吉本 祐子[1]、中曽 一裕[1]、中島 健二[1]
[1]鳥取大・医・脳神経内科 大学院医学系研究科

P2-211 初代培養マウス中脳神経細胞におけるミトコンドリア機能阻害の影響
Effect of mild mitochondrial dysfunction on primary cultured mouse mesencephalic neurons
今村 誠[1,2]、根岸 隆之[1,2]、田代 朋子[1,2]
[1]青山学院大・理工・化学生命科学、[2]独立行政法人 科学技術振興機構

P2-212 視床β帯域リズム活動は状況依存的にパーキンソン病固縮を誘起する
Thalamic β-band rhythmic activities contingently induce the parkinsonian rigidity
大島 知一[1]、楢林 洋介[1]
[1]楢林記念神経学ラボラトリー・楢林神経内科クリニック

P2-213 正常プリオン蛋白質安定発現HEK293における小脳変性関連遺伝子発現亢進
Upregulation of cerebellar degeneration-related genes in HEK293 following stable expression of the cellular prion protein PrPC
佐藤 準一[1]、尾上 祐行[1]、山村 隆[1]
[1]国立精神神経センター・神経研・免疫

P2-214 Hsp90βによる正常型プリオン蛋白質高次構造変換機構の解析
Hsp90β modifies the conformation of recombinant mouse prion protein *in vitro*
逆瀬川裕二[1,2]、八谷 如美[1,2]、金子 清俊[1,2]
[1]国立精神神経センター・神経研・疾病7部、[2]科学技術振興機構・CREST

P2-215 HD遺伝子プロモーター内の新規シスエレメントに結合する核－細胞質シャトルタンパク質の分子機能
Molecular function of nuclear-cytoplasmic shuttle proteins which bind to the novel *cis*-element in the *HD* gene promoter
田中　一則[1]、宮本なつき[2]、池田　穣衛[2,3]
[1]長寿科学振興財団、[2]科技団・SORST、[3]東海大・総医研

P2-216 変異GFAPの動態
Analysis of mutated GFAP dynamics
田中　謙二[1]、竹林　浩秀[1]、池中　一裕[1]
[1]生理学研究所・分子神経生理

P2-217 CMT病にみられるNFL変異の線維構造に与える影響
Analysis of neurofilament-L mutations linked to Charcot-Marie-Tooth disease on filament assembly
吉田　千枝[1]、佐々木孝寛[1]、浅田　明子[1]、斎藤　太郎[1]、久永　真市[1]
[1]東京都立大院・理・神経分子機能

P2-218 ポリグルタミンの細胞内導入により誘導される小胞体カルシウム応答の変化
Perturbed calcium regulation of endoplasmic reticulum in a peptide-delivery model of polyglutamine-induced neuronal death
鈴木　マリ[1]、小池　達郎[1]
[1]北海道大院・理・生物科学

P2-219 伸長 polyQ-EGFP 融合タンパク質凝集体を形成するハンチントン病トランスジェニックマウスにおける視床下部神経ペプチドの発現の低下
Decreased expression of hypothalamic neuropeptides in Huntington disease transgenic mice with expanded polyQ-EGFP fluorescent aggregates
小山　文隆[1]、黒澤　大[1]、宮崎　晴子[1]、土井　宏[1]、町田　陽子[1]、貫名　信行[1]
[1]理化学研究所・脳科学総合研究センター・構造神経病理研究チーム

P2-220 精製ポリグルタミン凝集体中におけるユビキチン結合タンパク質の同定
Identification of Ubiquitin-interacting proteins in the purified polyglutamine aggregates
土井　宏[1,2]、三井　健一[3]、黒沢　大[1]、町田　陽子[1]、黒岩　義之[2]、貫名　信行[1]
[1]理研・脳科学総合研究センター・構造神経病理研究チーム、[2]横浜市大院・医・神経内科学、[3]理研・脳科学総合研究センター・リサーチリソースセンター

P2-221 神経変性疾患におけるユビキチン陽性封入体の構成成分であるDorfinとVCPの機能的解析
Analysis of interaction between Dorfin and VCP, forming ubiquitylated inclusions in neurodegenerative disorders
石垣　診祐[1,2]、菱川　望[1]、丹羽　淳一[1]、垣塚　彰[3]、田中　啓二[2]、祖父江　元[1]
[1]名古屋大院・医・神経内科、[2]都臨床研・分子腫瘍、[3]京都大院・生命科学・高次生体統御

P2-222 PQBP-1 トランスジェニックマウスにおけるミトコンドリア異常
Mitochondrial dysfunction in PQBP-1 transgenic mice
丸淵　茂樹[1,2]、田川　一彦[1]、奥田　智博[2]、中川　昌也[2]、岡澤　均[1,3]
[1]東京医科歯科大学・難研・神経病理、[2]富山化学工業株式会社、[3]科学技術振興機構・戦略的創造研究推進事業・さきがけ21

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P2-223 リコピン経口摂取は運動ニューロン変性（*Mnd*）マウスの四肢麻痺の発現を遅延させる
Per oral plant-lycopene administration can delay the progression of paralysis of the limbs in motor neuron degeneration (*Mnd*) mutant mouse during postnatal growth
芳本　信子[1,2]、藤田　公和[2]、猪飼　弘子[1]、菅沼　大行[3]、稲熊　隆博[3]、今田　英己[2]、村上　洋子[1]、永田　豊[2]、宮地　栄一[2]
[1]名古屋文理短大、[2]藤田保健衛生大・医・生理学II、[3]カゴメ研究所

P2-224 Dishevelled-1（Dvl-1）に対する新規E3ユビキチンリガーゼNEDL1はスーパーオキシドジスムターゼ1（SOD1）変異体をも標的とするが、家族性筋萎縮性側索硬化症（FALS）において細胞内凝集体の形成原因となる
NEDL1, a novel E3 ubiquitin ligase for Dishevelled-1 (Dvl-1), targets mutant SOD1 to form intracellular aggregates in familial amyotrophic lateral sclerosis (FALS)
宮崎　耕[1]、尾崎　俊文[1]、加藤　信介[2]、糸山　泰人[3]、青木　正志[3]、中川原　章[1]
[1]千葉県がんセンター・生化学、[2]鳥取大・医・脳研・神経病理、[3]東北大院・医学研究科・神経内科

P2-225 AMPA受容体を介した神経細胞死によるALSモデルラットの開発
An ALS rat model by AMPA receptor-mediated excitotoxicity
郭　伸[1]、孫　慧[1]、河原　行郎[1]、伊藤　杏子[1]
[1]東京大院・医・神経内科

P2-226 脱髄におけるセリンプロテアーゼProtease M/neurosinの発現
Expression of serine protease protease M/neurosin in demyelination and remyelination
板東　良雄[1]、伊藤　慎治[1]、永井　美子[1]、寺山　隆司[1]、吉田　成孝[1]
[1]旭川医大・医・解剖学第1

P2-227 反復経頭蓋磁気刺激法による発現変動遺伝子の解析（No.2）
GeneChip analysis (Part.2) of mouse brain stimulated by repetitive Transcranial Magnetic Stimulation
池田　哲朗[1]、黒沢　大[1]、内川　千春[1]、貫名　信行[1]
[1]理研・脳研・構造神経病理チーム

P2-228 免疫抑制薬タクロリムスの神経保護作用における内在性結合タンパク質FKBPに関する研究
A role of FK506-related immunophilines for neuroprotective properties FKBP-binding immunophilin ligands
田中　健一[1]、片山布美子[1]、福原　薫子[1]、小川　紀雄[2]
[1]就実大・薬・薬物治療、[2]岡山大院・医歯・神経情報

P2-229 ReversAl of Orofacial Dyskinesia by FK506
Amanpreet Singh[1]、PS. Naidu[1]、S K. Kulkarni[1]
[1]UIPS Panjab University Chandigarh, India

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精神神経疾患
Neuropsychiatric and Neurological Diseases

P2-230 成体マウス海馬歯状回におけるBrdU取り込みのストレスによる調節
Modulation by stress of BrdU incorporation in adult mouse dentate gyrus
玉置 啓祐[1]、山田 清文[1]、米山 雅紀[1]、米田 幸雄[1]
[1]金沢大院・自然科学・病院薬学

P2-231 ワーキングメモリーのダイナミクスと精神疾患患者の認知障害との関係性について：計算実験を用いた研究
Relationship between the dynamics of working memory and the emergence of cognitive deficits in psychiatric patients: A computational study
野村 健介[1]、山下 幸樹[2]、加藤元一郎[1]、鹿島 晴雄[1]、田中 昌司[2]
[1]慶應大・医・精神神経科学、[2]上智大・理工・電気電子工学

P2-232 フェンシクリジン連続投与マウスに認められる認知障害とCa2+/calmodulin kinaseIIの関与
Repeated treatment with phencyclidine impairs latent learning in mice: association with the cortical Ca2+/calmdulin kinase II
毛利 彰宏[1]、野田 幸裕[1]、野田 明宏[1]、古川 宏[2]、鍋島 俊隆[1]
[1]名古屋大院・医療薬学・薬剤部、[2]名城大・薬・薬化学研究室

P2-233 phencyclidine (PCP) の全身性投与による効果は腹側海馬へのPCP局所潅流により再現される
Effects of systemically administered phencyclidine can be mimicked by the local perfusion in the ventral hippocampus
浄土 英一[1]、鈴木 喜明[2]、片山 規央[2]、香山 雪彦[1]
[1]福島県立医大・医・第2生理、[2]福島県立医大・医・神経精神科

P2-234 脳由来神経栄養因子 (BDNF) による伝達物質放出作用に抗うつ薬が与える影響
Antidepressants enhance BDNF-induced release of glutamate in cultured cortical neurons.
矢ヶ崎有希[1]、沼川 忠広[1]、功刀 浩[1]
[1]国立精神神経センター・神経研究所・疾病研究第三部

P2-235 ラット大脳皮質前頭葉モノアミン遊離に対するフルボキサミン、スルピリド併用投与の影響
Effect of co-administration of sulpiride and fluvoxamine on extracellular monoamine levels in rat prefrontal cortex
吾郷由希夫[1]、中村 茂生[1]、馬場 明道[2]、松田 敏夫[1]
[1]大阪大院・薬・複合薬物動態、[2]大阪大院・薬・神経薬理

P2-236 マウス強制水泳試験における塩酸セレギリンの抗うつ様作用の検討
The antidepressant-like effect of selegiline hydrochloride in the mouse forced swimming test
南 亜紀子[1]、楠本 治子[1]、島津誠一郎[1]、米田 文郎[1]
[1]藤本製薬・創薬研

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P2-237 健常者における末梢型ベンゾジアゼピン受容体遺伝子多型と不安との関係
Genomic polymorphism of the peripheral-type benzodiazepine receptor (PBR) and its relation to the trait anxiety
中本百合江[1]、中村　和彦[2]、三辺　義雄[2]、森　　則夫[2]、古川　愛造[3]、山田　和男[4]、吉川　武男[4]、麦島　　剛[1]、吉井　光信[1]
[1]東京都精神研・神経可塑性、[2]浜松医大・精神神経、[3]香川医大・生化、[4]理研・脳科学・分子精神

P2-238 不安惹起薬がマウス脳内のBTG2mRNAに及ぼす影響
Effects of anxiogenic drugs on BTG2 mRNA in the mouse brain
車地　暁生[1]、伊藤　　卓[1]、石井　澄和[1]、西川　　徹[1]
[1]東京医科歯科大院・精神行動医科学

P2-239 幼少期Interleukin-1 α末梢投与により成熟後に引き起こされるラットの行動異常
Peripheral Administration of Interleukin-1α To Rat Pups Impairs Cognitive /Behavioral Development
任海　　学[1]、津田　法子[2]、渡部雄一郎[2]、那波　宏之[2]
[1]新潟大・脳研究所・システム脳生理学分野、[2]新潟大・脳研究所・分子神経生物学分野

P2-240 リチウム増強治療による海馬神経細胞新生の増加
Enhancement of hippocampal neurogenesis by lithium augmentation
中川　　伸[1]、北市　雄士[1]、井上　　猛[1]、李　　暁白[1]、小山　　司[1]
[1]北海道大院・医・精神医学

P2-241 低用量覚せい剤投与により内側前頭前野にみられた神経毒性変化
Subchronic senstization induced by a low dose of methamphetamine develops with the progress of neurotoxic morphological changes in the medial prefrontal cortex of the rat
門田　朋子[1]、門田　　健[2]、大道　正義[2]
[1]千葉大院・医・環境生命医学、[2]千葉市環境保健研究所

P2-242 Group II代謝型グルタミン酸受容体拮抗物質（MGS0039）によるマウス海馬での細胞新生亢進と抗うつ作用について
Increased cell proliferation in the adult mouse hippocampus following chronic administration of mGluR2/3 antagonist (MGS0039) with antidepressant-like effects
吉水　孝緒[1]、島崎　聡立[1]、奥野一広田志帆[1]、茶木　茂之[1]
[1]大正製薬（株）・医薬研究所・創薬薬理

P2-243 Behavioral and molecular changes of olfactory bulbectomized rats: effects of a sigma$_1$ receptor agonist, SA-4503
Dayong Wang[1]、Yukihiro Noda[1]、Hiroko Tsunekawa[1]、Toshitaka Nabeshima[1]
[1]Dept. Neuropsychopharmacol. & Hosp. Pharm., Nagoya Univ. Grad. Sch. Med., Nagoya, Japan

P2-244 PTSDモデルラットを用いた扁桃体におけるシナプス可塑性と恐怖条件付けに関する研究
Synaptic plasticity in LA and fear conditioning in PTSD model rats
幸田　和久[1,2]、加藤　邦夫[3]、原田　勝也[4]、山路　隆之[4]、松岡　信也[4]、柚崎　通介[1]、加藤　進昌[2]
[1]慶應大・医・生理学教室、[2]東京大院・医・精神神経科、[3]高知医科大・神経精神科、[4]藤沢薬品・薬理研究所

P2-245 中脳皮質辺縁系システムの回路シミュレーション
Circuit simulation of the mesocorticolimbic system
永井 秀治[1]、田中 昌司[1]
[1]上智大院・理工・電気電子工学専攻

P2-246 神経性食思不振症の体重回復に伴う脳血流変化
Cerebral Blood Flow Change following body weight recovery in Anorexia Nervosa: A SPECT study analysis using statistical parametric mapping
松本 良平[1]、北林百合之介[1]、成本 迅[1]、和田 良久[1]、横山ちひろ[1]、上田 英樹[1]、福居 顯二[1]
[1]京都府立医科大院・精神機能病態学

P2-247 MK801による内側前頭前皮質ニューロンの活動性亢進は非NMDA型グルタミン酸受容体により媒介されている
Activation of medial prefrontal cortex neurons by MK801 is mediated via non-NMDA glutamate receptors
片山 規央[1]、浄土 英一[2]、鈴木 喜明[1]、香山 雪彦[2]
[1]福島県立医大・医・神経精神医学、[2]第2生理

P2-248 統合失調症におけるCalDAG-GEFI遺伝子の多型解析
Genetic Analysis of the human striatum-enriched CalDAG-GEFI gene with Japanese schizophrenia
川嵜 弘詔[1]、光安 博志[1]、前田 直子[1]、小林 祐樹[1]、後藤 玲央[1]、松尾 葉子[1]
[1]九州大院・医・精神病態

P2-249 DISC1は中心体タンパクkendrinと結合する
DISC1 interacts with a centrosomal protein, kendrin
三好 耕[1]、大西 一成[1]、浅沼 幹人[1]、宮崎 育子[1]、Francisco Diaz[1]、小川 紀雄[1]
[1]岡山大院・医歯・脳神経制御・神経情報

P2-250 yeast two hybrid法によるdysbindin結合蛋白のスクリーニング
Search for dysbindin binding partners by yeast two-hybrid systemSearch for dysbindin binding partners by yeast two-hybrid system
熊本奈都子[1]、山口 淳[1]、松崎 伸介[1]、遠山 正彌[1]
[1]大阪大院・医・ポストゲノム疾患解析学講座

P2-251 統合失調症関連遺伝子dysbindinを用いたyeast two-hybrid法による解析
Identification of a novel binding partner for schizophrenia susceptibility gene dysbindin
松崎 伸介[1,2,3]、山口 淳[1,3]、熊本奈都子[1,3]、遠山 正彌[1,3]
[1]大阪大院・医・ポストゲノム疾患解析、[2]研究成果活用プラザ、[3]21世紀COEプロジェクト

P2-252 統合失調症関連遺伝子dysbindinはタンパク輸送に関連する
Dysbindin, one of schizophrenia susceptibility genes, is involved in protein trafficking
山口 淳[1]、松崎 伸介[1]、熊本奈都子[1]、遠山 正彌[1]
[1]大阪大院・医・ポストゲノム疾患解析学講座・21世紀COEプログラム

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P2-253 モデル動物を用いた環境因子の気分障害（うつ病）に対する効果
Enriched environment influences depression-related behaviors and hippocampal neurogenesis
服部 聡子[1]、橋本 亮太[1]、宮川 剛[2]、前野 浩巳[3]、和田 圭司[3]、功刀 浩[1]
[1]国立精神・神経センター神経研究所・疾病研究第三部、[2]京都大院・医・先端領域融合医学研究機構、[3]国立精神・神経センター神経研究所・疾病研究第四部

P2-254 ラット前頭葉におけるセロトニン終末とノルアドレナリン終末の加齢に伴う相互作用
Age-Dependent Interactive Changes in Serotonergic and Noradrenergic Cortical Axon Terminals in F344 Rats
中井 定[1]、松永 渉[1]、磯部 健一[1]、城川 哲也[1]
[1]国立長寿医療センター・老化機構研究部

P2-255 ダウン症モデルマウスにおけるミトコンドリア機能異常の解析
Mitochondrial dysfunction in brain of the Down syndrome mouse model; Ts1Cje
下畑 充志[1]、A. S. Ebrahim[1]、山田 一之[3]、天野 賢治[1]、赤木 巧[2]、竹内 環[1]、左合 治彦[4]、C. J. Epstein[5]、端川 勉[2]、山川 和弘[1]
[1]理研・脳センター・神経遺伝、[2]理研・脳センター・神経構築、[3]理研・脳センター・リサーチリソースセンター、[4]国立成育医療センター・胎児診療科、[5]Dept. Pediatrics, UCSF, USA

P2-256 Studying the neurodegenerative phenotype in Ts1Cje, a partial Trisomy 16 mouse model for Down Syndrome
Ebrahim, A Shukkur[1]、A, Shimohata[1]、T, Takeuchi[1]、T, Akagi[2]、M, Murayama[3]、K, Amano[1]、HS Karthik[1]、T, Hashikawa[2]、H, Sago[4]、C.J Epstein[5]、A, Takashima[3]、K, Yamakawa[1]
[1]Lab for Neurogenetics, BSI-RIKEN、[2]Lab for Neural Architech. BSI-RIKEN、[3]Lab for Alzheimer's Disease, BSI-RIKEN、[4]Div. of Fetal Med. NCCHD、[5]Dept. of Ped. UCSF

P2-257 ダウン症マウスモデル（Ts1Cje）では、トリソミー領域内の遺伝子特異的な過剰発現が見られる
Dosage-dependent over-expression of genes in the trisomic region of Ts1Cje mouse model for Down syndrome
天野 賢治[1]、左合 治彦[2]、内川 千春[1]、鈴木 太始[3]、S. E. Kotliarova[3]、貫名 信行[3]、C. J. Epstein[4]、山川 和弘[1]
[1]理研・脳センター・神経遺伝、[2]国立成育医療センター・周産期診療部・胎児診療科、[3]理研・脳センター・構造神経病理、[4]Dept Pediatrics, UCSF, USA

P2-258 ポリグルタミン凝集によるオートファジー形成とERストレスの関与
Polyglutamine aggregates induces autophagy via activation of ER stress
高鹿 依子[1]、藤田恵里子[1]、内山 安男[2]、木南 英紀[3]、桃井 隆[1]
[1]国立精神神経セ・5部、[2]大阪大院・医・機能形態、[3]順天堂大・医・生化一

P2-259 小胞体ストレス細胞死を抑制する新規化合物
A new compound that inhibits the ER stress-mediated cell death
桃井 隆[1]、高鹿 依子[1]、藤田恵理子[1]
[1]国立精神神経セ・5部

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筋疾患
Muscle Disorders

P2-260 造血器型プロスタグランディンD合成酵素阻害剤を用いた筋変性疾患の治療
Prevention of muscular necrosis by inhibition of hematopoietic prostaglandin D synthase
佐藤　陽[1]、有竹　浩介[2]、毛利　育子[3]、谷池　雅子[3]、裏出　良博[2]
[1]理研・播磨研、[2]大阪バイオサイエンス研、[3]大阪大院・医・小児科

虚血
Ischemia

P2-261 6-ハイドロキシドパミン投与によるラット黒質障害のMRIを用いた可視化
Visualization of 6-hydroxydopamine-induced nigral lesions in rats by magnetic resonance imaging
近藤　高史[1]、坂内　慎[2]、高橋　迪雄[2]、西野　仁雄[2]、鳥居　邦夫[1]
[1]味の素（株）ライフサイエンス研、[2]名市大院・脳神経生理

P2-262 虚血負荷により誘起されるラット脊髄後角のGABAとグリシンの自発放出増加
Enhancement of the release of GABA and glycine during ischemia in rat spinal dorsal horn
熊本　栄一[1]、川崎　康彦[1]、松本　徳昭[1]、藤田　亜美[1]、古賀亜希子[1]、中塚　映政[1]
[1]佐賀大・医・生体構造機能学

P2-263 脳虚血急性期に発現するIL-6の神経細胞保護効果
Neuroprotective role of endogenous IL-6 signaling in the ischemic mouse brain
山下　徹[1,2,6]、澤本　和延[1,6]、鈴木　重明[3]、安達　一英[1,4,6]、三原　昌彦[5]、大杉　義征[5]、阿部　康二[2]、岡野　栄之[1,6]
[1]慶應大・医・生理、[2]岡山大・医・神内、[3]慶應大・医・神内、[4]慶應大・医・脳外、[5]中外製薬、[6]科技団・戦略

P2-264 17βエストラジオールは脳虚血後の海馬の遅発性神経細胞死を抑制する
17βestradiol can attenuate delayed neuronal death in gerbil hippocampus
藤田　公和[1]、加藤　寿章[1]、今田　英己[1]、永田　豊[1]、宮地　栄一[1]
[1]藤田保衛大・医・生理

P2-265 実験的脳虚血モデルにおけるポリ（ADP-リボース）合成酵素siRNAによるミトコンドリア傷害の軽減
Attenuation of mitochondrial injury by poly（ADP-ribose）synthetase siRNA in experimental model of cerebral ischemia
田中　静吾[1]、竹橋　正則[1]、上田　國寛[1]
[1]京都大院・化研・分子臨床化学

P2-266 ラット一過性脳虚血におけるヒドロキシラジカルのin vivo検出 －微小透析法の応用－
In vivo detection of the cerebral hydroxyl free radicals in a rat model of transient focal cerebral ischemia -a microdialysis study-
兵藤　行志[1]、Julie Keirsse[1]、加藤　徳之[2]、浅川　弘之[2]、谷中　清之[2]
[1]産総研・人間福祉医工学、[2]筑波大・脳神経外科

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P2-267 局所脳虚血の神経保護におけるG protein-coupled estrogen receptorを介したγPKCの関与
Involvement of γPKC in neruroprotection on focal brain ischemia through G protein-coupled estrogen receptor
林 成人[1]、上山 健彦[2]、八木 圭子[3]、梶本 武利[1]、甲村 英二[4]、斉藤 尚亮[2]
[1]高砂市民病院・脳神経外科、[2]バイオシグナル研究センター分子薬理学教室、[3]神戸薬科大学、[4]神戸大学・医・脳神経外科

外傷 Trauma

P2-268 ナロキソンの損傷前よりの持続投与は脊髄損傷後の早期運動機能回復を改善する
Preinjury administration of naloxone promotes early functional recovery after spinal cord injury in adult rats
伊藤 博隆[1]、飛田 秀樹[1]、西野 仁雄[1]
[1]名市大院・医・脳神経生理

P2-269 ラット運動神経切断後の機能的シナプス再構築
Synaptic re-organization after motoneuronal axotomy in the rat
池田 亮[1,2]、藤井 克之[2]、加藤 総夫[1]
[1]慈恵医大・神経生理学研究室、[2]慈恵医大・整形外科学講座

P2-270 外傷性脳損傷後の造血器型PGD合成酵素の誘導
Induction of hematopoietic prostaglandin D synthase after traumatic brain injury
有竹 浩介[1]、谷池 雅子[2]、毛利 育子[2]、黄 志力[1]、佐藤 陽[1]、角山 圭一[1]、永田奈々恵[1]、江口 直美[1]、裏出 良博[1]
[1]財団法人大阪バイオサイエンス研究所・分子行動生物学部門、[2]大阪大院・医・小児科

P2-271 ラット脊髄損傷モデルにおけるChondroitinase ABCの効果的な投与方法の検討
Effective application of Chondroitinase ABC to spinal cord injury
池上 健[1]、中村 雅也[1]、山根 淳一[1]、岡田 誠司[2]、岩波 明生[1]、渡邉 航太[1]、石井 賢[1]、高橋 響[4]、戸山 芳昭[1]、岡野 栄之[2,3]
[1]慶大・整形、[2]慶大・生理、[3]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、[4]生化学工業（株）・中央研究所

老化、痴呆、アルツハイマー Aging, Dementia and Alzheimer's

P2-272 ミクログリア活性化に対するβアミロイド凝集の影響
The influence of the aggregational state of amyloid-β-peptides on microglial activation.
門司 晃[1]、橋岡 禎征[1]、植田 正[2]、吉田 一郎[1]、神庭 重信[1]、中西 博[3]
[1]九州大院・医・精神病態医学、[2]九州大院・薬・免疫薬品化学、[3]九州大院・歯・加齢口腔科学

P2-273 老齢犬脳におけるアミロイド前駆タンパク蛋白（APP）に関する免疫組織化学的検討―年令、痴呆評点との相関
Immunohistochemical study of APP in the old dog brain
伊藤 龍生[1]、佐藤 隆夫[1]、古河 恵一[2]、伊藤 浩行[1]
[1]近畿大学・医・病理学、[2]近畿大学・医・基礎医学

P2-274 免疫沈降LC/MS/MSを用いたAβ関連ペプチドの高感度分析
Sensitive analysis of Aβ related peptide with immunoprecipitation and liquid chromatography ion trap mass spectrometer
亀谷富由樹[1]
[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

P2-275 MK801投与Alzheimer病モデルラット脳における神経ペプチド発現変化と治療薬の効果
Changes of neuropeptides expression in MK-801 treated Alzhimer's model rat brain and the effects of therapeutic drugs.
隈部有希子[1]、星野　玄[1]、Mahiuddin Ahmed[1]、知久馬敏幸[2]、加藤　武[1]
[1]横浜市立大院・総合理・システム機能科学、[2]昭和薬科大学・衛生化学

P2-276 シンタキシン5はホロ蛋白プレセニリンと結合してβアミロイド産生を抑制する
Syntaxin 5 interacts with Presenilin holoproteins and suppresses the production of Aβ peptides
須賀　圭[1]、富山　貴美[2]、森　啓[2]、赤川　公朗[1]
[1]杏林大・医・細胞生理、[2]大阪市立大・医・脳神経科学

P2-277 膜脂質環境におけるAβ重合
Amyloid β-protein aggregation in the membrane-mimicking environment
山本　直樹[1]、長谷川一浩[2]、松崎　勝巳[3]、内木　宏延[2]、柳澤　勝彦[1]
[1]国立長寿医療セ・アルツハイマー病、[2]福井大・医・病理、[3]京都大院・薬・薬品機能解析

P2-278 アミロイドβペプチドの分泌に与えるERストレスの影響
ER stress influences the production of amyloid beta peptides
奥村　雅代[1]、工藤　喬[2]、荒木　亘[3]、今泉　和則[1]
[1]奈良先端大・バイオ・細胞構造、[2]大阪大院・医・精神医学、[3]国立精神セ・神研

P2-279 アルツハイマー病モデルマウス（Tg2576）における老人斑とPGD2関連性炎症
The PGD_2-mediated inflammation is enhanced around amyloid plaques in the Tg2576 mouse model of Alzheimer's disease
兼清　貴久[1]、谷池　雅子[1]、角山　圭一[2]、工藤　喬[3]、大薗　恵一[1]、裏出　良博[2]
[1]大阪大院・医・小児科、[2]大阪バイオサイエンス研究所・分子行動生物学部門、[3]大阪大院・医・精神科

P2-280 アルツハイマー・βアミロイド蛋白およびプリオン蛋白による細胞内カルシウムホメオスタシスの異常
Disruption of calcium homeostasis by Alzheimer's β-amyloid protein and prion protein
川原　正博[1]、木葉　敬子[1]、定金　豊[1]
[1]九州保健福祉大・薬・分析学

P2-281 βアミロイド蛋白が長期増強を阻害する機構について
On the mechanisms by which β amyloid peptides inhibit long-term potentiation
野村　泉美[1]、武地　一[1]、加藤　伸郎[2]、北　徹[3]
[1]京都大院・医・加齢医学、[2]京都大院・医・認知行動、[3]京都大院・医・循環器内科

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P2-282 Presenilinによる FKBP38の抗アポトーシス活性に対する抑制機能
Presenilins negatively regulate antiapoptotic activity of FKBP38
西村 正樹[1]、王 華芹[1]、中谷 嘉文[1]、山根 拓也[1]、白根 道子[2]、中山 敬一[2]
[1]滋賀医大・分子神経科学研究センター、[2]九州大・生体防御医学研究所

P2-283 アルツハイマー病における老人斑周辺グリア細胞でのプロスタグランジン D_2 を介した炎症反応
Prostaglandin D_2-mediated inflammation in glial cells associated with senile plaques in Alzheimer's disease.
角山 圭一[1]、毛利 育子[2]、佐藤 陽[1]、下野九理子[2]、齋藤 祐子[3]、大薗 恵一[3]、
村山 繁雄[3]、谷池 雅子[2]、裏出 良博[1]
[1]（財）大阪バイオサイエンス研究所・分子行動生物学部門、[2]大阪大院・医・小児科、[3]東京都老人総合研究所・老化臨床神経科学研究グループ

P2-284 プレセニリンC末端配列の欠失による Aβ産生の促進
Deletion of Presenilin C-terminal tail enhances Aβ generation.
白石 博久[1,2]、王 華芹[3]、西村 正樹[3]、柳澤 勝彦[1]、駒野 宏人[1]
[1]国立長寿医療セ・アルツハイマー病、[2]独立行政法人・医薬品医療機器総合機構、[3]滋賀医科大・分子神経セ・脳疾患基礎

P2-285 MAP1BのN末に結合する物質の探索
Search for the proteins which interact with N-terminal 126 amino acids of Microtubule-associated protein (MAP) 1B
五味不二也[1]、内田 洋子[1]
[1]東京都老人総合研究所・老年病ゲノム研究グループ

P2-286 微小管結合蛋白質タウと14-3-3の蛋白複合体形成について。
Microtubule Binding Protein Tau and 14-3-3 Form a Multi-protein Complex.
橋口美津子[1]、Hemant K. Paudel[2]、橋口 利雄[1]
[1]東京医大・医・生理学第一、[2]Department of Neurology and Neurosurgery, McGill University, Montreal, Canada

P2-287 サイクリン依存性キナーゼ5によるタウのリン酸化ーFTDP-17型変異の影響ー
Effect of FTDP-17 mutations on tau phosphorylation by cyclin-dependent kinase 5
坂上 史佳[1]、長谷川成人[2]、石黒 幸一[3]、浅田 明子[1]、斎藤 太郎[1]、久永 真市[1]
[1]都立大院・理・生物科学、[2]都精神研・分子神経生物学、[3]三菱化学生命科学研究所

P2-288 アミロイド前駆体蛋白質を含むマウス脳由来膜ラフトのプロテオーム解析
Proteomic analysis of mouse brain lipid rafts rich in amyloid precursor protein
櫻井 隆[1]、奥野弥佐子[1]、金子 貢巳[1]、和田 浩司[1]、貫名 信行[1]
[1]理化学研究所・脳科学総合研究センター・構造神経病理研究チーム

P2-289 ApoEはHDLによる神経細胞からのコレステロール搬出作用を抑制する
A Novel Action of Apolipoprotein E (ApoE) : ApoE Inhibits HDL-Mediated Cholesterol Release
龔 建生[1,2]、森田 真也[3]、服部 俊秀[4]、高 美姫[1]、于 文新[1]、Zou Kun[1]、
半田 哲郎[3]、柳澤 勝彦[1]、道川 誠[1]
[1]国立長寿医療センター研究所・アルツハイマー病研究部、[2]（独）医薬品医療機器総合機構、[3]京都大院・薬、[4]名古屋大院・生命農

P2-290 ネプリライシン遺伝子欠損によるAPPトランスジェニックマウスのin VIVO 海馬LTPの障害
Impairment of LTP in the Hippocampus of APP Transgenic Mice in VIVO by Neprilysin-Deficiency
黄　樹明[1]、岩田　修永[1]、関口みさき[1]、西道　隆臣[1]
[1]理化学研究所・脳研究センター・神経蛋白制御

P2-291 アルツハイマー型痴呆患者の前頭葉皮質における遺伝子発現の解析
Gene expression in frontal cortices of patients of Alzheimer-type dementia
梅村　賢[1]、山下　宣之[1,2]、有馬　邦正[1]、朝田　隆[3]、巻渕　隆夫[4]、村山　繁雄[5]、後藤　雄一[1]、高坂　新一[1]、金澤　一郎[1]、木村　英雄[1]
[1]国立精神神経センター、[2]明治製菓、[3]筑波大、[4]国立療養所犀潟病院、[5]東京都老人総研

P2-292 前頭葉と海馬回における青斑核由来ノルアドレナリン性神経末端の加齢変化
Different age-dependent changes of noradrenergic innervations in the hippocampus and frontal cortex
松永　涉[1]、城川　哲也[1]、磯部　健一[1]
[1]国立長寿医療センター・老化機構研究部

P2-293 cGMPによるAβ誘発性グルタミン酸取り込み上昇の抑制
Exogenous and endogenous cGMP inhibits upregulation of GLAST induced by β-amyloid in cultured astrocytes
三守　和彦[1]、山田　麻紀[1]、松木　則夫[1]
[1]東京大院・薬・薬品作用

P2-294 アセチルコリン依存性脳循環自動調節と記憶障害
Lost of acetylcholine dependent autoregulation of cerebral blood flow and memory disturbance in mice
野口　孝則[1,5]、結城　宗浩[2]、北村　尚士[2]、新家　瑠奈[2]、関　健二郎[1]、糸原　重美[1]、種村健太郎[3]、高島　明彦[3]、Jurgen Wess[4]、山田　真久[2]
[1]理研・脳科学・行動遺伝学、[2]理研・脳科学・山田研究ユニット、[3]理研・脳科学・アルツハイマー病、[4]Bioorganic Chemistry, NIDDKD, USA、[5]神戸学院大・栄養・人間栄養学

P2-295 アルツハイマー型痴呆モデルラット脳内のグリア活性化，神経ペプチドと代謝酵素の変化に対するメマンチンの保護効果
Memantine protective effects on the glia activation and the changes of neuropeptides and peptide-degrading enzymes in NBM-lesioned Alzheimer dementia model rat brain regions
加藤　武[1]、Md. Mahiuddin Ahmed[1]、星野　玄[2]、知久馬敏幸[3]
[1]横浜市大院・総合理・自然情報科学/分子認識、[2]横浜市大・理、[3]昭和薬大・衛生化

P2-296 ラット大脳皮質NMDA受容体におけるアルツハイマー病治療薬Galantamineの作用機序
Mechanism of Action of Galantamine on N-Methyl-D-Aspartate Receptors in Rat Cortical Neurons
森口　茂樹[1]、楢橋　敏夫[2]、福永　浩司[1]
[1]東北大院・薬・薬理学、[2]Department of Molecular Pharmacology and Biological Chemistry, Northwestern University, Chicago, U.S.A.

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P2-297 記憶遺伝子 *amnesiac* と寿命遺伝子 *methuselah* の相互作用によるの記憶と寿命の制御
Interaction of the memory gene *amnesiac* and the aging gene *methuselah* in longevity and memory
齊藤 実[1,2]、堀内純二郎[1,2]、宮下 知之[1]、伊東 直美[1,2]
[1]東京都神経科学総合研究所、[2]科学技術振興機構さきがけ21

P2-298 タウ発現NG108-15細胞におけるcAMPによるカスパーゼ誘導、及びタウの蓄積とリン酸化
Induction of Caspase 3 activity and accumulation and phosphorylation of tau in NG108-15 cells expressing tau by cAMP
塚根まり子[1]、吉崎 千尋[1]、山内 卓[1]
[1]徳大院・薬・生化学

P2-299 高齢者における記銘時の脳活動と記憶獲得能；前痴呆スクリーニング法開発の試み
Brain activation and memory acquisition during picture encoding in the elderly; an approach to screening system of pre-dementia state
平野 好幸[1]、藤田 雅文[2]、丹羽 政美[3]、高橋 尚弘[3]、檜山 征也[4]、井戸 靖司[4]、渡邊 和子[5]、小野塚 実[1]
[1]神奈川歯科大・歯・生体機能、[2]岐阜大・医・高次神経形態、[3]西美濃厚生病院・放射線、[4]木沢記念病院・放射線、[5]岐阜大・医・生理

P2-300 アラキドン酸による老齢ラットのカルシウム流入経路の修飾
Modification of calcium entry channel in the aged rat supplemented with arachidonic acid
深谷 泰亮[1]、小谷 進[3]、堀越 哲郎[2]、石倉 義之[4]、木曽 良信[4]、榊原 学[2]
[1]東海大院・開発工学・生物工、[2]東海大学・開発工学・生物工、[3]東海大院・医、[4]サントリー健康科学研究所

薬物依存、精神薬理
Drug Abuse and Psychopharmacology (Addiction)

P2-301 メタンフェタミンによる脳内モノアミン代謝回転変化に対するモノアミン酸化酵素阻害薬の効果
Effect of monoamine oxidase inhibitor pretreatment on methamphetamine-induced changes in brain monoamine metabolism in mice
北中 純一[1]、北中 順惠[1]、竹村 基彦[1]
[1]兵庫医大・薬理

P2-302 モルヒネ依存におけるLeu-Ileの抑制効果
Inhibitory effects of Leu-Ile on morphine-induced dependence
丹羽 美苗[1]、新田 淳美[1]、山田裕一郎[1]、斎藤 邦明[2]、清島 満[2]、Shen Liya[3]、野田 幸裕[1]、鍋島 俊隆[1]
[1]名古屋大院・医・医療薬学、[2]岐阜大・医・臨床検査医学、[3]Lab. of Mammalian Genes and Devel., NIH

P2-303 メタンフェタミン誘発行動障害における脳内 MMP/TIMP システムの役割
The MMP/TIMP system is involved in the sensitization induced by Methamphetamine in the rat
溝口 博之[1]、毛利 彰宏[1]、丹羽 美苗[1]、水野 朋子[1]、山田 清文[1,2]、野田 幸裕[1]、新田 淳美[1]、鍋島 俊隆[1]
[1]名古屋大院・医・医療薬学・附属病院薬剤部、[2]金沢大学・薬・病院薬学

P2-304 GIRKチャネルに対するいろいろな抗うつ薬の抑制効果
Inhibition of GIRK channels by various antidepressants
小林　徹[1]、鷲山　和雄[1]、池田　和隆[2]
[1]新潟大学・脳研究所・分子神経病理、[2]東京都精神医学総合研究所・分子精神医学

P2-305 5MeO-DIPTによるドパミン神経細胞毒性
5MeO-DIPT-induced dopaminergic cytotoxicity
宮崎　育子[1]、浅沼　幹人[1]、小川　紀雄[1]、舩田　正彦[2]
[1]岡山大院・医歯・神経情報学、[2]国立精神・神経センター・精神保健研究所

P2-306 モルヒネ耐性・依存性形成における神経可塑性の研究ーエレクトロポレーションによる遺伝子欠損マウス脳への特異的遺伝子レスキューによる新規戦略
Study of neuronal plasticity in morphine-induced tolerance and dependence - new strategy of locus-specific rescue of gene into knock-out mouse brain by electroporation
井上　誠[1]、植田　弘師[1]
[1]長崎大院・医歯薬・分子薬理

P2-307 ノシセプチン受容体欠損マウスのエタノール嗜好について
Nociceptin receptor knockout mice show reduced preferences for ethanol
佐郡　和人[1]、Niall Murphy[1]
[1]理化学研究所・BSI

中毒症
Neurotoxicity

P2-308 エタノール摂取に応答する脳内遺伝子の探索
cDNA subtractive screening for ethanol-responsive genes in the rat brain
大友　隆之[1]、高木　充弘[1]、野水　基義[1]、山田　純司[1]
[1]東薬大・薬・病態生化学

てんかん
Epilepsy

P2-309 大脳皮質形成異常を伴うヒトてんかん摘出病巣におけるKCC2発現の減少
KCC2 expression is reduced in human epileptogenic cortical dysplasia
清水一岡部千草[1]、田中　正樹[2]、松田　一己[2]、三原ただひろ[2]、岡部　明仁[1]、佐藤　康二[3]、八木　和一[2]、藤原　建樹[2]、井上　有史[2]、福田　敦夫[1]
[1]浜松医大・医・生理第一、[2]静岡医療センター、[3]浜松医大・医・解剖第一

P2-310 ウサギ海馬における発作波誘発閾値に対するレーザー照射効果
Effects of laser irradiation on the threshold of paroxysmal discharge in the rabbit hippocampus
木暮　信一[1]、吉岡　学[1]、丹羽麻由香[1]、本城　信行[2]、小松　光昭[2]、石井　良夫[2]、渡辺　一弘[2]
[1]創価大院・工・生物工学、[2]創価大院・工・情報システム学

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P2-311 Ca^{2+}チャネルミュータントtottering マウスにおける欠伸発作のネットワークメカニズム
A network mechanism of absence epilepsy in Ca^{2+} channel mutant mice tottering
井本 敬二[1,2]、佐々木幸恵[1,2]、宮田麻里子[1,2]、下野 健[3]
[1]生理学研究所・神経シグナル部門、[2]総合研究大院・生命科学科、[3]アルファメッドサイエンス

P2-312 重篤な知能障害を伴う難治てんかん患者に観察された異常ナトリウムチャネルの電気生理学的解析
Biophysical Analyses of Voltage-gated Na^+-Channel Gene Mutations Identified in Intractable Epilepsies with Severe Mental Decline
荻原 郁夫[1]、神谷 和作[1]、真崎 恵美[1]、岡村 奈美[1]、菅原 隆[1]、Mauricio Montal[2]、蒔田 直昌[3]、藤原 建樹[4]、井上 有史[4]、金田 誠[5]、山川 和弘[1]
[1]理化学研究所・脳科学総合研究センター・神経遺伝研究チーム、[2]Section of Neurobiol, Div of Biological Sciences, UCSD, USA、[3]北海道大院・医・循環病態内科学、[4]静岡てんかん・神経医療センター、[5]慶応大学・医・生理学

P2-313 vigabatrinとtiagabineの組み合わせ投与による抗てんかん効果
Antiepileptic action induced by a combination of vigabatrin and tiagabine
笛田由紀子[1]、欅田 尚樹[1]、Schwarz Wolfgang[2]
[1]産業医大・産業保健、[2]Max-Planck Institute for Biophysics, Frankfurt/Main, Germany

P2-314 若年性ミオクローヌスてんかん原因遺伝子の単離に向けて
Towards the identification of genes responsible for juvenile myoclonic epilepsy
鈴木 俊光[1]、Av Delgado-Escueta[2]、K Aguan[1]、原 雄二[3]、西田 基宏[3]、T Numata[3]、竹内 環[1]、D Bai[2]、井上 有史[4]、大沢真木子[5]、兼子 直[6]、小国 弘量[5]、森 泰生[1]、山川 和弘[1]
[1]理研・脳科学・神経遺伝、[2]UCLA、[3]京都大、[4]国立静岡てんかんセンター、[5]東京女子医大、[6]弘前大

P2-315 PTZ誘発キンドリング動物におけるNOとnNOSの関係
Magnetic Resonance and Biochemical Studies in Pentylenetetrazole-induced Epileptogenesis.-the relationship between NO, nNOS and seizures
伊藤 康一[1]、渡邊 正知[1]、藤井 博匡[2]
[1]徳島文理大・香川薬・分子薬理、[2]札幌医科・保健医療

脳血流、代謝
Cerebral Blood Flow and Metabolism

P2-316 走運動時のラット海馬局所血流量の調節機構を探る新たな方法の提案：レーザードップラー血流測定法とマイクロダイアリシス法を用いた研究
New approach examining enhanced cerebral blood flow in rat hippocampus during treadmill running with laser-Doppler flowmetry combined with microdialysis.
西島 壮[1]、征矢 英昭[1]
[1]筑波大院・人間総合・体育

P2-317 機能的磁気共鳴画像法によって明らかとなった疲労の神経メカニズム
Neural mechanism of central fatigue, as revealed by fMRI study
田中 雅彰[1]、定藤 規弘[2]、岡田 知久[2]、水野 敬[1]、笹部 哲也[1]、齋藤 大輔[2]、尾上 弘隆[3]、倉恒 弘彦[4]、渡辺 恭良[1]
[1]大阪市大院・医・システム神経科学、[2]岡崎国立共同研究機構生理学研究所・大脳皮質機能研究系・心理生理学研究部門、[3]東京都神経科学総合研究所、[4]大阪大院・医・分子病態内科学講座

P2-318 ラット海馬スライスのATP含量に対する近赤外レーザー照射効果の波長依存性
Wavelength-dependent effect of near-infrared laser on ATP content in rat hippocampal slices
小田一望月紀子[1,2]、片岡 洋祐[2]、田村 泰久[2]、山田 久夫[2]、山田 廣成[1]
[1]立命館大学・COE機構・放射光生命科学研究センター、[2]関西医大・解剖学第一

P2-319 ラット視床下部におけるグルコキナーゼの分布
Localization of Glucokinase in Rat Hypothalamus
西尾 崇[1]、豊田 行康[2]、平松 正行[1]、三輪 一智[2]、前川 文彦[3]、矢田 俊彦[3]、千葉 拓[4]
[1]名城大院・総合学術・生体機能制御学、[2]名城大学・薬・病態生化学研究室、[3]自治医科大学・医、[4]名古屋市立大学・薬・生体防御機能学

P2-320 ニューロンはブドウ糖よりもグルタミンをエネルギー基質とする
Glutamine is a preferential energy substrate over glucose for neurons
小山 晶子[1]、中嶋 一行[1]、中臺 武人[1]、金松 知幸[1]
[1]創価大・工・生物工

P2-321 カルシウム，PKC依存性代謝経路を介したシナプスでの乳酸の利用
Calcium and PKC dependent metabolic process for synaptic utilization of lactate in hippocampus
櫻井 孝[1]、明嵜 太一[1]、楊 波[1]、横野 浩一[1]
[1]神戸大学・医・老年内科

P2-322 血液希釈によるラット賦活脳血流と神経活動への影響
The effect of hemodilution on changes in local cerebral blood flow and field potential in rat somatosensory cortex evoked by hindpaw stimulation.
嬉 正勝[1]、菅野 巌[1]
[1]秋田県立脳血管研究センター・放射線医学

P2-323 精神作業疲労により惹起される血液中アミノ酸レベルの変化
Changes in amino acids levels caused by mental fatigue
水野 敬[1]、野崎 聡[1]、山口 浩二[1,2]、水間 広[1]、田中 雅彰[1]、笹部 哲也[1,3]、杉野 友啓[4]、乾 富士男[4]、白井 智子[4]、須賀 裕子[4]、梶本 佳孝[4]、梶本 修身[4,5]、倉恒 弘彦[2,6]、渡辺 恭良[1]
[1]大阪市大院・医・システム神経科学、[2]大阪大院・医・分子病態内科学、[3]大阪大・院・歯・矯正歯科、[4]（株）総合医科学研究所、[5]大阪外国語大学・保健管理センター、[6]関西福祉科学大学・健康福祉

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P2-324 フルスルチアミンによる抗疲労効果
Recovery and prevention from physical and mental fatigue: effect of fursultiamine on behaviors in an animal model of fatigue
野崎 聡[1]、田中 雅彰[1]、水野 敬[1]、中島 英樹[1]、水間 広[1]、中村夫左央[1]、秋元 浩二[2]、野村 之博[2]、中村 正敏[2]、森山 耕[2]、渡辺 恭良[1]
[1]大阪市大院・医・システム神経科学、[2]武田薬品工業株式会社 ヘルスケア カンパニー

イメージング Imaging

P2-325 サルとヒトにおける論理思考の脳機能マッピング
Functional brain mapping of thinking in monkeys and humans
大林 茂[1]、秋根 良英[1]、須原 哲也[1]、永井 裕司[1]、岡内 隆[1]、池平 博夫[1]、前田 純[1]
[1]放医研・脳機能イメージング

P2-326 ニューロン活動の光学的長時間多部位連続記録システムの開発
Long time multiple-site optical recording system for neural activity
廣田 秋彦[1]、伊藤 眞一[1]
[1]島根大学・医・生理学

P2-327 代数的計算の脳活動 − fMRI 研究
Brain Activation during Algebraic Calculation - an fMRI study
渡邉 丈夫[1,2]、三浦 直樹[2,4]、岩田 一樹[2,3]、佐々 祐子[2,3]、浪川 幸彦[5]、川島 隆太[2]
[1]科学技術振興機構、[2]東北大学・未来科学技術共同研究センター、[3]東北大学・工、[4]東北大学・21世紀COEプログラム、[5]名古屋大院・多元数理科学

P2-328 [^{11}C]McN5652と[^{11}C]DASBのセロトニントランスポーターに対する結合能に及ぼす脳内セロトニン濃度の影響;サルを用いたPET実験
Effects of serotonin concentration on *in vivo* binding of two PET tracers [^{11}C]McN5652 and [^{11}C]DASB to serotonin transporters
山本 茂幸[1]、尾上 浩隆[2]、尾上 嘉代[1]、塚田 秀夫[3]、渡辺 恭良[1]
[1]大阪市立大院・医・システム神経科学、[2]東京都神経科学総合研究所・心理学研究部門、[3]浜松ホトニクス（株）・中央研究所

P2-329 語音によって誘発された脳磁場反応に対する幼少時の言語環境の影響
Remains of infant days: Cortical magnetic responses from first generation American English speakers
小山紗智子[1]、山田 玲子[2]、Roberts Timothy[3]、John Houde[1]、Michael Merzenich[1]
[1]北海道大・電子研、[2]University of California, San Francisco, USA、[3]ATR人間情報科学研究所、[4]Department of Medical Imaging, University of Toronto, Toronto, Canada

P2-330 シータリズムの位相同期に関連したグローバルな皮質ネットワークの同定:fMRIとEEGの同時計測を用いて
Phase synchronization in theta rhythm indexes global cortical networks: A study of simultaneous fMRI and EEG
水原 啓暁[1]、王 力群[2]、小林宏一郎[1,3,4]、山口 陽子[1,4]
[1]理研脳センター、[2]東京電機大学、[3]岩手大学、[4]JST, CREST

P2-331 過去の経験に基づいた推論過程に関する神経活動：fMRIによる検討
Neural activation during memory-dependent reasoning: An fMRI study
鈴木 千里[1,2]、月浦 崇[1]、望月 寛子[1]、藤原 寿理[1,2]、飯島 敏夫[2]
[1]産業技術総合研究所・脳神経情報研究部門、[2]東北大院・生命科学研究科・脳情報処理

P2-332 グループ1代謝型グルタミン酸受容体による海馬CA1錐体神経細胞におけるカルシウムオッシレーション
Group I mGluR-induced calcium oscillation of CA1 pyramidal neuron in rat hippocampal slice culture preparation
工藤 佳久[1]、鳴海 栄[1]、森田 光洋[1]
[1]東京薬科大・生命科学・生体高次

P2-333 嘘をつくこととその重要性に関した神経活動：fMRIによる研究
Brain activations associated with telling a lie and its importance: An fMRI study
藤原 寿理[1,2]、月浦 崇[1]、望月 寛子[1]、鈴木 千里[1,2]、飯島 敏夫[2]
[1]産業技術総合研究所・脳神経情報技術研究部門、[2]東北大院・生命科学・脳情報処理

P2-334 特定のmRNAからの翻訳をリアルタイムに可視化するシステムの構築
Visualizing system for ongoing translation from a specific mRNA using translation termination complex.
森 泰丈[1,2]、宮田 信吾[1,2]、藤原 達司[1,2]、池中 建介[1,2]、遠山 正彌[1,2]
[1]大阪大院・医・ポストゲノム疾患解析学、[2]21世紀COEプログラム

P2-335 ラット海馬神経細胞の樹状突起における局所的翻訳のリアルタイムな可視化システムの構築
A novel approach for visualizing spatiotemporal regulation of localized translation in dendrite of rat hippocampal neuron.
宮田 信吾[1,2]、森 泰丈[1,2]、藤原 達司[1,2]、遠山 正彌[1,2]
[1]大阪大院・医・ポストゲノム疾患解析学、[2]21世紀COE program

P2-336 サルの一次運動野における光シグナルの成分解析
Analysis of the origin of optical signal recorded in the primary motor cortex of the monkey brain.
大場 健嗣[1]、廣瀬 秀顕[1]、筧 慎治[1]、飯島 敏夫[1]
[1]東北大院・生命科学・脳情報処理

P2-337 香り刺激時における呼吸に同期したアルファ波起源 －双極子追跡法による推定－
Inspiration-related alpha band synchronization in human olfaction: locations of source generators estimated by a dipole tracing method.
政岡 ゆり[1]、本間 生夫[1]
[1]昭和大学・医・第二生理

P2-338 自家蛍光イメージングによって捉えたラット体性感覚野切片におけるS1-S2間機能的結合
Autofluorescence imaging of functional connections between S1 and S2 in rat somatosensory slices
鎌谷 大樹[1]、工藤 雅治[1]、菱田 竜一[1]、澁木 克栄[1]
[1]新潟大・脳研・システム脳生理

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P2-339 NOとプロスタノイドはマウス体性感覚野の活動依存性の脳血管拡張に関与する
NO and prostanoids mediate activity-dependent cerebral vasodilation in mouse somatosensory cortex
北浦 弘樹[1]、工藤 雅治[1]、渋木 克栄[1]
[1]新潟大学・脳研究所・システム脳生理学

P2-340 fMRIおよびNIRSデータの融合：ボトムアップアプローチに基づく動的モデル
fMRI and NIRs data fusion: a dynamical model based on a bottom-up approach
岩田 一樹[1,2]、Jorge Riera[1,2]、川島 隆太[2]
[1]東北大学未来科学技術共同研究センター、[2]東北大学未来科学技術共同研究センター

P2-341 FMRI時系列データにおける不均一分散補正の検討
Impact of modeling heteroscedastity on fMRI time series analysis
河内山隆紀[1]、岡田 智久[2]、呉 景龍[1]、米倉 義晴[4]、定藤 規弘[3]
[1]香川大・工、[2]先端医療センター、[3]生理研・心理生理、[4]福井大・医・高エネ研

P2-342 覚醒マウス皮質活動のフラビン蛋白蛍光を用いた経頭蓋イメージング
Trans-cranial imaging of awaking mouse cortical activities using flavoprotein autofluorescence
澁木 克栄[1]、菱田 竜一[1]、工藤 雅治[1]、手塚 恵子[1]
[1]新潟大・脳研・生理

P2-343 等価双極子の推定位置の信頼性 ーてんかん間けつ発作波ー
Validity of source localization of interictal epileptic spike potentials estimated by the dipole tracing method.
本間 生夫[1]、岡本 良夫[2]、政岡 ゆり[1]
[1]昭和大学・医・第二生理学、[2]千葉工業大学・工・電気工学科

P2-344 microPET P4でのラット脳用視野外放射能シールドの評価
Evaluation of a shield for extra-FOV activity for a rat brain study with microPET P4
和田 康弘[1]、水間 広[1]、渡辺 恭良[1]
[1]大阪市大院・医・システム神経科学

P2-345 コンプトンカメラによる複数核種同時ガンマ線イメージング
Concurrent imaging of multiple radioactive tracers with a Compton camera
竹市 博臣[1]、本村 信治[1]、郷農 靖之[1]、矢野 安重[1]、榎本 秀一[1]
[1]理化学研究所・加速器基盤研究部

分子生物学的、細胞生物学的方法
Molecular and Cellular Biological Techniques

P2-346 時空間分解能を有する簡便な分子機能解析システムの開発
Development of the simple analysis system for neuronal molecular function with spatial and temporal resolutions
佐藤 泰史[1,2]、五嶋 良郎[1,2]、竹居光太郎[1,2]
[1]横浜市大院・医・薬理、[2]科学技術振興機構・戦略的創造事業

P2-347 タンパク質を構成要素とする膜電位感受性プローブと神経選択性ウイルスベクターを用いた神経細胞の光学的膜電位測定
Optical monitoring of membrane potential changes of neurons using protein-based voltage-sensitive probe and neuron specific vectors
井上 謙一[1]、一條 宏[1]、大原 慎也[1]、筧 慎治[1]、市川 道教[2]、飯島 敏夫[1]
[1]東北大院・生命科学・脳情報処理、[2]理研BSI・脳創成デバイス

P2-348 改変狂犬病ウィルスベクターの神経回路トレーサーとしての適用
Recombinant rabies virus vectors for neuroanatomical tracers.
大原 慎也[1]、井上 謙一[1]、一條 宏[1]、筧 慎治[1]、飯島 敏夫[1]
[1]東北大学・生命科学・脳情報処理

P2-349 新規蛋白質Amidaによる細胞周期制御
Cell cycle regulation of by Amida
入江 康至[1]、甘 業華[2]、平 英一[1]、三木 直正[1]
[1]大阪大院・医・情報薬理、[2]Department of Surgery, Baylor College of Medicine, Houston, USA

P2-350 無血清培養液を用いたラット海馬スライス培養方法の確立
A novel culture method for rat hippocampal slice using a serum-free culture medium
糸総るり香[1]、工藤 佳久[1]、森田 光洋[1]
[1]東京薬科大学院・生命科学・生体高次機能学

Sep 23 (Thu) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

突起伸展、回路網形成
Axonal Outgrowth and Network Formation

P3-001 膜電位の光学的測定による鶏胚前庭動眼反射回路網の機能イメージング
OPTICAL IMAGING OF NEURAL NETWORK ACTIVITY IN THE VESTIBULO-OCULOMOTOR REFLEX OF THE CHICK EMBRYO
佐藤　勝重[1]、Joel Glover[2]、佐藤　容子[1]
[1]東京医科歯科大院・医・機能協関システム医学、[2]Dept Physiol, Univ Oslo Sch Med, Oslo, Norway

P3-002 膜電位の光学的イメージングによる鶏胚脳幹迷走神経回路網の機能的構築過程の解析
OPTICAL MAPPING OF NEURAL CIRCUIT FORMATION IN THE CHICK VAGAL PATHWAY DURING EARLY EMBRYOGENESIS
宮川　尚久[1]、佐藤　勝重[1]、佐藤　容子[1]
[1]東京医科歯科大院・医・機能協関システム医学

P3-003 マウス前脳における小胞性グルタミン酸輸送体1（VGLUT1）およびVGLUT2の生後発達
Postnatal development of vesicular glutamate transporter 1 (VGLUT1) and VGLUT2 in the mouse forebrain
中村　公一[1]、日置　寛之[1]、藤山　文乃[1]、金子　武嗣[1]
[1]京都大院・医・高次脳形態学

P3-004 ショウジョウバエ触角葉の投射神経形成におけるeylessとhomothoraxの役割
Homeobox genes, eyeless and homothorax, control differentiation of projection and local-inter neurons in the Drosophila antennal lobes.
戸谷　洋子[1]、来栖　光彦[2]
[1]筑波大院・生命環境・構造生物、[2]Division of Biology, Caltech, USA

P3-005 神経細胞由来RPTPβ/phosphacanの発現と局在
Neuronal expression of chondroitin sulfate proteoglycan RPTPβ/phosphacan
林　　謹子[1]、宮田　清司[1]、亀井加恵子[1]、大平　敦彦[2]
[1]京工繊大・応生、[2]愛知県心身者コロニー研究所

P3-006 セマフォリン3Fノックアウトマウスの作成と解析
Generation and analysis of Semaphorin 3F knockout mice
松田　育雄[1]、中尾　晴美[1]、深谷　昌弘[2]、渡辺　雅彦[2]、中尾　和貴[3]、饗場　　篤[1]
[1]神戸大院・医・細胞生物学、[2]北海道大院・医・解剖発生学、[3]理研・発生再生科学総合研究センター

P3-007 リーラーマウスにおける橋小脳路の三次元分布
Three-dimensional computerized distribution of pontocerebellar projection neurons in the normal and reeler mice
田中　靖史[1]、寺島　俊雄[1]
[1]神戸大院・医・神経発生学

P3-008 N-WASPと相互作用し神経突起分枝化を引き起こすRnd2エフェクター、Rapostlinのsplicing variantsの同定
Identification of Splicing Variants of Rapostlin, a Rnd2 Effector that Interacts with N-WASP and Induces Neurite Branching
柿本 哲宏[1]、加藤 裕教[1]、根岸 学[1]
[1]京都大院・生命・生体システム学

P3-009 低分子量G蛋白質Rnd1はセマフォリン4Dが引き起こす海馬神経細胞の成長円錐退縮に不可欠である
Small GTPase Rnd1 is essential for the semaphorin 4D-induced growth cone collapse in hippocampal neurons
石川 幸雄[1]、加藤 裕教[1]、生沼 泉[1]、根岸 学[1]
[1]京都大院・生命科学・生体システム学

P3-010 NGF 誘発突起伸展における NADPH oxidase の役割
The role of NADPH oxidase to neurite outgrowth induced by NGF in PC12
衣斐 督和[1]、勝山 真人[1]、矢部 千尋[1]
[1]京都府立医科大学・医・薬理

P3-011 大脳皮質ニューロンのspine形成及びbranch形成における軸索ガイド分子Sema3Aの役割
Role of Sema3A in dendritic branching and spine formation of mouse cortical neurons
森田 麻[1]、竹居光太郎[1,2]、佐々木幸生[3]、内田 穣[1]、中島 央美[1]、中村 史雄[1]、五嶋 良郎[1,2]
[1]横浜市立大院・医・分子薬理神経生物学、[2]科学技術振興機構・戦略的基礎研究推進事業、[3]Dept. of Neurosci., Albert Einstein College of Medicine, NY, USA

P3-012 大脳皮質非錐体細胞の軸索・樹状突起の分岐パターン
Axon and dendrite branching patterns in GABAergic nonpyramidal cells
苅部 冬紀[1]、窪田 芳之[1]、川口 泰雄[1]
[1]生理研・大脳神経回路論

P3-013 脳特異的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のラット大脳皮質ニューロンに対する神経突起伸長促進
Neuroglycan C, a brain-specific chondroitin sulfate protenglycan, promotes neurite outgrowth from rat neocortical neurons.
中西 圭子[1]、青野 幸子[1]、平野佳奈子[1]、周尾 卓也[1]、伊田みちる[1]、時田 義人[1]、松井ふみ子[1]、大平 敦彦[1]
[1]愛知県コロニー発達障害研・周生期

P3-014 Stathminによるプルキンエ細胞樹状突起内微小管の制御と登上繊維・プルキンエ細胞間シナプス除去への関与の可能性
Stathmin regulates dendritic microtubule stability of developing Purkinje cells that might be important for the olivocerebellar synapse elimination
大川 宜昭[1]、右島 理可[1]、日野 敏昭[1]、高部 美穂[1]、横山 峯介[1]、井ノ口 馨[1]
[1]三菱化学生命科学研究所・井ノ口グループ

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P3-015 摘出小脳一橋一延髄標本における下オリーブ核および橋核から小脳への投射様式の発達
Development of cerebellar network connection from inferior olive and pontine nucleus in neonatal rats
荒田 晶子[1]、籔中 厚生[1]、伊藤 正男[1]
[1]理化学研究所・脳科学センター・記憶学習チーム

P3-016 神経成長円錐におけるカルシウム依存性局所タンパク質新生
Calcium-Regulated Local Protein Synthesis Within Nerve Growth Cones
仙石くみこ[1]、松下 正之[2]、Angus Nairn[3]、五嶋 良郎[1,4]、竹居光太郎[1,4]
[1]横浜市大院・医・分子薬理神経生物、[2]岡山大学・医・生理、[3]Dept. of Psychiat., Yale Univ. Sch. Med., New Haven, USA、[4]科学技術振興機構・戦略的創造事業

P3-017 新規セマフォリンのクローニング
Identification of novel members of semaphorin family
谷口 雅彦[1]、清水 孝雄[1]
[1]東京大院・医・生化学分子生物学

P3-018 ゼブラフィッシュ視覚運動変換に関わる視蓋延髄路の発生と構造
Development and Organization of Tectobulbar Tracts involved in Visuomotor Transformation in Zebrafish
佐藤 智美[1]、浜岡 崇憲[1]、相澤 秀紀[1]、岡本 仁[1]
[1]理化学研究所・脳科学総合研究センター・発生遺伝子制御研究チーム

P3-019 新規 Nogo-66 receptor/p75 シグナリング複合体コンポーネント LINGO-1 のニワトリホモログの解析
Analysis of a chick homologue of LINGO-1, a novel component of the Nogo-66 receptor/p75 signaling complex
岡藤 辰也[1]、田中 英明[1]
[1]熊本大院・医薬・神経分化学

P3-020 Plexin-B1 の Ras-GAP 様ドメインは低分子量G蛋白質 Rnd1 により機能的制御をうける
The Ras GAP-homologous domain of Plexin-B1 is functionally regulated by the small GTPase Rnd1
生沼 泉[1]、加藤 裕教[1]、石川 幸雄[1]、根岸 学[1]
[1]京都大院・生命・生体システム学

P3-021 胎仔期X線照射によるラット大脳皮質における神経路の形成異常
Multiple defects in the pathway formation of rat cerebral cortex after prenatal X-irradiation
李 洪鵬[1]、本間 静[1]、三木 崇範[2]、竹内 義喜[2]、川野 仁[1]
[1]東京都神経研・発生形態、[2]香川大・医・神経機能形態

P3-022 発生期のニワトリ網膜において領域特異的に発現する分子の網羅的同定と解析
Large-scale identification and characterization of genes with asymmetric expression patterns in the developing chick retina
新谷 隆史[1,2]、加藤 彰[1,2]、湯浅 純一[1,2]、作田 拓[1,2]、高橋 正和[1,2]、鈴木 亮子[1,2]、大河原 剛[1,2]、高橋 弘雄[1,2]、野田 昌晴[1,2]
[1]自然科学研究機構・基礎生物学研究所・感覚情報処理、[2]戦略的創造研究・科学技術振興機構

P3-023 KIF2の軸索成長円錐における突起伸長の制御
Kinesin superfamily protein 2A (KIF2A) functions in suppression of collateral branch extension
本間 典子[1]、武井 陽介[1]、田中 庸介[1]、中田 隆夫[1]、寺田 純雄[1]、吉川 雅英[1]、野田 泰子[1]、広川 信隆[1]
[1]東京大院・医・細胞生物

P3-024 NGFによるRac1/Cdc42の活性化と神経突起伸展におけるVav2/Vav3の役割
An essential role of Vav2/Vav3 in NGF-induced Rac1/Cdc42 activation and neuritogenesis in PC12 cells
青木 一洋[1]、中村 岳史[1]、松田 道行[1]
[1]大阪大院・微研・腫瘍ウイルス

P3-025 小麦胚芽凝集素 (WGA) トランスジーンを用いたホヤ幼生の視覚神経経路の可視化 (2)
Visualization of visual pathways in ascidian larvae using wheat germ agglutinin (WGA) transgene (II)
川上 功[1]、堀江 健生[1]、吉原 良浩[2]、日下部岳広[1]、津田 基之[1]
[1]兵庫県立大院・生命理学・生命、[2]理研・脳科学総合・臨界期機構・シナプス分子機構

P3-026 アリルハイドロカーボン受容体過剰発現Neuro-2aにおける神経突起伸展
Expression of aryl hydrocarbon receptor induces neurite outgrowth in Neuro-2a cells
菅野美津子[1]、吉村 斎湖[1]、赤星 英一[1]
[1] (株) 東芝 研究開発センター・環境技術ラボラトリー

P3-027 神経突起伸長に関わるNeuro-p24の役割とマウス脳内におけるその分布
Role of Neuro-p24 in neurite extension and its distribution in the mouse brain
鈴木 春野[1]、井田 瑞穂[1]、森 望[3]、竹谷 茂[2]、荒木 正介[1]
[1]奈良女子大院・人間文化研究・生物科学、[2]京都工芸繊維大・繊維・応用生物、[3]国立長寿医療研究センター・分子遺伝学研究部

P3-028 ホヤ幼生の運動神経回路網の可視化
Motor nervous system of the ascidian larva
堀江 健生[1]、中川 将司[1]、日下部岳広[1]、津田 基之[1]
[1]兵庫県立大院・生命理

P3-029 プルキンエ細胞の樹状突起とスパイン形成におけるホスホリパーゼA2-リポキシゲナーゼ経路の役割
Role of phospholipase A_2 and lipoxygenase pathway in formation of dendrites and spines of Purkinje cells
佐藤 衛[1]、島田 厚良[1]、河村 則子[1]、千葉 陽一[1]、慶野 裕美[1]、細川 昌則[1]、魚住 尚紀[2]、清水 孝雄[2]
[1]愛知県心身障害者コロニー発達障害研究所・病理、[2]東大・院・医・細胞情報

P3-030 神経系Igスーパーファミリー接着分子群の機能進化：最も原始的なプラナリア脳・IgCAMsの解析
The functional evolution of neural Ig superfamily cell adhesion molecules (IgCAMs) : The analysis of the most primary IgCAMs in planarian brain
房岡 恵理[1]、岡本 圭司[1,2]、阿形 清和[2]、峯田 克彦[3]、五條堀 孝[4]、武内 恒成[1]
[1]名古屋大院・理・生命理学、[2]理研・発生再生センター、[3]北海道大院・情報科学・生命人間情報科学、[4]遺伝研・生命情報センター

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P3-031 樹状突起のパターンの多様性を支える分子機構
Development of morphological diversity of dendrites of Drosophila sensory neurons by the BTB-zinc finger protein Abrupt
杉村　薫[1]、佐藤　大祐[1]、Stephen Crews[2]、上村　匡[3]
[1]京都大院・理・生物物理、[2]Biochemistry and Biophysics, School of Medicine, The University of North Carolina at Chapel Hill, USA、[3]京都大院・生命科学

P3-032 ゼブラフィッシュ後脳背外側交連神経細胞の可視化による結合様式の解析
Visualization of dorsolateral commissural neurons and their connections in zebrafish hindbrain
佐々　貴之[1]、岡本　仁[1,2]
[1]理研・脳科学センター・発生遺伝子制御、[2]科学技術振興機構・CREST

P3-033 マイクロアレイ法を用いた側枝形成因子の解析
Microarray analysis of the axon branching factor genes
岡田　拓也[1]、桝　和子[1]、塩見　健輔[1]、桝　正幸[1]
[1]筑波大院・人間総合科学・感性認知脳

P3-034 海馬神経細胞の樹状突起形成におけるデルターカテニンの役割
δ-catenin-induced filopodial elaboration is mediated by interactions with its PDZ domain targets
落石　知世[1,2]、Andrea Witt[2]、Maria-Cruz Martinez-De-Arrieta[2]、Michael Majewski[2]、Miguel Medina[2]、Kenneth Kosik[2]
[1]産総研・脳神経情報、[2]Department of Neurology, Brigham and Women's Hospital and Harvard Medical School, Boston, USA

P3-035 インターフェイス型脳スライス共培養のためのコンパートメントチェンバー作成と皮質-脊髄投射系研究への適用
Compartmented chamber for interface type brain slice co-culture and its use for studying corticospinal projection
吉岡　昇[1]、桜井　正樹[1]
[1]帝京大学・医・生理学

P3-036 スラミンによる神経細胞突起伸展作用はMAPキナーゼカスケードを介する
Suramin mediates neurite outgrowth via MAP kinase-signaling pathways in a neuronal cell line
中田　裕康[1]
[1]東京都神経研・生体機能分子

P3-037 BDNFによる海馬歯状回神経回路の再編成
BDNF induces dentate neural network reorganization.
小山　隆太[1]、山田　麻紀[1]、藤澤　茂義[1]、仙波りつ子[2]、西山　信好[1]、松木　則夫[1]、池谷　裕二[1]
[1]東京大院・薬・薬品作用学、[2]愛知県心身障害者コロニー・発達障害研究所・周生期学部

P3-038 Sema3Fに対する海馬歯状回顆粒細胞成長円錐の崩壊応答：細胞内cAMPシグナル伝達系による軸策・樹状突起の応答差異
Sema3F-mediated growth cone collapse: differential actions of cAMP on axons and dendrites of dentate granule cells
山田 隆二[1]、山田 麻紀[1]、松木 則夫[1]、池谷 裕二[1]
[1]東京大院・薬・薬品作用学

P3-039 成長円錐蛋白質SCG10の機能とリン酸化の関連性の解析
Significance of the phosphorylation sites in SCG10, a growth cone protein depolymerizing microbutules
梅野 哲哉[1]、倉知 正[3]、加藤 薫[4]、Gabriele Grenningloh[5]、五十嵐道弘[1]
[1]新潟大院・医歯学・分子細胞機能学（医・生化2）、[2]新潟大院・医歯学・眼科学、[3]群馬大・医・分子病態学、[4]産総研・脳機能調節因子、[5]Ctr Neurosci Psychiatr, Univ Lausanne, Prilly, Switzerland

P3-040 網膜軸索ガイダンスに及ぼすchondroitin sulfateの構造多様性の効果
Effects of strucural diversity in chondroitin sulfates on retinal axon guidance
一條 裕之[1]
[1]筑波大・基礎医学系・解剖学

P3-041 ゼブラフィッシュ・ニューロピリン-1・ホモログの発現調節機構の解明
Identification of cis-acting elements regulating tissue-specific expression of *neuropilin-1* (*nrp-1*) in zebrafish
俵山 寛司[1]、帯刀 益夫[1]、東海林 亙[1]
[1]東北大・加齢研・分子発生

P3-042 FGF8によって誘導されたSema3fは中脳ドーパミン作動性ニューロンの軸索ガイダンスに関与する
Fgf8-induced Sema3F is involved in axon guidance of the midbrain dopaminergic neurons
山内 健太[1]、水嶋 茂樹[1]、六車 恵子[1]、玉田 篤史[1]、高島 成二[2]、村上富士夫[1,3]
[1]大阪大院・生命機能・脳発生生理、[2]大阪大院・医・第一内科、[3]SORST and CREST, JST

P3-043 ショウジョウバエ嗅覚神経の軸索投射に異常を示す突然変異体の表現型の解析
Phenotypic analyses of the mutants that have defects in the axonal projection of the olfactory sensory neurons in *Drosophila*
遠藤 啓太[1]、依田 有香[1]、木村 賢一[2]、浜 千尋[1]
[1]理研・CDB・神経回路発生、[2]北教大・岩見沢・生物

P3-044 特異的シナプス形成の可視化：標的認識分子カプリシャスの役割
Live imaging of synaptic matchmaking and synaptogenesis: the role of target recognition molecule Capricious
高坂 洋史[1]、能瀬 聡直[1]
[1]東京大院・理・物理学

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P3-045 線虫C. elegansの嗅覚可塑性に関わる転写因子MBR-1の軸索除去における機能解析
Analysis of the function of MBR-1, a C. elegans transcription factor involved in olfactory plasticity, in axon pruning
林　悠[1]、鹿毛枝里子[1]、広津　崇亮[2]、國友　博文[2]、竹内　秀明[1]、飯野　雄一[2]、久保　健雄[1]
[1]東京大院・理・生物科学、[2]東京大・遺伝子実験施設

P3-046 培養小脳組織におけるK252aの神経回路形成効果
The effects of K252a to cell cycle and Purkinje cell development
森島　寿貴[1]、石川　稔[1]、大下　美雪[1]、上松　正和[1]、吉田　祥子[1]
[1]豊橋技術科学大・物質工学系

P3-047 RhoG標的蛋白質Elmoとその結合蛋白質Dock180のマウス脳の発達過程における発現分布
Distribution of the RhoG effector Elmo and its binding protein Dock180 in the developing mouse brain
加藤　裕教[1]、石田智早希[1]、藤本　聡志[1]、石川　幸雄[1]、根岸　学[1]
[1]京都大院・生命科学・生体システム

P3-048 軸索分子netrin-Gによる投射経路選択的なシナプス機能制御
Axonal netrin-G proteins regulate local postsynaptic properties
西村　幸子[1]、関　健二郎[1]、新美　君枝[2]、糸原　重美[1]
[1]理研・脳総研・行動遺伝、[2]リサーチリソースセンター・理研・脳総研

P3-049 ニワトリ皮質（Wulst）に対称的および非対称的に投射する視床領域
Thalamic regions symmetrically and asymmetrically connected to chick Wulst
小柴満美子[1,2]、中村　俊[1]
[1]国立精神セ・診断、[2]東京農工大・生命工

P3-050 NGF突起伸展時のPC12細胞におけるセロトニンの効果
5-HT enhances NGF-induced neurite outgrowth via intracellular Ca^{2+} increase in PC12 cells
本間　耕平[1]、新野　祐介[1]、小川　宏人[2]、北村美一郎[1]、岡　浩太郎[1]
[1]慶應大院・基礎理工・生命理工、[2]埼玉医科大・医学基礎部門・医学生物

P3-051 視床－subplate neuron間の胎生期におけるシナプスの活動
synaptic transmission between thalamic axons and subplate neurons during embryonic periods
東　秀二[1]
[1]京都府立医大・医・神経生理

P3-052 神経特異的膜型セマフォリン分子Sema4Cの発現とその性状解析
Characterization of expression of brain-specific transmembrane semaphorin, Sema4C
大岡　嘉治[1]、稲垣　忍[1]
[1]大阪大院・医・保健

P3-053 発生過程大脳皮質における多極性細胞の振る舞い
The behavior of multipolar cells in the developing cortex
田畑　秀典[1,2]、仲嶋　一範[1,2]
[1]慶應大院・医・解剖、[2]慈恵会医大・DNA医学研究所・分子神経

P3-054 Polymer Hydrogel Electrodes for the Study of Neural Network Formation
T. Nyberg[1]、N. Kasai[1]、A. Shimada[1]、C. Han[1]、I. Fujimoto[2]、K. Torimitsu[1]
[1]NTT Basic Research Lab., NTT Corp., Japan、[2]The University of Tokyo, NTT-IMSUT

P3-055 Selective inhibition of chondroitin sulfate to neurite growth from mouse retina
Joyce Sy Lam[1]、S. O. Chan[1]
[1]Department of Anatomy, The Chinese University of Hong Kong

P3-056 Expression of Nogo-A in the chiasm of mouse embryos
Jun Wang[1]、S. O. Chan[1]
[1]Department of Anatomy, The Chinese University of HK

栄養因子、サイトカイン
Trophic Factors and Cytokines

P3-057 未分化PC12細胞の無血清継代培養
Passage culture of undifferentiated PC12 cells in serum-free medium
大沼　清[1]、林　洋平[1]、古江　美保[2]、浅島　誠[1]
[1]東大院・総分化・広域、[2]神奈川歯科大

P3-058 PEDFはCREB及びNF-kappaBの活性化を介して新生児由来アストロサイトにおける炎症誘導遺伝子を誘導する
PEDF induces pro-inflammatory genes in neonatal astrocytes through activation of NFκB and CREB
佐柳　友規[1]、西尾　昌子[1]、矢部　武士[1,2]、山田　陽城[1,2]
[1]北里大院・生命科学研、[2]北里研・東洋医学総合研

P3-059 シナプスの発達と成熟におけるEGF/ErbB1シグナルの抑制的機能
Negative Function of EGF/ErbB1 Signaling in Synaptic Development and Maturation
横幕　大作[1]、永野　忠聖[1]、柿田　明美[2]、水野　誠[1]、青木　弘行[1]、Jourdi Hussam[1]、武井　延之[1]、那波　宏之[1]
[1]新潟大・脳研・分子神経生物、[2]新潟大・脳研・脳疾患リソース解析

P3-060 活動依存的なPACAP遺伝子の転写および転写後調節機構の解析
Activity-dependent transcriptional and post-transcriptional regulation of pituitary adenylate cyclase-activating polypeptide (PACAP) coordinated by calcium and cAMP signals in neurons
福地　守[1]、田渕　明子[1,2]、津田　正明[1,2]
[1]富山医薬大・薬・分子神経生物、[2]CREST, JST

P3-061 転写因子NF-kBを介した、インターロイキン1によるグリア細胞の機能変換
NF-kB-dependent Activation of Glutamate Uptake by Interleukin-1 in Rat Primary Astrocytes
川村　名子[1]、武井　延之[1]、那波　宏之[1]
[1]新潟大・脳研・分子神経生物

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P3-062 パーキンソン病線条体におけるEGF/EGFレセプターシグナルの変化
Decreased EGF/ErbB1 receptor signal in the brain of patients with Parkinsons disease
岩倉百合子[1]、柿田　明美[2]、朴　　英善[1]、武井　延之[1]、那波　宏之[1]
[1]新潟大・脳研・分子神経生物、[2]新潟大・脳研・脳疾患標本資源解析学

P3-063 神経栄養因子の分泌に関わる新規遺伝子CAPS2の解析
The secretory granule-associated protein CAPS2 regulates neurotrophin release and cell survival
定方　哲史[1,5]、溝口　　明[2]、佐藤　友美[1]、仙波りつ子[3]、福田　光則[4]、御子柴克彦[5]、古市　貞一[1]
[1]理研・脳センター・分子神経形成研究チーム、[2]三重大・医・神経再生、細胞情報、[3]愛知県コロニー・発達障害研究所、[4]理研・福田主幹プロジェクト、[5]東大・医科研・脳神経

P3-064 リルゾールによるラット海馬のBDNF合成促進：p38MAPKおよびERK1の下流にあって活性化される細胞内シグナル
Mechanisms of BDNF synthesis in the rat hippocampus: intracellular signals activated in the downstream of p38 MAPK and/or ERK1
仙波　りつ子[1]、北島　哲子[1]、一坂　史志[2]、畠　　義郎[3]、続木　雅子[1]、加藤　兼房[1]
[1]愛知県コロニー・研究所、[2]鳥取大院・医・生命・神経生物、[3]鳥取大院・医・機能再生・生体高次機能

P3-065 ミクログリアのGDNF産生能について
Secretion of glial cell line-derived neurotrophic factor (GDNF) from rat cultured microglia
松下　雄一[1]、遠山　陽子[1]、栗原　　正[1]、高坂　新一[2]、中嶋　一行[1,2]
[1]創価大・工・生命情報工学、[2]国立精神・神経センター、神経研

P3-066 LECT2のマウス脳における発現と局在
Expression and localization of LECT2 in mouse brain
奥水　洋平[1]、山越　　智[2]、鈴木　和男[2]、大冨美智子[1]
[1]東邦大院・理学研究科・生物分子科学専攻科、[2]国立感染症研

P3-067 マカクサル小脳における神経栄養因子タンパク量、免疫陽性構造の発達にともなう変化
Developmental changes in concentrations and distributions of neurotrophins in the cerebellum of macaque monkeys
託見　　健[1]、森　　琢磨[1]、清水　慶子[1]、林　　基治[1]
[1]京都大・霊長類研・分子生理

P3-068 新生仔期の上皮成長因子/ErbB1活性は中脳ドパミン神経の興奮性シナプス伝達を亢進する
Perinatal EGF/ErbB1 activity potentiates excitatory synaptic transmission to midbrain dopaminergic neurons
難波　寿明[1]、須藤　晃佑[1]、武井　延之[1]、那波　宏之[1]
[1]新潟大・脳研・分子神経生物

P3-069 生後及び成体の中枢神経系におけるTROY/TNFRSF19の局在
Localization of TROY/TNFRSF19 in the postnatal and adult CNS
久岡　朋子[1]、森川　吉博[1]、北村　俊雄[2]、仙波恵美子[1]
[1]和歌山医大・医・第二解剖、[2]東大・医科研・先端医療・細胞療法

P3-070 ラット大脳皮質神経細胞における脳由来神経栄養因子（BDNF）の自律的発現制御系の解析
Autoregulation of Brain-derived neurotrophic factor gene expression in rat cortical neurons
安田　誠[1]、川原　正博[2]、今村　理佐[1]、田渕　明子[1]、津田　正明[1]
[1]富山医薬大・薬・分子神経生物、[2]九州保健福祉大・薬・分析化学

P3-071 ラット中脳培養切片における（-）-BPAPの神経栄養因子の発現およびスーパーオキシドジスムターゼ活性に対する効果
Effect of (-) -1- (benzofuran-2-yl) -2-propylaminopentane on the expression of neurotrophins and superoxide dismutase activity in mesencephalic slice cultures
高畑　和恵[1]、平見智映子[1]、島津誠一郎[1]、米田　文郎[1]、林　恭三[2]、香月　博志[3]、赤池　昭紀[3]
[1]藤本製薬・創薬研、[2]岐阜薬科大、[3]京都大院・薬・薬品作用解析

P3-072 3-Phosphoglycerate Dehydrogenase遺伝子ノックアウトマウスの解析
Mouse 3-phosphoglycerate dehydrogenase is essential for brain morphogenesis
吉田　一之[1,2]、古屋　茂樹[1]、大須賀　壮[1]、三苫　純也[3]、篠田　陽子[1]、渡辺　雅彦[4]、東　徳洋[5]、田中　秀幸[5]、端川　勉[1]、糸原　重美[1]、平林　義雄[1]
[1]理研・脳研、[2]宇都宮大・ベンチャービジネスラボラトリー、[3]バーナム研、[4]北海道大院・医・生体構造解析、[5]宇都宮大・農・生物生産

P3-073 行動への周産期IL-1と遺伝的背景の相互作用
Interplay of perinatal interleukin-1 and mouse genetic backgrounds in neurobehavioral consequences
津田　法子[1]、任海　学[1]、水野　誠[1]、那波　宏之[1]
[1]新潟大・脳研・分子神経生物学分野

P3-074 ラット大脳初代神経培養系におけるニューロトロフィン－3の転写調節機構の解析
Transcriptional mechanisms of neurotrophin-3 (NT-3) gene promoter IB by Sp3 and Sp4 in cultured rat cortical neuron
石丸　直樹[1]、林　裕之[1]、安原　昌宏[1]、田渕　明子[1,2]、津田　正明[1,2]
[1]富山医科薬科大・薬・分子神経生物、[2]CREST, JST

P3-075 NRSFを介するBDNF遺伝子転写制御の解析
Regulation of brain-derived neurotrophic factor gene transcription mediated by neuron-restrictive silencer factor (NRSF)
原　大智[1,2]、宮下　敏秀[1,2]、田渕　明子[1,2]、津田　正明[1,2]
[1]富山医科薬科大・薬・分子神経生物、[2]CREST, JST

P3-076 BDNFによるグルタミン酸開口放出増強作用における神経活動と細胞内シグナル経路の相互作用
Involvement of neuronal activity in intracellular signaling, which is important for BDNF-potentiated glutamate release.
松本　知也[1]、沼川　忠広[2]、横幕　大作[3]、安達　直樹[4]、田口　隆久[1]
[1]産総研・セルエンジニアリング・ニューロニクス、[2]国立精神神経センター・神経研・第三部、[3]新潟大・脳研・分子神経生物、[4]大阪大院・医・神経生理

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P3-077 腹腔マクロファージの活性化に対するグリア細胞株由来神経栄養因子の影響
Glial cell line-derived neurotrophic factor influences the activation of peritoneal macrophages in autocrine/paracrine manners
橋本　学[1]、西村祐一郎[1]、古川　昭栄[1]
[1]岐阜薬科大・分子生物学

P3-078 大脳皮質視覚野でのBDNF（脳由来神経栄養因子）によるGABA$_A$受容体応答修飾作用の発達変化および視覚経験依存性
The developmental shift of the BDNF actions on GABA$_A$ receptor responses by the rapid up- or down-regulation of cell surface functional GABA$_A$ receptors in rat visual cortex
溝口　義人[1,2]、鍋倉　淳一[2]
[1]九州大院・歯・口腔細胞工学、[2]岡崎生理学研・発達生理

P3-079 グルココルチコイドは培養大脳皮質ニューロンにおいてBDNFによるグルタミン酸放出を抑制する
Glucocorticoid suppresses brain-derived neurotrophic factor (BDNF) -regulated glutamatergic transmission in cultured cortical neurons.
沼川　忠広[1]、矢ケ崎有希[1]、橋本　亮太[1]、功刀　浩[1]
[1]国立精神神経センター・神経研・第三部

P3-080 BDNF Val68Met変異体による抑制性シナプスの機能亢進
BDNF Val68Met Mutant Increases an Inhibitory Synaptic Machinery
池田　隆光[1]、池谷　裕二[1]、松木　則夫[1]、山田　麻紀[1]
[1]東京大院・薬・薬品作用学

P3-081 活性化ミクログリアにおけるケモカイン受容体CCR7の発現
Induction of chemokine receptor CCR7 mRNA in activated Microglia
小池　達郎[1]、中森　文洋[1]
[1]北海道大・理・生物科学

P3-082 Growth hormone receptor modulates adult neural stem cell proliferation.
Rodney L. Rietze[1]、Brent A. Reynolds[1]、Michael J. Waters[2]
[1]The Queensland Brain Institute, Brisbane, Australia、[2]Institute for Molecular Biology, University of Queensland, Brisbane, Australia

細胞接着因子
Cell Adhesion Molecules

P3-083 中枢神経系におけるコンドロイチン硫酸プロテオグリカンPhosphacanのPerineuronal netにおける局在と可塑性
Perineuronal net of chondroitin sulfate proteoglycan phosphacan and plasticity in the central nervous system
宮田　清司[1]、大平　敦彦[2]
[1]京都工芸繊維大・応用生物、[2]愛知県発達障害研

P3-084 細胞接着因子OBCAMの樹状突起スパインにおけるinternalization
Internalization of cell adhesion molecule OBCAM at dendritic spines
山田　真弓[1]、宮田　清司[1]、前川　昌平[2]
[1]京都工芸繊維大・応用生物、[2]神戸大院・自然科学

P3-085 スパインおよびシナプス接着の安定性はαN-カテニンによって制御される
Stability of dendritic spines and synaptic contacts is controlled by αN-catenin
安部健太郎[1,2]、千坂　修[2]、Frans Van Roy[3]、竹市　雅俊[1,2]
[1]理研・発生再生科学総合研究センター・高次構造形成研究グループ、[2]京都大院・生命科学、[3]VIB-Ghent University

P3-086 ゼブラフィッシュプロトカドヘリン遺伝子クラスターの解析
Zebrafish Pcdh family genes: genomic organization and expression during brain development
多田　基紀[1]、杉野　英彦[1,2]、八木　健[1,2]
[1]大阪大院・生命機能・時空生物、[2]CREST, JST

P3-087 RA175/TSLC1/SynCAM の選択スプライシング産物とプロセッシングによる機能調節
Splicing products of RA175/TSLC1/SynCAM and regulation of their cell adhesion activity by processing
藤田恵理子[1]、村上　依子[1]、桃井　隆[1]
[1]国立精神神経センター・5部

P3-088 CNR/Pcdhαアイソフォームの発現パターンは、ゲノムのメチル化状態と関連する
The expression pattern of CNR/Pcdhα isoforms correlates with genomic methylation status.
川口　将史[1]、遠山　知子[1]、杉野　英彦[1]、八木　健[1,2]
[1]大阪大院・生命機能・心生物学、[2]CREST, JST

P3-089 マウス成熟脳における細胞接着分子L1-cam mRNAの分布
Distribution of L1-cam mRNA in the adult mouse brain.
堀之内和広[1]、中村　雪子[1]、塩坂　貞夫[1]
[1]奈良先端科学技術大学院大・バイオサイエンス・細胞構造学

P3-090 CNR/Pcdhαファミリータンパク質の構造ー機能連関解析
Structural and functional characterization of CNR/Pcdhα family protein
海津　正賢[1,2,6]、森下　博文[1,2,6]、村田　陽二[1]、草野　清輔[3]、樋口　芳樹[3]、池上 貴久[1,4]、八木　健[1,2,5,6]
[1]大阪大院・生命機能・心生物学グループ、[2]大阪大院・医・分子遺伝学、[3]姫路工業大・理・生命科学、[4]大阪大・蛋白研、[5]生理学研、[6]CREST, JST

P3-091 単一神経細胞における *CNR / Pcdh* α遺伝子の発現解析
Variation of *CNR / Pcdh* αtranscripts in a single neuron
江角　重行[1,2]、平山　晃斉[1,2]、遠山　知子[1,2]、嘉数　直樹[5]、木津川尚史[4]、濱田　俊[1,2]、八木　健[1,2,3]
[1]大阪大院・生命機能・時空生物学・心生物学、[2]CREST, JST、[3]生理学研・高次神経機構、[4]基礎生物学研・種分化第一、[5]京都府立医科大院・医・分子標的癌予防医学

P3-092 神経回路形成期におけるCNR/Pcdh αファミリー蛋白質の解析
CNR/Pcdhαfamily proteins in the developing cortical connectivity
森下　博文[1,2,4]、村田　陽二[1]、江角　重行[1,4]、濱田　俊[1,4]、八木　健[1,2,3,4]
[1]大阪大院・生命機能研究科、[2]大阪大院・医、[3]生理学研、[4]CREST,JST

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P3-093 ニューロプシン遺伝子欠損マウスにおけるL1免疫陽性シナプスの数および形態変化
Numerical and morphological abnormalities of L1-immunoreactive synapse in neuropsin-deficient mice
中村　雪子[1]、堀之内和広[1]、塩坂　貞夫[1]
[1]奈良先端科学技術大学院大・バイオサイエンス・細胞構造学

P3-094 成熟マウス海馬におけるL1CAMの相手分子のスクリーニング
Screening of possible postsynaptic binding partners for presynaptic L1CAM in the hippocampus
山崎　浩誠[1]、中村　雪子[1]、塩坂　貞夫[1]
[1]奈良先端科学技術大学院大学・バイオサイエンス・細胞構造学

P3-095 神経幹細胞における複合型糖鎖の役割
Role of complex N-linked oligosaccharides on embryonic neural stem cells
浜之上　誠[1]、高松　研[1]
[1]東邦大・医・第2生理

P3-096 嗅神経/鋤鼻神経におけるCNR/Pcdhαファミリー蛋白質の発現
Differential expression of CNR/Pcdhα family proteins in the olfactory and vomeronasal nerves
長谷川園子[1]、村田　陽二[2]、濱田　俊[1,3]、八木　健[1,3]
[1]大阪大院・生命機能、[2]東京医科歯科大、[3]CREST, JST

P3-097 CNR/Pcdhαスプライシングバリアント特異的遺伝子変換マウスの表現型解析
Phenotype analysis of CNR/Pcdhα splicing variant-specific gene-targeted mice
福田　絵美[1,3]、濱田　俊[1,3]、平林　敬浩[2,3]、八木　健[1,2,3]
[1]大阪大院・生命機能、[2]岡崎生理学研、[3]CREST, JST

P3-098 神経活動によって誘導される神経接着分子arcadlinの細胞内情報伝達機構
Activation of the signal cascade by the activity-regulated adhesion molecule Arcadlin in brain
安田　新[1]、杉浦　弘子[1]、鈴木　香子[1]、前野　愉香[1]、竹宮　孝子[1]、山形　要人[1]
[1]東京都神経研・神経薬理

再生、移植
Regeneration, Transplantation

P3-099 In vitro ischemiaにおける神経幹細胞の活性化機構
In vitro ischemia regulates proliferation and differentiation of murine neural stem cells.
堀江　信貴[1]、守屋　孝洋[1]、平石　敬三[1]、北川　直毅[2]、永田　泉[2]、篠原　一之[1]
[1]長崎大・院・医歯薬・神経機能、[2]長崎大・脳神経外科

P3-100 低濃度NOによるネコ網膜神経節細胞の軸索再生の促進
Low-dose NO promotes axonal regeneration of cat retinal ganglion cells
渡部　眞三[1]、時田　義人[1]
[1]発達障害研・周生期

P3-101 ドパミン入力の欠如した線条体に発現増加するpleiotrophinの神経移植における効果
Effect of pleiotrophin, up-regulating in dopamine-depleted striatum, on neural transplantation
飛田 秀樹[1]、増田 匡[1]、浦川 将[1]、熊崎 路子[1]、櫻井 輝美[1]、西野 仁雄[1]
[1]名古屋市立大院・医・脳神経生理学

P3-102 Nogo受容体とそのホモログのNogo結合特性の比較解析
Comparison of the Nogo binding specificity between Nogo receptor and its homolog
佐藤 理行[1,2]、藤井 博[1]、五十嵐道弘[1]
[1]新潟大院・医歯学・分子細胞機能学（医・生化2）、[2]新潟大院・医歯学・整形外科学

P3-103 LacZ標識マウス，ラット培養シュワン細胞株の樹立と移植に関する検討
Establishment of LacZ-labeled mouse and rat Schwann cell lines for neural transplantation
渡部 和彦[1]、林 祐一[1]、川添 陽子[1]、伊藤聰一郎[2]
[1]東京都神経研・分子神経病理、[2]東京医科歯科大・疾患遺伝子実験センター

P3-104 軸索再生したラット網膜神経節細胞において、再生後に起こる減少
Decrease of larger retinal ganglion cells regenerating axons into a peripheral nerve graft of rats
加藤 美幸[1]、渡部 眞三[1]
[1]愛知県コロニー・発達障害研究所

P3-105 神経幹細胞の分裂能におけるベンゾジアゼピン受容体の役割について
The role of Benzodiazepine receptor in the neural stem cells proliferation
平石 敬三[1]、守屋 孝洋[1]、堀江 信貴[1]、篠原 一之[1]
[1]長崎大院・医歯薬・神経機能

P3-106 培養系におけるマイクログリア前駆細胞、MGBとProMGB
Putative precursor cells for microglia in vitro; microglioblasts and promicroglioblasts.
田中 潤也[1]、横山 彰子[1]、高橋 寿明[1]、玉井 浩二[1]、今井 嘉紀[1]
[1]愛媛大・医・分子細胞生理

P3-107 成熟ラット脳内の移動中の神経前駆細胞はドレブリンEを発現しているがドレブリンAは発現していない
Migrating neuronal precursors in adult rat brain express drebrin E but not drebrin A
宋 明橋[1,2]、花村 健次[1]、三國 雅彦[2]、白尾 智明[1]
[1]群馬大院・医・高次細胞機能学、[2]群馬大院・医・脳神経精神行動学

P3-108 ラット脊髄損傷後のp21投与による軸索再生と機能回復
Cytoplasmic p21$^{Cip1/WAF1}$ enhances axonal regeneration and functional recovery after spinal cord injury in rats
田中 啓之[1]、谷内 孝次[1]、遠山 正彌[1]
[1]大阪大院・医・ポストゲノム疾患解析学

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P3-109 マウスES細胞から運動ニューロンへの分化誘導：ES細胞由来神経系前駆細胞の特性の解析
Generation of motor neurons from mouse embryonic stem cells: analysis of ES-cell-derived neural cells
岡田 洋平[1,2,3]、島崎 琢也[1,3]、祖父江 元[2]、岡野 栄之[1,3]
[1]慶応義塾大・医・生理学、[2]名古屋大院・医・神経内科、[3]CREST, JST

P3-110 運動神経損傷時におけるRegファミリーメンバーの発現変動
Expressions of Reg/PAP family members are differentially regulated after nerve injury
濤川 一彦[1]、村上 公一[1]、福嶋 宗久[1]、鈴木 亨暢[1]、高沢 伸[2]、岡本 宏[3]、木山 博資[1]
[1]大阪市大院・医・機能細胞形態学、[2]東北大院・医・医学生物化学、[3]東北大院・医・先端再生生命科学

P3-111 マウス脊髄損傷モデルに対する顆粒球コロニー刺激因子の効果
Effects of granulocyte colony-stimulating factor (G-CSF) on spinal cord injury in mice
国府田正雄[1,2]、西尾 豊[1]、鎌田 尊人[1]、染谷 幸雄[1]、守屋 秀繁[1]、大河 昭彦[1]、山崎 正志[1]
[1]千葉大院・医・整形外科、[2]千葉大院・医・SRL環境健康医学

P3-112 脱随モデルにおける神経幹細胞のオリゴデンドサイトへの分化
Oligodendroglia differentiation of neural stem cells in a demyelination model
東 幹人[1]、等 誠司[1]、池中 一裕[1]
[1]三重大・医・医科学

P3-113 海馬歯状回顆粒細胞の生存と分化において細胞外環境の調節
Lamina-specific Morphogenesis of Dentate Granule Neurons
金 貞娥[1]、小山 隆太[1]、山田 隆二[1]、山田 麻紀[1]、西山 信好[1]、松木 則夫[1]、池谷 裕二[1]
[1]東京大院・薬・薬品作用

P3-114 キンギョ視神経切断後発現の網膜で増加する新規遺伝子について
A new cDNA clone of which expression incresed after optic nerve transection in goldfish retina
松川 通[1]、劉 中武[1]、永島 幹子[2]、加藤 聖[1]
[1]金沢大・医・脳情報分子、[2]金沢大・医・保健・検査

P3-115 中枢性線維芽細胞の増殖を介したFGF-2による脊髄損傷修復効果
FGF-2 facilitates axonal regeneration of the injured spinal cord via inducing CNS-derived fibroblasts proliferation
宗宮 仁美[1]、慈幸 貴洋[1]、福光 秀文[1]、古川 昭栄[1]
[1]岐阜薬科大・分子生物学

P3-116 レチノイン酸は金魚視神経再生をトリガーするか?
Does retinoic acid trigger axonal regeneration of fish optic nerve after injury?
馬渡 一浩[1]、松川 通[2]、杉谷 加代[1]、加藤 聖[2]
[1]金沢大・医・保健・検査、[2]金沢大院・医・脳情報分子

P3-117 新規Semaphorin 3A阻害剤のin vitroおよびin vivoにおける解析
In vitro and in vivo characterization of novel semaphorin 3A inhibitors
菊地 薫[1]、岸野 晶祥[1]、小西 修[1]、熊谷 和夫[1]、細谷 宜生[1]、佐治幾太郎[1]、中山智加男[1]、木村 徹[1]
[1]住友製薬・研究本部

P3-118 トリメチルスズ誘発性神経障害後の海馬CA3領域における神経新生
Possible neurogenesis in the hippocampal CA3 subfield after trimethyltin-induced neural damage
西山 徳人[1]、米田 幸雄[2]、荻田喜代一[1]
[1]摂南大・薬・薬理、[2]金沢大院・自然科学・薬物

P3-119 パーキンソン病モデルにおける神経幹細胞の解析
Analysis of progenitor cells in the striatum and midbrain by MPTP administration
大泉 英樹[1]、山口 正洋[2]、望月 秀樹[1]、水野 美邦[1]
[1]順天堂大・医・脳神経内科、[2]東京大院・医・細胞分子生理

P3-120 骨髄移植後初期に脳実質中へ移行する未分化骨髄細胞の性質に関する解析
Characterization of immature bone marrow cells migrating into the brain parenchyma after bone marrow transplantation
小野 健治[1]、鈴木 弘美[1]、澤田 誠[1]
[1]藤田保健衛生大・総医研・難病治療

P3-121 上鰓プラコードは鼻プラコードを補完する
Epibranchial placodes have a developmental potential of the olfactory placode.
丹一竹内京子[1]、河野 真子[1]、小川 正晴[1]
[1]理研・脳研・細胞培養技術開発

P3-122 RGMは脊髄損傷後の組織で発現上昇し、軸索再生・伸展阻害因子として働く
RGM is a neurite outgrowth inhibitor upregulated in response to central nervous system injury
羽田 克彦[1]、山下 俊英[1]
[1]千葉大院・医・神経生物

P3-123 Characterization of EGF and FGF responsive human embryonic neural stem/progenitor cells
Mohammed O. Islam[5]、Yonehiro Kanemura[1,3]、Jesmin Tajria[1]、Satoshi Kobayashi[1]、Hideki Mori[1]、Masayuki Hara[1]、Mami Yamasaki[3,4]、Hideyuki Okano[5]、Jun Miyake[1]
[1]Research Institute of Cell Engineering (RICE), AIST-Kansai、[2]Insititue of Biomedical Research and Innovation、[3]Institute for Clinical Research、[4]Department of Neurosurgery, Osaka National Hospital、[5]Keio University School of Medicine

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神経細胞死、アポトーシス
Neuronal Death and Apoptosis

P3-124 Keap1/Nrf2/ARE/HO-1を介したニューロン死制御
Role of Keap1/Nrf2/Antioxidant-Responsive Element/Hemeoxygenase-1 System in the Neuroprotective Effects by Cyclopentenone Prostaglandin Derivatives
佐藤　拓己[1,2]、岡本　秀一[2]、渡辺　恭良[3]、古田　享史[4]、鈴木　正昭[4]、Stuart Lipton[2]
[1]岩手大・工・福祉システム・福祉生体工学、[2]The Burnham Institute, La Jolla, USA、[3]大阪市大院・医・システム神経科学、[4]岐阜大院・医・再生医学

P3-125 新規小胞体ストレスセンサーOASISの機能解析
The regulation of unfolded protein response by OASIS, a transmembrane bZIP transcription factor, in astrocytes.
近藤　慎一[1]、村上　智彦[1]、和中　明生[2]、今泉　和則[1]
[1]奈良先端大・バイオサイエンス・細胞構造、[2]奈良県立医科大・第二解剖

P3-126 ショウジョウバエE2F1/DP過剰発現による分裂終了ニューロンでのアポトーシス誘導
Overexpression of dE2F1 and dDP induces apoptosis in Drosophila postmitotic neurons
荒木　健児[1]、西村伊三男[1]、吉川　和明[1]
[1]大阪大院・蛋白研・蛋白質機能制御

P3-127 新生児期ラット大脳皮質より精製したprocaspase-3結合蛋白の解析
Purification of protein that interacts with procaspase-3 from rat neonatal cortex
岡田　和久[1]、佐伯万騎男[1]、入江　康至[2]、寺尾　豊[3]、川端　重忠[3]、上崎　善規[1]
[1]大阪大院・歯・薬理、[2]大阪大院・医・薬理、[3]大阪大院・口腔細菌

P3-128 カルシニューリンによる低酸素応答性因子（HIF-1α）の安定化の解析
Calcineurin stablizes hypoxia-inducible factor（HIF-1α）.
加藤一玉水しおり[1]、加藤　裕之[1]、芝崎　太[1]
[1]東京都臨床研・細胞生理

P3-129 カイニン酸によるマウス脳におけるDNAの酸化損傷と酸化プリンヌクレオシド三リン酸分解酵素MTH1による神経保護作用の検討
Kainic acid-induced oxidative DNA damage in mouse brain and neuro-protection by MTH1 encoding an oxidized purine nucleoside triphosphatase
梶谷　康介[1]、作見　邦彦[1]、山口　浩雄[1]、中別府雄作[1]
[1]九州大・医・生体防御医学研・脳機能制御学

P3-130 Rho/Rho-kinaseシグナル伝達系による運動神経細胞の生存の制御
Survival of developing motor neurons mediated by Rho/Rho-kinase signaling pathway
小林　憲太[1]、高橋　将文[2]、松下　夏樹[3]、宮崎　純一[4]、小池　正人[5]、八木沼洋行[5]、大隅　典子[2]、貝淵　弘三[6]、小林　和人[1]
[1]福島医大・医・生体機能、[2]東北大院・医・器官構築、[3]愛知県コロニー、[4]大阪大院・医・幹細胞制御、[5]福島医大・医・解剖、[6]名古屋大院・医・細胞情報薬理

P3-131 ラット大脳皮質培養細胞におけるglycogen synthase kinase-3阻害剤によるエタノール誘導アポトーシスの阻止
Glycogen synthase kinase-3 inhibitors prevent caspase-dependent apoptosis induced by ethanol in cultured rat cortical neurons
高寺 恒雄[1]、大屋敷孝雄[1]
[1]北陸大・薬・臨床化学

P3-132 セクレトグラニンII由来新規神経ペプチド"マンセリン"
Manserin, a secretogranin II-derived peptide, is a novel neuropeptide
成田 正明[1]、前島 徹[2]、成田奈緒子[1]
[1]筑波大・基礎医学系・解剖学、[2]筑波技術短期大・理学療法学科

P3-133 カスパーゼ-3結合タンパク質の2つのスプライシング・バリアントによるカスパーゼ-3の相互調節
Reciprocal regulation of caspase-3 by two splice variants of caspase-3 binding protein
渡辺 崇[1]、武笠 毅[1]、野中 隆[2]、長谷川成人[2]、額田 敏秀[1]
[1]東京都精神医学研・脳細胞情報伝達、[2]東京都精神医学研・分子神経生物学

P3-134 経角膜電気刺激の変性網膜神経節細胞に対する生存促進効果
Survival-promoting effect of transcorneal electrical stimulation on axotomized RGCs *in vivo*
森本 壮[1]、三好 智満[1]、松田 理[3]、不二門 尚[2]、田野 保雄[2]、福田 淳[1]
[1]大阪大院・医・情報生理、[2]大阪大院・医・感覚機能形成学、[3]大阪大院・医・眼科学

P3-135 PKAとPKCの同時活性化によるERKとp38 MAP kinaseを介した過酸化水素誘発ネクローシス抑制作用
The prevention of H_2O_2-induced necrosis by coactivation of PKA and PKC is mediated by ERK and p38 MAP kinase activation in PC12 cells.
吉岡 靖啓[1]、竹島 直子[1]、北尾 達哉[1]、山室 晶子[1]、前田 定秋[1]
[1]摂南大・薬・薬物治療

P3-136 神経細胞型グルタミン酸トランスポーターEAAC1/EAAT3による新たな細胞死防御機構
Neuronal glutamate transporter, EAAC1/EAAT3, prevents neuronal death with novel anti-apoptotic function
桐生一瀬尾寿美子[1]、加藤 隆一[1]、平山 光久[1]、木山 博資[1]
[1]大阪市立大院・医・機能細胞形態学

P3-137 ペルオキシナイトライトにより誘導される小胞体ストレスにおけるATF6の活性化
Activation of ATF6 in response to peroxynitrite-induced endoplasmic reticulum stress in human neuroblastoma SH-SY5Y cells
森田 恵子[1]、山室 晶子[1]、河口 義隆[1]、丸山 直岳[1]、吉岡 靖啓[1]、前田 定秋[1]
[1]摂南大・薬・薬物治療

P3-138 大脳皮質初代培養ニューロンの過酸化水素誘発細胞死にはTRPM2チャネルが関与する
TRPM2 channels are involved in the H_2O_2-induced cell death in cultured cerebral cortical neurons
金子 周司[1]、川上 聖子[1]、伊藤 悦子[1]、高田 悠記[2]、久米 利明[2]、赤池 昭紀[2]
[1]京都大院・薬・生体機能解析、[2]京都大院・薬・薬品作用解析

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P3-139 神経成長因子除去による神経突起変性過程におけるCRMP-2の変化
Dysregulated protein expression of CRMP-2 in neurite degeneration following trophic factor withdrawal
加藤 聡子[1]、池上 浩司[1]、小池 達郎[1]
[1]北海道大院・理・生物科学

P3-140 NMDA受容体の活性化はマウス海馬におけるカイニン酸神経毒を抑制する
Activation of NMDA receptors suppresses kainate-induced neurotoxicity in murine hippocampus
杉山 千絵[1]、渡邉 真未[2]、米田 幸雄[2]、荻田喜代一[1]
[1]摂南大・薬・薬理、[2]金沢大院・自然科学・薬物

P3-141 トリメチルスズによるマウス海馬ミトコンドリアDNAの変異
Mitochondrial DNA mutation induced by trimethyltin in murine hippocampus
長嶋 玲子[1]、藩守 一朗[2]、後藤 由佳[1]、杉山 千絵[1]、伊藤 文昭[2]、荻田喜代一[1]
[1]摂南大・薬・薬理、[2]摂南大・薬・生化

P3-142 マウス脳ミトコンドリア画分におけるグルココルチコイド受容体のデキサメタゾンによる変化
Altered levels of glucocorticoid receptors in the mitochondrial fractions by dexamethasone in murine brain
中谷 有希[1]、黒岩 仁美[1]、杉山 千絵[1]、荻田喜代一[1]
[1]摂南大・薬・薬理

P3-143 トリメチルスズ誘発性神経細胞傷害に対する内在性グルタチオンの抑制効果
Possible protective effect of endogenous glutathione against trimethyltin-induced neuronal cell damage
世古 敬一[1]、西山 徳人[1]、樋口 桂[1]、米田 幸雄[2]、荻田喜代一[1]
[1]摂南大・薬・薬理、[2]金沢大院：自然科学・薬物

P3-144 初代培養神経細胞における転写抑制による遺伝子発現プロファイル
Gene expression profile change by transcriptional suppression in primary culture neurons: a model of polyglutamine diseases.
星野 将隆[1,2]、田川 一彦[1]、丸淵 茂樹[1,3]、戚 美玲[1,4]、岡澤 均[1,2,4]
[1]東京医科歯科大・難治研・神経病理学、[2]21世紀COEプログラム、[3]富山化学工業、[4]CREST、さきがけ研究21、PRESTO, JST

P3-145 マウス海馬におけるトリメチルスズによる高分子熱ショック蛋白質の発現増強
Enhanced expression of the high-molecular-weigth heat shock proteins in murine hippocampus after trimethyltin treatment
岩本 直子[1]、後藤 由佳[1]、伊藤 文明[2]、米田 幸雄[3]、荻田喜代一[1]
[1]摂南大・薬・薬理、[2]摂南大・薬・生化、[3]金沢大院・自然科学・薬物

P3-146 海馬細胞の可塑性に与えるニコチンの影響
Effects of nicotine on neural development and plasticity in the hippocampus
鬼頭 昭三[1]
[1]兵庫大・健康科学部・脳科研

P3-147 神経特異的分子SFAP75のアポトーシス抑制作用の解析
Characterization of inhibitory effects of brain-specific, SemaF (Sema4C) -associating protein, SFAP75, on serum-deprivation-induced apoptosis in CHO cells
酒井 学[1]、大岡 嘉治[1]、小林 千恵[2]、松本 浩[2]、稲垣 忍[1]
[1]大阪大院・医・保健、[2]大阪大院・歯・一口外

P3-148 ラットの海馬スライス培養においてCA2-CA3aはbicucullineの刺激によって最も損傷を受けやすい領域の一つである
The CA2-CA3a is one of the most vulnerable regions to bicuculline stimulation in the rat hippocampal slice cultures
韓 春錫[1]、河西奈保子[1]、鳥光 慶一[1]
[1]NTT物性科学基礎研究所

P3-149 ラット運動ニューロン短期培養系によるグルタミン酸毒性の評価
Glutamate toxicity in short-term cultures of rat motoneurons
濱野 史子[1,2]、宮崎 真[1]、日比野有美[1]、岡 淳一郎[2]、金井 文彦[1]、小俣 政男[1]、荒川 義弘[1]
[1]東大病院・臨床試験部、[2]東京理科大院・薬・薬理

P3-150 大脳半球由来因子の神経細胞死抑制作用
Action of a neuroprotective factor derived from cerebral hemispheres
本田 岳夫[1]、田畑 秀典[1,2]、仲嶋 一範[1,3]
[1]慶應大・医・解剖、[2]CREST, JST、[3]慈恵医大・DNA研・分子神経

P3-151 硫化水素は神経細胞に対する酸化的ストレスを防御する
Hydrogen sulfide protects neurons from oxidatieve stress
木村 英雄[1]、木村 由佳[1]
[1]国立精神神経センター、神経研究所、遺伝子工学

P3-152 小胞体におけるBcl-2ファミリーによるカスパーゼ-7活性化制御機構の解析
ER stress induced caspase-7 activation is regulated by Bcl-2 family at endoplasmic reticulum
中川 敏幸[1]、Ross Tomaino[2]、光田 輝彦[1]、Steven Gygi[2]、Junying Yuan[2]
[1]岐阜大院・医・神経生物、[2]Department of Cell Biology, Harvard Medical School

P3-153 プリオン蛋白質過剰発現老齢トランスジェニックマウスに於けるミトコンドリア由来アポトーシス機構
Age-dependent mitochondria-mediated neuronal apoptosis in transgenic mice overexpressing cellular prion protein
金子 清俊[1,2]、山田真紀子[1,2]、定塚 昌子[1,2]、大久保卓哉[1,3]、逆瀬川裕二[1,2]、八谷 如美[1,2]
[1]国立精神神経センター・疾病研究第7部、[2]科学技術振興機構、[3]東京医科歯科大、神経内科

自律神経系
Autonomic Nervous System

P3-154 マウス小腸におけるカハールの介在細胞の機能的神経支配
Functional innervation of the interstitial cells of Cajal in the mouse small intestine
飯野 哲[1]、野条 良彰[1]
[1]福井大・医・形態機能医科学講座

Sep 23 (Thu) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

P3-155 麻酔下の新生ラットにおける腹壁筋の呼息性活動
Abdominal expiratory motor activity in the anesthetized neonatal rat
飯塚眞喜人[1]
[1]茨城県立医療大・医科学センター・生理学

P3-156 排尿収縮には正帰還ループのほかにBarrington核からのフィードフォワード制御が必要である
Feedback and feed-forward regulation of micturition contraction by Barrington's nucleus
佐々木光美[1]
[1]東京医大・第二生理

P3-157 ラット胸髄で交感神経節前ニューロンにシナプスするVGluT3陽性終末の電顕的証明
Ultrastructual evidence of the synaptic contact between VGluT3 positive terminals and sympathetic preganglionic neurons in rat thoracic spinal cord.
伊藤 哲史[1]、堀口 和秀[1]、飯野 哲[1]、野条 良彰[1]
[1]福井大・医・人体解剖学・神経科学

P3-158 麻酔下ラットにおける吻側延髄腹外側部に投射する小細胞性網様体ニューロンの電気生理学的同定
Electrophysiological identification of the parvicellular reticular formation (RFp) neurons that project to RVLM in anaesthetized rats
Diana Oskutyte[1]、石塚 健一[1]、佐藤 義英[1]、村上 俊樹[1]
[1]日本歯科大・新潟歯・生理学

P3-159 皮膚触刺激によるラット脊髄血流の反応
Responses of spinal blood flow to cutaneous brushing in rats
黒澤美枝子[1]、渡邉 織江[1,2]、Brian Budgell[3]
[1]国際医療福祉大・基礎医学研究センター、[2]国際医療福祉大院・医療福祉学、[3]京都大・医・保健学科

P3-160 モルモット精管のα_1収縮成分に対するT型カルシウムチャネルとカルシウム依存性Kチャネルの関与
Involvement of T-type Ca^{2+} channels and Ca^{2+}-activated K^+ channels in the α_1-adrenoceptor-mediated contraction of the guinea-pig vas deferens
坂井 朗子[1]、登坂 恒夫[1]
[1]東京医大・医・第二生理

P3-161 ラット皮質線維および扁桃体線維の外側視床下部ニューロンへの収束
Convergent inputs from the cortical and amygdaloid fibers to the lateral hypothalamic neurons in the rat
津森登志子[1]、横田 茂文[1]、岡 達郎[1]、岸 敏郎[1]、安井 幸彦[1]
[1]島根大・医・解剖・神経形態

P3-162 三叉神経降圧反応を誘導する神経基盤
Neural substrates inducing trigeminal depressor response
大下 修弘[1]、江口 覚[1]、中條 信義[1]、竹村 元秀[2]
[1]徳島大・歯・歯科麻酔学講座、[2]大阪大院・歯学研究科 高次脳口腔機能学講座

P3-163 外側傍小脳脚核セロトニン作動性神経の塩分および水分摂取調節への関与
Participation of serotonergic systems in the lateral parabrachial nucleus in sodium and water intake in rats
宮久保浩子[1,2]、田中　淳一[2]、林　　泰資[3]、山戸　浩一[2]、野村　正彦[4]
[1]兵庫教育大院・連合学校教育、[2]鳴門教育大・授業開発、[3]ノートルダム清心女子大・人間生活・食品栄養、[4]埼玉医大・生理

P3-164 ラット孤束核からニューロキニン１レセプター発現pre-Botzinger complexニューロンへのGABA性入力
Pre-Botzinger complex neurons expressing neurokinin-1 receptors receive GABAergic inputs from the nucleus of the solitary tract in the rat
横田　茂文[1]、津森登志子[1]、岸　　敏郎[1]、岡　　達郎[1]、安井　幸彦[1]
[1]島根大・医・神経形態学

P3-165 脳弓下器官領域におけるGABA受容体を介するセロトニンの放出調節
GABAergic control of serotonin release in the rat subfornical organ area
牛込　彰彦[1]、田中　淳一[2]、野村　正彦[1]
[1]埼玉医大・生理、[2]鳴門教育大・授業開発

P3-166 ドパミンによる呼吸リズムの調節：新生ラット摘出脳幹標本を用いた解析
Dopaminergic effects on respiratory rhythm generation in rat brainstem-spinal cord preparation
藤井　盛光[1,2]、梅澤　一夫[2]、荒田　晶子[1]
[1]理研・脳センター・記憶学習、[2]慶応大・理工

P3-167 ラット消化管壁内神経叢内のL-アスパラギン酸含有ニューロン
L-aspartate immunoreactive neurons in the rat enteric nervous system
長浜　真人[1]、馬　　寧[1]、仙波　禮治[2]
[1]三重大・医・解剖学第二、[2] 愛知県心身障害者コロニー・発達障害研究所

P3-168 脳弓下器官のアンギオテンシン受容による外側傍小脳脚核領域のセロトニン放出の減少
Reduced serotonin release in the lateral parabrachial nucleus area elicited by angotensin II activation of the subfornical organ in rats
畠中　智子[1,2]、宮久保浩子[1,2]、田中　淳一[2]、林　　泰資[3]、服部　幸雄[3]、野村　正彦[4]
[1]兵庫教育大院・連合学校教育、[2]鳴門教育大・授業開発 、[3]ノートルダム清心女子大・人間生活・食品栄養、[4]埼玉医大・生理

P3-169 自由行動ザリガニをもちいた心臓調節神経制御下の心電図の状態空間表示解析
State space representation of ECG in freely moving crayfish
矢澤　　徹[1,2]、田中　克典[2]、勝山　智男[2,3]
[1]東京都立大院・理・生物、[2]東京都立大学理学部Bipac、[3]沼津高専・物理

P3-170 映像から抽出した動きベクトル合成映像の生体影響
Biomedical effects evoked by a random-dot image composed of the global motion vector
板東　武彦[1]、木竜　　徹[2]、小林　倫丈[2]、野村　恵理[2]、飯島　敦彦[1]、小山田　浩[1]、戸田　春男[1]、鵜飼　一彦[3]、千葉　　滋[4]
[1]新潟大院・医・統合生理、[2]新潟大院・自然科学・情報理工、[3]早稲田大・理工・応用物理、[4]シャープ次世代商品開発センター

P3-171 Vasomotion が物質交換におよぼす影響
Effects of sympathetically induced vasomotion on capillary-tissue fluid exchange
櫻井 照美[1]、照井 直人[1]
[1]筑波大・基礎医学系・生理

P3-172 Allopregnanoloneは孤束核交連亜核のGABA$_A$受容体に作用して化学受容器反射を抑制する
Allopregnanolone inhibits the chemoreceptor reflex by acting on GABA$_A$ recetors in the commissural subnucleus of the nucleus tractus solitarius in rats
鈴木 正彦[1]、中村 信一[2]、丸山 敬[1]
[1]埼玉医大・薬理、[2]埼玉医大・麻酔

P3-173 ラット脳弓下器官に対するニューロペプタイドY免疫陽性投射の起始
Origins of neuropeptide Y-immunoreacive projections to the rat subfornical organ
河野 史[1]、増子 貞彦[1]
[1]佐賀大・医・生体構造機能

P3-174 嘔吐を誘発する神経回路；イヌとフェレットにおける相同性
The neural circuit to induce vomiting; homology between the ferret and dog
大西 貴子[1]、柳原 衛[2]、古川 直裕[3]、福田 博之[1]
[1]川崎医療福祉大学・臨床栄養学科、[2]岡山県立大・短期大学部、[3]川崎医科大・生理学

P3-175 ウサギ延髄縫線核における呼吸性ニューロンの発射様式と脊髄への投射
Firing pattern and projection to spinal cord of respiratory neurons in rabbit medullary raphe nuclei
細貝 正江[1]、松尾 聡[1]
[1]鳥取大・医・機能形態統御学・適応生理学

P3-176 真骨魚類における視床下部に投射する主要な終脳背側部領域
MAJOR PORTIONS OF THE DORSAL TELENCEPHALON PROJECTING TO THE HYPOTHALAMUS IN A TELEOST, *OREOCHROMIS NILOTICUS*.
澤井 信彦[1]、伊藤 博信[1]
[1]日本医科大・医・第二解剖

P3-177 仙骨神経節におけるNOS, ENK陽性神経線維の分布
A characteristic distributional pattern of NOS-containing and ENK-containing nerve fibers in the sacral sympathetic ganglia.
李 明子[1]、村田 祐造[1]、増子 貞彦[1]
[1]佐賀医科大・医・生体構造機能

P3-178 マウスのCO_2換気応答に対する背内側延髄のセロトニンの効果
Effects of serotonin in the dorsomedial medulla oblongata on ventilatory responses to CO_2 in mice
金丸みつ子[1]、岩瀬みち子[1]、本間 生夫[1]
[1]昭和大・医・第二生理

P3-179 正常及び脳梗塞ラットの吻側橋網様体刺激による膀胱活動と脊髄グリシン濃度の変化
Effect of rostral pontine reticular formation stimulation on bladder activity and the spinal glycine level in rats with or without cerebral infarction
西島さおり[1]、菅谷　公男[1]、宮里　　実[1]、諸角　誠人[1]、小川　由英[1]
[1]琉球大・医・泌尿器科

P3-180 L-carnosine による自律神経活動及び血圧の変化における視交叉上核とヒスタミン受容体の役割
Involvement of suprachiasmatic nucleus and histaminergic sysytem in effect of L-carnosine autonomic nervous and cardiovascular systems
谷田　　守[1]、新島　　旭[2]、澤井　　元[3]、福田　　淳[3]、木曽　良信[4]、永井　克也[1]
[1]大阪大・蛋白研・代謝、[2]新潟大・医、[3]大阪大・医・情報生理、[4]サントリー（株）・健康科学

P3-181 カメの脊髄神経節交感神経節複合体について
The spinal sympathetic ganglia of the chelonian *Trachemys scripta elegans*
門田　哲夫[1]、中野　真人[1]、久島　達也[1]、跡部　好敏[1]、R-C. Goris[1]、船越　健悟[1]
[1]横浜市立大・医・解剖学第二

P3-182 ラット背側縫線核への8-OH-DPATの微量注入は呼吸抑制と脳波の速波化を引き起こす
Microinjection of 8-OH-DPAT into dorsal raphe nucleus produces respiratory inhibition and faster rhythms on electrocorticogram in rat.
中谷　康司[1]、関　　由成[1]、麓　　正樹[1]、鈴木　郁子[1]、小栗　　貢[1]、有田　秀穂[1]
[1]東邦大・医・第1生理

P3-183 意識下のラットにおける2-deoxy-D-gulcose投与時の腎と腰部交感神経活動の地域差
Non-uniform regional sympathetic nerve responses to 2-deoxy-D-gulcose in conscious rats
永田　恵子[1]、吉本　光佐[1]、三木　健寿[1]
[1]奈良女子大院・人間文化・生活環境・生活健康

P3-184 ノルアドレナリンのラット静脈内または脊髄髄腔内投与が排尿反射と脊髄グリシン濃度に及ぼす効果
Effects of intravenous or intrathecal noradrenalin on the micturition reflex and the spinal glycine level in rats
菅谷　公男[1]、西島さおり[1]、宮里　　実[1]、諸角　誠人[1]、小川　由英[1]
[1]琉球大・医・泌尿器科

P3-185 ラット部分閉塞膀胱後の膀胱機能と腰仙髄内及び血清中グリシン濃度の変化
Changes of bladder activity and the glycine level in the lumbosacral cord and serum after partial bladder outlet obstruction in rats
宮里　　実[1]、菅谷　公男[1]、西島さおり[1]、諸角　誠人[1]、小川　由英[1]
[1]琉球大・医・泌尿器科

P3-186 血圧反射実験における延髄心血管運動ニューロン単一ユニット記録法の改良
An improved method of recording a single-unit activity from medullary cardiovascular neurons in baroreflex experiments
三枝　岳志[1]、有田　　順[1]
[1]山梨大・医・第一生理

Sep 23 (Thu) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

神経内分泌
Neuroendocrine System

P3-187 ラット脳室内に投与したCRHは脳内アセチルコリン含有神経系および脳内ノルアドレナリン含有神経系の賦活を介して血漿コルチコステロンを増加させる
Centrally administered CRH-induced elevation of plasma corticosterone is mediated by activation of the brain stem noradrenergic neurons in rats
岡田尚志郎[1]、清水　孝洋[1]、横谷　邦彦[1]
[1]高知大・医・神経薬理

P3-188 ラット脳室内投与ボンベシンによる副腎ノルアドレナリン遊離には脳内トロンボキサンA2が関与する
Centrally administered bombesin elicits adrenal noradrenaline release by brain thromboxane A2-mediated mechanisms in rats
横谷　邦彦[1]、横谷　恵子[1]、中村久美子[1]、岡田尚志郎[1]
[1]高知大・医・神経薬理学

P3-189 L-TRYPTOPHAN及び&BETA-ENDORPHINの投与が中枢性疲労に及ぼす影響
Control of central fatigue by using a combination of injected L-tryptophan and &beta-endorphin
山本　隆宣[1]、Eric A. Newsholme[2]
[1]帝塚山大．心理福祉．大脳生理、[2]Merton College, University of Oxford, Oxford, U.K.

P3-190 ラット脳海馬における性ステロイド合成酵素及び受容体の発現解析
mRNA analysis of enzymes and receptors for neurosteroids in an adult male rat hippocampus
石井　寛高[1,2,3]、園木　康大[2,3]、古川　愛造[3,4]、北條　泰嗣[3]、川戸　佳[1,2,3]
[1]東京大院・理・生物物理、[2]東京大院・総合文化・生命環境、[3]CREST、[4]国立療養所久里浜病院

P3-191 エストロゲンの小脳顆粒細胞生存支持効果と雌雄差
17-beta estradiol promotes the survival of cultured male rat cerebellar granule neurons.
年綱　志保[1]、富永一吉野恵子[1]、小倉　明彦[1]
[1]大阪大院・生命機能・脳神経工学

P3-192 副腎皮質刺激ホルモン放出ホルモン受容体拮抗剤の脳室内投与は、ニューロメジンUによるパルス状LH分泌抑制を阻止する
Injection of corticotropin-releasing hormone (CRH) receptor antagonist blocks the suppression of pulsatile LH secretion induced by neuromedin U (NMU) in ovariectomized (OVX) rats after 48 hours of fasting
全　紅[1]、舩橋　利也[1]、貴邑冨久子[1]
[1]横浜市大院・医・神経内分泌学

P3-193 ob/obマウス視床下部におけるGLUT4トランスローケーションの検討
Glucose transporter 4 (GLUT4) translocation system in the hypothalamic arcuate nucleus of ob/ob mice
小森　忠祐[1]、森川　吉博[1]、田村　志宣[1]、土井　麻子[2]、南條輝志男[2]、仙波恵美子[1]
[1]和歌山県立医科大・第二解剖、[2]和歌山県立医科大・第一内科

P3-194 ラット脳におけるオレキシンニューロンの性差について
Sexual difference in the orexin neurons in rat brain.
小澤 一史[1]、松田 賢一[1]、上田 陽一[2]、中里 雅光[3]、河田 光博[1]
[1]京都府立医科大院・医・解剖学生体構造科学、[2]産業医科大学生理学教室、
[3]宮崎大・医・第3内科

P3-195 ラット海馬におけるニューロステロイド合成
Neurosteroid Synthesis in the Rat Hippocampus.
木本 哲也[1,2]、北條 泰嗣[1,2]、榎並 太平[1,2]、中嶋 浩平[1,2]、村上 元[1,2]、山崎 岳[3]、
小南 思郎[3]、原田 信広[4]、川戸 佳[1,2]
[1]東京大院・総合文化・広域科学・生命物理、[2]CREST, JST、[3]広島大・総合科学、[4]藤田保健衛生大・医・生化学

P3-196 視床下部大細胞性ニューロンに特異的に存在する新規E3ユビキチンリガーゼの同定
Identification of a novel E3 ubiquitin ligase specifically exists in hypothalamic magnocellular neurons
高森 康晴[1]、宇都宮一泰[1]、片岡 洋祐[1]、黒川 清[1]、渡辺 淳[1]、山田 久夫[1]
[1]関西医科大・解剖学第一

P3-197 GnRHサージ発生機構におけるfos発現の経時的変化とGnRH分泌能の左右差
Temporal changes in fos expression of GnRH surge-generating system and possibility of asymmetrical GnRH-secreting activity in female rats
塚原 伸治[1]、星 信彦[1]、北川 浩[1]
[1]神戸大院・自然科学

P3-198 Green Fluorescent Protein-Glucocorticoid receptor (GR) knockin mouseを用いた脳内GRの可視化
Imaging of Glucocorticoid Receptor (GR) in the Brain of Green Fluorescent Protein-GR Knockin Mouse
宇宿 智裕[1,2]、西 真弓[1]、森本 昌史[2]、杉本 徹[2]、Judson A. Brewer[3]、
Louis J. Muglia[3]、河田 光博[1]
[1]京都府立医大院・医・生体構造、[2]京都府立医大院・医・小児発達、[3]Department of Pediatics, Molecular Biology, and Pharmacology, Washington University School of Medicine, St. Louis, MO

P3-199 神経幹細胞の分化過程とステロイドレセプターコアクチベーターの発現
Expression of the steroid receptor coactivator during differentiation of neural stem cells
西原 永潤[1]、守屋 孝洋[1]、篠原 一之[1]
[1]長崎大学院・医歯薬・神経機能

P3-200 ステロイドホルモンによるマウス海馬Neuropsin (KLK8) の発現上昇
Regulation of hippocampal neuropsin (KLK8) mRNA by steroid hormones
原田 暁子[1]、石川 保幸[1]、塩坂 貞夫[1]
[1]奈良先端科学技術大学院大学・バイオサイエンス研究科・細胞構造学講座

Sep 23 (Thu) Poster Session

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P3-201 ラット海馬におけるストレスステロイド合成経路の解析
Analysis of Neurosteroidogenic Enzymes in Stress Steroid Synthesis in Adult Rat Hippocampus
園木 康大[1,2]、石井 寛高[1,2]、古川 愛造[3]、川戸 佳[1,2]
[1]東京大院・総合文化・広域科学、[2]科学技術振興機構、CREST、[3]国立久里浜病院

P3-202 マウス脳内チロシン水酸化酵素産生細胞におけるメラノコルチン受容体の発現
Expression of the melanocortin receptor (MC-R) family genes in tyrosine hydroxylase (TH) -producing neurons in the mouse brain
中村 浩章[1]、内田 克哉[1]、本間 裕[1]、佐藤 靖史[2]、小林 和人[3]、井樋 慶一[1]
[1]東北大院・情報科学・情報生物、[2]東北大・加齢医学研究所、[3]福島県立医大・生体情報伝達研究所・生体機能研究部門

P3-203 Estrogen and ovariectomy regulate expression of glutamic acid decarboxylases and cation-chloride cotransporters in adult female rat hippocampus.
Nozomu H. Nakamura[1]、Daniel R. Rosell[1]、Keith T. Akama[1]、Bruce S. McEwen[1]
[1]Neuroendocrinology, The Rockefeller University, New York

神経免疫
Neuroimmunology

P3-204 LPS投与ラット脳血管におけるプロスタグランジンE_2産生増大とその酵素機構の解析
Enhanced PGE_2 production in brain blood vessels of LPS-treated rat and its enzymatic mechanism
堀 あいこ[1]、山本 知子[1]、松村 潔[1]、水島由美子[1]、細川 浩[1]、小林 茂夫[1]
[1]京都大院・情報・知能情報・生体情報処理

P3-205 リポポリサッカライドの末梢投与により、マウス嗅球においてアポトーシスが誘導される
Peripheral lipopolysaccharide injection induces apoptosis in the murine olfactory bulb
森 啓至[1]、金子 葉子[1]、中島 昭[1]、永津 郁子[2]、太田 明[1]
[1]藤田保健衛生大学・医・生理、[2]藤田保健衛生大学医学部 解剖学

P3-206 リンパ球における非神経性コリン作動系の発現と免疫調節における役割
Expression of an independent, non-neuronal cholinergic system in lymphocytes and its roles in regulation of immune activity
藤井 健志[1]、川島紘一郎[1]
[1]共立薬大・薬理

P3-207 炎症性サイトカインの発現に及ぼすリポポリサッカライドとノルエピネフリンの影響
Effect of lipopolysaccharide and norepinephrine on the expression of proinflammatory cytokines
金子 葉子[1]、森 啓至[1]、中島 昭[1]、永津 郁子[2]、太田 明[1]
[1]藤田保健衛生大・医・生理学1、[2]藤田保健衛生大・医・解剖学2

P3-208 マイクログリアで発現するCCケモカインCCL6の解析
Characterization of CC chemokine CCL6 in microglia
鈴木 俊二[1]、菅野 素子[1]、藤原 崇志[1]、横山 彰子[1]、高橋 寿明[1]、今井 嘉紀[1]、田中 潤也[1]
[1]愛媛大・医・分子細胞生命科学講座・分子細胞生理学分野

P3-209 ミエリンP0タンパク質に対するヒト末梢神経障害患者血清中抗体の解析
Analysis of antibodies to peripheral myelin P0 in sera from human peripheral inflammatory neuropathies
林　明子[1]、大西　雅人[1]、中島圭慧子[1]、山口　宜秀[1]、藤井　香南[1]、薄　敬一郎[2]、結城　信泰[2]、馬場　広子[1]
[1]東京薬科大学・薬・機能形態、[2]獨協医科大学・神経内科

本能と情動行動
Instinct and Emotional Behavior

P3-210 初期応答遺伝子を用いたミツバチのダンス言語に関わる脳領野同定の試み
Attempt to map the honeybee brain regions involved in the dance language by using the immediate early gene
木矢　剛智[1]、國枝　武和[1]、久保　健雄[1]
[1]東大・院理・生物科学

P3-211 PrRPの不安行動における働き
Role of prolactin-releasing peptide in the control of anxiety behaviour in rats
尾仲　達史[1]、矢田　俊彦[1]
[1]自治医科大・医・生理

P3-212 ストレスとポリアミンの関連性：プトレッシンとα-ジフルオロメチルオルニチンの効果
Involvement of brain polyamines in stressful conditions: effects of putrescine and α-difluoromethylornithine
林　泰資[1]、田中　淳一[2]、北村　弥生[1]、長谷　沙織[1]、平田　智絵[1]、向井　恵子[1]、丹原香菜子[1]、服部　幸雄[1]
[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

P3-213 摂食関連ペプチドによる摂食行動制御におけるヒスタミン神経系の役割：オレキシン-A，ニューロペプチドY，グレリンの比較
A role of the histaminergic system for the regulatory mechanism of feeding by orexigenic peptides: orexin-A, neuropeptide Y, and ghrelin
石塚　智子[1]、野村　幸子[1]、細田　洋司[2]、寒川　賢治[2]、渡邊　武[3]、山本　隆[4]、大和谷　厚[1]
[1]大阪大院・医・保健・生体情報科学、[2]国循セ・研・生化、[3]理研・免疫アレルギー科学総合研究センター、[4]大阪大院・人間科学・行動生理

P3-214 不安情動行動におけるアクチビンの役割
Activin is involved in anxiety-related brain function
上田　洋司[1]、右島　理可[1]、茂手木淑子[1]、日野　敏昭[1]、高部　美穂[1]、喜田　聡[2]、杉野　弘[3]、土田　邦博[3]、横山　峯介[1]、井ノ口　馨[1]
[1]三菱化学生命科学研究所 井ノ口グループ、[2]東京農大、[3]徳島大学分子酵素学研究センター

P3-215 新規神経活性物質：7α-ヒドロキシプレグネノロンの機能と作用機構
A novel neuronal activator: function and mode of action of 7α-hydroxypregnenolone
松永　昌宏[1,2]、浮穴　和義[1,2]、筒井　和義[1,2]
[1]広島大・総合科学部脳科学・統合脳科学プロジェクト研究センター、[2]科学技術振興機構

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P3-216 ウィルソン病モデルラットでは運動機能の増大とプレパルス抑制の低下が認められる
Increased motor function and reduced prepulse inhibition in Wilson disease model rats
藤原 範子[1]、磯 博行[2]、北中 順惠[3]、北中 純一[3]、尾澤敬一郎[2]、下田 重朗[2]、
江口 裕伸[1]、大河原知水[1]、竹村 基彦[3]、鈴木敬一郎[1]
[1]兵庫医科大学・生化学、[2]兵庫医科大学・行動学、[3]兵庫医科大学・薬理学

P3-217 前脳特異的エストロゲン受容体過剰発現マウスを用いた情動行動解析
Analysis of transgenic mice overexpressing estrogen receptor in forebrain
内田 周作[1]、舛重 正一[1]、喜田 聡[1]
[1]東京農大・農・農化

P3-218 視床下部外側野メラニン凝集ホルモン（MCH）ニューロンのグルコース反応性の性差に対するエストロジェンおよびテストステロンの効果
Effects of testosterone (T) or estrogen (E) replacement on sex differences in the response of melanin-concentrating hormone (MCH) neurons to glucose in the LHA
舩橋 利也[1]、茂木 一孝[1]、貴邑冨久子[1]
[1]横浜市大院・医・神経内分泌学

P3-219 飢餓ラット視床下部におけるヒスタミンH_1受容体mRNAレベルの上昇
Increase in histamine H_1 receptor mRNA level in hypothalamus of starved rats
福井 裕行[1]、吉村 祥穂[1]、三島 涼子[1]、熊谷 恭子[1]、錦織 充[1]
[1]徳島大院・ヘルスバイオサイエンス研究部・分子薬理学

P3-220 疼痛関連情動反応における扁桃体でのノルアドレナリン神経伝達の関与
Roles of the noradrenergic neurotransmission at the amygdala in pain-related aversion
南 雅文[1]、中川 貴之[1]、瀧下あずさ[1]、谷本 幸[1]、金子 周司[1]、佐藤 公道[1]
[1]京都大院・薬・生体機能解析

P3-221 サル扁桃体における亜鉛の分布
Vesicular zinc distribution in the monkey amygdaloid complex
一戸 紀孝[1]、Kathleen Rockland[1]
[1]理研・BSI・脳皮質機能構造研究チーム

P3-222 絶食による視床下部腹内側核内のGABAとグルタミン酸分泌の変化
Fasting induced changes in GABA and glutamate release in the ventromedial hypothalamus using in vivo microdialysis
成田 和巳[1]、村田 拓哉[1]、樋口 隆[1]
[1]福井大学・医・統合生理学

P3-223 求愛行動発現におけるプロラクチンの中枢作用
CENTRAL ACTION OF PROLACTIN (PRL) ON THE INDUCTION OF COURTSHIP BEHAVIOR IN THE MALE NEWT
豊田ふみよ[1]、蓮沼 至[2]、山本 和俊[2]、山下 勝幸[1]、菊山 榮[2]
[1]奈良県立医科大学・第一生理、[2]早稲田大学・教育・生物

P3-224 囀り中の鳥における中脳VTAニューロンの活動は社会的文脈に依存した修飾を受ける
ACTIVITY OF VTA NEURONS IN SINGING BIRDS IS MODULATED BY SOCIAL CONTEXT.
柳原　真[1]、Neal A. Hessler[1]
[1]理研・BSI

P3-225 鳴鳥における求愛行動とその解剖学的解析
Investigation of behavioral and anatomical lateralization in courtshipsinging.
原　衣利奈[1]、小柴満美子[1]、Isabble George[1]、Neal A. Hessler[1]
[1]理化学研究所・BSI・発声行動機構

P3-226 味覚嗜好性摂取行動における腹側淡蒼球内神経調節の関与
Intake of palatable tastants is mediated by neuromodulation in the ventral pallidum
今岡　寛之[1]、志村　剛[1]、山本　隆[1]
[1]大阪大院・人間科学・行動生理

P3-227 子宮内環境が性的二型核（SDN-POA）の構造とERαの分布に与える影響
Organization of Sexually Dimorphic Nucleus and the Distribution of ERαReceptor Are Affected by the Intrauterine Position
裴　敏娟[1]、松田　賢一[1]、坂本　浩隆[1]、河田　光博[1]
[1]京都府立医大・生体構造科学

睡眠、生体リズム
Sleep and Biological Rhythms

P3-228 イソアワモチ神経節における眼外光受容器細胞の介在ニューロンとしての膜特性
Membrane properties as interneurons of the extra-ocular photoreceptors in the *Onchidium* ganglia
後藤　司[1]、西　孝子[2]、中河　志朗[1]
[1]鹿児島大院・医歯学総研・神経解剖、[2]Inst. Nati Sci, Senshu Univ., Kawasaki, Japan

P3-229 PACAP遺伝子欠損マウスにおけるSCNの光誘導性c-Fos発現およびERK，CREBのリン酸化解析
Light-induced c-Fos expression and phosphorylation of ERK and CREB in the SCN in mice lacking PACAP
川口ちひろ[1]、礒島　康史[2]、橋本　均[1]、新谷　紀人[1]、永井　克也[2]、馬場　明道[1]
[1]大阪大院・薬・神経薬理、[2]大阪大・蛋白研・代謝

P3-230 レム睡眠期に観測される橋の相動性活動に伴って生じる海馬θ波加速現象
Instantaneous acceleration of hippocampal theta wave associated with pontine phasic activity during REM sleep in rats
辛島　彰洋[1]、中尾　光之[1]、本多　和樹[2]、岩崎直穂子[1]、片山　統裕[1]、山本　光璋[1]
[1]東北大院・情報科学・バイオモデリング論分野、[2]東京医科歯科大・生体材料工学研究所

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P3-231 mPER1はmPER2タンパクを制御することで生物時計を環境の明暗サイクルに同調させている
mPER1 acts as a circadian adaptor to entrain theoscillator to environmental light-dark cycles by regulating mPER2 protein
増渕　悟[1]、片岡　憲稔[1]、Paolo Sassone-Corsi[2]、岡村　均[1]
[1]神戸大院・医・分子脳科学、[2]Institut de Génétique et de Biologie Moléculaire et Cellulaire, CNRS-INSERM-Université Louis Pasteur, Strasbourg, France.

P3-232 網膜光照射に対するマウス視交叉上核ニューロンと視交叉線維の発火応答感度
Different sensitivity of the suprachiasmatic nucleus neurons and optic fibers to light stimulation in mice
藤村　幸一[1]、中村　孝博[2]、海老原史樹文[2]、篠原　一之[1]
[1]長崎大院・医歯薬・神経機能、[2]名古屋大院・生命農・動物行動統御

P3-233 モルモット海馬スライスにおける、自発神経振動によるpEPSPの抑制
Spontaneous neuronal oscillation suppresses the evoked pEPSP in guinea-pig hippocampal slices
冨田　裕二[1]、夏目季代久[1]
[1]九州工業大院・生命体工学研究科

P3-234 転写調節因子CLOCK/BMAL1の新規相互作用因子の検索及び機能解析：NSP-C (Neuroendocrine specific protein-C) のCLOCK/BMAL1による転写に対する役割の解析の
Identification and functional analysis of new interacting proteins with transcription factor CLOCK and BMAL1
Function of NSP-C (Neuroendocrine specific protein-C) in CLOCK/BMAL1-mediated transcription
細田　浩司[1]、松井　勇輔[1]、亀田　明美[1]、舛重　正一[1]、喜田　聡[1]
[1]東京農大・応生科・バイオ

P3-235 視交叉上核における細胞間相互作用
Cell-to-cell interaction on the rat suprachiasmatic nucleus
野口　貴子[1,2]、渡辺　和人[1]、山岡　貞夫[1]
[1]獨協医大・生理学（生体制御）、[2]大阪大院・理学研究科・生物科学専攻

P3-236 末梢グリシンの松果体への蓄積
Accumulation of Peripheral Glycine in the Pineal Body
関　忍[1]、小野一武居　郁[1]、吉田真太郎[1]、稲川健太郎[1]、村上　昇[2]、高橋　迪雄[1]
[1]味の素株式会社健康基盤研究所、[2]宮崎大学・農・獣医学科・家畜生理学講座

P3-237 Enhancement of sleep by IL-1β is not mediated by prostaglandin D2 in mice
WM Qu[1]、ZL Huang[1]、N Eguchi[1]、S Narumiya[2]、Y Urade[1]、O Hayaishi[1]
[1]Dept Mol Behav Biol, Osaka Bioscience Institute、[2]Dept Pharmacol, Kyoto Univ Sch Med

P3-238 アセチルコリンによる神経調節を考慮した視床の振動現象に対するニューラルネットワークモデル
Neural Network Model for Oscillatory Phenomena in Thalamus with Neuromodulation Due to Acetylcholine
大森 敏明[1,2,3]、堀口 剛[3]
[1]科学技術振興機構 さきがけ研究21「協調と制御」研究領域、[2]理化学研究所・脳科学総合研究センター・脳数理研究チーム、[3]東北大院・情報科学研究科

P3-239 線虫 *Caenorhabditis elegans* による新たな概日時計機構の解析
Caenorhabditis elegans as a tool opening up new insights into the circadian clock mechanism.
三枝 徹[1]、田中館明博[2]、岡村 均[4]、長谷川建治[1,3]
[1]北里大院・医療系研究科、[2]北里大学・医療衛生、[3]北里大学・医、[4]神戸大院・医学系研究科

P3-240 哺乳類体内時計振動系のDELAY因子に関与する細胞内シグナリング
Intracellular signaling involved in the crucial time-delay of the mammalian clock-oscillating system
田丸 輝也[1]、礒島 康史[2]、高松 研[1]
[1]東邦大・医・第2生理、[2]阪大・蛋白研・代謝

P3-241 視交叉上核での時計関連遺伝子Per2とBmal1の生後発達：vasopressinとの関係
Postnatal development of rPer2, rBmal1 and Arg-vasopressin mRNA circadian rhythms in the suprachiasmatic nucleus of rat pups under the light-dark cycle and constant dim light.
磯部 芳明[1]、田内 春見[1]、川口 毅恒[1]
[1]名古屋市立大院・医・脳神経生理

P3-242 Altered behavior of sleep-wake cycle and histaminergic systems in mice lacking histamine H_1 receptor
黄 志力[1]
[1]大阪バイオサイエンス研究所

P3-243 ゾウリムシの核・ミトコンドリアDNAにおける損傷修復の概日変動
Circadian fluctuations in the activity of repairing DNA damaged in Paramecium nuclear and mitochondria.
田中 千貴[1]、三枝 徹[1]、博田 竜史[1]、山本 基栄[1]、長谷川建治[2]
[1]北里大院・医療系・脳機能、[2]北里大学・医・生理

P3-244 脳の「酸化」は睡眠発現の初期プロセスである
Brain oxidation is an initial process in sleep induction
本多 和樹[1]、相良 昌美[1]、岡田 哲也[2]、Clement Pirre[2]、裏出 良博[2]、杉山 崇[3]、吉岡 享[3]、井上昌次郎[1]、池田 真行[2]
[1]東京医科歯科大学・生体材料工学研究所・制御分野、[2]大阪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

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P3-245 FMR1 ノックアウトマウスにおける睡眠と体温リズム
Sleep and body temperature profiles in FMR1 knockout mice
佐野 敦子[1]、勢井 宏義[1]、北岡 和義[1]、近久 幸子[1]、上江洲香代子[1]、武井 延之[2]、森田 雄介[1]
[1]徳島大院・情報統合医・統合生理、[2]新潟大・脳研究所・分子神経生物学

P3-246 視交叉上核光同調機能促進因子としてのCCK-A受容体
CCK-A receptors as Photic Entrainable Signal Transducers in the Mouse Suprachiasmatic Nucleus
島添 隆雄[1]、森田 光貴[1]、渡辺 繁紀[1]、守屋 孝洋[3]、篠原 一之[3]、池田 真行[4]、河野 彬[2]、瀧口 総一[2]、宮坂 京子[5]、L.D. Mercer[6]、P.M. Beart[6]、船越 顕博[2]
[1]九州大院・薬・薬効解析、[2]九州がんセンター、[3]長崎大学大院・医歯薬総合・神経機能学分野、[4]大阪バイオサイエンス研、[5]東京都老人研・臨床生理、[6]Howard Florey Inst, Univ Melbourne, VIC3010 Australia

P3-247 30分間の音読が脳波に与える影響
Effects of 30 minutes oral reading on human EEG
麓 正樹[1]、関 由成[1]、中谷 康司[1]、毛利 右子[1]、中里 茜[1]、鈴木 郁子[1]、有田 秀穂[1]
[1]東邦大学・医・第一生理

P3-248 老齢ラット脳幹のアセチルコリン、モノアミン、及びGABA作動性ニューロンについて
Aging of cholinergic, monoaminergic and GABAeregic neurons in the brainstem of old rats
二宮冶重子[1]、小山 純正[1]、香山 雪彦[1]
[1]福島県立医大・医：第二生理

学習、記憶
Learning and Memory

P3-249 胎生期MAM投与による実験的小頭（脳）症ラットの離乳期および成体期における水迷路学習と海馬CA1の発生学的形態異常
Spatial cognition deficit in a water maze during both weaning and adult periods in experimentally-induced microencephalic rats with an abnormal morphogenesis of the CA1 hippocampus
杉岡 幸三[1]、薛 富義[1]、寺島 俊雄[1]
[1]神戸大院・医・脳科学・神経発生

P3-250 前頭前野－腹側被蓋野間閉回路を介した皮質内ドーパミン放出安定化の理論的研究
Theoretical implications for stable cortical dopamine release: contribution of the fronto-mesofrontal system
山下 幸樹[1]、田中 昌司[1]
[1]上智大院・理工・電気電子工学

P3-251 においの嫌悪学習と嗅球LTPの共通特性
Common properties of aversive olfactory learning and LTP in the OB
奥谷 文乃[1]、張 敬姫[1]、椛 秀人[1,2]
[1]高知大・医・統合生理、[2]生理研・環境適応機能発達

P3-252 鳩背外側皮質野の線維連絡
Afferent and efferent connections of the area corticoidea dorsolateralis in the pigeon
阿閉 泰郎[1]
[1]岐阜大・応用生物・獣医解剖学

P3-253 前頭前野内側部における二次的報酬による行動の評価
Action evaluation by secondary rewards in the medial prefrontal cortex
松元まどか[1]、松元 健二[1]、田中 啓治[1]
[1]理研・脳センター・認知機能表現

P3-254 神経活動に伴ってナメクジ脳で発現誘導される新規kruppel-like factorの解析
A novel kruppel-like factor induced by neuronal activity in slug brain
松尾 亮太[1]、福永 悟史[1]、星野 彰平[1]、桐野 豊[1]
[1]東京大院・薬・神経生物物理

P3-255 マウスの水平性視機性眼球反応の長期適応と長期抑圧との因果関係
Role of cerebellar LTD in the long-term adaptation of horizontal optokinetic response in mice
永雄 総一[1]、首藤 文洋[1]、大木 雅文[1,3]、北澤 宏理[1]、糸原 重美[2]
[1]理研BSI・運動学習制御、[2]理研BSI行動遺伝、[3]東大・医・耳鼻科

P3-256 瞬目反射条件づけ学習における海馬NMDA受容体の役割
Role of hippocampal NMDA receptors in trace eyeblink conditioning
坂本 敏郎[1,3]、高月香菜子[2]、川原 茂敬[2]、桐野 豊[2]、二木 宏明[3]、三品 昌美[1]
[1]東京大院・医・分子神経生物、SORST/JST、[2]東京大院・薬・神経生物物理学、CREST/JST、[3]理研・脳センター・情動機構

P3-257 単一神経細胞における多安定発火状態の実現メカニズム、及びワーキングメモリー機能との関連
A novel cellular mechanism for graded persistent activity in a model neuron and its implications in working memory
寺前順之介[1]、深井 朋樹[1,2]
[1]玉川大学・学術研究所、[2]玉川大学・工・情報通信工学科

P3-258 マウス前庭神経核の視機性眼球反応長期学習による電気生理学的変化
Changes of electrical responsiveness of vestibular nuclei correlated with the long-term adaptation of horizontal optokinetic responses in mice
大木 雅文[1,2]、首藤 文洋[2]、永雄 総一[2]
[1]東京大院・医・耳鼻咽喉科、[2]理研・BSI運動学習制御研究チーム

P3-259 メカミラミン誘発学習・記憶障害に対するノシセプチンの作用
Reversion of mecamylamine-induced acetylcholine decrease in rat hippocampus and learning and memory impairment by nociceptin
平松 正行[1,2]、三輪 将也[1]、橋本 和樹[2]、野村 奈央[2]
[1]名城大院・総合学術・生体機能制御、[2]名城大・薬・薬品作用

P3-260 C57BL老若男女マウスの神経幹細胞分布と行動特性
Behavioral performance and brain nestin distribution in the C57BL old mice.
黒川 衛[1]
[1]長崎大学・医・神経解剖（大学院医歯薬総合研究科発生分化機能再建学）

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P3-261 マウスにおけるリアクションタイム課題の獲得と遂行
Acquisition and performance of the reaction time task in mice
筒井 雄二[1]、小林 和人[2]
[1]福島大学・生涯学習教育研究センター、[2]福島県立医科大学・医・生体機能研究部門

P3-262 ラット背側帯状回皮質は２つの領域に分けられる
The rat dorsal cingulate cortex is subdivided into two distinct areas
松川 睦[1]、酒匂 裕子[1]、志水 巌[1]、有國 富夫[1]、今田 正人[1]
[1]日大・医・解剖

P3-263 小鳥の歌の可塑性は、歌制御中枢ニューロンの樹状突起スパインにおけるFアクチン集積と相関する。
Birdsong plasticity is correlated with cytoskeletal F-actin content within the dendritic spine in zebra finch song control nuclei.
坂口 博信[1]
[1]獨協医大・医・生理（生体情報）

P3-264 前頭前皮質と扁桃体からラット腹側海馬ニューロンへの逆行性発火
Antidromic activation of rat ventral hippocampal neurons from prefrontal cortex and amygdala
石川 晃教[1]、中村 彰治[1]
[1]山口大・医・高次神経科学

P3-265 海馬と前頭前野のlocal field potentialの関係に対する神経可塑性の影響
Effects of hippocampus-prefrontal LTP/LTD induction on cross correlation between hippocampal and prefrontal spontaneous local field potential powers in anesthetized rats
伊崎 義憲[1]、明間 立雄[1]
[1]聖マリアンナ医科大・生理

P3-266 ジュウシマツの大脳歌産生システムの雌雄差
Sexual Difference of Song Motor Connections in Bengalese Finch Telencepalon
戸張 靖子[1]、奥村 哲[2]、谷 淳[2]、岡ノ谷一夫[1,3,4]
[1]千葉大院・自然科学・多様性科学、[2]理研・BSI動的認知行動研究チーム、[3]理研・BSI生物言語研究チーム、[4]科学技術振興機構 さきがけ21

P3-267 ミミズ非連合学習におけるNO-cGMP経路の効果
Effect of NO-cGMP signaling in non-associative learning of the earthworm
北村美一郎[1]、青沼 仁志[2]、丹羽 光一[3]、水谷 賢史[1]、小川 宏人[4]、岡 浩太郎[1]
[1]慶大・理工・生命情報、[2]北大・電子研・神経情報、[3]北大・電子研・自律調節、[4]埼玉医大・生物

P3-268 時系列学習における事象関連電位研究
On-going sequence learning: evidence from event-related potentials study
Dilshat Abla[1,2]、岡ノ谷一夫[1,2,3]
[1]理研・脳科学センター・高次脳・生物言語、[2]科学技術振興機構・さきがけ21、[3]千葉大・文・認知情報

P3-269 味覚嫌悪学習の想起に対する扁桃体ノルアドレナリン伝達の促進作用
Noradrenaline transmission in the amygdala enhances memory retrieval of conditioned taste aversion
八十島安伸[1]、山本　隆[2]、小林　和人[1]
[1]福島医大・医・生体機能、[2]大阪大院・人間科学・行動生理

P3-270 海馬におけるドパミンD2レセプターのアセチルコリンを介した記憶への影響について
acetylcholine-dopamine interaction in the ventral hippocampus on memory in rats.
藤城　弘樹[1]、梅垣　宏行[1]、鈴木　裕介[1]、大原しのぶ[1]、山口　容子[1]、井口　昭久[1]
[1]名古屋大院・医・老年科学

P3-271 記憶保持回路の再構成には学習後の大脳皮質NMDA受容体の活性化が必須である
Systems consolidation requires the post-learning activation of cortical NMDA receptors
竹原　可織[1]、川原　茂敬[1,2]、桐野　豊[1]
[1]東京大院・薬・神経生物物理、[2]CREST, JST

P3-272 膜翅目（ハチ目）昆虫と脊椎動物で共通する遺伝子*Mahya*の解析
A novel gene, *Mahya*, is shared between Hymenoptera and vertebrates
門脇　辰彦[1]、船田　真広[1]、土本麻友美[1]、北川　泰雄[1]、吉村　崇[2]
[1]名大院・生命農・生命技術科学、[2]名大院・生命農・バイオモデリング

P3-273 サルの前頭連合野背外側部及び前頭眼窩野の活動に対する報酬スケジュールの影響
Effect of reward schedules on monkey dorsolateral prefrontal and orbitofrontal activities
竹田　里江[1,2]、船橋新太郎[2]
[1]札幌医科大・保・臨床作業療法、[2]京都大院・人間環境学・認知行動科学

P3-274 新規タンパク質合成の必要性を指標とした記憶再固定化と消失との関係の解析
Characterization of the relationship between memory reconsolidation and extinction
鈴木　章円[1]、Josselyn Sheena A.[2]、Frankland Paul W.[2]、舛重　正一[1]、Silva Alcino J.[3]、喜田　聡[1]
[1]東農大院・バイオ、[2]HSC, Canada、[3]UCLA, USA

P3-275 グルタミン酸局所投与により賦活される海馬神経回路ダイナミクス
Neuronal dynamics activated by local glutamate application in CA3 region of hippocampal slices
菊池　修[1]、片山　統裕[1]、中尾　光之[1]、山本　光璋[1]
[1]東北大院・情報科学・応用生命情報・バイオモデリング論

P3-276 HPC-1/syntaxin 1A欠損マウスの解析
Phenotype analysis of HPC-1/syntaxin 1A knock-out mice
藤原　智徳[1]、三嶋　竜弥[1]、小藤　剛史[1]、千葉　智樹[2]、田中　啓二[2]、赤川　公朗[1]
[1]杏林大学・医・細胞生理、[2]東京都臨床医学総合研究所・分子腫瘍

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P3-277 GluRδ2ノックアウトマウスの瞬目反射条件付けにおけるNMDA受容体の関与
Different contribution of NMDA receptors to the eyeblink conditioning between GluRδ2 mutant mice and wild-type mice
加藤 良子[1]、高月香菜子[1]、川原 茂敬[1]、森 寿[2]、三品 昌美[2]、桐野 豊[1]
[1]東京大院・薬・神経生物物理、[2]東京大院・医・薬理学・分子神経生物学

P3-278 マウス視機性眼球反応の長期適応に対する小脳片葉遮断と損傷での効果の相違
Neural mechanism of long-term motor learning revealed by different effects of floccular lesion and shutdown on the long-term adaptation of mouse ocular reflex
首藤 文洋[1]、大木 雅文[1]、北澤 宏理[1]、糸原 重美[2]、永雄 総一[1]
[1]理化学研究所・BSI・運動学習制御、[2]理化学研究所・BSI・行動遺伝

P3-279 前脳領域にCAMKIVを過剰発現させたマウスを用いた老齢時における学習・記憶能力の解析
Enhancement of memory consolidation in aged mutant mice overexpressing CaMKIV in forebrain
福島 穂高[1]、前田 良太[1]、鈴木 良祐[1]、遠藤 健悟[1]、舛重 正一[1]、喜田 聡[1]
[1]東京農業大学・動物分子生物学

P3-280 転写調節因子SRFの学習・記憶に対する役割の解析
Analysis of a role of transcription factor SRF in learning and memory
佐度 恵[1]、鈴木 章円[1]、細田 浩司[1]、舛重 正一[1]、喜田 聡[1]
[1]東京農大院・農・バイオサイエンス

P3-281 離乳後の粉餌による飼育が海馬体内へのアセチルコリン分泌の性差に及ぼす影響
Soft diet feeding after weaning affects sex difference in the acetylcholine release in the hippocampus
高瀬 堅吉[1]、美津島 大[1]、茂木 一孝[1]、貴邑冨久子[1]
[1]横浜市大院・医・神経内分泌学

P3-282 島皮質における報酬予測と関連した単一ニューロン反応
Single neuronal responses related to reward expectancy in insular cortex
水挽 貴至[1,3]、Barry J Richmond[2]、設楽 宗孝[3]
[1]筑波大学大学院、[2]Lab. of Neuropsychol., NIMH, Bethesda, MD, USA、[3]産業技術総合研究所・脳神経情報研究部門

P3-283 ショウジョウバエ幼虫においてCREB依存性記憶はキノコ体に局在している
CREB-dependent memory localizes in the mushroom bodies in *Drosophila* Larvae
本庄 賢[1]、齊藤 実[2]、古久保一徳永克男[1]
[1]筑波大学・生物科学系、[2]東京都神経科学総合研究所・分子神経生理研究部門

P3-284 ラットの物体弁別および物体再認に及ぼす嗅周皮質内NMDA受容体またはアセチルコリン受容体遮断薬投与の効果
NMDA and muscarinic blockade in the perirhinal cortex impairs object discrimination and recognition in rats
安部 博史[1]、石田 康[1]、岩崎 庸男[2]
[1]宮崎大・医・精神医学、[2]目白大・院・心理

P3-285 扁桃体から海馬への神経投射 －光学的計測法を用いた研究－
The neural projection from the amygdala to the hippocampus studied with an optical recording method
小金澤紀子[1]、大場 健嗣[1]、梶原 利一[2]、市川 道教[3]、飯島 敏夫[1]
[1]東北大院・生命科学・脳情報処理、[2]産業技術総合研究所・脳神経情報研究部門、[3]理化学研究所・脳科学総合研究センター

P3-286 海馬体内における一日のアセチルコリン分泌動態とテストステロンの役割
Testosterone maintains daily acetylcholine release in the hippocampus in male rats
美津島 大[1]、高瀬 堅吉[1]、貴邑冨久子[1]
[1]横浜市大院・医・神経内分泌

P3-287 eight-arms maze working memory task 遂行中のラット側座核神経活動
Neuronal activities of rat nucleus accumbens during eight-arms maze working memory task
星野 真人[1]、古舘 宏之[2]、関口 達彦[1]、Anthony G. Phillips[3]、松本 元[1]、木村 哲也[1]
[1]理研・BSI・脳創成表現、[2]埼玉大学、[3]Univ. British Columbia, Canada

P3-288 ヨーロッパ・モノアラガイにおけるショ糖、接触刺激による味覚嫌悪条件付け
Taste aversive conditioning in *Lymnaea*
榊原 学[1]、砂田 寛司[2]、川合 亮[2]、堀越 哲郎[1]
[1]東海大・開発工学部・生物工学科、[2]東海大院・開発工学研究科

P3-289 マカクザルにおける海馬CA1の皮質投射終末の検討
Cortical terminations of hippocampal projections from area CA1 in macaque monkeys
鞠負 正雄[1]、鐘 咏梅[2]、小長谷初枝[1]、Kathleen Rockland[2]
[1]東京都神経研・認知行動、[2]理研・脳研センター・脳皮質機能構造研究チーム

P3-290 海馬および嗅内野におけるcontextの学習および表現に関連した神経細胞活動
Population activities related to contextual learning and coding in rat hippoocampal formation
庄野 修[1]、木村 哲也[1]、松本 元[1]
[1]理化学研究所・脳科学総合研究センター・脳創成表現

P3-291 D1受容体アゴニスト（SKF38393）投与は小脳Golgi細胞に発現するUbiquitin-activating enzyme（Ube1x）mRNAのメタンフェタミン反復投与による発現の減少を回復させた
Effects of SKF38393 (D1 receptor agonist) injection on inhibition of Ubiquitin-activating enzyme (Ube1x) mRNA induced by methamphetamine (MAP) administration in rat vermis
濱村みつ子[1]、澤田 和彦[4]、島添 隆雄[3]、寺田 善弘[3]、服巻 保幸[3]
[1]九州大院・歯・口腔機能修復学咀嚼機能制御、[2]九州大・生医研・遺伝情報・ゲノム機能、[3]九州大院・薬・薬効解析、[4]徳島大・医・機能解剖

P3-292 海馬シータ位相コードを用いた物体－場所連合記憶と物体移動の記憶の神経回路モデル
Memory of object-place association and object relocation formed as a hierarchical associative network with theta phase coding
佐藤 直行[1,2]、山口 陽子[1,2]
[1]科学技術振興機構CREST、[2]理化学研究所・脳総合研究センター・創発知能ダイナミクス研究チーム

Sep 23 (Thu) Poster Session

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P3-293 報酬を手掛かりとした音順序弁別学習の聴覚野へのドーパミン入力依存性
Dopaminergic inputs to the rat auditory cortex required for sound sequence discrimination learning
工藤 雅治[1]、澁木 克栄[1]
[1]新潟大・脳研・システム脳生理

P3-294 ラットのwin-shift/stay放射状迷路行動と迷路内手がかり利用における内側前頭前野－線条体回路の役割
A double dissociation within prefrontal-striatal circuits between win-shift/stay radial maze performance and retention of intra-maze cue in rats
高濱 祥子[1]、熊田 孝恒[1]、瀧田 正寿[1]
[1]産総研・人間福祉医工学

P3-295 恐怖条件付けモデルにおける扁桃体内のアミノ酸、ドーパミンの変化
Conditioned stimulus and amygdalic amino acids and dopamine
横山 正宗[1]、佐藤 拓[1]、鈴木 映二[1]、渡辺 滋[1]、稲田 健[1]、丸田 修史[1]、宮岡 等[1]
[1]北里大学院・医療系研究科・精神科学

P3-296 腹側淡蒼球へのGABA$_A$受容体阻害薬投与によって味覚嫌悪学習の表出が減弱した
The microinjections of GABA$_A$ receptor antagonist into the ventral pallidum attenuate the expression of conditioned taste aversion in rats
乾 賢[1]、今岡 寛之[1]、志村 剛[1]、山本 隆[1]
[1]大阪大・人間科学・行動生理

P3-297 単純交代系列課題および光追随課題時におけるラット海馬CA1領域の神経活動計測
Neuronal property in the hippocampal CA1 area during the same behavioral context by memory retrieved and visual guided performances in the rat
高橋 宗良[1]、相原 威[2,3,4]、塚田 稔[2,4]、安西祐一郎[1]
[1]慶應義塾大院・理工・計算機科学、[2]玉川大・工、[3]科学技術振興機構・CREST、[4]日本学術振興会・COE

P3-298 記憶誘導性の感覚運動変換におけるサル前頭前皮質と前頭眼野ニューロンの機能的差異
Functional differences between primate prefrontal and frontal eye field neurons in a memory-guided sensorimotor transformation
雨森 賢一[1,2]、澤口 俊之[1,2]
[1]北海道大院・医・高次脳機能、[2]CREST, JST

P3-299 シェファー側枝刺激による海馬CA1錐体細胞の膜電位θ波周期振動の位相変化
Phase Shift of Theta Oscillation in Hippocampal CA1 Pyramidal Cell Membrane with the Stimuli to the Shaffer Collaterals
渡辺 秀典[1,2]、長宗 伸朋[1]、相原 威[1]、塚田 稔[1]
[1]玉川大学・工・学習記憶システム研究室、[2]日本学術振興会特別研究員

P3-300 ラットの放射状迷路課題遂行に及ぼすAP5およびSCH23390前頭前野局所投与の効果
Effects of local injection of AP5 and SCH23390 into the prefrontal cortex on radial arm maze performance in rats
林 和子[1]、一谷 幸男[1]
[1]筑波大院・人間総合科学

P3-301 陸産軟体動物チャコウラナメクジにおける逆行感性的予備条件づけ
Backward sensory preconditioning in the terrestrial mollusc, *Limax marginatus*
三浦 治己[1]、木曽 和毅[1]、牧野 悌也[1]、矢野 雅文[1]
[1]東北大学・電気通信研究所

P3-302 機能的MRIを用いた聴覚－視覚刺激対連合学習における脳活動変化の解析
Change in brain activity during arbitrary auditory-visual association learning as revealed by functional MRI
田邊 宏樹[1,2]、本田 学[1,2]、定藤 規弘[1,2]
[1]生理研・心理生理、[2]科技振

P3-303 マウス味覚嫌悪記憶の獲得と保持に対する疑似微少重力環境の影響
Effect of pseudo-microgravity environment on acquisition and retention of mouse conditioned taste memory
宮本 武典[1]、藤原 悠美[1]、坪井 香苗[1]、藤原 宏子[1]
[1]日本女子大・理・物質生物

認知
Cognition

P3-304 複数の空間ワーキングメモリ処理時の中脳皮質系ドーパミン放出量の経時変化
Dopamine release time course of the mesocortical system during multiple spatial working memory processing
矢田 寛樹[1]、田中 昌司[1]
[1]上智大院・理工・電気電子工学

P3-305 味覚弁別遅延GO./NOGOタスクサル大脳皮質味覚領域の味覚関連ニューロンの他イベントに対する応答特性および同期性の相違
Difference in firing synchronicity of task-related neurons between primary and secondary gustatory cortices in monkey
中村 民生[1]、井福 裕俊[1]、平田 真一[1]、小川 尚[1]
[1]熊本大院・医薬・知覚生理

P3-306 楽音に対する大脳皮質応答：音楽経験との関係
Cortical Magnetic Responses to Musical Tones: Comparison between Musicians and Non-Musicians
栗城 真也[1]、神田 聡[1]、平田 恵啓[1]、小山紗智子[1]
[1]北海道大学・電子科学研究所

P3-307 fMRIによる文理解に関わる脳活動の研究
Brain activation during the sentence comprehension: an fMRI study
生田 奈穂[1,2]、秋月 祐子[1]、佐々 祐子[1,2]、渡邉 丈夫[1]、岩田 一樹[1]、三浦 直樹[1]、岡本 英行[1]、佐藤 滋[2]、松江 克彦[3]、川島 隆太[1]
[1]東北大・未来科学技術共同研究センター・新素材創製、[2]東北大院・国際文化・異文化、[3]東北福祉大・感性福祉研究所

P3-308 線条体における報酬の履歴に基づいた予測的な行動の計画
Predictive action planning in the striatum based on the history of rewarding but not aversive events.
山田 洋[1]、松本 直幸[1]、木村 實[1]
[1]京都府立医科大学・神経生理学

Sep 23 (Thu) Poster Session

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P3-309 行動の企画過程における最終および目前の行動目標を表現する前頭前野の神経細胞活動
Representation of immediate and final behavior goals in the prefrontal cortex
斎藤 尚宏[1,2]、虫明 元[1,2]、坂本 一寛[3]、丹治 順[1,2]
[1]東北大・医・生体システム生理、[2]科学技術振興機構、[3]東北大・電気通信研究所

P3-310 行為の意味をコードする頭頂葉皮質ニューロン
Purposeful meanings of behaviors coded by parietal neurons
横地 博子[1]、田中美智雄[1]、入來 篤史[1,2]
[1]東京医科歯科大・院・認知神経生物、[2]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

P3-311 空間認識課題における頭頂－前頭領域の結合関係の変化
Changes of functional connectivity between the superior parietal and prefrontal cortex during a spatial coding task: a fMRI study
松本絵理子[1]、宮内 哲[1]
[1]独立行政法人情報通信研究機構・脳情報グループ

P3-312 ノルアドレナリンによる空間ワーキングメモリの制御
Noradrenergic control of multi-target spatial working memory
西島 康夫[1]、田中 昌司[1]
[1]上智大院・理工・電気電子

P3-313 サル前頭前野において時間弁別に関わる神経活動
Neuronal activity for duration discrimination in monkey prefrontal cortex
生塩 研一[1]、千葉 惇[1]、稲瀬 正彦[1]
[1]近畿大・医・第一生理

P3-314 サル前頭前皮質ニューロンによる行動文脈のモニタリング
Retrospective monitoring of behavioral context in monkey prefrontal neurons
辻本 悟史[1,2]、澤口 俊之[1,2]
[1]北海道大院・医・高次脳機能、[2]CREST, JST

P3-315 発話運動における島皮質の活動とその役割について
Activation of the left anterior insula in speech
能田由紀子[1]、本多 清志[1]
[1]ATR 人間情報科学研究所

P3-316 黒質入力を受け前頭前野へ投射するサル視床大脳皮質ニューロンの認知機能
Cognitive functions of thalamocortical neurons with nigral input projecting to prefrontal cortex in monkeys
谷渕 育夫[1]、陣内皓之祐[1]、北野 浩之[2]
[1]滋賀医科大学・医・生理学第一、[2]滋賀医科大学・医・脳神経外科

P3-317 固執性反応の抑制におけるサル前頭前皮質$GABA_A$受容体の役割
The role of GABAergic inhibiton in suppressing perseverative responses in the monkey prefrontal cortex
和島 佳世[1,2]、澤口 俊之[1,2]
[1]北海道大院・医・高次脳機能、[2]CREST, JST

P3-318 周期変動静電気刺激による認知調整
Cognitive calibration by periodically altering static electricity stimulus
安田 達弘[1]
[1]Free

P3-319 視床線条体系における計画した運動の切り替えに関わる神経活動
A neural basis for switching action plans in the thalamo-striatal system
堀 由紀子[1]、南本 敬史[1]、木村 實[1]
[1]京都府立医科大院・神経生理学

P3-320 ドーパミン細胞は報酬や意思決定の不確実性を表現する
Dopamine neurons encode uncertainty of reward and of decision
松本 直幸[1]、榎本 一紀[1]、木村 實[1]
[1]京都府立医科大院・神経生理学

P3-321 第一言語・第二言語の理解に関わる脳活動領域の特定
A shared network of cortical regions involved in a first and second language comprehension
横山 悟[1,2]、内田 信也[2,5]、岩田 一樹[2,3]、金 情浩[6]、岡本 英行[2,4]、
Jeong Hyeonjeong[1,2]、中村 渉[1]、堀江 薫[1]、佐藤 滋[1]、川島 隆太[2]
[1]東北大院・国際・異文化、[2]東北大学・未来科学技術共同研究センター、[3]東北大学・COEプログラム、[4]東北大学・医学研究科、[5]東北大学・加齢医学研究所、[6]東北大学・文学研究科

P3-322 サル視床背内側核における情報処理：ポピュレーションベクトル解析法による解析
Information processing in the primate mediodorsal thalamic nucleus: population vector analysis
渡辺由美子[1]、船橋新太郎[2]
[1]産総研・人間福祉医工学・ニューロバイオニクス、[2]京都大院・人間／環境学研究科

P3-323 意思決定に関わるサル帯状皮質神経活動の解析とモデル化
Analysis and modelling of neuronal activation in the monkey cingulate cortex during decision processes
岡本 洋[1]、礒村 宣和[2]、高田 昌彦[2]、深井 朋樹[3]
[1]富士ゼロックス（株）中央研究所、[2]東京都神経科学総合研究所、[3]玉川大学・工・情報通信工学科

P3-324 片手３本指の時間順序判断
Temporal order judgment of three stimuli delivered to unimanual fingers
渋谷 賢[1,2]、高橋 俊光[1,2]、北澤 茂[1,2]
[1]順天堂大学・医・生理、[2]科学技術振興機構

P3-325 視線のシフトの積極的な抑制へのサル外側前頭前皮質の関与：局所機能脱落法による研究
Involvement of the monkey lateral prefrontal cortex in active suppression of gaze shift: a local inactivation study with muscimol
桑島真里子[1,2]、澤口 俊之[1,2]
[1]北海道大院・医・高次脳機能学、[2]CREST, JST

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P3-326 時間認知課題に応答するサル前頭前野の神経活動
Prefrontal activity related to time coding
湯本 直杉[1,2]、陸 暁峰[2,3]、南部 篤[3]、岡本 洋[4]、深井 朋樹[1]、高田 昌彦[5]
[1]玉川大院・工学研究科・数理情報工学、[2]東京都神経科学総合研究所・統合生理、[3]順天堂大・医・生理学第一、[4]富士ゼロックス中央研究所、[5]国立生理学研究所・生体システム

P3-327 メタンフェタミン誘発性認知障害におけるドパミン及びNMDA受容体の関与
Involvement of dopamine and N-methyl-D-aspartate receptors in methamphetamine-induced cognitive impairment
東岸由里子[1]、亀井 浩行[1]、永井 拓[1]、田熊 一敞[1]、中野 寛子[1]、山田 清文[1]、鍋島 俊隆[2]
[1]金沢大院・自然・病院薬学、[2]名大院・医・医療薬学・付属病院薬剤部

P3-328 補足眼野および前補足運動野へ投射する前頭前野細胞の分布
Spatial distribution of prefrontal cortical cells projecting to the supplementary eye field and pre-supplementary motor area in the monkey
王 艷[1,2,3]、磯田 昌岐[1]、嶋 啓節[1]、丹治 順[1,2]
[1]東北大院・医・生体システム生理、[2]CREST, JST、[3]中国医科大学

P3-329 ルール切替え課題遂行中のサル前頭前皮質におけるルールの逆行性および前向性符号化
Retrospective and prospective coding of rules in the monkey prefrontal cortex during a rule-shifting task
雨森 智子[1,2]、澤口 俊之[1,2]
[1]北海道大院・医・高次脳機能、[2]CREST, JST

P3-330 ジュウシマツの歌神経核HVCにおける視覚応答の欠如
Lack of visual responses in song nucleus HVC of Bengalese finches
関 義正[1]、岡ノ谷一夫[2,3,4]
[1]千葉大院・文・人文科学、[2]千葉大学・文、[3]理化学研究所、[4]科学技術振興機構

P3-331 マカクザル前頭前野における正の報酬に特異的な神経活動
Prefrontal activity related to positive, but not negative, reward outcome in macaque
小林 俊輔[1]、野元 謙作[1]、渡辺 正孝[2]、彦坂 興秀[3]、Wolfram Schultz[4]、坂上 雅道[1]
[1]玉川大・学術研究所・脳科学研究施設、[2]東京都神経研、[3]National Eye Institute, NIH, Bethesda, USA、[4]Dept. Anatomy, Univ. Cambridge, UK

P3-332 大脳皮質神経回路活動の調節における二状態間膜電位遷移の役割：モデル研究
Role of the two-state membrane potential fluctuations in regulating cortical network activity: Modeling study
姜 時友[1,3]、北野 勝則[2]、深井 朋樹[1]
[1]玉川大院・工・計算神経科学、[2]立命館大・理工学情報学科、[3]日本学術振興会特別研究員

P3-333 眼球運動方向の決定に関わる前頭連合野神経機構
Neural process for decision of saccade direction in the prefrontal cortex
渡邉 慶[1]、居垣 紗織[1]、船橋新太郎[1]
[1]京都大院・人間環境学・認知行動科学

P3-334 L1，L2及びL3間の言語学的類似性と関連する神経機構
Neural correlates of linguistic similarity among L1, L2 and L3 : An fMRI study
Jeong Hyeongjeong[1]、岩田　一樹[2,3]、渡邊　丈夫[2,4]、佐々　裕子[2,3]、生田　奈緒[2,3]、秋月　裕子[2,5]、三浦　直樹[2,6]、岡本　英行[2,7]、横山　悟[1,2]、Jorge Riera[2,3]、土師　知己[8]、臼井　信男[4,8]、泰羅　雅登[8]、佐藤　滋[1]、川島　隆太[2]
[1]東北大院・国際文化研究科、[2]東北大学・未来科学技術共同研究センター、[3]東北大学・言語／認知総合科学戦略研究教育拠点、[4]科学技術振興機構、[5]東北大学・医学系研究科、[6]東北大学・工学研究科、[7]東北大学・医学系研究科、[8]日本大学 先端医学総合研究センター

P3-335 日本語音韻刺激に対する脳反応の発達的変化 －近赤外分光法による検討－
Developmental changes of cerebral responses to a Japanese phonemic contrast measured with NIRS
皆川　泰代[1,2,3]、森　浩一[2]、佐藤　裕[2]、田中　章浩[2]
[1]慶應義塾大学・文、[2]国立身障者リハビリセンター研究所、[3]科学技術振興機構

P3-336 情動関連質問時の脳波変化
EEG observation under thinking on questions relating to emotional affairs
青木　亮三[1]、佐々木　仁[1]
[1]大阪大院・医・情報生理、[2]産業技術短大

P3-337 作業記憶課題における刺激呈示速度変化に伴う脳活動量の検討
Brain activation with the changes of presentation rate of stimulus during a working memory task.
内田　信也[1,2]、岩田　一樹[2,3]、渡辺　丈夫[2,3]、佐々　祐子[3,4]、三浦　直樹[2,5]、木之村重男[1]、佐藤　和則[1]、福田　寛[1]、川島　隆太[2]
[1]東北大院・医・機能画像医学、[2]東北大・未来科学技術共同研究センター、[3]東北大・言語認知総合科学戦略研究教育拠点、[4]東北大院・国際文化研究科、[5]東北大院・工学研究科

P3-338 仮想道具に生じる知覚
Sensation at the tips of virtual tools
茂泉俊次郎[1,2,3]、山本　慎也[2,3]、北澤　茂[1,2,3]
[1]順天堂大院・医・生理学第一講座、[2]科学技術振興機構、[3]産業技術総合研究所

P3-339 反復プライミングにおける意識的処理と無意識的処理の神経機構
The neural basis of conscious and unconscious repetition priming revealed by functional MRI.
松本　敦[1]、飯高　哲也[1]、山本　幸子[2]、定籐　規弘[2]
[1]名古屋大院・環境・心理学、[2]自然科学研究機構生理学研究所

P3-340 サルの頭頂葉内側面の場所選択性ニューロン：人工現実空間内のナビゲーションによる研究
Location selective response in the mesial parietal cortex of monkeys during navigation task in virtual environment
泰羅　雅登[1,2]、佐藤　暢哉[2,3]、酒田　英夫[4]
[1]日本大学 総合科学研究所、[2]日本大学医学部 応用システム神経科学、[3]日本学術振興機構、[4]聖徳栄養短期大学

P3-341 Visual pattern-dependent reward-predicting activity by primate prefrontal neurons
Xiaochuan Pan[1,2]、Masamichi Sakagami[1,2]
[1]BSRC Tamagawa University、[2]PREST, JST

Sep 23 (Thu) Poster Session

Room P

9:00～16:00
discussion / an odd number 14:00～15:00
an even number 15:00～16:00

コンピュータ科学、ロボット
Computer Science and Robot

P3-342 パルス結合されたResonate-and-Fireモデル
Pulse-coupled resonate-and-fire models
三浦 佳二[1,2]、岡田 真人[2,3]
[1]京大・理、[2]理研・脳総研、[3]科学技術振興機構

P3-343 E-CALIシステムによる神経成長制御のシミュレーション解析
Simulation analysis of signaling dynamics regulating nerve growth in computational model using E-CALI system
関山 和秀[1]、菊地 進一[1]、諸戸 麻衣[1]、村田 真也[1]、五嶋 良郎[2]、冨田 勝[1]、竹居光太郎[1,2]
[1]慶應義塾大学・先端生命科学研究所、[2]横浜市立大学・医・分子薬理神経生物学講座

P3-344 海馬長期増強作用におけるサイクリン依存性キナーゼ5のシミュレーション解析
Simulation analysis of cyclin-dependent kinase 5 in hippocampal long-term potentiation
菊地 進一[1]、谷 紗妙佳[1]、武道相以子[1]、合田 智子[1]、岡 浩太郎[1,2]、竹居光太郎[1,3]、冨田 勝[1]
[1]慶大・先端生命研、[2]慶大・理工・生命情報、[3]横市大・医・薬理

◆◇◆ Luncheon Seminar ◆◇◆

12:30～13:30　Room C

LS-1　　カールツァイス（株）

座長：石館　文善（カールツァイス（株）マイクロスコープディビジョン）

観ることは知ることーツァイスは昔からイメージングの最先端ー
What you see you know. – Carl Zeiss the Imaging Leader for Decades –
市川　謙
カールツァイス（株）マイクロスコープディビジョン
矢口　晶
カールツァイス（株）マイクロスコープディビジョン

12:30～13:30　Room D

LS-2　　アルファメッドサイエンス（株）

座長：森　泰生（京都大学大学院　工学研究科　合成生物化学専攻）

MEDシステムを用いた脳機能解析
下野　健
アルファメッドサイエンス（株）

12:30～13:30　Room H

LS-3　　ライカマイクロシステムズ（株）

座長：鈴木　博（ライカマイクロシステムズ（株））

共焦点顕微鏡使用の解析例と最新の顕微法
Analytical example by Confocal Microscope and the latest microscopy
古谷一清木　誠
科学技術振興機構
鈴木　博
ライカマイクロシステムズ（株）

Sep 22 (Wed) Luncheon Seminar

12:30～13:30　Room C

LS-4　ネッパジーン（株）「In Vivo・In Vitro　Electroporation:非ウィルス性遺伝子導入・薬物送達のニューテクノロジー」

座長：植田　弘師（長崎大学大学院　医歯薬学総合研究科　分子薬理学分野）

海馬神経細胞へのエレクトロポレーション法を用いたシナプス機能解析
岡部　繁男
東京医科歯科大学　医歯学総合研究科　細胞相関機構学

エレクトロポレーション法を用いた遺伝子レスキューによる薬物依存の責任脳領域の同定
植田　弘師
長崎大学大学院　医歯薬学総合研究科　分子薬理学分野

＜特別企画＞
ECIS A MORPHOLOGICAL BIOSENSOR FOR CELL RESEARCH
Dr Ivar Giaever（1973年　ノーベル物理学賞受賞者）
President, Applied BioPhysics, Inc.

12:30～13:30　Room D

LS-5　田辺製薬（株）

座長：吉峰　俊樹（大阪大学大学院　医学系研究科　神経機能制御外科）

エビデンスに基づいた慢性期脳梗塞のクリティカルケア－脳卒中治療ガイドライン2004を踏まえて－
永山　正雄
東海大学　医学部　内科学系神経内科学

12:30～13:30　Room H

LS-6　エーザイ（株）

座長：武田　雅俊（大阪大学大学院　医学系研究科　精神医学）

アルツハイマー病－治療のターゲットとしてのAβオリゴマー－
東海林幹夫
岡山大学大学院　医歯学総合研究科　神経病態内科学

Sep 23 (Thu) Luncheon Seminar

12:30～13:30　Room D

LS-7　日本メジフィジックス（株）／第一ラジオアイソトープ研究所／日本脳神経核医学研究会
画像による神経機能・病態研究の最前線

座長：畑澤　順（大阪大学大学院・医・生体情報医学）

グリアの代謝・機能解析
井上　修
大阪大学大学院・医・医用物理工学

パーキンソン病
桑原　康雄
九州大学・医・附属病院放射線部

発表抄録集

プレナリーレクチャー
特別講演
時　実　賞

PL-1 Basic And Clinical Neurobiology of Alzheimer Disease.

Peter St. George-Hyslop
Centre for Research in Neurodegenerative Diseases, Toronto Western Research Institute, Toronto Western Hospital, University of Toronto, Ontario, Canada

Although many of the adult onset neurodegenerative diseases, such as Alzheimer Disease (AD), have both genetic and non-genetic causes, a common emerging theme is that many of these diseases represent disorders of protein folding. For instance, genetic studies have identified four genes associated with inherited risk for AD (presenilin1 - PS1; presenilin 2 - PS2; amyloid precursor protein - APP; apolipoprotein E - APOE). There is tentative evidence for additional, unidentified AD susceptibility genes on chromosomes 12, 10, and possibly 9 and 18. Several 'candidate' genes have also been implicated on the basis of positive case:control allelic association studies, but have not generally been as widely replicated. Nevertheless, analysis of the functional effects of mutations/polymorphims in the four known AD genes suggest that they alter the production and/or clearance of Aβ peptide - a proteolytic derivative of APP. This commonality has led to the hypothesis that the accumulation of Aβ within the brain is an important initiating event, at least in genetic cases, and probably also in sporadic cases. It remains unclear exactly how the accumulation of Aβ is related to the accumulation of phosphorylated tau in neurofibrillary tangles (although both are neuropathological hallmarks of AD). However, the discovery that polymorphisms/mutations in the tau gene are also associated with inherited forms of neurodegenerative disease (frontotemporal dementia, progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD)) implies that misprocessing of the tau microtubule associated protein can also cause neurodegeneration.

These results provide several important practical and theoretical insights. First, knowledge of the pathways generating Aβ and causing accumulation of tau protein provide rational therapeutic targets. Second, these observations have also uncovered basic biological mechanisms (e.g. γ-secretase-mediated cleavage of Type 1 transmembrane proteins - regulated intramembranous proteolysis). This latter process is essential in diverse biological events including Notch signal transduction during development, and the production of Aβ in AD.

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PL-2 Modification of Developing Neural Circuits by Sensory Experience

Mu-ming Poo
Division of Neurobiology, Department of Molecular and Cell Biology, University of California, Berkeley USA

Correlated spiking of pre- and postsynaptic neurons can result in synaptic strengthening or weakening, depending on the order of spiking and the time interval between the pre- and postsynaptic spiking. Recent findings indicate that correlated spiking also induces persistent changes in the global intrinsic excitability of the presynaptic neuron and in the properties of spatial summation of synaptic potentials in the postsynaptic dendrite. These spike timing-dependent modifications of synapses and neurons are likely to play an important role in the development and plasticity of neural circuits.

In the developing visual system, early visual experience is critical for the refinement of developing nerve connections. *In vivo* whole-cell recording from the tectum of *Xenopus* tadpoles showed that correlated spiking of retinal ganglion cells and tectal neurons can induce persistent strengthening or weakening of retinotectal synapses. Such synaptic modifications appear to underlie activity-dependent refinement of the receptive field properties of the tectal neuron. During development, there is a progressive reduction in the size of receptive field and the precision of retinotopic projections. This refinement involves a progressive matching of the excitatory (retinotectal) and inhibitory (intratectal) inputs to the tectal neuron, a process that requires an appropriate level of inhibitory activity. Furthermore, we found that following repetitive application of moving "conditioning" visual stimuli of a particular direction across the receptive field of a tectal neuron for a few minutes, the response of the tectal cell became sensitive to light stimuli of the conditioned direction for a period of at least 60 minutes. This rapid "learning" requires spiking of the postsynaptic neurons and is accompanied by persistent synaptic potentiation and depression of different retinotectal synapses. Interestingly, activity-induced synaptic modifications in the developing *Xenopus* retinotectal system can be quickly reversed by either subsequent spontaneous activity in the tectum or exposure to random visual inputs. This reversal depends on the burst spiking and activation of the NMDA subtype of glutamate receptors. Stabilization of synaptic modifications can be achieved by an appropriately spaced pattern of induction stimuli. Taken together, these findings underscore the importance of spike timing- dependent plasticity in the refinement of developing circuits and suggest a temporal constraint on the pattern of visual inputs for effective induction of stable synaptic modifications.

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Neuro 2004

PL-3 Cortical plasticity contributing to child development and skill learning

Michael M. Merzenich
W.M. Keck Center for Integrative Neurosciences, University of California at San Francisco, USA

Cortical recording and imaging studies in developing and adult animals and humans have provided us with an increasingly clear understanding of the phenomenology of cortical plasticity, as it accounts for the development of the specific skills and abilities of the developing child and for the acquisition of new skills and abilities in the adult brain. They also provide us with an increasingly clear understanding of the contributions of experience and learning to variations in child and adult achievement, to the complex interplay between genetic and environmental contributions to human performance variation, and to the origin of the expressions of specific developmental and adult impairments. We have used this growing understanding of neurogenetics and brain plasticity phenomenology to develop a new class of models of human disabilities (e.g., *in development*, impairments in language and reading acquisition, autism, cerebral palsy, schizophrenia; *in adults*, acquired focal dystonias, Parkinsonism, memory/cognitive losses in aging), and to design brain plasticity-based therapeutic strategies designed to ameliorate or reverse them. Several of these treatment models have now demonstrated that key behavioral and neurological expressions of developmental and adult impairments can be substantially re-normalized by this integrative neuroscience-based therapeutic approach.

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SL-1 Reward, prediction and uncertainty

Wolfram Schultz
Department of Anatomy, University of Cambridge, UK

Survival in uncertain environments requires individuals to maximize the intake of liquid and food rewards. This task involves multiple levels of decision-making and goal-directed behavior which is based on the detection of rewards and the use of predictions for obtaining advance information and reducing subjective uncertainty. Based on theoretical considerations from learning theory and game theory, we investigated the activity of individual neurons in major reward structures including the dopamine systems, striatum and orbitofrontal cortex. We studied how neurons detected and discriminated between different rewards, reward quantities and reward probabilities, adapted their processing to the predictions about rewards, and explicitly signalled uncertainty. These results revealed efficient neural mechanisms underlying the coding of basic reward variables for decision-making and goal-directed behavior.

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SL-2 From Peppers to Peppermints: The Molecular Biology of Thermosensation

David Julius
Department of Cellular and Molecular Pharmacology University of California, San Francisco .USA

We are interested in determining the molecular basis of somatosensation - the process whereby we experience touch and temperature - with an emphasis on identifying molecules that detect noxious (pain-producing) stimuli. We are also interested in understanding how somatosensation is altered in response to tissue or nerve injury. Our approach has been to identify molecular targets for drugs or natural products that mimic psychophysical effects of commonly encountered somatosensory stimuli, such as heat or cold. Using this strategy, we have identified two members of the TRP channel family, TRPV1 and TRPM8, which are activated by capsaicin and heat or menthol and cold, respectively.

One of our main interests has been to determine the molecular mechanism(s) by which tissue injury and inflammation produces hypersensitivity of primary afferent neurons to heat (thermal hyperalgesia). We have elucidated two basic mechanisms that contribute to this process: one involving the direct interaction of inflammatory mediators with TRPV1 such that they serve as positive allosteric modulators of this heat-activated channel, and another involving the potentiation of TRPV1 gating downstream of phospholipase C-coupled receptors. Insights into these mechanisms will be discussed.

We have also examined the properties of another TRP channel, TRPA1, which we believe will play an interesting and significant role in inflammatory pain. Our recent studies have shown that TRPA1 is a major site of action of mustard oil (allyl isothiocyanate), the main pungent ingredient in wasabi. Mustard oil excites a subpopulation of capsaicin-sensitive, peptidergic trigeminal neurons, consistent with its ability to produce robust neurogenic inflammation. Our in vitro studies further suggest that TRPA1 functions as a receptor-operated channel, depolarizing sensory neurons in response to pro-algesic agents (such as bradykinin or ATP) that activate PLC-coupled receptors. These findings have significant implications for understanding mechanisms of inflammatory pain and identifying potential therapeutic targets.

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Neuro 2004

SL-3 The multipotent neural stem cell of the CNS, the matrix cell, and the molecular mechanism of corticogenesis

Setsuya Fujita
Louis Pasteur Center for Medical Research

In the present talk, I would like to sketch development of the cerebral cortex from the standpoint of the matrix cell theory; the basic concept of the theory has only recently been reconfirmed after a long period (more than 40 years) of controversy and neglect.

In the early 1960s I applied ^{3}H-thymidine autoradiography to the study of the cells constituting the neural tube, and found that its wall was composed solely of one kind of single-layered epithelial cells, which performed an elevator movement synchronous with the cell cycle. Dynamic behavior of N-cadherin, beta-and alpha-catenins of the matrix cell disclosed that the adherens junctions as well as junctions to the basement membrane by integrin systems disintegrate during mitosis, releasing the cell from the neighboring cells and the basement membrane. As the consequence, the mitotic matrix cell is rounded off and attracted to the ventricular surface where the junctional complexes remain unchanged during mitosis. After division, the daughter matrix cells recover their adhesion apparatuses and, extending their cytoplasmic processes, resume their downward elevator movement. The matrix cells in early stages of development were homogeneous and identified as multipotent stem cells of the CNS to which I gave the name of *matrix cells* (1962).

^{3}H-thymidine autoradiography revealed the sequential stages in development of the matrix cells: At first they proliferate only to expand the population (stage I), then switch to differentiate specific neuroblasts in given sequences (stage II). In human fetal cerebral hemisphere, stage II begins at 7th and continues up to 15th fetal week. When a neuroblast is produced in stage II, the perikaryon of the postmitotic neuroblast starts to migrate in the matrix layer by the same mechanism of the downward elevator movement of the daughter matrix cell. Cellular events that announce differentiation of a neuroblast are (1) absolute repression of DNA replication, (2) loss of junctional complexes, (3) down-regulation of adhesions to the basement membrane and (4) emergence of the axon. In some early neuroblasts like Cajal-Retzius cells, the adhesions to ECM remain conserved and, together with Reelin production, play an important role in the corticogenesis. Since all the neuroblasts lose their ventricular attachment as a result of down-regulation of the junctional complex, their perikarya can migrate beyond the limit of the matrix layer to form the mantle layer. As the distance between the matrix layer and the cortical plate widens, the migration of the neuroblasts in the intermediate zone to cortical plate is carried out along the bundles of the matrix cell basal processes that play a pivotal role in corticogenesis. If the bundles are not formed, the neuroblast migration to the cortical plate becomes abortive and the cortex is not formed, generating non-corticogenetic pattern.

When neuroblast production ceases in respective sectors of the brain, stage III begins and the matrix cells change themselves into ependymoglioblasts which are soon differentiated into ependymal cells and progenitors of neuroglia called glioblasts. The glioblasts are freely migrating cells moving out from the birth-place. After the out-migration of glioblasts, empty radial spaces are left in the axial core of the cortical columns the ependymoglioblasts have occupied. In the cortex, afferent axons having come from thalamus and other regions of the cerebral cortex and waited in the layer VII now dash into the spaces and form synapses resulting in a rapid local enlargement of the cortical volume. The gyri are thus formed from 19th to 28th fetal week (i.e., during stage III), in the midst of glioblast formation.

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Tokizane

Experimental analysis of cognitive functions in chimpanzees

Tetsuro Matsuzawa
Primate Research Institute, Kyoto University, Inuyama, Japan

The present study aims to clarify the evolutionary basis of cognitive functions through the comparative study of humans and chimpanzees. Neuroscience has illuminated various aspects of human cognition. Higher cognitive functions such as language and thought distinguish humans from the rest of the animal kingdom. Since the brain is not preserved in fossils, we can not discern details of brain functions from fossil skulls. Therefore, comparative studies among living primates including humans become particularly valuable. From this perspective, the most important species is the chimpanzee: the closest relative of humans, with a genomic difference of only 1.2 %. In their natural habitat, chimpanzees use various kinds of tools, perform cooperative hunting, and possess a set of cultural traditions unique to each community. These characteristics can not be extrapolated from the cognitive functions of macaques and other monkeys. I have been carrying out a long-term comparative study of cognitive functions in humans and chimpanzees. First, I investigated the fundamental characteristics of visual perception in chimpanzees, studying color categorization, form perception, visual acuity, and so forth. To measure thresholds, I applied psychophysical techniques such as the titrating up-down method invented in studies of human psychophysics. Second, I also used multidimensional scaling and cluster analysis to represent the perceptual world of chimpanzees based on the confusion matrix in discrimination tasks such as matching-to-sample. Third, thanks to the unique capability of chimpanzees, I trained them to use visual symbols such as Arabic numerals, letters of the alphabet, and Kanji characters to represent what they recognized. In this paradigm, a chimpanzee subject faces a fully automated computer-controlled system. Using visual symbols as the communication medium, I can access chimpanzee cognition. My main subject has been a female chimpanzee named Ai. Ai was the first chimpanzee who learned to use Arabic numerals to represent number, and she has shown a rudimentary understanding of both cardinal and ordinal aspects of the number system. A recent study using Arabic numerals from 0 to 9 showed that Ai could memorize up to five numerals that appeared randomly on the monitor within a brief period. The chimpanzee's memory span for numerals was thus comparable to that of human adults. Taken together, these series of studies combined techniques of behavioral control and psychophysics to examine visual perception and cognition in chimpanzees. To explore developmental changes in the cognitive functions of chimpanzees, my colleagues and I have begun a new project in 2000. This project focuses on the transmission of knowledge and skills across generations, in other words, on social aspects of cognitive functions. Our results include the finding that chimpanzee infants, just like human infants, show neonatal smiling from right after birth, and that this transforms into social smiling at around three months of age. We have used magnetic resonance imaging to study the development of three infant chimpanzees and found that their larynges descend during infancy, much like they do in human infants. We have also elucidated the learning processes underlying the acquisition of tool use. Chimpanzee infants began to use their first probing tool at the age of one year and ten months, after having had the opportunity for long-term observation of their mothers' behavior from just after birth. Through my search over the past two decades for the evolutionary basis of human cognition in chimpanzees, I also recognized a marked difference between the two species in terms of cognitive functions. Human cognition can be characterized by a hierarchical organization in which one thing or event is related to another. This hierarchy can have a self-embedding or recursive structure. When we measure the depths of nodes in such hierarchical organizations, differences between humans and chimpanzees become evident in object-object combinations during tool use, event-symbol combinations in symbolic matching tasks, as well as in terms of understanding others' minds. Human cognition can be characterized by triadic relationships in nature from the beginning. Take the case of an infant facing a task in the presence of the mother. So-called social referencing is marked in human cases. In contrast, chimpanzees focus on the dyadic relationship and seldom look back at the mother.

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シンポジウム

SY01-1 ～ SY36-5

SY01-01 Genetic basis of host plant preference and speciation in *Drosophila*

Takashi Matsuo[1]
[1]Department of Biology, Tokyo Metropolitan University, Tokyo, Japan

Unlike its morphological similarity to the other species in the *melanogaster* species subgroup, *Drosophila sechellia* has evolved distinctive physiological and behavioral characters adapting to its host plant *Morinda citrifolia*, known as the Tahitian Noni fruit. The ripe fruit of *M. citriforia* contains hexanoic acid and octanoic acid, the main components of the bad smell from the fruit. *D. sechellia* is specifically attracted to hexanoic acid, while the other species are repelled. By genetic mapping using inter-species hybrid with *D. melanogaster* mutants, *Odorant binding protein 57e* was identified as the gene corresponding to the species difference in the hexanoic acid sensation. *Obp57e* forms a gene cluster with *Obp57d*, and these two genes are expressed in the same cells associated with the chemosensory organ. The dynamic history of *Obp57d/e* evolution was revealed by comparison of the genomic sequences of the *Obp57d/e* cluster region obtained from 30 species phylogenetically located between *D. melanogaster* and *D. pseudoobscula*.

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SY01-02 The Drosophila fruitless locus is required for male-typical development of the nervous system and male sexual behavior

Daisuke Yamamoto[1]
[1]Genetics, School of Science and Engineering, Waseda University, Tokyo, Japan

Mutations in the fruitless (fru) locus of Drosophila enhance male-to-male courtship activity and interfere with the formation of male-specific muscle of Lawrence (MOL). The fru gene encodes a series of putative transcription factors with the BTB domain and zinc finger motif. Although the fru transcripts are expressed in both the male and female central nervous system (CNS), the Fru protein is only detectable in the male CNS. Reporter assays with the transfected Drosophila cells suggest that fru mRNA seems to be prevented from translation by binding to the mRNA of Transformer (Tra), a feminizing factor present only in female. Artificial expression of Fru in motoneurons of fru mutants rescues the formation of MOL. The critical period of Fru expression for rescue is in the pupal stage. These observations indicate that Fru is a mascalinizing factor for certain neurons in the CNS. Mutations in the fru locus likely lead to failure in male-typical development of these neurons during the pupal stage, resulting in enhanced male-to-male courtship and the loss of MOL in the adult.

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SY01-03 Maternal influence on neuroendocrine development of offspring

Kumi Kuroda[1], Michael Meaney[2], Tadafumi Kato[1]
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Inappropriate maternal care affects the children's stress reactivity and their parental behavior. This has been confirmed in animal models, and was found to be mediated by alteration of postnatal development of the hypothalamo-pituitary-adrenal (HPA) axis. On the other hand, epidemiological studies recognized the correlation between prenatal malnutrition and the higher incidence of cardiovascular diseases (CVD). As explanation, "the thrifty phenotype hypothesis" postulates the active mechanism that adjusts the fetal metabolic system for better survival in future malnutrition. If food is not as scarce as predicted, however, this adaptation leads to the metabolic syndrome (hypertention, insulin resistance, obesity) and increases the risk of CVD. These two phenomena are now converging into a unified concept of "maternal programming", in which adaptation to the environment during a critical period of development causes life-long alteration of the neuroendocrine system. Its molecular mechanism will be discussed.

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SY01-04 QTL studies for understanding genetic mechanism of diversity on spontaneous activity in mice

Tsuyoshi Koide[1,2]
[1]Mouse Genomics Resource Laboratory, National Institute of Genetics, Mishima, Japan, [2]The Graduate University for Advanced Studies

Recent dramatic progress of molecular genetics clarified causative genes of many human diseases. In these studies, it was found that mutation in single gene cause aberrant phenotype. However, most of the complex diseases are caused by not single gene but multiple genes. Based on the hypothesis that disease is just one of the extreme case of natural variation of the phenotype, understanding genetic mechanism of producing general diversity of the phenotype is getting more important after the genome sequencing. In order to understand genetic mechanism of behavioral diversity, we are currently conducting QTL analyses of spontaneous locomotion behavior in mice, which are involved in survival strategy of many animals. Although we were able to find genetic loci involved in spontaneous activity, it is still hard to find the actual genes. So, I will talk about future prospect of the QTL research to study genes and behavior.

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SY01-05 Neuro-behavioral changes induced by precocious weaning.

Takefumi Kikusui[1], Yuji Mori[1]
[1]Veterinary Ethology, The University of Tokyo, Tokyo, Japan

Social environmental stimuli during the early life stage are shown to affect various aspects of physiological and behavioral development. In this study, long-lasting influences of early weaning upon adulthood behavioral as well as neurochemical changes were investigated in rats and mice. The early-weaned rats had higher anxiety level as assessed by various anxiety tests, and they had higher autonomic responses under a novel environment. Social behavior tests carried out in other sets of mice revealed that early weaning resulted in increased number of attacks towards their cagemates when co-housed. In addition, maternal and sexual behaviors were found to be repressed in early weaned mice. The early-weaned mice had a higher neuroendocrine response to stress, as assessed by corticosterone secretion. Various neurochemical changes by early weaning were observed in the brain, including lower expression of glucocorticoid receptor mRNA in the hippocampus. These results indicate that early weaning affects brain development in rodent species and thus modifies behavior patterns in their adulthood.

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SY02-01 Epidemiological trends in autism and developmental disorders

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After a brief background on concepts and diagnostic criteria for autistic spectrum disorders, and on etiologic factors, we will review epidemiological studies of autism. Over 40 epidemiological studies have now been conducted. Prevalence rates are much higher than thought before and current estimates suggest a figure around 0.6%, a threefold increase compared to prior estimates. The causes of this increase are not well understood. The increased rates are nevertheless alarming and might suggest that some environmental triggers could have modified the incidence of the disorder, perhaps in interaction with genetic susceptibility factors. We will conclude in providing some additional data about the epidemiology and the time trends for other developmental disorders, including attention deficit hyperactivity disorders (ADHD) and specific learning disorders (dyslexia, language disorders, etc.). These data will also be presented in the context of possible gene environment interactions to explain time trends in the incidence of these developmental disabilities.

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SY02-02 COMORBIDITY OF LD, ADHD AND AUTISM SPECTRUM CAN BE CAUSED BY SPATIO-TEMPORAL DISRUPTION OF DEVELOPING CIRCUITS (SYNAPSE) FORMATION WITH CASCADES OF GENE EXPRESSION BY ENVIRONMENTAL TOXIC CHEMICALS.

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Autism spectrum disorders (ASD), including learning disorder (LD), attention defficit hyperactivity disorder (ADHD), children show comorbidity, having complex combination of various symptoms, which makes difficult to diagnosis them under a single name of disorder. Environmental (chemical and social) and genetic factors interact and determine human cognitive development and behavior, through synaptogenesis and plastic changes of formed neural circuits both to which are subserved by cascades of gene expression. Hormone-dependent and activity-dependent gene expressions in the fetal and newborn brain can be disrupted by spatio-temporal exposure of environmental chemicals such as endocrine disrupters, resulting abnormal synaptogenesis and malformation of neural circuits depending on their sensitive periods. (Kuroda, Y. Environ. Sci. 10:23, 2003)

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SY02-03 Effect of environmental disruptors on the activity-dependent expression of brain-derived neurotrophic factor (BDNF) gene in neurons

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Brain-derived neurotrophic factor (BDNF) is a member of neurotrphin family, which contributes not only to survival and differentiation of neurons but also to synaptic plasticity. The expression of BDNF gene is controlled in an activity-dependent manner, and we and other groups have already demonstrated that the calcium (Ca2+) signal-dependent activation of BDNF gene promoters is mediated by the transcriptional factors CREB and USF. In rat cortical neuronal cultures, we have recently found that the BDNF mRNA expression can be activated in an autocrine fashion and maintained being elevated for at least 3 days after stimulation. In this culturing systems, administration of deltamethlin, a pyrethroid insecticide, which increases an exitability of neurons through an interaction with sodium (Na+) channels, elevated the BDNF mRNA expression and maintained its highly expressed level. We are now analysing the mechanisms for this deltametlin-induced elevation of BDNF mRNA expression and the effects of other chemical compounds on BDNF mRNA expression.

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SY02-04 Suppression of thyroid hormone receptor-mediated transcription by polychlorinated biphenyls.

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Thyroid hormone (TH) plays a critical role in brain development. Its deficiency causes abnormal brain development. On the other hand, exposure of polychlorinated biphenyls (PCBs) during perinatal period also causes a mild retardation of brain development. Thus, part of PCB action has been considered to be exerted through TH system. However, the molecular mechanism of PCB action on TH system has not yet fully understood. We have examined the effect of PCB compounds on TH-receptor (TR)-mediated transcription. In CV-1 cell, a fibroblast-derived clonal cell, PCB suppressed TH-induced transcription of TR as low as 10^{-10} M. The effect of PCB may not be a competitive inhibition of TH binding to TR, since dose dependency of PCB action was not so evident. When different clonal cells were used, the effect was greatest in neuron-derived cells. These results indicate that PCB may affect brain development at least in part by inhibiting TR action.

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SY02-05 Development of a quantitative oligonuceotide microarray "Synaptoarray" to monitor the effect of environmental chemicals on brain development

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By selecting the relevant genes and designing oligonucleotide probes of specified molecular properties, we developed a quantitative DNA microarray "Synaptoarray" to monitor gene expression changes during brain development. In addition to the synaptic genes, genes were selected experimentally by analyzing changes due to hypothyroidism, inhibition of neural activity, and mitochondrial dysfunction. Reproducibility of array data was further improved by employing multiple spots/probe and multiple probes/gene on one slide, and by the use of a gold-coated slide with probes attached via a new 5'-terminal linker. As a result, ±20% difference in gene expression ratio could be detected. Gene expression changes during postnatal development of the rodent cerebellum examined with the Synaptoarray clearly demonstrated the alterations due to hypothyroidism. Effect of perinatal exposure to hydroxy-PCB is analyzed using these data.

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SY03-01 Properties of activity-dependent synaptic plasticity induced by 0.5-10 Hz low-frequency stimulations

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We have investigated the effects of low-frequency stimulations (LFS: 40, 80, 120, 200, 500 or 1,000 pulses at 0.5, 1, 2, 5 or 10 Hz) on the synaptic transmission in CA1 neurons of guinea pig hippocampal slices. LFS with each of these parameters was applied to a naive inputs or 20 min after tetanus (100 Hz, 100 pulses). LFS of 40-120 pulses at 1-10 Hz and 200-1,000 pulses at 5-10 Hz produced long-term potentiation (LTP) whereas the LFS of 200-1,000 pulses at 1 Hz produced long-term depression (LTD) in the responses. When tetanus was applied prior to the delivery of LFS, LFS reduced the potentiated synaptic efficacy (Depotentiation). Then, we applied an antagonistfor NMDA receptors or mGluRs during LFS with each parameters and observed the changes in the LTP, LTD and depotentiation. Based on the results, the roles of NMDA receptors and mGluRs in the dynamics of the activity-dependent synaptic plasticity are duiscussed.

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SY03-02 Activity-driven changes in neuronal actin cytoskeleton

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Actin cytoskeleton constitutes the predominant cytoskeletal component in the postsynaptic density of excitatory synapses. Actin assembly and disassembly are thought to play a critical role in the appropriate reorganization of the synaptic signaling scaffold bound to glutamate receptors. Actin rearrangement is further essential for triggering changes in dendritic spine morphology. However, the precise interdependence between synaptic activity and actin cytoskeletal dynamics remains largely unexplored. Here we present our recent results from time lapse GFP-actin imaging indicating strong spatial correlation between strong Ca^{2+} influx sites and activity-driven actin remodeling. Together with other Ca^{2+}-mediated plasticity mechanisms, Ca^{2+}-triggered synaptic actin reorganization may thus participate in efficient storage of information both at the synaptic and cellular levels.

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SY03-03 Organization of PSD scaffolding proteins revealed by live cell imaging

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A variety of proteins are recruited to the PSD, an electron dense thickening of the postsynaptic membrane, and are thought to play critical roles in the modulation of synaptic functions. Transmembrane proteins, such as glutamate receptors, and lipid-anchored scaffolding proteins, such as PSD-95, are thought to function as linkers between the postsynaptic membrane and the cytoplasmic matrix and can potentially stabilize the PSD structure. However, analyses of culture preparations from NMDAR and mGluR -/- mice and those manipulated pharmacologically to remove PSD-95 indicated that these molecules are dispensable for the PSD stability. In contrast, manipulation that disrupts F-actin induced rapid disassembly of multiple PSD scaffolding proteins. The fractions of these actin-dependent components were comparable with rapid-exchangeable fractions measured by fluorescence recovery after photobleaching analysis. We conclude that integrity of F-actin in dendritic spines is indispensable for the maintenance of the molecular architecture of the PSDs.

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SY03-04 Two forms of long-term synaptic plasticity produced in cultured brain slice

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Due mainly to the lack of good model system for analysis, the mechanism of long-term synaptic plasticity in mammalian brain remains unrevealed, as compared with that of short-term one. We propose here that the cultured brain slice meets the requirements for the wanted model system. When we repeatedly induced LTP (long-term potentiation, a typical form of short-term plasticity) in the cultured rat hippocampal slice by chemical means, it showed a slowly-developing persistent (lasting for days-weeks) enhancement in synaptic strength accompanied by an increase in the number of synapses. This repetition-induced synaptic enhancement (RISE) depended on the activity of protein kinases (PKA and MAPK). In an apparent symmetry, when we repeatedly provoked LTD (long-term depression), the cultured slice showed a long-term reduction in synaptic strength accompanied by a decrease in the number of synapses. This LTD-operated synaptic suppression (LOSS) depended on the activity of protein phosphatase.

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SY03-05 Molecular mechanisms of memory consolidation, reconsolidation and extinction

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A new memory is converted from labile to stable state through a process; consolidation. Previous studies showed that memory retrieval may initiate two dissociable but opposite processes; reconsolidation and extinction. Reconsolidation acts to stabilize, whereas extinction weakens, the expression of the original memory. Importantly, these three processes require new gene expression. To understand the molecular mechanisms of these memory processes requiring gene expression, we are investigating molecular signatures of them. Our recent transgenic studies indicated that CREB may function as a molecular switch of consolidation and reconsolidation. We also examined the relationship between reconsolidation and extinction, and found three distinct time-dependent memory phases following retrieval: While retrieved memory is stable, extending retrieval triggers a reconsolidation, and further prolonged retrieval induces the formation of a new extinction trace. We will also present the results showing the roles of CB1 and LVGCCs in these processes.

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SY04-01 Membrane Excitability in Hypothalamic GnRH Neurons: Roles of G Protein-Coupled Receptors and Second Messengers

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The characteristic pulsatile secretion of gonadotropin-releasing hormone (GnRH) from hypothalamic neurons is dependent on an autocrine interaction between GnRH and its receptors expressed in GnRH-producing neurons. GnRH neurons expressed in native and immortalized GnRH neurons initiate diverse signaling pathways by coupling to multiple G proteins. Such coupling is time- and dose-dependent, and switches between Gq, Gs, and Gi/o, in GnRH-stimulated GT1-7 neurons. Both native and immortalized GnRH neurons exhibit spontaneous electrical activity that is stimulated by GnRH agonist treatment. The co-expression of GnRH-R and GnRH, GnRH-dependent modulation of spontaneous electrical activity, activation of Ca^{2+} signaling and autocrine regulation of GnRH release, are characteristics of early embryonic GnRH neurons and provide a mechanism for regulated GnRH secretion during embryonic migration.

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SY04-02 Voltage-gated Calcium Channels in Rat GnRH Neurons

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To investigate cellular functions of GnRH neurons, we have established the transgenic rat line in which GnRH neurons express GFP. Dispersed GnRH neurons were prepared and cultured overnight for perforated patch-clamp experiment. In neonatal GnRH neurons, high voltage-activated calcium currents were clearly observed, but low voltage-activated calcium current was negligible. Pharmacological examination revealed that the total current was composed with ~20% of L-type, ~20% of N-type and ~55% of R-type. P/Q-type and T-type were negligible. Around puberty, however, T-type and P/Q-type currents were clearly observed in addition to other calcium currents. A similar expression pattern was observed in GnRH neurons from adult rats. There was no difference between males and females. These results indicate that high expression of the R-type channel especially in young animals may relate to certain functions of the GnRH neuron and the T-type and P/Q-type channels are involved in certain cellular functions including membrane excitability after pubterty.

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SY04-03 Physiological properties of neuromodulatory GnRH peptidergic neurons

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Animals flexibly adjust their endocrine, autonomic, and behavioral functions in response to ever-changing environments, and the peptidergic nervous system is believed to play an important role. Here, I introduce our recent studies on an extrahypothalamic GnRH peptidergic neuron system, the terminal nerve (TN) GnRH system, which we suggest is neuromodulatory and is involved in the control of motivational state of the animal. By using a fish model system, we examined electrophysiological properties of TN-GnRH neurons, focusing on the voltage-gated Ca^{2+} channels, which are thought to be coupled to several cellular events such as GnRH release, and the glutamate receptors, which are involved in the synaptic control of GnRH neuronal activities. They showed low-voltage activated (LVA) and three types of high-voltage activated (HVA; L-, N-, and R-) Ca^{2+} currents. The HVA currents were inhibited (preferentially N- and R-) by the application of GnRH via an auto-/paracrine mechanism. We also identified non-NMDAR and NMDAR, group 3 mGluRs, and a novel type of ionotropic GluR.

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SY04-04 Novel hypothesis for sexual differentiation of the control of GnRH secretion: Why females have reproductive cycles and males do not

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Neuroendocrine regulation of ovulatory cycles is complex and requires four steroid feedback mechanisms to control GnRH secretion, whereas spermatogenesis is continuous and requires only one feedback mechanism. In the female, two feedbacks regulate the preovulatory surge mode of GnRH secretion, and two control the pulsatile mode. In the male, three controls are not necessary, and only a simple negative feedback regulation of pulsatile GnRH secretion remains operative. We propose that all four feedbacks are inherent in both sexes, and they require no further developmental input to function appropriately during adulthood. The developing male brain is programmed by exposure to androgen and estrogen actions of testicular steroids. Depending upon the species, programming begins before birth but can continue through postnatal life.

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SY04-05 Lactational anestrus: Mechanism mediating suppression of pulsaitle luteinizing hormone (LH) release in lactating rats

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Ovarian cyclicity is strongly inhibited during lactation. This inhibition is mainly due to the suppression of the pulse mode of gonadotropin-releasing hormone (GnRH)/ LH release, which is responsible for follicular development and steroid production. The surge mode of LH secretion seems to remain intact in lactating mothers. In the lactating rat model, neural inputs to the hypothalamus derived from the suckling stimulus predominantly suppresses LH pulses during the first half of lactation, without other humoral factors such as ovarian steroids and prolactin. On the other hand, LH pulse is suppressed by suckling stimulus in an estrogen dependent manner during the second half of lactation. Thus, the brain mechanisms mediating suppression of LH pulses could be different between the phases of lactation in rats. The present paper will discuss the estrogen-dependent and -independent mechanisms mediating the suppression of LH pulses during lactation.

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SY05-01 *Therapeutic strategy for the polyglutamine diseases targeting the toxic conformational transition and aggregation of the polyglutamine protein*

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The polyglutamine (polyQ) diseases are a group of inherited neurodegenerative diseases including Huntington's disease, in which the expanded polyQ stretch is thought to undergo a conformational transition, leading to aggregation. We previously identified QBP1 (PolyQ Binding Peptide 1) and showed that QBP1 suppresses polyQ protein aggregation *in vitro* and neurodegeneration in *Drosophila* models. We show here that QBP1 prevents the expanded polyQ protein from undergoing a conformational transition to a β-sheet-rich structure, resulting in inhibition of amyloid-like fibril formation. We established a high-throughput screen for polyQ aggregate inhibitor chemicals. We conclude that therapeutic targets for the polyQ diseases are the toxic conformational transition and aggregation of the expanded polyQ protein.

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SY05-02 Prion disease and Unfoldin: A protein with robust ATP-dependent unfoldase activity and a novel *grapple-like* structure in vitro

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We have isolated and characterized a novel robust protein unfolding activity from S. cerevisiae membrane fractions. The factor, denoted Unfoldin, has no obvious substrate specificity, it disrupted the tertiary structure of a variety of appropriately and stably-folded substrate proteins as determined in vitro even when pathogenic polypeptides such as the prion protein in the β-sheet form, α-synuclein or the Amyloid β (1-42) peptide were tested as substrates. The binding of ATP to Unfoldin regulates both of the substrate-binding and protein-unfolding activities. Unfoldin forms an oligomeric grapple-like structure with an opening and ATP regulates the opening and closing of the gate that forms the opening within Unfoldin.

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SY05-03 Metabolic mechanism of amyloid-β peptide

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Neprilysin is a rate-limiting peptidase participating in Aβ catabolism in brain. Neprilysin gene-disruption caused a gene dose-dependent elevation of the endogenous Aβ levels in the mouse brain, suggesting that even a subtle reduction of neprilysin activity will contribute to Alzheimer's disease (AD) development. Recent evidence that expression levels of neprilysin were reduced in the hippocampus of sporadic AD patients and aged laboratory mice suggests a close association of neprilysin with the etiology and pathogenesis of AD. Viral vector-mediated neprilysin gene transfer reduced Aβ levels and could retard amyloid deposition in the brains of AD model mice. Therefore, up-regulation of neprilysin would represent a promising strategy for therapy and prevention via the reduction in Aβ levels. Recently, we have found that a certain neuropeptide regulates the expression of neprilysin in primary neurons. Since a number of receptors for neuropeptides are G-protein-coupled receptors, we may be able to control brain Aβ levels pharmacologically by the manipulation of neprilysin activity.

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SY05-04 Neurodegenerative disease protein, APTX, is a new DNA repair protein.

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Aprataxin (APTX), a causative gene product for early-onset ataxia with ocular motor apraxia and hypoalbuminemia, interacts with X-ray repair cross-complementing group 1 (XRCC1), which plays an important role in single-strand DNA break repair (SSBR). In addition, the N-terminus of aprataxin is highly homologous to polynucleotide kinase 3' phosphatase, which is also important for SSBR. This homologous region is considered to be the forkhead- associated (FHA) domain. The FHA domain of APTX is important for its interaction with the BRCT II motif of XRCC1. Moreover, PARP-1 was also coimmunoprecipitated as a component of the APTX-XRCC1 protein complex. These results indicate that APTX may contribute to SSBR. Several neurodegenerative disorders were caused by impairment of DNA repair. However the mechanisms underlying neurodegeneration in these disorders is not clear. To elucidate the mechanism of selective neurodegeneration, further studies of the physiological functions of APTX are necessary.

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SY05-05 Development of siRNA expression vector for gene therapy

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RNAi is the process of post-transcriptional gene silencing in animals and plants. Guides of sequence-specific mRNA degradation are 21 nt small interfering RNAs (siRNAs) generated by Dicer-dependent cleavage from longer dsRNAs. In mammalian cells, siRNAs can induce RNAi and it occurs only in the cytoplasm. In this study, we constructed expression vectors for tRNAVal-based short hairpin RNA (tRNA-shRNA) that can promote the cytoplasmic localization of the dsRNAs. Stably expressed tRNA-shRNAs targeted to erbB2 transcripts induced significant RNAi-mediated silencing of expression of erbB2 gene in MCF-7 cells. In addition, our tRNA-shRNA expression system also functioned when PML-RAR alpha and telomerase mRNA were made to be a target. Thus, the tRNA-shRNA technology should be a powerful tool for studies of gene functions, with additional potential utility as a therapeutic tool.

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SY05-06 Development of HSV amplicon vector system: a versatile gene delivery tool for gene therapy and functional studies

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Herpes simplex virus type-1 (HSV) amplicon vectors are bacterial-mammalian shuttle plasmids amplified as ~150-kb head-to-tail concatemers and packaged within HSV virions. Infectious delivery of vector DNA is possible due to HSV virion function, but the vector DNA itself does not mediate any viral gene expression. The vectors possess several unique features that make them promising gene transfer vehicles, including: (i) their large transgene capacity up to 150 kb; (ii) they can infect a wide variety of cell types across a broad range of species; (iii) the ease of vector construction; (iv) their limited cytotoxicity and immunogenicity because of their lack of viral coding sequences. The large packaging capacity enables HSV amplicons to carry entire genomic loci, multiple transcriptional units, and virtually any DNA components from other viruses and/or organisms. The characterization and potential applications of this unique vector system will be discussed.

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SY06-01 Posing a problem for neuroimaging: Relationship between changes in hemodynamic parameters and BOLD effects

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Blood oxygenation level-dependent functional magnetic resonance imaging (BOLD- fMRI) is widely used for the basis on functional brain mapping. During brain activation, increases in the regional blood flow lead to an increase in blood oxygenation and a decrease in paramagnetic deoxygenated hemoglobin (deoxy-Hb), causing an increase in the MR signal intensity. Not a few studies, however, have reported failures to detect activation of areas where ought to be functioned. Here, we assigned BOLD-positive (increase in the signal intensity), -negative (decrease in the signal intensity), and -silent (no change) brain activation to respective circulatory conditions through a description of the fMRI signal as a function of the concentration of oxy-Hb and deoxy-Hb obtained with near-infrared optical imaging. Using the above model, we succeeded to solve the problem of the sensory motor paradox as the presence of BOLD-negative and -silent brain activation as well as BOLD-positive.

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SY06-02 Examination of a biophysical model of the BOLD signal using MRI measurements and concurrent optical imaging spectroscopy and laser Doppler flowmetry.

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BOLD fMRI measurements are a confound of changes in blood volume and oxygenation. Optical imaging spectroscopy (OIS) can be used to estimate changes in blood volume (CBV) and oxygenation dependent on estimates of the baseline blood volume fraction. It is possible using intra-vascular super paramagnetic contrast agents to estimate both changes in blood volume and the baseline concentration of blood (CBV0) in tissue using MRI techniques (cbv-MRI). Hence BOLD MRI together with cbv-MRI, and OIS are complementary methods to investigate the hemodynamic response. This study investigates the adequacy of the models needed to link these different measurement methodologies. Experiments combining BOLD-fMRI and cbv-MRI to estimate the values of the parameters in the biophysical model of the BOLD signal following activation will be described. We compare the MRI results with data from a comparable study using identical procedures in which OIS and laser Doppler flowmetry (LDF) are used to measure the hemodynamic response, estimate changes in CMRO2 and the exponent in the power law relating changes between blood volume and flow.

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SY06-03 Functional signal- and paradigm-dependent linear relationships between synaptic activity and hemodynamic responses

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When imaging stimulus-related neuronal activity by subtracting control-state signals from stimulus-state signals, we rely on two assumptions: no interaction between the neuronal activity of interest and the control, and linear relationships between functional signals and neuronal activity. Here we investigated how linearly functional signals, especially cerebral blood volume (CBV) and oxygenation changes, correlate with underlying synaptic activity. We simultaneously measured local field potentials (FP) and optical intrinsic signals in rat somatosensory cortex. The results showed CBV-related signals had higher fidelity to summed evoked FP (ΣFP) than oxygenation-derived signals. Moreover, CBV and late oxygenation signals increased linearly with ΣFP while varying stimulus number, but they exhibited a threshold and steeper gradient while varying stimulus intensity. These nonlinearities should be considered in the application of hemodynamic signals.

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SY06-04 Functional Brain Imaging with Light Scattering Changes Elicited by Neural Activities

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Intrinsic optical signals (IOSs) enable us to visualize functional structures near the cortical surface. Circumstantial evidence suggests that changes in light scattering (LS) as well as hemoglobin absorption changes are the sources of IOSs. Recently, we found that IOSs are elicited in retina even at the region where no capillaries exist, providing concrete evidence that changes in LS is one of the sources of IOSs. Taking LS changes as the signal to be detected, we succeeded in visualization of depth resolved functional structure by using Optical Coherence Tomography (OCT). OCT is the technique to measure the first order light reflection from the deep structure in a tissue without exposure. Since changes in LS strongly affect the first order light reflection, active regions in deep structure are revealed as changes in OCT signals. In this talk, we will show depth resolved functional structures in visual cortex obtained by this technique as well as IOSs in retina.

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SY06-05 A Model for the Regulation of Local Cerebral Blood Flow Evoked by Neuronal Activation

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Linearity of the local cerebral blood flow (LCBF) with respect to neuronal activation is essential for analyzing functional images obtained with paradigms containing overlapping multiple tasks. Using perturbation of physiological conditions, we have demonstrated that the relative LCBF, rather than the absolute LCBF, properly reflects and is linearly coupled to neuronal activation. Also, we showed the LCBF latency was <1s. This presentation aims to describe a model for vascular regulation that explains these observations. The model will be that: 1) local vascular resistance is serially connected with the systemic vessel circuit; 2) imprinting neuronal activity on vascular response should be in a linear manner with respect to activation strength; 3) the mechanism should operate in a deterministic, rather than a feedback, manner because of a rapid balance (<1s) between them. Although morphological and biochemical linkage between neuronal activity and vascular response is still to open-discussion, a feasible and useful model will be presented.

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SY06-06 Stimulus induced changes in cerebral microcirculation measured by optical coherence tomography

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Optical coherence tomography (OCT) is a technique to obtain cross-sectional images of highly scattering media like biological tissues to a few millimeters depth with around 10 micrometers spatial resolution. It can also detect the Doppler frequency shift due to the blood flow in microvessels with the same spatial resolution. In this study, velocity profiles in pial microvessels as well as cross-sectional images of the somatosensory cortex were measured with OCT during sensory stimulation using rats. Temporal changes in the OCT signal distribution and the velocity profiles are discussed in relation to the stimulation. Delayed swelling of the cortical surface appeared in the somatosensory cortex following the stimulation in the case of dura removal. It was restricted to a thin surface layer with less than several 10 micrometers, and is considered to reflect the reactive hyperemia accompanying the neuronal activation.

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SY07-01 Responses of single neurons coding emotional expressions in the amygdala of monkeys

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To investigate the functions of the amygdala in emotional processing, the activity of single neurons was recorded while video clips of emotional expressions were presented. A stimulus set consisted of 9 video clips, 3 monkeys by 3 emotional expressions (scream, grunt, and coo). Of the neurons tested in the amygdala, 55% responded to the emotional stimuli. A two-way analysis of variance, in which the variables were individual and emotional expression, revealed that the effect of emotional expression on the activity was significant in most neurons while that of individual was significant in fewer neurons. Further properties of neuronal responses in the amygdale will be discussed in this talk.

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SY07-02 Context-dependent cortical activation in response to financial reward and penalty: An event-related fMRI study

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An event-related fMRI technique was used to assess neural responses to financial reward and penalty during a simple gambling task. Thirty-six healthy volunteers participated in the study. The task was to guess the color of the suit of a card on each trial and to respond by pressing a button. Every correct response (win) and incorrect response (loss) was associated with financial reward and penalty, respectively. We found that the bilateral anterior cingulate and medial prefrontal cortices were specifically activated when the subjects experienced the fourth win of four wins in a row and the fourth loss of four losses in a row. When the subjects experienced a win following four losses in a row or a loss following four wins in a row, the right dorsolateral prefrontal cortex was specifically activated. Our data indicate that there exists brain activities associated with the event-sequence context in which abstract reward or penalty is received.

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SY07-03 Functional neuroimaging in visceral perception, emotion and irritable bowel syndrome

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Clarifying functional module of the brain in response to visceral stimulation ("gut feeling") is one of keys to understand the mechanism of emotion and irritable bowel syndrome (IBS). In a study of positron emission tomography (PET) with injection of $H_2[^{15}O]$, colonic distention pressure-dependently induced visceral perception and anxiety, which significantly correlated with activation of specific regions of the brain including the prefrontal, anterior cingulate, parietal cortices, insula, pons, and the cerebellum. Specific triphasic cerebral evoked potential of N1, P1, and N2 was obtained with the rectal electrical stimulation. Colonic distention induced a significant decrease in the alpha power and a significant increase in the beta power of electroencephalogram (EEG). These data suggest that the functional module of the brain in response to the visceral perception is present and that the abnormal consequence of stimulation-evoked arousal and emotion relates to pathophysiology of IBS.

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SY07-04 Characteristics of frontal lobe function in depression, bipolar disorder, and posttraumatic stress disorder: A multichannel near-infrared spectroscopy study

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Characteristics of frontal lobe function in depression, bipolar disorder, and posttraumatic stress disorder (PTSD) were investigated during a word fluency task by monitoring regional cerebral blood volume changes as oxy-hemoglobin concentration ([oxy-Hb]) changes using two sets of 24-channel multichannel near-infrared spectroscopy (NIRS) machines. [oxy-Hb] change patterns were characterized by a rapid increase in the control subjects, lack of increase in depression, a gradual increase in bipolar disorder, and a paradoxical decrease in PTSD. The observed differences in the [oxy-Hb] change patterns in three patient groups are assumed to reflect reduced, delayed, and suppressed function of the frontal lobe, respectively. The present study has been approved by the Institute Research Board of Gunma University Hospital.(Collaborators: Drs. Suto T, Ito M, Kameyama M, Yamagishi Y)

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SY07-05 Structural and functional neuroimaging in posttraumatic stress disorder

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First, a combined structural MRI and event-related potential study in victims of Tokyo subway sarin attack revealed a significant volume reduction in left anterior cingulate cortex (ACC) in PTSD patients which was significantly correlated with P300 amplitude. Second, an fMRI study using subliminally presented threatening stimuli demonstrated that, while association cortices are activated in the control group, left parahippocampal gyrus was activated in the PTSD group. Finally, in a monozygotic twin study of US combat veterans, we found a significant volume reduction in the hippocampus, ACC, and insula in both combat-exposed PTSD subjects and their co-twins compared with both combat-exposed non-PTSD subjects and their co-twins. In conclusion, cortical abnormalities in PTSD may be characterized by those in limbic regions and paralimbic regions that regulate the former. These abnormalities may at least in part represent pre-existing vulnerability to PTSD, rather than post-traumatic toxic change.

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SY07-06 Neuroimaging and the traumatized brain: PTSD and emotion

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The goal of this study was to compare areas of brain activation and inter-regional brain activity correlations during two different types of response (flashback/reliving and dissociative) to the recall of traumatic memories in traumatized subjects with posttraumatic stress disorder (PTSD) using standard subtraction- and functional connectivity analyses.Traumatic memory recall in PTSD was studied using the traumatic script-driven symptom provocation paradigm adapted to fMRI at a 4 Tesla field strength. PTSD subjects with flashback/reliving responses showed greater activation of the prefrontal cortex bilaterally, the left inferior parietal lobe, and the left precentral gyrus. In contrast, dissociated PTSD subjects showed greater activation in the occipital lobes and bilateral thalami. Comparison of connectivity maps of PTSD patients with flashback/reliving and dissociative responses showed significant differences in brain areas involved in autobiographical memory and emotional processing.

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SY08-01 Alternative poly(A) site selection confers neural activity-dependent regulation of RNA splicing of vesl-1/homer-1

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The vesl-1 (homer-1) gene encodes a scaffold protein that interacts with mGluR1/5 and RyRs to modulate synaptic functions. The gene also encodes two shorter forms that counteract the functions of the long form Vesl. Expression of the shorter forms is driven by neural activities such as LTP, which modulate the alternative splicing of vesl-1. Each functional poly(A) site forms a different part of the 3'-terminal exon, with promoter-proximal and promoter-distal sites at the end of exons corresponding to the short and long form Vesl proteins, respectively. In this symposium I provide data that demonstrate that the promoter-proximal poly(A) region plays an indispensable role in the switching of alternative pre-mRNA splicing that is triggered by extracellular stimuli. I propose that a polyadenylation event regulates the neural activity-dependent alternative splicing of vesl-1 pre-mRNA.

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SY08-02 Hzf binds to IP3R1 mRNA and is required for dendritic mRNA localization and the motor learning in cerebellum

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The localization of certain mRNAs to dendrites in synaptic regions are proposed to be involved in synaptic plasticity. IP3R1 mRNA is a dendritically localized mRNA. However, the mechanisms underlying the dendritic targeting of IP3R1 mRNA have been unknown. Here we identified Hzf as a trans-acting factor for this dendritic targeting. Hzf protein is predominantly expressed in Purkinje cells, and localized to their dendrites. The finding that the dendritic localization of the IP3R1 mRNA in Purkinje cells and motor learning were impaired in Hzf-deficient mice suggested that dendritic localization of mRNAs is involved in motor learning.

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SY08-03 RNG105: A Novel RNA-Binding Protein in Neuronal RNA Granules, Regulatory Machinery for Synaptic Stimulation-Dependent Local Translation

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Local translation in neuronal dendrites is an important basis for long-term synaptic plasticity. RNA granules are reportedly involved in local translational control, but the constituents of the granules have not been fully identified. Here we identify RNG105, a novel RNA-binding protein, as a component of the RNA granules in dendrites of hippocampal neurons. RNG105 has an ability to repress translation, which is consistent with the finding that the RNA granules are translationally dormant in the basal conditions. Dissociation of RNG105 from the RNA granules is induced by BDNF, which is significantly correlated with the induction of local translation near the RNA granules. These findings indicate that the RNA granule is a control site for stimulation-dependent translation, and provide insight into the link between RNG105 dynamics and local translational regulation.

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SY08-04 Mouse Larks activate post-transcriptional expression of the Period1 gene.

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Two mammalian Lark genes, termed Lark1 and Lark2, have been identified as structural homologues of the Drosophila circadian clock gene, lark.. Both the mouse Lark (mLark) genes encode polypeptides of two RNA recognition motifs (RRMs) and a CCHC zinc finger motif as dLark. mLark activated the translation of the mouse Period1 (mPer1) mRNA by the direct interaction with the 3' untrasnlated region (3'UTR) of the mPer1 mRNA. Whereas the mLark mRNAs were kept at a constant level, a strong circadian cycling of mLark expression was observed in the mouse suprachiasmatic nuclei (SCN), and the expression phase of mLark was consistent with that of mPer1. To elucidate the function of the mLark gene in vitro, we analyzed the expression of Per1 in a mouse NIH3T3 cell line transfected with the Per1::luc reporter gene. A knock-down analyses by Lark-specific siRNA resulted in an approximately 0.3 hr shorter circadian period in the reporter gene expression rhythm than that obtained with a control siRNA. On the other hand, overexpression of mLark showed 0.4 hr longer circadian period. These data indicate that mLark has a certain role in the mammalian circadian system.

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SY08-05 mTOR-dependent translational control in neurons induced by BDNF and neural activity

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Local translation in dendrites is implicated in synapse-wide alterations of function and morphology triggered by neural activity. However the molecular mechanisms of local translational control is still unclear. Neurons have all the equipment for protein synthesis in their dendrites. Local protein synthesis and translational activation in neuronal dendrites were up-regulated by brain-derived neurotrophic factor (BDNF). In parallel, BDNF activated the cascade of mammalian target of rapamycin (mTOR) signaling in dendrites and the synaptoneurosome fraction. The observed mTOR activation led to the stimulation of translation initiation and elongation processes. In addition to pharmacological manipulation, mTOR siRNA transfection also blocked protein synthesis and translational activation induced by BDNF. Further evidence that mTOR is involved in activity-dependent neural plasticity such as LTP, spatial learning and sensory-deprivation will be discussed.

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SY09-01 The nucleus of tractus solitarius: The local network and its developmental regulation

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In the caudal nucleus of tractus solitarius (NTS), both glutamatergic and GABAergic neurons are interconnected synaptically with each other in a highly organized manner. The morphological and electrophysiological characteristics of this local network of the NTS will be presented with the classification of NTS neurons. These highly differentiated local networks in adult rats are established at around postnatal days 6-8 by distinct eliminations of both glutamatergic and GABAergic synapses from specific types of the NTS neurons. The former would involve activity-dependent mechanisms through an activation of NMDA type glutamate receptors, while the latter gene regulation of specific $GABA_A$ receptor subunit(s). The molecular mechanisms and functional significance of this developmental differentiation of NTS local networks will be presented and discussed in the second half of this session.

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SY09-02 Optical analysis of developmental organization of functional synaptic networks in the NTS

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The multiple-site optical recording with a voltage-sensitive dye was used to study the developmental organization of functional synaptic networks in the NTS. Glutamatergic EPSPs evoked by N.X stimulation were first detected from the NTS at E7 in chick embryos and E15 in rat embryos, when morphological differentiation of pre- and postsynaptic neurons has yet to be completed. When extracellular Mg^{2+} was removed, small EPSPs were elicited at E6 in chick embryos and E14 in rat embryos. These results suggest that synaptic function mediated by NMDA receptors is latently generated one day before the expression of the glutamatergic EPSPs. Functional synapses related to N.IX appeared to be generated at the same time as N.X, but their spatial distribution was different from that of N.X. We further investigated the development of the second synaptic pathway from the NTS to the higher centers, and found that neuronal circuits from the NTS are already generated when the primary afferents make functional synapses on the NTS neurons.

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SY09-03 DOPA, as a neurotransmitter candidate in the nucleus tractus solitarii

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L-3,4-dihydroxyphenylalanine (DOPA) is a neutrotransmitter candidate of the primary baroreceptor afferents terminating in the necleus tractus solitarii (NTS). The depressor responses to DOPA microinjected into depressor sites of the NTS are sensitive to DOPA methyl ester (DOPA ME), a competitive DOPA antagonist. Antagonism by DOPA ME in the NTS elicits hypertension and tachycardia, thereby suggesting the tonic function of DOPA recognition sites. However, either kynurenic acid or DOPA ME abolished the depressor response to aortic depressor nerve (ADN) stimulation. Further, prior injection of NG-monomethyl-L-arginine or antisense oligos for neuronal nitric oxide (nNO) synthase reversibly reduced the responses to DOPA. A NO precursor L-arginine induced depressor responses, but these responses were not affected by kynurenic acid. DOPA may release glutamate via recognition sites for DOPA, and probably produces depressor responses via activation of kynurenic acid-sensitive ionotropic glutamate receptors and nNOS. We herein propose a new pathway that DOPA is a neurotransmitter of the primary ADN, and glutamate is a neurotransmitter of primary and/or secondary neurons in neuronal microcircuits of the NTS.

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SY09-04 Synaptic organization of cough reflex in the nucleus tractus solitarius

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Cough is a protective reflex that expels mucus or particles from the airways with large airflows generated by abdominal muscle contractions. Fictive cough is characterized by a rapid, large burst in the expiratory nerve combined tightly with a preceding increase in the inspiratory nerve discharge. I will present evidence that the nucleus tractus solitarius (NTS) is an essential part of the cough reflex pathway. A fictive cough is inducible by microstimulation of the NTS, and it is inhibited by microinjection of codeine into the NTS and abolished by the NTS lesion. The NTS contains cough-related neurons that are active during inspiration in normal respiration and respond to lung inflation by depolarization. During the expulsive phase of fictive cough, the NTS neurons display a rapid, large depolarization and burst discharge as similar to augmenting expiratory neurons. Codeine inhibits this depolarization. The cough reflex pathway and the relation to the respiratory network will be discussed.

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SY09-05 Transmitter release regulation in the nucleus of the solitary tract

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The second-order neurons in the caudal part of the nucleus tractus solitarii (cNTS) receive glutamatergic inputs from the primary afferents (TS) and from interneurons. N-type voltage-dependent Ca^{2+} channels (VDCCs) mediate ~45% of the glutamate release from TS, whereas a half of that is resistant to most of the known VDCC blockers except Cd^{2+} (Yamazaki, Shigetomi and Kato, 2003). Activation of presynaptic adenosine A1 receptors significantly decreases the release from TS in rats and mice (Tsuji and Kato, 2003), an effect suppressed by blockade of N-type VDCCs in wild type mice, whereas in the mice lacking CaV2.2, neither N- nor P/Q-type VDCC underlies this effect (Shigetomi, Yamazaki, Nishida, Mori and Kato, 2004). A large part of the spontaneous release in the cNTS does not depend on Ca^{2+} (Yamazaki, Shigetomi and Kato, 2003), but is strongly facilitated by presynaptic Ca^{2+} entry through P2X receptor channels (Shigetomi and Kato, 2004). The physiological relevance of the various forms of Ca^{2+}-dependent and independent transmitter release in the cNTS is discussed.

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SY10-01 Overview of the symposium: Simple system approaches to the higher brain functions

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In this symposium, we focus on the two brain systems: honeybee mushroom body and rodent cerebellum, to clarify the higher brain functions. These systems appear to be simple, but play some important and common roles underlying recognition, learning and memory, and social behavior. The comparison of the studies using these two systems, therefore, illuminates the molecular and cellular bases of the higher brain functions. The following four investigators will discuss these systems from various approaches at molecular to behavioral levels. Dr. Hideaki Takeuchi shows a transcription factor involved in social behavior in honeybees. Dr. Uli Mueller indicates cellular signaling pathways for memory formation in honeybees. Dr. Shogo Endo exhibits a downstream component of PKG in LTD. Finally, Dr. Shigenori Kawahara discusses glutamate receptors involved in eyeblink conditioning.

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SY10-02 Future aspect of molecular neurobiology using honeybee mushroom body as a model system.

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Honeybees perform exquisite social behaviors such as communications and division of labor. Mushroom body (MB) is one of insect brain structures, which is involved in higher-order sensory processing. Honeybee MBs function might be closely associated with their social behaviors. To clarify the molecular basis underlying MB function, we identified over 30 genes selectively expressed in the MBs in the honeybee brain using differential display and DNA microarray method. Several genes may act in intracellular signalling including Ca^{2+}-signaling pathway. We also identified a novel gene encoding a transcription factor (Mblk-1). These genes are good candidates for those modulating the advanced social behaviors of honeybee. Now we construct the gene expression database of these MB-selective genes, showing *in situ* hybridization results, microarray data and genome structure. Furthermore we established visual learning assay to examine involvement of the genes in the honeybee behaviors.

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SY10-03 What bees can teach us: Networks and dynamic molecular mechanisms underyling memory formation

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Memory formation is a highly dynamic process that critically depends on the training procedure. It is mostly unknown how the different training procedures modulate the molecular processes underlying memory formation and how they relate to distinct behavioural changes. We investigate learning-induced modulations of molecular processes underlying the induction and maintenance of memory in honeybees spanning time-windows from seconds up to several days. Our analysis shows that different cellular signalling pathways in distinct neuronal circuits take part in various aspects of memory formation. In addition to parallel-acting molecular processes during training, we identified processes required in very distinct time-windows after olfactory conditioning. The identification of the molecular network underlying memory formation in the bee provides the basis for understanding memory formation in general, and allows us to search for mechanisms known to influence memory formation. Supported by the German Research Council

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SY10-04 Molecular dissection of cerebellar-dependent memory

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The neuronal plasticity underlying cerebellar-dependent memories is called long-term depression (LTD) that is an activity dependent depression of synaptic transmission between parallel fiber and Purkinje cell. A variety of signal transduction molecules are shown to be involved in cerebellar LTD and cerebellar-dependent behaviors. NO-soluble guanylate cyclase-cGMP-cGMP-dependent protein kinase (PKG) pathway was shown to be involved in LTD, however, the downstream component of PKG was unknown. We have examined the role of G-substrate, an excellent substrate of PKG in Purkinje cell, using gene-targeting technique. The mice lacking G-substrate gene are vital and normal in general movement. However, the mice lacked cerebellar LTD in slice and in cell culture preparations. Furthermore, the mice are deficient in long-term adaptation in optokinetic reflex of eye movement without any effect on short-term one. Recently, similar results was reported in the mice lacking PKG in Purkinje cells. The results suggest a specific role of G-substrate and NO pathway in long-term memory.

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SY10-05 Eyeblink conditioning as a window for the higher nervous system

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Classical conditioning of the eyeblink response is an associative leaning that critically depends on the cerebellum. The higher nervous system such as the hippocampus also plays an important role in certain situations. Because its essential neural circuit is well characterized, this learning task offers a good model for studying hierarchical mnemonic systems. Mutant mice lacking the glutamate receptor subunit δ2 have several deficiencies specific to the cerebellar cortex. As expected, these mutant mice exhibit severe impairment in delay paradigm, but normally learn in short-trace interval paradigm. Pharmacological studies revealed that learning in the mutant mice depends on mACh and NMDA receptors. Lesion study indicated that the hippocampus plays an important role for acquisition but not for retention or expression. These results suggest that when the task is difficult for the lower system, the contribution of the higher system increases. Further study will reveal the detail of the function of the higher nervous system. Supported by CREST, JST.

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SY11-01 Development of customized software GeneFIS for analysis of gene expression profiles

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DNA microarray studies have recently shown that gene expression signatures can be used to classify heterogeneous cancer patients according to the predicted chemotherapy response. Such microarray data are also useful in disease diagnosis and prognosis. Most approaches to computational analysis of gene expression data attempt functionally significant classification of genes in an unsupervised fashion. We developed a Fuzzy Neural Network model to analyze gene expression profiling data from microarray. In the case of diffuse Large B-cell Lymphoma (DLBCL), this model identified four genes that could be used to predict prognosis with 93% accuracy. In the case of breast cancer, the model consisting of 5 genes was able to predict metastases of breast cancer with 94% accuracy. To disseminate widely this software to medical doctor, we developed the customized software, GeneFIS® (Fuzzy Inference System for Gene expression analysis), which is incorporated in FNN modeling for prognostic prediction from gene expression data. In the present paper, we will introduce the performance of this packaged software.

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SY11-02 Discovery of ghrelin and its physiological significance

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In 1999, we discovered the natural ligand for the orphan G-protein coupled receptor, growth hormone secretagogue receptor (GHS-R). The peptide was consisted of 28 amino acids, in which the Ser-3 residue was n-octanoylated. Surprisingly, this n-octanoyl modification at Ser-3 was essential to the activity. We named the GH-releasing peptide "ghrelin". Ghrelin induced GH release in a dose-dependent manner both in rats and humans. Ghrelin immunoreactive cells in the stomach were found in X/A-like cells, an endocrine cell in stomach. In rat brain, ghrelin immunoreactive neurons were found to be localized in the hypothalamic arcuate nucleus, a region where regulates GH release and food intake. In fact, ghrelin induced potent appetite-stimulating effects in rats. In human studies, IV injection of ghrelin elicited a potent GH release and had beneficial hemodynamic effects. Thus, the occurrence of ghrelin in both stomach and hypothalamus will give a new dimension to the regulation of GH release and feeding. Ghrelin also have multifaceted roles in the cardiovascular system and metabolism.

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SY11-03 Therapeutic approach for spinal and bulbar muscular atrophy based on pathogenesis

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Spinal and bulbar muscular atrophy(SBMA) is a late-onset motor neuron disease caused by the expansion of a polyglutamine(polyQ) tract in androgen receptor(AR). SBMA exclusively affects males, while females are usually asymptomatic. Pathogenic AR accumulates in the nucleus, leading to transcriptonal dysregulation. The ligand, testosterone, accelerates nuclear translocation of AR. Transgenic mouse model carrying full-length human AR with expanded polyQ demonstrates testosterone-dependent phenotypes, which are rescued by castration or leuprorelin, an LHRH agonist. In affected mice, neurofilament and synaptophysin accumulated at the distal motor axon due to impaired retrograde axonal transport resulting from pathogenic AR-induced transcriptional dysregulation of the p150 subunit of dynactin. These pathological events are observed in the early stage of the disease, but reversibly improved by castration. We have been carrying out a clinical trial of leuprorelin for SBMA patients, as a randomized, double-blind, placebo-controlled study

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SY11-04 Apoptosis based remedy for amyotrophic lateral sclerosis (ALS) and upper motor neuron dominant ALS

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Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disease that manifests as selective upper and lower motor neuron degeneration, whose molecular base is not delineated yet, but apoptosis as major causative is implicated. A number of anti-apoptotic proteins, such as Bcl-2, neuronal apoptosis inhibitory protein (NAIP) and XIAP, have been found to be up-regulated in neurons under degeneration. Among these anti-apoptosis proteins, NAIP reveals an anti-apoptosis unique to oxidative stress. Ectopic- and over-expression of NAIP have shown to attenuate ischemic neuron damage, adequately rescue motoneurons in peripheral nerve axotomy, preserve nigrostriatal dopaminergic function in an intrastriatal 6-OHDA Parkinson's disease rat model, and save motor neuron through intracellular signaling of glial cell-derived neurotrophic factor. These findings favor to that NAIP plays a key role in conferring resistance to several types of apoptotic insults, and up-regulation of endogenous NAIP mitigates neurodegeneration. Thus, NAIP based exploitation of the small compounds provides new clue to delineate a molecular base and new remedy to confer for neuronal cell death in ALS and UMND-ALS.

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SY11-05 Line-up of behavioral methods in development of CNS drugs

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Several model animals of neuropsychological disorders such as Alzheimer, Parkinson, schizophrenia, depression and anxiety have been developed by using drug-induced and gene targeted mice. Especially, various gene targeted mice such as knockout and/or transgenic mice are developed by using molecular biological techniques. The knockout and transgenic mice are a powerful tool with which to investigate the causal relationship between molecular and phenotype aspects of disease. The developed model animals show various abnormalities such as behavioral, biochemical and electrophysiological dysfunctions. Behavioral studies are very important to evaluate symptoms in model animals and therapeutic effects of new CNS drugs for neuropsychological disorders. I will here report and discuss the line-up of behavioral methods in development of CNS drugs.

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SY12-01 Contextual modulation in the primary visual cortex

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Contextual response modulation is the property that stimulus presented at the receptive field surround (SRF) modulates responses to the receptive field (CRF) stimulation depending on the relationship of the stimulus parameters between CRF and SRF. This modulation is generally suppressive, and suggested to be a neuronal correlate of the perceptual figure-ground segregation. Because the modulation is most often suppressive, it has been attributed to the intracortical inhibition. In our results, however, a blockade of the intracortical inhibition did not change the strength of surround suppression, and excitatory and inhibitory response tunings to the stimulus size exhibited profiles quite similar to each other. Also, LGN neurons showed a surround suppression which is comparable to that in V1 in terms of strength and properties. These results suggest that a reduction of thalamocortical inputs, which drive the cortical excitation and inhibition, and a subsequent decrease in the cortical excitatory interactions underlie the contextual modulation.

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SY12-02 Organizational rules for the specificity of neuronal connections in visual cortex

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Visual cortex is composed of a complex network of connections that can be categorized as feedforward, recurrent, feedback, excitatory and inhibitory. But we lack a clear understanding of the basic features of each type of connection. We tested the extent of common input to pairs of layer 2/3 (L2/3) pyramidal cells in rat visual cortex based on cross-correlation analysis of photostimulation-evoked synaptic currents. For most neuron pairs there was very little common feedforward (from L4) or recurrent (within L2/3) excitatory input. However, when the L2/3 pyramids were connected they shared extensive common feedforward and recurrent excitatory inputs. These connections selectively target interconnected pyramidal cells. In contrast, L2/3 cells shared common inhibitory and feedback (from L5) excitatory input regardless of whether they were interconnected. Thus, the organizational rules governing the specificity of connections are fundamentally different for feedforward and recurrent excitatory circuits versus inhibitory and feedback excitatory circuits.

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SY12-03 Additional receptive field properties of neurons in the early visual cortex

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As a first approximation, receptive fields of typical simple cells in area 17 may be modeled as a spatial Gabor filter with temporal dynamics. Complex cells, in turn, may be modeled by a hierarchically combination of multiple simple cell input. However, there are additional properties of neurons in areas 17 and 18 of the cat that are not predictable from these models. In this talk, I will present an overview of these additional properties of neurons in the early visual cortical areas, and attempt to provide a cohesive view on the type of information represented in these areas. Among these properties, dynamics of spatial frequency tuning and responses to texture or envelope stimuli are of substantial interest. Optimal spatial frequency of neurons tend to increase during the course of response to briefly flashed stimuli. These cortical areas are also known to contain cells that respond to stimuli with texture borders as well as to luminance-defined borders. Binocular and monocular properties of these responses and their implications will be discussed.

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SY12-04 Bridging gaps at V1: neural responses for filling-in and completion

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There is no retinal input at the blind spot (BS), However, we do not perceive a hole in the visual field. Instead, the same color, contour or texture as the stimuli surrounding the BS is perceived in it. This is called perceptual filling-in or completion at the BS. Filling-in and completion occurs not only at the BS but also in the retinal scotoma and in various phenomena in the normal visual field. This suggests that there are some mechanisms in our visual system that could interpolate incomplete retinal signals to form contiguous surface and contour. To elucidate the role of V1 in filling-in and completion, we analyzed neural responses at the retinotopic representation of the BS in V1. We addressed two questions: (1) Do V1 neurons respond to stimuli inducing filling-in or completion even though there is no direct retinal input to this region ? (2) Do the responses of V1 neurons to such stimuli correlate with perception ? Our results suggest that V1 plays an important role in the occurrence of filling-in and completion at the BS.

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SY12-05 Seeing higher cortical functions in early visual systems: Shape representation in area V2.

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Fundamental to our understanding of the neuronal basis of object recognition is the idea that attribute-specific and location-specific information is gradually integrated into a single object representation along the hierarchy of the visual pathways. Because of high dimension and extremely wide variety of shapes, object recognition is thought be mainly achieved within higher stages of the ventral visual pathway such as the inferotemporal cortex. Recently accumulating results, however, demonstrate that response selectivity is far more complicated than simple line detection even in the early stages. Thus, studying shape representation in the early stages provides us an opportunity to reveal neuronal mechanisms underlying object recognition. Here, I discuss the case of angle representation in area V2 of macaque monkeys, in which the angle selectivity was measured by using a set of angle stimuli formed by combining two half lines systematically.

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SY13-01 Dynamic regulation of microtubules and cytoplasmic dynein in migrating neurons.

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Regulation of cytoplasmic dynein and microtubule dynamics is crucial for both mitosis and neuronal migration. NUDEL was identified as an interacting protein with LIS1, the protein product of a gene mutated in the neuronal migration defect lissencephaly. NUDEL and LIS1 together regulate cytoplasmic dynein (CDHC). In addition, we have identified katanin p60 as a target of NUDEL. NUDEL is specifically phosphorylated at centrosomes during mitotic prophase and in migrating neurons. Katanin p60 had much higher affinity for phosphorylated NUDEL (P-NUDEL) than unphosphorylated NUDEL (NUDEL), suggesting that P-NUDEL regulates the distribution of katanin p60. Embryonic lethality and abnormal accumulation of p60 in nucleus of Nudel null mutants support an essential role of NUDEL in p60 regulation. Furthermore, Lis1-Nudel double heterozygotes display more severe neuronal migration defects than Lis1 heterozygotes. Our results indicate that coordinated regulation of cytoplasmic dynein and katanin p60 by LIS1 and NUDEL is essential for mitotic cell division and neuronal migration.

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SY13-02 Regulation of cell polarity and shape by Filamin A in the subventricular and intermediate zones during radial migration

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In the developing neocortex, radial migration from the ventricular zone through the subventricular and intermediate zones to the cortical plate is essential for the proper formation of its precise architecture. Quite recently neurons in the subventricular and intermediate zones has attracted much attention, since fundamental phenomena, such as cell division and differentiation, happen there. Also, it has been reported some migrating neurons exhibit specific cell shape there. Despite such importance, molecular mechanisms underlying the regulation of such cell shape and migration have not been elucidated. We then hypothesized the involvement of Filamin A, a well-known actin-binding protein, and investigated this issue by suppressing and/or enhancing Filamin A function. We observed that Filamin A regulates the polarity and motility of neocortical neurons in the subventricular and intermediate zones during radial migration.

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SY13-03 Dynamics of cerebral cortical development

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Cortical projection neurons are born in the ventricular zone (VZ) and migrate to beneath the marginal zone (MZ), where they form the cortical plate (CP). Since the most of the neurons invariably stop migrating just beneath the MZ and begin to mature, the late-born neurons migrate past the early-born neurons and come to be aligned in the superficial layers, resulting in the "inside-out" progression of CP development. Despite the remarkable recent progress in identification of new molecules that function in the mechanism of neuronal layering, the biological aspects of cortical layer formation are not well understood. In this talk, we will visualize the neuronal migration and show how the three modes of migration, locomotion, somal translocation, and multipolar migration are used during corticogenesis. In addition, we will introduce our recent effort to address whether the neurons need to migrate from the VZ to beneath the MZ to segregate into layers.

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SY13-04 CORTICAL INTERNEURON MIGRATION

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Cortical interneurons arise in the medial ganglionic eminence (MGE) of the ventral telencephalon. These neurons use tangential migratory paths, mainly within the IZ and MZ, to reach the cortex. Their expression of the transcription factor Lhx6 has suggested a role for it in the neurochemical identity and migration of cortical interneurons. We performed loss of function studies in mouse brain slices and dissociated MGE cell cultures using Lhx6-targeted siRNA. We found that silencing Lhx6 impeded the migration of interneurons into the cortex, although it did not obstruct their dispersion within the ganglionic eminence. Blocking Lhx6 expression in dissociated MGE cultures did not interfere with the production of GABA. These results indicate that Lhx6 does not specify the neurochemical identity of cortical interneurons, but regulates their migration from the MGE. We followed the migration of cortical interneurons using time-lapse imaging and observed that, upon enetering the cortex, a significant population moved towards the VZ. Upon reaching the ventricle, cells ceased moving before migrating radially towards the CP. We speculate that interneurons generated in the ventral telencephalon enter the cortical VZ to receive positional cues before migrating to their destinations in the developing cortex.

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SY13-05 The involvement of cortical hem in neocortical histogenesis through being a major source of Cajal-Retzius cells

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Reelin-expressing Cajal-Retzius cells (CR cells) in the marginal zone (MZ) help orchestrate layer formation in the neocortex and hippocampus. However, the origin of CR cells is poorly understood. We provide evidence that the Wnt3a-expressing cortical hem is a major source of CR cells. When the cortical hem was genetically labeled by generating a Wnt3aCre mouse, we observed derivates spreading from the cortical hem into the MZ of neocortex and hippocampus. They expressed REELIN and P73, both markers of CR cells, but not GABA. In embryonic slice culture, CR cells migrated tangentially from the hem into the medial neocortex. Finally, when we ablated the cortical hem genetically in living mouse embryos using the diphtheria toxin-A gene, CR cells were almost absent in the neocortical MZ, and layer formation in the neocortex appeared disordered. These findings suggest the involvement of the cortical hem in cortical histogenesis, in addition to the role in area patterning already proposed.

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SY14-01 Involvement of dopamine system in reward-based decision and learning

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Decision making is generally based on the reward value of alternatives and on motivation. To study involvement of midbrain dopamine (DA) system in this process, we recorded activity of DA neurons of macaque monkeys performing trial and error decisions for reward. DA neurons responded to reinforcers after behavioral decisions, and the responses precisely encoded reward expectation errors. This complies with teaching signal in reinforcement learning theories. DA neurons responded also to start cue of trial acted as first predictor of reward availability. The responses had properties of motivation and uncertainty rather than reward expectation, because they were largest at 50 to 80% of reward probability and decreased at higher and lower probabilities, and because the response magnitude was negatively correlated with reaction time after the start cue. These observations indicate crucial and unique involvement of DA system in reward-based decision and learning.

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SY14-02 Dopamine neurons represent context-dependent reward prediction error

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Midbrain dopamine (DA) neurons are thought to encode reward prediction error. Reward prediction can be improved if any relevant context is taken into account. We now show that monkey DA neurons can encode a context-dependent prediction error. In the first non-contextual task, a light stimulus was randomly followed by reward, with a fixed equal probability. The DA neural response was positively correlated with the number of preceding unrewarded trials and could be simulated by a conventional temporal difference (TD) model. In the second contextual task a reward-indicating light stimulus was presented with the probability that was incremented as a function of the number of preceding unrewarded trials. The DA neuronal response then was negatively correlated with this number. This history effect corresponded to the prediction error based on the conditional probability of reward and could be simulated only by implementing the relevant context into the TD model. We discuss the implication of this finding in relation to motivation and motor control.

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SY14-03 Selection and evaluation of actions in the medial prefrontal cortex

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In exploring rules that guide behaviors, we interact with the environment by selecting actions based on goals and evaluating actions based on external feedbacks. We trained monkeys either with a visually cued, asymmetrically rewarded go/no-go selection task (selection task) or with an action evaluation task by visual feedbacks (evaluation task). In the selection task, distinguishing effects of visual, motor, and reward factors on neuron activity, we found that some medial prefrontal cortex (MPFC) cells represented expectation of a particular reward condition and others represented a particular combination of action and reward condition during the cue presentation. In the evaluation task, MPFC cells selectively responded to either visual secondary rewards or punishments, and the responses to the visual rewards were prominent only when the executed action was evaluated. These results suggest that MPFC contributes to both goal-based action selection and action evaluation by secondary rewards.

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SY14-04 Prefrontal cortical mechanisms for selecting behavioral goals

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To investigate the involvement of the prefrontal cortex in selecting behavioral goals, we recorded neuronal activity from the dorso-lateral prefrontal cortex (PF) while monkeys performed a path-planning task. The monkey was required to move a cursor from a start to a final goal by controlling two manipulanda with each hand. We trained the monkey to perform this task with three different hand-cursor assignments. First, behavioral analyses showed that the animal adopted a strategy of selecting a fixed immediate point to reach the final goal. This result meant that the animal selected hand-movements appropriately to reach the fixed immediate goal, depending on three hand-cursor assignments. Next, a majority of PF neurons showed activity changes related to final or immediate goal. We also found that neuronal activity representing the final goal appeared first, followed by the representation of the immediate goal. Our data demonstrated that the PF was involved in processing information of the final goal to generate information for the immediate goal.

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SY14-05 Reward-based learning in dopamine neurons, striatum and orbitofrontal cortex

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Information about rewards is processed in a limited number of brain structures. Many forms of learning can be conceptualized as a change in the prediction of outcome. We found in formal learning paradigms that dopamine neurons detect the extent to which rewards occur differently than predicted, thus coding an 'error' in the prediction of reward. Both behavioral and neuronal learning occurs predominantly when dopamine neurons register a reward prediction error. Some neurons in the striatum and orbitofrontal cortex process information about the prediction, expectation and reception of liquid and food rewards. The different forms of reward expectation activity adapt to changes in reward expectation during the learning of new reward-predicting stimuli. The neuronal changes occur in parallel with the adaptation of behavioral reactions to the new reward contingencies. These data reveal neuronal mechanisms involved in mediating behavioral changes to modified expectations of future outcomes.

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SY15-01 Mechanism underlying the regulation of presenilin-mediated γ-secretase activity

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In the brains of patients with Alzheimer's disease (AD), the fundamental neuropathological change is the abnormal deposition of Aβ. Aβ is generated from APP through its sequential proteolytic cleavage catalyzed by β- and γ-secretases. Presenilin is a multipass transmembrane protein involved in many case of early-onset familial AD. Very interestingly, the molecules responsible for γ-secretase were found to be the PS complex, including nicastrin, PEN-2, and APH-1. Although accumulating evidence showed that the formation of the active γ-secretase requires PS, NCT, PEN-2 and APH-1, the precise roles of these components remain to be clarified. PS is also required for several intramembranous cleavages, including those of Notch, Cadherin, ErB4 and CD44. These results suggest that PS-mediated intramembranous cleavage plays a critical role in several biological functions. In this symposium, we like to present our study of how γ-secretase activity is regulated by the components in the PS complex or other modulators.

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SY15-02 Regulation of APP metabolism and function by X11L and Alcadein

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The β-amyloid protein (Aβ) precursor (APP) and Alcadein (Alc) associate with each other in brain by cytoplasmic interaction through X11L. The APP-X11L-Alc complex suppresses the metabolic cleavages of APP and Alc. In the absence of X11L, however, APP and Alc are subjected to a primary and then secondary presenilin-dependent cleavages that leads to the secretion of Aβ and β-Alc, and the liberation of APP intracellular domain (AICD) and Alc intracellular domain (AlcICD) fragments. AICD binds to FE65 and involves gene transactivation. AlcICD suppressed the FE65-dependent gene transactivation activity of AICD because AlcICD competes with AICD for binding to FE65. Thus, the metabolism of APP and Alc are coordinately regulated and reciprocally involved in regulation of gene transactivation. Any imbalance in the metabolism of the both proteins may influence the gene transactivation together with increased generation of Aβ in Alzheimer's disease.

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SY15-03 Analysis of neuron-specific phosphorylation of APP cytoplasmic domain in neural functions

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Amyloid-β protein precursor (APP) is one of causative factors of Alzheimer's disease (AD). However the physiological functions of this protein in brain are still unclear. Previously we have reported that Thr668 reside in APP cytoplasmic domain was phosphorylated in brain. This phosphorylation regulates the neurite out-growth in PC12 cells and binding to FE65. Another group reported that this phosphorylation might be linked to generation of β-amyloid (Aβ) peptide in brains of AD patients. To analyze the function of the phosphorylation of APP at Thr668, we generated knock-in mice which carried a substitution of Thr for Ala or Asp at the position of 668 amino acid residue. Employing these mice, we analyzed the effects of those substitutions with behavioral, electrophysiological and biochemical methods and would demonstrate that the importance of the phosphorylation at Thr668 residue in APP function.

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SY15-04 Biological roles of presenilin 1

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Presenilin 1 (PS1) is a transmembrane protein responsible for the development of early-onset familial Alzheimer's disease. This protein regulates intramembrane proteolysis of the amyloid precursor protein, Notch-1, ErbB4, E-cadherin and N-cadherin. To explore the biological roles of PS1, we extensively examined mice lacking PS1. The PS1-deficient mice exhibited vascular defects and cardiac anomaly, in addition to CNS defect and impaired somite segmentation. CNS neurons cultured from the PS1-mutant mice exhibited increased vulnerability to oxidative stress in calcium/NO-dependent manners. These findings suggest that PS1 plays essential roles in angiogenesis and cardiac morphogenesis, as well as neurogenesis and somitogenesis, and confers oxidative stress-resistance on neurons.

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SY15-05 γ-Secretase associated and unassociated roles of presenilin in cadherin-dependent signaling pathways

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Mutations in Presenilin1 (PS1) accounts for most cases of familial Alzheimer's disease (FAD), and PS1 is a critical component of the γ-secretase complex which catalyzes cleavage of APP producing amyloid β. PS1 mediates also cleavage of cadherins, Ephrin B2 receptor and Ephrin B2 ligand, all single transmembrane proteins. The produced cytoplasmic fragment of N-Cadherin promotes proteosomal degradation of CBP, thus inhibiting CREB transcriptional activity. PS1 has also a γ-secretase independent function in cadherin/PI3K cell survival signaling pathway under conditions of cell-cell adhesion. PS1 forms complexes with the p85 subunit of PI3K and promotes cadherin/PI3K association. Elimination of PS1 suppresses Akt and GSK3 phosphorylation leading to tau overphosphorylation and apoptosis. PS1 FAD mutations inhibit the PS1-dependent PI3K/Akt activation thus promoting GSK3 activity and tau overphosphorylation. FAD mutations may promote AD pathology by inhibiting the PI3K/Akt signaling pathway.

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SY16-01 The involvment of microglia and ATP receptors in neuropathic pain

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Two weeks after L5 spinal nerve injury, rats displayed a marked mechanical allodynia. In the rats, activated microglia were detected in the injury side of the dorsal horn where the level of the phosphorylated p38MAPK (phospho-p38MAPK) was increased. We found that phospho-p38MAPK-positive cells were microglia. Moreover, intraspinal administration of p38MAPK inhibitor, SB203580, suppressed the allodynia. We also found that the expression level of P2X4 was increased strikingly in spinal cord microgila after nerve injury and that pharmacological blockade of P2X4 reversed the allodynia. Intraspinal administration of P2X4 antisense oligodeoxynucleotide reduced the expression of P2X4 and suppressed tactile allodynia. Taken together our results demonstrate that activation of P2X4 or p38 MAPK in spinal cord microglia is necessary for tactile allodynia following nerve injury.

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SY16-02 Lysophosphatidic acid as the initiator of neuropathic pain

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Peripheral nerve injury in human can lead to a pain state referred to as neuropathic pain, with symptoms that include continuous burning pain, and abnormal sensory sensations such as allodynia and hyperalgesia. Lysophosphatidic acid (LPA) is bioactive lipid that is released after tissue injury. Intrathecal injection of LPA in mice mimics the sciatic nerve injury-induced behavioral, morphological and biochemical changes, which are representative features for neuropathic pain. Partial sciatic nerve injury in mice caused robust mechanical allodynia and thermal hyperalgesia paralleled by demyelination in the dorsal root and increased expression of both PKCg within the spinal cord and Caa2d1 in the DRG. The mice lacking a LPA1 receptor that activates the Rho-ROCK pathway did not develop all of these signs. Furthermore, inhibitors of Rho and ROCK also prevented these signs of neuropathic pain. These results implicate a critical role for receptor-mediated LPA signaling through Rho-ROCK pathway in neuropathic pain.

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SY16-03 Changes in serine protease and cell adhesion molecule in nociceptive pathway as novel pathomechanisms of neuropathic pain

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Pathomechanism of neuropathic pain has been thought to include synaptic reorganization associated with morphological changes. First, we found the tissue type plasminogen activator (tPA) in the rat dorsal root ganglion (DRG) was induced following sciatic nerve transection and transported to the dorsal horn. The tPA has a role in increasing the proteolytic activity and excitability of neurons in dorsal horn after peripheral nerve injury. Second, we revealed that cell adhesion molecule L1-CAM was increased in cell membrane of injured DRG neurons somata and in presynaptic button of ipsilateral dorsal horn. Administration of neutralization antibody for L1-CAM significantly improved the pain behavior in neuropathic pain model rats. These results indicate that the tPA-mediated hypersensitivity in dorsal horn neurons and also the post-transcriptional regulation of L1-CAM may be involved in the onset and maintenance of neuropathic pain

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SY16-04 Change in the emotionality under a chronic pain state: Focused on the synaptic plasticity related to the ascending pain processing

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The present study was designed to investigate whether a chronic pain state induced by nerve injury or peripheral inflammation could cause a long-lasting change in the CNS. Here we found that rat injured by sciatic nerve ligation failed to exhibit the morphine-induced place preference and caused a significant inhibition of μ-opioid receptor (MOR)-mediated G-protein activation in the ventral tegmental area (VTA). These effects were reversed by pre-microinjection of the MOR antagonist into the VTA. It should be mentioned that nerve injury leads to the expression of anxiogenic effects in rats. Under these conditions, the G-protein activation induced by μ/δ-opioid receptor agonist was decreased in the frontal cortex. These findings support the idea that a robust increase of the released endogenous opioids in the brain under a chronic pain state, may lead to changes in opioid-related emotionality

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SY16-05 Glial control of LTP induction in superficial dorsal horn revealed by optical imaging of pre- and postsynaptic excitation

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We visualized excitation of individual projection neurons (PNs) in the superficial dorsal horn of spinal cord slices by retrograde staining with a voltage-sensitive dye, and excitation of presynaptic axons and terminals by anterograde staining from the dorsal root. After conditioning, excitation newly elicited in previously silent PNs by dorsal root stimulation, and also in presynaptic elements that had been silent. Experimental results with pharmacological agents indicated that this presynaptic facilitation was mediated by nitric oxide liberated from glial cells via the activation of glial metabotropic glutamate receptors. Such increase of spike-generating presynaptic elements provides important cue to understand functional aspects of LTP, such as the spread of receptive field in hyperalgesia where long-term potentiation (LTP) of afferent synapses plays a pivotal role. Supported by JSPS and Yazaki Foundation.

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SY16-06 In vivo patch-clamp analysis of the pain processing in the primary sensory cortex

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Although the sensory information is finally transmitted to the primary sensory cortex, the sensory processing is largely unknown. To address this issue, we have developed an in vivo patch-clamp recording from the cortex that enable us to analyze synaptic responses evoked by peripheral stimulation. Differ from the spinal cord, the most neurons exhibited an oscillation at frequency of 0.5 to 5 Hz that had a clear correlation with EEG, but not with breathing or heat beat. The oscillation was eliminated when the surface of the cortex was perfused with CNQX, suggesting that it was mediated by glutamatergic inputs, presumably from thalamus. Some cortex neurons responded to the hind paw stimulation with a brush that was consisted of first EPSCs, no slow current was observed in all neurons tested, suggesting that glutamate plays a critical role in the sensory transmission.

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SY17-01 *Origin of the subventricular neuron-progenitors in the adult mouse neocortex*

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The neural stem cells are found in the subependymal layer of the adult mouse telencephalon. It is not known how the neural stem cells appear in the subependymal layer after maturation. In order to know the origin of the subventricular neural stem cells, we have investigated the characteristics of subependymal cells. We mated Emx1-Cre knockin mouse with Cre-mediated lacZ, AP double reporter mouse and examined the expression of the two reporters in the subependymal cells. They were not homogenous but were positive for either reporter. Patch-like lacZ-positive (Emx1-Cre negative cell linage) cell clusters were distributed in the subependyma. Similar patch-like GFP-positive cell clusters were found in the subependymal layer of the GAD67-GFP knockin mouse brain. They were negative for GFAP but positive for GAD67, GABA, nestin, and often somatostatin. We conclude that subependymal cells of the mouse telencephalon are heterogeneous. Some of them would originate in the subpallium and are in GABAergic neuron lineage.

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SY17-02 Mechanisms for long-distance directional migration of neuroblasts in the adult mouse subventricular zone

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Neuroblasts continue to be born in the subventricular zone (SVZ) of the lateral ventricles of adult mice. Newborn cells migrate through a complex network of chains within the SVZ and the rostral migratory stream (RMS) into the olfactory bulb (OB). In the olfactory bulb, the new neurons differentiate into granule and periglomerular neurons. Previous studies identified some candidate molecules that might be involved in directional chain migration of neuroblasts. However, the regulatory mechanism of the directional neuroblast migration in SVZ and RMS is largely unknown. We found that a repulsive activity secreted from choroid plexus into the ventricular cerebrospinal fluid plays an important role in directional control of neuronal migration in the adult mouse SVZ and RMS. Based on these results, a novel model of cell migration in the adult mammalian brain will be proposed.

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SY17-03 Sensory experience-dependent survival of newly-generated granule cells in the olfactory bulb of adult mice

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Newly-generated cells in the adult subvenricular zone migrate rostrally and become interneurons in the olfactory bulb (OB). Some of new granule cells are integrated into neuronal circuit and others eliminated. Effect of olfactory input on the granule cell survival was examined by unilateral odor deprivation. Number of BrdU-labeled granule cells decreased in the odor-deprived OB compared to the undeprived OB. Odor deprivation from day 14 to 28 after BrdU labeling effectively decreased the number of labeled cells, while deprivation before or after this period gave little effect. Diazepam administration could reproduce these effects. Odor deprivation and diazepam enhanced the apoptosis of granule cells. Detection for the activated caspase and BrdU indicated that granule cells at the age of day 14-20 were most susceptive to cell death. These results indicate the presence of a critical period when new granule cells determine the survival or death in an odor-dependent and activity-dependent manner.

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SY17-04 The early steps in adult neurogenesis

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Adult neurogenesis in the dentate gyrus of hippocampus has been proved in rodents, non-human primates, and human; however, the origin of new neurons and its differentiation process has remained to be clarified. We developed a GFP-guided detection for progenitor cells in a fresh hippocampal slice by means of nestin-GFP mice and identified the two steps of cell-differentiation process: from early radial-glia like progenitors to late progenitors with neuronal excitability (S. Fukuda et al., (2003) J. Neurosci. 23:9357). Recent studies have suggested that activity-dependent control of adult neurogenesis would occur during the early steps in adult neurogenesis. Recently, we found that late progenitor cells receive excitatory neural inputs from hippocampal interneurons through their GABAA-R. A systemic injections of a GABAA-R agonist, phenobarbital, promoted the rate of neuronal differentiation, and subsequently increased the number of new neurons in phenobarbital-treated animals. These results clearly demonstrate the control of adult neurogenesis by GABAergic system in the early steps in adult neurogenesis.

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SY17-05 Cell-cell interaction in hippocampal adult neurogenesis

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Neurogenesis continues into adulthood in the dentate granule cell layer. We have studied the early developmental events of the adult neurogenesis. To detect proliferating cells and trace the fate of them we used bromodeoxyuridine or enhanced green fluorescent protein-retroviral labeling. Neuronal precursor cells appeared to divide at least 3-4 times and formed clusters in the subgranular zone. Some clusters contained immature neurons expressing Hu or polysialic acid. Thereafter a part of newly generated cells migrated from the clusters. A majority of the migrating cells have tangentially oriented long processes that frequently made contact with the clusters. However some newly generated cells remained within the cluster at least for 1 week. We assume that the cell to cell interaction among newly generated cells at different stages of development, such as the clustering and the contact by the tangentially oriented long processes contributes to the proliferation and neuronal differentiation of the precursor cells.

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SY17-06 Neurogenesis in a model of Parkinson's disease

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Neurogenesis is increased in certain acute neurological disorder, such as ischemia and epilepsy, but the effect of more chronic neurodegeneration such as Parkinson's disease is uncertain. To identify how neurogenesis is affected in Parkinson's disease, we investigated the population dynamics of newly formed neurons of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) treated mouse as a Parkinson's diseased model using an improved retrovirus vector and BrdU treatment. We found increased progenitor cell proliferation in the striatum and the olfactoly bulb after MPTP application. Stimulating neurogenesis may provide a new treatment strategy in Parkinson's disease.

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SY18-01 Origins and subsequent evolution of lateralization

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A standard pattern of lateralisation occurs from fish to mammals, originating perhaps in early chordates. The right eye system (RES) mediates visual control of response (VCR: originally guiding mouth). The left eye system (LES) assesses novelty and spatial layout ('examination'), and is important in intense emotion. However, there are also cases of apparent absence of a consistent pattern across individuals. This is best explained by varying selection across a range of behavioural phenotypes, which shifts in direction with changes in the environment. There is evidence for selection of this sort in wild populations of fish. Such phenotypes are produced by reversal of some, but not all mechanisms, rather than by complete reversal. In the zebrafish, reversal of dorsal diencephalon results in shift of VCR to the LES. Competition with examination (also requiring LES) results. This abbreviates examination of a potential danger from a refuge and allows persistence in emergence. The amygdala is lateralised for the facilitation of fear and attack in lizard and chick. If this were also open to reversal, it would explain other variations between individuals

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SY18-02 ADVANTAGES AND DISADVANTAGES OF LATERALIZATION, AND ITS DEVELOPMENT

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Exposure of chick embryos to light shortly prior to hatching leads to the development of asymmetry in the organization of the visual projections, and to lateralization of both attack behaviour and the ability to find grains of food scattered on a background of pebbles. The sensitivity of the embryos to this effect of light is modulated by the steroid hormones. This paper will report further on the interaction between genetic, hormonal and experiential (light) influences on the development of lateralization and discuss ways in which lateralization might be modified by parental behaviour and other social interactions. The ubiquity of lateralization in vertebrate species suggests that it is advantageous. This paper will also report recent evidence showing that having a lateralized brain enhances the individual's ability to carry out two tasks simultaneously, each demanding use of opposite hemispheres (foraging, using the left hemisphere, plus monitoring for predators, using the right hemisphere).

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SY18-03 Left-right asymmetry of habenular projection in zebrafish brain

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We show the evidence of lateralized circuit in the habenulo-interpeduncular (IPN) tract that is the pathway conserved phylogenetically. Neurotracing unraveled that left habenula was mainly connected with the dorsal IPN, while right habenula with the ventral IPN. The results indicated that the asymmetric projection reflected the left-right difference in the size ratio of the two subnuclei of habenulae each of which projects either to the dorsal or ventral IPN. The finding reduces the asymmetric axonal projection to the problem of the topographic projection from different subgroups of neurons. By using the lefty1: GFP transgenic zebrafish, we revealed that the laterality of habenular projection was determined by asymmetric expression pattern of Nodal signals. Our results have uncovered the asymmetry of neural circuits embedded in the vertebrate CNS and the mechanisms underlying this.

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SY18-04 Role of midbrain cholinergic and dopaminergic systems in social modulation of forebrain activity

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Male zebra finches sing a similar song to females during courtship, and while alone. Singing-related activity and gene expression in several forebrain / basal ganglia regions is strongly dependent on the social context in which a bird sings. Here, I will present recent data suggesting that midbrain dopaminergic and cholinergic systems could be a source of this modulation. Single-unit activity of 2 types of neurons in the dopaminergic nucleus VTA is specifically modulated only during courtship singing. In the medial habenula and interpeduncular nuclei, expression of the immediate early gene egr-1 is markedly increased only after courtship singing. Given the past studies of brain lateralization in songbirds, we are examining behavioral and anatomical lateralization of courtship singing.

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SY18-05 Development of asymmetric thalamofugal visual pathway in chick

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We are interested in development of social behavior like social hierarchy and grouping (imprinting to mother, reciprocal contact etc.). These behaviors may emerge from the interaction among animals that would couple visual cues with sensory-motor reactions. To understand thus embodied dynamics, we need to know neuronal base. Chick visual pathway consists of tectofugal and thalamofugal pathway. The latter develops asymmetrically by light just before hatch. Anatomical, electrophysiological, and behavioral studies have suggested an important role of the pathway, not simply on vision itself, but as part of integration system in multimodal sensory-motor reaction. It is uncertain, however, how and what extent it contributes to visually guided behavior. Thus, we have anatomically characterized the development of the pathway by retrograde tracers and expression pattern of molecular markers. We found several regions symmetrically and asymmetrically connected to distinct Wulst regions and will discuss functional relevance of the regions based on neuroimaging.

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SY18-06 Molecular basis for the structural and functional asymmetry of the brain: A study on mouse hippocampus

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The asymmetrical hemispheric specialization is a fundamental concept of brain science and has been described mainly in relation to higher-order functions of the brain. Recently we have found that synaptic distribution of NMDA receptor GluRε2 (NR2B) subunits in the adult mouse hippocampus is asymmetrical between the left and right, and between the apical and basal dendrites of single neurons. These asymmetrical allocations of ε2 subunits differentiate the properties of NMDA receptors and synaptic plasticity between the left and right hippocampus. This indicates that left-right asymmetry is found not only in higher-order functions of the brain but also in basic structures and functions of brain neuronal networks. Our findings also suggest that brain can involve asymmetries at each level of its structural hierarchy, namely neuron, synapse, neuronal network and left-right hemispheres of the brain.

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SY18-07 Functional asymmetry in the inferior frontal cortex for verbal and nonverbal information processing.

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With fMRI, we measured brain activity while right-handed normal volunteers performed verbal and nonverbal face tasks. All of the face stimuli were 2-s video clips. In the verbal (lip-reading) task, the subjects discriminated silent, visual lip-read stimuli. We prepared four vowel stimuli, that is, "a", "i", "u", and "o". In the nonverbal (facial expression) task, they assessed the emotion displayed by each facial expression as positive or negative. In a control task, they answered the gender of each face. The lip-reading task activated the bilateral superior temporal gyri as previously reported. The inferior frontal and middle temporal gyri in the left hemisphere were also activated. In contrast, the facial expression task activated the inferior frontal gyrus, superior temporal sulcus, and middle temporal gyrus in the right hemisphere. These data suggest that the right hemisphere predominantly processed nonverbal information whereas the left hemisphere processed verbal information. Perhaps, in the inferior frontal, there is functional asymmetry for verbal and nonverbal communication.

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SY20-01 Cell polarity and asymmetric divisions in neurogenesis

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During neurogenesis, neural progenitor cells generate a large number of neurons of different fates at appropriate points in time. Asymmetric cell division plays essential roles in producing more differentiated precursors and in differentiating neuronal fates. An excellent model for investigating such processes is Drosophila neural progenitors called neuroblasts, which repetitively produce sibling ganglion mother cells that devide into two neurons. Recent extensive studies revealed that the neuroblast asymmetric division is regulated by two polarized intracellular signals, which are orthogonal to each other in parental epithelial cells; one is the aPKC-PAR3-PAR6 complex regulating the apical-basal polarity, and the other is the Galpha-Pins complex that orients the mitotic spindle in epithelia. We show here how these two signals operate differentially and coordinately to ensure the asymmetric division. Comparison of the neural precursor divisions between Drosophila and vertebrates gives us insights to the mechanisms controlling neurogenesis.

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SY20-02 Dynamic movement of cellular components during asymmetric cell division of neuroepithelial cells

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In development, asymmetric division of neuroepithelial cells (NEs) appears to play a key role for generating neurons, while undergoing self-renewal. Previously, we showed that Numb, a Notch antagonist, localizes in the basal cortex of mitotic NEs of avian embryos. We thus proposed that Numb would be segregated to one of the two daughter cells, thereby affecting the cell fate. Recently, we identified Transitin, an intermediate filament protein, co-localizes with Numb in the basal cortex of mitotic NEs. We also found that Transitin directly binds to Numb, providing an anchor site. Since Transitin-EGFP fusion protein also localizes in the basal cortex, we observed the localization of the fusion protein during mitosis. We found that the fusion protein initially localizes basally, but at the late metaphase, the fusion protein moves laterally. Further analyses indicated that Actin-Myosin system is required for the basal localization and the subsequent lateral shift of Transitin.

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SY20-03 Neuronal polarity and axon elongation

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Neurons are one of the most highly polarized cells, comprised of two structurally and functionally distinct parts, axon and dendrites. We previously showed that collapsin response mediator protein-2 (CRMP-2) is enriched in growing axon of cultured hippocampal neurons. The overexpression of CRMP-2 induces the formation of multiple axons, whereas truncated fragment of CRMP-2 inhibits the formation of the primary axon. Thus, CRMP-2 appears to play a crucial role in the determination of axon/dendrite fate. We have recently found that CRMP-2 interacts with tubulin dimers to promote microtubule-assembly for axon growth, and that CRMP-2 is involved in Numb-mediated polarized endocytosis of certain adhesion molecules such as L1. More recently, we have found that the Par complex, the small GTPase Rac and GSK-3beta are also involved in neuronal polarization. This time, we summarize and discuss functions of these polarity molecules in neuronal polarity and axon elongation.

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SY20-04 Extrinsic and intrinsic control of dendritic shaping

Tadashi Uemura[1,2], Yasuyuki Shima[1], Kaoru Sugimura[3], Daisuke Satoh[3], Misato Yamamoto[1], Hiroshi Kimura[3], Tadao Usui[1], Stephen Crews[4], Masatoshi Takeichi[5]
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In contrast to extensive studies on axonal outgrowth and pathfinding, molecular mechanisms that control dendritic patterning are much less understood. We and others previously showed that a seven-pass transmembrane cadherin Flamingo plays multiple roles in outgrowth of both axons and dendrites in Drosophila. We have taken an approach of RNA interference using mammalian maturing neurons and shown that a Flamingo homolog plays a pivotal role in development and/or maintenance of dendritic arbors. Besides a potentially extrinsic control through the cell surface receptor, we will also discuss a transcriptional factor that is expressed in one specific subclass of dendritic arborization neurons in Drosophila and responsible for its subclass-specific dendritic design.

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SY20-05 Intra-axonal patterning: its mechanism and implications

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An critical feature of the neuron is its long cellular processes, axon and dendrite, that cover a long distance within the nervous system. While their main function is to transmit the information on neuronal activity, long cellular processes can also carry developmental signals over a long distance, by transporting and localizing molecules to specific sub-axonal segments. We have shown that an intra-axonal localization of a receptor protein can concentrate its ligand molecule within the nervous system, thereby generating a positional cue for the guidance of other axons. Thus the fundamental positional information on the global axonal organization of the nervous system can originate from intracellular events within a neuron. We also show that the localization of molecules to intra-axonal segments can occur in isolated neurons in culture, suggesting that neurons possess an autonomous intracellular pattering ability.

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SY21-01 Rescue of knockout mice by introducing transgenes using a tissue specific promoter: mGluR1-rescue mice

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Metabotropic glutamate receptors (mGluRs) are implicated in modulation of synaptic transmission and plasticity. We showed mGluR subtype 1 null-mutant (KO) mice have characteristic cerebellar symptoms such as ataxic gait and motor discoordination. mGluR1 KO mice also showed impaired cerebellar climbing fiber synapse elimination. We introduced two different alternative splice variants of mGluR1, mGluR1a and mGluR1b, into mGluR1 KO mice using a Purknje cell specific promoter and asked whether the transgenes could be able to complement the KO phenotypes. mGluR1b lacks the long carboxyl-terminal domain specific for mGluR1a, which could interact with Homer/Vesl and other scaffold proteins. mGluR1a transgene could rescue both motor discoordination and impaired synapse elimination, whereas mGluR1b transgene could rescue motor discoordinaiton but not impaired synapse elimination. These results suggest the important role of carboxyl-terminal domain specific for mGluR1a in synapse elimination.

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SY21-02 Analysis of neural function by virus-mediated gene transfer into mutant mice brain

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Virus-mediated gene delivery is increasingly showing promise for the study of brain functions. In addition, recent identification of neuron-specific promoters allowed us to express genes specifically in neurons. Using transgenic mice that express the tetracycline-dependent transcription activator (tTA) under control of neuron specific enolase (NSE) promoter, we show that Purkinje cell-specific gene expression is achieved by targeted delivery of a lentivirus carrying a GFP driven by a tTA-responsive promoter. Injection of the lentivirus into cerebellar cortex caused an efficient expression of GFP in Purkinje cells without significant damage to the neurons. These results suggest that the virus-mediated gene expression in a NSE promoter-tTA mouse is useful to study the pathophysiology of cerebellum.

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SY21-03 Propagation of γPKC translocation in the Purkinje cell dendrites revealed by γPKC-GFP transgenic mice

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To investigate spatial and temporal profiles of the PKC activation in relation to neuronal functions, we developed brain region-specific and inducible γPKC-GFP transgenic mice, and combination of intracellular recording and GFP fluorescence imaging was applied to Purkinje cells in cerebellar slices. Gamma-PKC-GFP was uniformly distributed throughout the cell. When mGluR agonists were applied to the dendrite, GFP transiently moved to membrane regions, indicating that γ PKC translocated to its target protein. Electrical stimulation of parallel fibers also induced the translocation, however, the event propagated along the dendritic tree. Time course of the propagation was slower than electrical signal and Ca^{2+} waves. The present results suggest that in Purkinje neuron local mGluR signals could propagate through dendrites by PKC activation.

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SY21-04 Behavioral analysis of the bitransgenic mice with a region-specific promoter in combination with the tetracycline transactivator.

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Studies with genetically modified animals have sought to relate specific genes to specific forms of cognition. However, current methodology does not allow one to distinguish between a direct effect of the gene and an indirect effect on the development of the neuronal circuits. Thus, it is essential not only to restrict the expression to appropriate cell populations but also to control the timing of expression. We used a region-specific promoter in combination with the tetracycline transactivator and examined the role of PKC γ. When the bitransgenic mice with a high level of expression of PKC γ to the restricted regions were treated with a doxycycline, the expression was suppressed to wild level. Behavioral analysis revealed that doxycycline-treated mice were impaired in water maze test but not in other tests. These data indicate that PKC γ altered spatial memory formation directly and not by effects on neuronal development.

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SY22-01 Infantile autism : recent understanding on epidemiological data, basic clinical symptoms and neurobiological background

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Infantile autism is rather popular disease in many countries, now. In Japan,important epidemiological reports suggest that the incidence will be around one per 100 children. The reason of such increase is still unknown, though there are various reports discussing the possible reason of this increase. Recent results on this topic will be discussed first.
Early intensive behavioral interventions for autistic infants are really useful for improving the clinical symptoms. Evaluation of the actual effects of such interventions was useful for selecting important theory on basic clinical symptoms of autism. The basic clinical symptoms which are useful for early detection and early intervention will be discussed.
Most of the specialists for this disorder believe that brain damages in the early life of the patients might be etiological causes of this disorder. History of important neurobiological findings regarding autism and related disorders will be discussed.

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SY22-02 The relationship between dopaminergic neurons and amino acids in an animal model of autism

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Through our PET studies on infant autistic patients and amino acid analysis in their cerebrospinal fluid (CSF), we hypothesize that autistic symptoms may be caused by abnormalities in development and plasticity of mainly monoaminergic systems in CNS during pre- or postnatal stage. To extend this hypothesis, we here examined the behaviors, brain monoamines and amino acids in CSF in immature rats with neonatal dopamine (DA) depletion induced by 6-hydroxydopamine (i.c.v.). By the treatment, locomotion activities were increased, and DA level in the striatum was dramatically decreased. In this model, eleven amino acids in CSF were significantly elevated, and glutamate level in CSF was negatively correlated with DA level in the striatum. These results suggest that the dysfunction of DAergic system in early life stage may cause the abnormal behaviors not only DA depletion but also possibly through the change of amino acids in CSF in immature period.

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SY22-03 Clinical characteristics of ADHD - recent advance in the pathophysiological investigation

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ADHD is a developmental neuropsychiatric disorder characterized by impulsiveness, inattention and hyperactivity. Reduction in right prefrontal gray matter and left occipital gray and white matter, observed in MRI of both patients and their unaffected siblings suggests familial consistency. The central deficit of ADHD is postulated as deficiencies of the monoaminergic neurotransmitters and the fronto-striatal circuitries, for executive dysfunction, the fronto-dorsal striatal circuit and for altered reward processes, the fronto-ventral striatal circuits terminating in especially the nucleus accumbens. Altered midbrain dopamine (DA) system in the brain is suggested to play a major role for these abnormalities. Elevation of the dopamine transporter (DAT) density in the striatum shown in SPECT and increased DA receptor availability observed in PET in ADHD patients support originally hypofunction of the DA neuron as a cause of ADHD.

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SY22-04 Multiple evidences for the dopamine hypothesis of Attention Deficit Hyperactivity Disorder in two animal models

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A dysregulated mesocorticolimbic (MCL) dopamine (DA) system is involved in different variants of Attention Deficit Hyperactivity Disorder (ADHD). Behavioral, pharmacological, neuroanatomical and molecular biology evidence in two animal models of ADHD, the Spontaneously Hypertensive (SHR) and Naples High Excitability (NHE) rats, suggest an involvement of different branches of the MCL DA system in ADHD variants.The SHR show hypoactive limbic and cortical branches as assessed by higher basal DA tone, density of DA transporter (DAT) and DA D1 and D2 receptors and lower DA reuptake. Conversely, the NHE show hyperactive mesocortical DA branch only as assessed by hypertrophic DA neurons, higher DAT density and down-regulation of DA D1 receptors in prefrontal cortex. In both models methylphenidate reverses the basal profile. Thus, hypo or hyperactive DA neurons may lead to a dysregulated system with behavioral consequences on response inhibition, attention and control of executive functions

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SY22-05 Analysis of attention-deficit hyperactivity disorder (ADHD) model rats

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To understand the mechanisms of motor hyperactivity, we examined animal models for ADHD. Spontaneous motor activities were significantly increased in rats with neonatal 6-hydroxydopamine (6-OHDA) lesions at 4-5 weeks of age, which was attenuated by methamphetamine. Motor hyperactivity was also evident in Wig rats recently generated by Nagashima et al. (Hokkaido Univ.). However, methamphetamine accelerated activity levels in Wig rats. Both groups showed a decrease in tyrosine hydroxylase immunoreactivities and GABA transporter gene expression in the striatum and midbrain. After 6-OHDA lesions, gene expression was increased in NMDA receptor 1 and tachykinins in the striatum at 4 weeks. Gene expression profile in Wig rats was almost completely different from that in 6-OHDA-lesioned rats. These results suggest that the deficit in the development of dopaminergic neurons may reflect motor hyperactivity. Other factors additionally altered in Wig rats might cause different response to methamphetamine.

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SY22-06 Endocrine Disrupters Impair Sexual Differentiation of Higher Brain Functions

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The effects of perinatal exposure of low dose (50 microgram/L) of endocrine disrupters, bisphenol A (BPA), diethylhexyl phthalate (DEHP) and tributyltin (TBT) on sexually dimorphic behaviors in the open field test and the passive avoidance test, and the volume of the locus coeruleus (LC). In the open field test, control female rats were more active and explored more frequently than male controls. In the passive avoidance test at 7 weeks of age, control male rats learned better than females. All treated groups did not show sexual dimorphism. The sex difference in the volume of the LC (female > male) was abolished in all treated groups. The impairment of the sexual differentiation in the LC may become a useful endpoint in the assessment of the effect of endocrine disrupters on the central nervous system.

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SY22-07 Endocrine disruptors cause neurodevelopmental disorder, leading to hyperactivity in the rat.

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Attention-deficit hyperactivity disorder (ADHD) and autism are neurodevelopmental disorders, whose feature contains uncontrolled hyperacticvity. The animal model for hyperactivity was produced by Shaywitz et al. (1976) who demonstrated that rat pups treated with 6-hydroxydopamine via intracisternal administration at 5 day of age developed increased motor activity, leading to cognitive difficulties in shuttle-box learning between 2 and 4 weeks of age. These observations were strikingly similar to the clinical syndrome of minimal brain dysfunction, called ADHD at present, found in children.Following the protocol of Shaywitz, we examined behavioral and cellular effects of intracisternal administration of 20 kind of endocrine-disrupting chemicals (87 nmol) into the 5-day Wistar rats. We conclude that many endocrine disruptors affected the developing rat brain, resulting in hyperactivity, probably due to alterations of both the level of gene expression and the level of proteins involved in the dopaminergic neurones.

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SY23-01 Discrete stochastic process and neural fluctuations underlying visual consciousness

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When we observe ambiguous figures such as the Necker cube, we experience spontaneous alternations of two conscious percepts. Our psychophysical studies showed that stochastic transitions among a certain number of discrete substeps cause the conscious alternation and that this type of discrete stochastic process is general in various kinds of ambiguous perception. In our fMRI studies it was shown that common activations over frontoparietal cortical areas as well as visual areas are associated with conscious alternations in the various kinds of ambiguous perception, suggesting that nonlinear interactions among these multiple cortical areas are possible cause of the psychophysically observed discrete stochasticity. We also discuss possible relationships between fluctuations of MEG signals and stochastic dynamics of perceptual alternations. Our findings suggest that stochastic dynamics of neural fluctuations may play important roles in the flexibility of human visual perception.

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SY23-02 Property of transient synchrony related to perceptual switching

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Prolonged presentation of ambiguous figures results in perceptual switching between alternative interpretations. According to recent fMRI studies, areas related to perceptual switching are widely-distributed in the brain. Their coordination may require some form of temporal synchronization of activity. We investigated the temporal structure of synchronized activity between distant electrodes in human scalp EEG. A Necker cube was presented continuously. We assumed that there are at least two different aspects to perceptual switching. One might be a controllable, top-down, process and the other might be an uncontrollable, bottom-up, process. As different processes are assumed to be related to different neural activity, conventional event-locked averaging techniques are not appropriate to detect perceptual-switching related activity. We developed a method to detect and classify event related activity. We found that there are at least two distinct patterns of transient synchrony between frontal and parietal areas that are related to perceptual switching.

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SY23-03 Noise-induced entrainment of human brain waves: Stochastic Synchronization

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We experimentally demonstrate the stochastic synchronization (SS) in the human brain through the noise effect of the alpha-wave. In order to show a clear evidence of the SS in the central nervous systems, the measurement has been carried out under the following conditions; the periodic and noisy photic stimuli are respectively applied to both eyes of a subject separately. When the periodic stimulus is applied with a sub-threshold condition and the amplitude of noise stimulus is varied, strong entrainment occurs for certain range of the noise amplitude. Then the curve for the amplitude of the peak against the noise amplitude shows a bell-type shape, i.e. the maximum signal amplitude at an optimum noise. Thus the SS occurs on the visual processing area after the optic chiasma, that is no mixing occurs before primary visual cortex. This result suggests that the human brain may exploit internal and/or external noise for the higher order activities such as perception and cognition.

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SY23-04 Noise-induced sensitization of human brain

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The beneficial role of noise in biological systems has been extensively studied over the past decade. One of the fundamental phenomena considered in this respect is that of stochastic resonance (SR), which is generally defined as the optimization of responses of a nonlinear system to a weak input signal caused by external noise. It has recently been demonstrated that externally added noise can enhance input-output coherence within the human brain. The improved responses were observed at the level of the brain stem where the neurological noise can help trigger changes in heart rate in response to small changes in blood pressure, as well as at the level of the cortices where visual noise can enhance behavioral output and responses in the brain waves to weak visual signals. In my talk, I summarize our experimental observations on SR in the human brain and discuss about a possible mechanism for this noise-induced sensitization of human brain based on the effects of externally added noise on nonlinear dynamical networks.

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SY23-05 Why nerves are bundles of fibers ? - Thermal noise sensitivity of neuron and parallel structures of the nervous system

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It is a very naive question that why parallel structures are so common in the nervous system. This ingenuous question has not yet been answered ultimately. Further extensions are why has no high-performance single fiber evolved, and is the parallelism a consequence of multi-cellular organization ? Based on my two measurements that cricket wind receptor elicits a neuronal pulse in response to mechanical energy of the order of k_BT, thermal Brownian motion of single molecule, and that a single neuronal spike carries about 3 bit of information, I will show that the working principle of neuron is signal recovery or information extraction by summation average from heavy noise contamination. I will further discuss the thermal noise sensitivity of neuron as the constraint from the origin of life, where energies of the order of k_BT are converted into information or orderliness, i.e. negative entropy principle of information, in reference to Maxwellian daemon.

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SY24-01 Functional analysis of *Disrupted-In-Schizophenia 1*, novel candidate gene for schizophrenia

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Schizophrenia is a debilitating mental disease and is thought to involve the combined effects of multiple genetic components. *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 and affective disorders in a Scottish family. Using yeast two hybrid system, we identified interacting partners of DISC1. In PC12 cells, the interaction between DISC1 and these proteins was regulated by neurite-extending stimuli such as NGF or PACAP. In the developing rat brain *DISC1* and these interacting partners were synchronously expressed, suggesting the involvement of DISC1 in the development of the mammalian nervous system through the binding to interacting partners. Our results will help the elucidation of pathophysiological role of DISC1 and a better understanding of the general etiology of schizophrenia.

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SY24-02 Netrin-G deficient mice have deficits associated with NMDA receptor hypofunction

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Cumulative evidence suggests that neural circuit deficits associated with NMDA receptor dysfunction underlie schizophrenia pathophysiology. Netrin-G1 and netrin-G2 compose a subfamily within the UNC-6/netrin family. Here, we show that mutant mice devoid of either of these proteins have pathway-specific deficits in NMDA-receptor function. Netrin-G1 mutants have deficits in sensory motor gating and increased locomotor activity, and netrin-G2 mutants have distinct cognitive deficits. Furthermore, netrin-G1 mutants have abnormal responses to repeated administration of a non-competitive NMDA antagonist dizocilpine (MK801). The results indicate that netrin-G proteins are involved in the formation and/or maintenance of functional neuronal circuits, and netrin-G deficient mice might be useful to characterize neural circuits underlying schizophrenia-related behaviors.

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SY24-03 Molecular mechanisms of cognitive dysfunction in schizophrenic animal models: approaches from glutamatergic systems

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We demonstrated here that the phencyclidine (PCP: 10 mg/kg/day s.c for 14 days)-treated and N-methyl-D-aspartate (NMDA) receptor ε1 subunit (NR2A)-knockout mice showed the impairment of associated learning in a Pavlovian fear conditioning and of latent learning in a water finding test. The activations of Ca^{2+}/calmodulin kinase II and extracellular signaling-regulated kinase were not observed after training in both tests in PCP-treated and NR2A-knockout mice, compared to saline-treated mice. Atypical antipsychotics, but not typical antipsychotics, attenuated cognitive dysfunction in PCP-treated mice. These results suggest that the PCP-treated and/or NR2A-knockout mice are useful models for studying cognitive deficits of schizophrenia and their treatments.

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SY24-04 Cognitive dysfunction of the forebrain specific calcineurin knockout mice: Relation to schizophrenia pathogenesis

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Calcineurin (=CN) is a calcium/calmodulin-dependent serine/threonine protein phosphatase. To characterize the behavioral significance of CN, we subjected forebrain specific CN knockout mice to a comprehensive behavioral test battery. The mutant mice showed severe working memory deficit, increased locomotor activity, decreased social interaction, and impairments in prepulse inhibition and latent inhibition. The abnormalities of CN mutant mice were strikingly similar to those described for schizophrenic patients (Zeng et al, 2001; Miyakawa et al., 2003). Consistent with these findings, transmission disequilibrium studies in a large sample of affected families detected significant association of the PPP3CC gene, which encodes the calcineurin gamma catalytic subunit, with the disease (Gerber at al., 2003). The potential impact of our findings on the studies of schizophrenia and its related mental disorders will be discussed.

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SY24-05 Perinatal Neurotrophic Perturbation and Later Cognitive Deficits; Implication in schizophrenia

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EGF, BDNF and IL-1/2/6 regulate DA synthesis and receptor expression. Their expressions are impaired in the brain and blood from schizophrenic patients. To test their contribution to cognitive development, we subcutaneously administered various cytokines to rat pups to transiently perturb their signaling. During and after development, a variety of cognitive/behavioral abnormalities emerged, depending on age as well as on the cytokine species. Abnormal sensorimotor gating was apparent in EGF-, LIF- and IL-1-treated rats at adult stages. EGF- and IL-1-treated rats exhibited severe cognitive abnormality in latent inhibition and working memory task. There was persistent abnormality in dopamine metabolism or signaling in the frontal cortex of these animals. These results indicate that abnormal neurotrophic signals given to the immature dopaminergic system produce persistent cognitive dysfunctions in adulthood.

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SY25-01 The risk for Alzheimer's disease is related to a cluster of genes involved in brain cholesterol metabolism.

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Cholesterol is related to pathological hallmarks of AD and genes related to cholesterol homeostasis have been reported to be associated with AD risk.Objective: We investigated the simultaneous impact of cholesterol-related polymorphisms on the risk for developing AD and controlled for genetic heterogeneity of the study population.Results: The APOE epsilon 4 haplotype accounted for 75% of the variability of the risk associated with this genetic set. The majority of the remaining genes also contributed significantly to AD risk (APOE > > > SOAT1 > APOE promoter > OLR1 > CYP46 > LPL > CH25H > APOA4). Only LDLR, LRP1 and PPARA failed to exert significant risk. Receiver operating characteristics (ROC) analysis revealed that this cholesterol gene-cluster reached a diagnostic accuracy of 0.75. Conclusion: We conclude that a cluster of cholesterol-related genes is strongly associated with AD risk.

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SY25-02 Glial lipoproteins promote axon growth of CNS neurons

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We investigated the role of lipoproteins (LP) secreted by glial cells in the regeneration and growth of axons in compartmented cultures. Addition of glial cell-conditioned medium (GCM) to distal axons increased the rate of axon extension by ~50%. Inhibition of cholesterol synthesis in glial cells reduced the secretion of cholesterol and apolipoprotein E (apoE), and prevented the growth stimulatory effect of GCM. Glial cell-derived LP containing cholesterol and apoE were provided to distal axons and stimulated axon extension to the same extent as GCM. In contrast, provision of the LP to cell bodies did not enhance axon extension. Addition of receptor-associated protein abrogated the growth stimulatory effect indicating that the stimulation was mediated by low density lipoprotein receptor family member(s). These observations suggest that glial cells stimulate axon growth of CNS neurons by providing LP containing cholesterol and apoE to distal axons.

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SY25-03 Cholesterol homeostasis and function in CNS neurons

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Proper regulation of the cholesterol content is critical for the development of neurons, their survival and functional integrity. Accordingly, defects in cholesterol-handling components cause neurodegenerative diseases including Niemann-Pick disease type C or Alzheimer's disease. So far, surprisingly little is known about cholesterol homeostasis in the central nervous system. Our observation that glia-derived cholesterol promotes synapse formation in cultures of CNS neurons (Mauch et al., 2001) provoked the hypothesis that neurons depend in an obligatory manner on astrocyte-derived cholesterol (Pfrieger, 2003). In my talk, I will briefly summarize the findings of Mauch et al. and its implications and provide an overview of our ongoing research. Currently, we focus on three questions. First, how do glial factors including cholesterol promote synaptogenesis? Second, how do neurons regulate their cholesterol level and are their regional differences in cholesterol homeostasis ? Third, is there a cholesterol exchange between astrocytes and neurons in vivo ?

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SY25-04 Roles of apoE and it's receptors in the central nervous system

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In the CNS, apoE plays important roles in neurite outgrowth, synaptic maintenance and neuronal repair. The interaction of apoE with lipids forms lipoprotein particles, which are subsequently recognized and catabolized by lipoprotein receptors. Recently, the analysis of mice lacking both of VLDLR and ApoER2 revealed that VLDLR and ApoER2 function as receptors for reelin, which is involved in the signaling pathway that control neuronal cell positioning. The binding of reelin to the receptors is inhibited by apoE in vitro. To further define the function of apoE in the CNS, we generated the triple KO mice lacking apoE, VLDLR and ApoER2. An additional defect of apoE in VLDLR/ApoER2 double KO mice could not improve the abnormalities of neuronal migration, rather made worse. Although 20% of the double KO mice survived more than 3 months, all of the triple KO mice died by three weeks after the birth. The triple KO mice also showed elevated serum cholesterol levels and fatty liver, but no change of cholesterol contents in the brain.

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SY25-05 ApoE-isoform-specific cholesterol transport in the central nervous system

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Studies on the metabolism of cholesterol have been performed for decades; however, the knowledge on cholesterol metabolism in the central nervous system (CNS) has been limited. Since cholesterol transport in the CNS is segregated from the systemic circulation by the blood-brain barrier, one should obtain a better understanding of cholesterol metabolism and its significant roles in the CNS before discussing the relationship between cholesterol and neurodegenerative diseases. Another important point is that the morphological characteristics of neurons are markedly different from those of other cell types, which may allow cholesterol to play a specific and unique role in neurons. In this symposium, taking these neuron- and CNS-specific characteristics into account, apolipoprotein E (apoE)-isoform-specific cholesterol transport in CNS and putative role of cholesterol in the pathogenesis of Alzheimer's disease and Niemann-Pick disease type C is discussed.

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SY26-01 Activin, a TGF-β superfamily protein, regulates maintenance of hippocampal LTP through modulating the dendritic spine morphology

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Long-lasting modifications of synaptic transmission depend on *de novo* gene expression in neurons. The expression of activin is up-regulated during LTP. Here we show that inhibition of activin function blocked the late LTP maintenance (L-LTP) without affecting the induction and early LTP (E-LTP) expression in the hippocampal dentate gyrus *in vivo*. Activin administration facilitated the LTP maintenance elicited by a weak tetanus that in the control case produced E-LTP. In cultured neuron, activin lengthened the dendritic spine via MAP kinase pathway and enhanced the contact number of presynaptic terminal per spine. Inhibition of cytoskeletal actin dynamics blocked not only the activin-induced change of spine morphology but also the activin-induced enhancement of LTP persistence. We suggest that activin plays an essential role in the shift from E-LTP to L-LTP through modulating the spine actin dynamics and resulting increase in the synaptic contact number.

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SY26-02 Differentiation of Climbing Fiber Synapses during Cerebellar Development

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Climbing fiber (CF) synapses onto cerebellar Purkinje cells (PCs) undergo dynamic changes during development. Redundant CF synapses formed at early stages are eliminated subsequently. In mice, most PCs become mono-innervated by single CFs by P21. We found that PCs were innervated by multiple CFs with similar strengths at P2. Then, the difference in their strengths became progressively larger. Around P7, each PC was either mono-innervated by one strong CF or multiply innervated by one strong CF plus a few relatively weak CFs. We found that these transitions involved the changes in the simultaneous release of multiple vesicles per site and the extent of CF innervation area over PC dendrites. Besides, we presented evidence suggesting that weaker CFs with low probability of multivesicular release would be withdrawn preferentially. These results suggest that the functional differentiation preceding the synapse elimination is a key step for the selection of the CFs that persist into adulthood.

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SY26-03 The role of NMDA receptor tyrosine phosphorylation in molecular and cellular mechanisms of selective synaptic potentiation in the central nervous system

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Excitatory synaptic transmission in the hippocampus is mediated by the glutamate receptors, among which the NMDA receptor plays a crucial role in synaptic plasticity such as long-term potentiation (LTP). Normal synaptic transmission is mostly mediated by AMPA receptors, while the Ca^{2+} influx through NMDA receptors in the postsynaptic cell induces LTP, which is expressed by the modification of AMPA receptors. The single channel properties of NMDA receptors, including channel openings, are regulated by tyrosine phosphorylation of NMDA receptor NR2 (GluRε) subunits, and thus, this regulation is critical for the modulation of synaptic plasticity. In this presentation, I will focus on molecular and cellular mechanisms of synaptic regulation by the NR2 tyrosine phosphorylation in the hippocampus and amygdala, and on their implication in memory formation in the whole animal.

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SY26-04 Neuroligin-associated proteins, S-SCAM and PSD-95

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Synapses have cell adhesion molecules including cadherins and neuroligin. At synapses, cell adhesion and neurotransmission are related to each other. The expression of cadherin and the interaction of cadherin with β-catenin are affected by neuronal activation. Mutations of neuroligin genes impair brain function and cause autism or mental retardation. These findings suggest that synapses have molecular links between cell adhesion and neurotransmission. In this context, we are focused on S-SCAM and PSD-95 that interact with NMDA receptors and neuroligin. They belong to a membrane-associated guanylate kinase family and their molecular structures are similar. They also share MAGUIN and GKAP/SAPAP as binding partners. Their localizations are overlapped. These similarities led us to raise a question whether S-SCAM and PSD-95 play redundant or distinct roles in the synaptic organization. I will report here how S-SCAM, PSD-95 and neuroligin interact among them and discuss the possibility that S-SCAM has a specific role in the assembly of synapses.

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SY26-05 Regulation of synaptogenesis by nectin cell adhesion molecule

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Synapses have been regarded as one of the specialized cell-cell junctions whose specificity and plasticity provide neurons with structural and functional basis for the formation of neural network. The nectin is an immunoglobulin-like adhesion molecule which has four isoforms (-1, -2, -3 and -4). We have shown that at the synapses between the mossy fiber terminals and the dendrites of pyramidal cells in the CA3 area of mouse hippocampus, nectin-1 and -3 asymmetrically localize at the presynaptic and postsynaptic sides of puncta adherentia junctions, respectively. Inhibition of the nectin-based adhesion by an inhibitor of nectin-1 in cultured rat hippocampal neurons results in marked decrease in size and concomitant increase in number of synapses. These results indicate a key role of the nectin-afadin system in formation of synapses.

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SY26-06 Characterization of AMPA receptor-containing membrane vesicles in rat brain

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The activity-dependent insertion and extrusion of AMPA receptor at the synaptic site play important roles in modulating synaptic strength in long-term potentiation as well as in long-term depression. In order to elucidate the activity-dependent AMPA receptor recycling, we isolated AMPA receptor-containing vesicles from rat brain homogenate by using antibody-conjugated magnetic beads, and their protein components were characterized by immunoblotting and by liquid chromatographic-tandem mass spectrometry (LC-MS/MS). We found that there are several different kinds of AMPA receptor-containing vesicles having distinct sets of protein components, and membrane traffic of these vesicles could be regulated by various ptotein kinases and phosphtases.

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SY27-01 ORP150, an ER-chaperone, suppresses neuronal cell death.

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Cells subject to environmental change express stress proteins, which contribute importantly to maintenance of metabolic homeostasis and viability. As a most abundant cell type in the central nervous system, astrocytes are shows a phenotype resistant to environmental alterations. Based upon this ability of astrocytes we have purified and cloned a novel stress protein, ORP150, a 150 kDa oxygen regulated protein. We have shown that ORP150 is an inducible chaperone present in endoplasmic reticulum (ER), which regulates cytosolic free calcium and activation of proteolytic pathways causing cell death in neurons subject to excitatory stress. Using mice heterozygous for ORP150 deficiency, exposure to excitatory stimuli caused hippocampal neurons to display exaggerated elevation of cytosolic calcium accompanied by an increased vulnerability to glutamate-induced cell death in vitro and decreased survival to kainate in vivo. In contrast, targeted neuronal overexpression of ORP150 suppressed each of these events, and enhanced neuronal and animal survival in parallel with diminished seizure intensity. Our data underscore a pivotal role for ER stress in glutamate toxicity, and pinpoint a key ER chaperone, ORP150, which orchestrates the protective stress response critical for neuronal survival.

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SY27-02 Vaccination therapy for Alzheimer's disease.

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Alzheimer's disease (AD) is a dementing disorder characterized by senile plaques with b amyloid deposits. Active and passive immunization of amyloid precursor protein (APP) gene transgenic mice with aggregated amyloid b protein (Ab)1-42 and adjuvants reduced the amyloid burden in the brain. Since the vaccinated mice showed improvement in cognitive functions, a clinical trial was started in US and Europe. The vaccine AN-1792 was given to patients intramuscularly and repeatedly. However, the clinical trial was halted, because about 6% of the patients developed acute meningoencephalitis as a side effect, which was thought to be induced by CD4+ Th1-type T cells reactive to Ab. Although the trial was suspended, a follow-up study of the vaccinated patients who developed antibodies to b amyloid showed milder clinical decline and an autopsy case who had had meningoencephalitis after vaccination suggested the disappearance of senile plaques. We have developed a safe vaccine using the adeno-associated virus vector carrying Ab cDNA. When this vaccine was given once in APP transgenic mice (tg2576) by an oro-gastric tube induced antibodies to b amyloid and the vaccinated mice showed a significant reduction in Ab burden in the brain. There were no infiltrated T cells in the brain as well as in other organs.

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SY27-03 Brain regions and neural circuits for fatigue and sensation of fatigue

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Fatigue is a state introduced after stressful events, and might be causative for a variety of diseases. We investigated neural substrates for fatigue and fatigue sensation by using functional neuroimaging. By activation studies with PET on normal volunteers during mental task (advanced trail making test on touch-panel screen display), Brodmann's areas (BA) 10/47 and 11 were activated in correlation with their Visual Analogue Scale of fatigue. From PET studies with the Chronic Fatigue Syndrome (CFS) patients, regional cerebral blood flow decreased significantly in several brain regions including BA24 and cerebellum, the uptake of the acetyl moiety of [2-^{11}C]acetyl-L-carnitine, a direct precursor for releasable glutamate, was reduced mostly in BA24/33 and BA9/46d, and the density of serotonin transporter decreased in the rostral subsegment of BA24. These results, together with the data from recent fMRI studies, could illustrate the hypothetic mechanisms of fatigue transmission and sensation in the brain.

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SY27-04 Stress - neurobiology of psychogenic mental disorders

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Post-traumatic stress disorders (PTSD) has been introduced in DSM on 1980, after the experience of many patients among Vietnam veterans in USA and now the concept has been widely accepted. PTSD is characterized by recurrent recall of bad memory, nightmare with insomnia and emotional withdrawal, all of which are long-lasting and very resistant to any therapeutic approach. We started a nation-wide collaborative research on PTSD and related stress disorders since 2000. Clinical surveys are targeted to the victims of Tokyo subway sarin attack in 1995, since PTSD may provide a useful model to understand brain mechanisms underlying psychogenic reactions, for long thought as the matter of psychoanalysis. In this Symposium, we will overview the current status of our basic and clinical research of stress related disorders, and then focus on the structural and functional imaging studies of PTSD due to terrorism occurred in Tokyo, some of which have already been published. Based on these results, we further are in progress to investigate the problem of nature vs. nurture in PTSD.

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SY27-05 Pathophysiology of schizophrenia; DISC1 and cerebral cortical development

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In this presentation, I will overview current genetic and biological approach towards schizophrenia. Following the brief overview, I will introduce our research on DISC1 for the pathophysiology of schizophrenia. Our recent observation indicates that loss of DISC1 functions impairs cerebral cortical development, providing evidence that a genetic susceptibility factor of schizophrenia directly links to neurodevelopmental aspects of schizophrenia.

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SY27-06 Molecular basis of bipolar disorder

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Bipolar disorder is characterized by recurrent episodes of mania and depression. Monoamine hypothesis was established based on the effects of antidepressants and antipsychotics. However, this hypothesis may not be relevant to the molecular basis of recurrence. Three mood stabilizers, lithium, valproate, and carbamazepine, are known to have prophylactic effects on bipolar disorder. Among them, lithium inhibits inositol monophosphatase, a key enzyme in the phosphoinositide pathway. In addition, altered phosphoinositide-linked calcium signaling was reported in peripheral blood cells of patients with bipolar disorder. These findings suggest that calcium signaling is altered in bipolar disorder. We have been studying the molecular pathology of bipolar disorder using neuroimaging and gene expression analysis. The results suggested the role of mitochondrial dysfunction and impaired endoplasmic reticulum (ER) stress signaling, both of which relate to calcium signaling. Accumulated data suggest that altered neuroplasticity associated with dysfunction of mitochondrial and ER-calcium signaling may be related to molecular pathophysiology of bipolar disorder.

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SY28-01 Mechanism behind Neuroprotection by Propargylamines

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Hitherto therapy for Parkinson disease (PD) is the supplement of deficient dopamine, and a novel therapy to delay/halt the progression of the neural degeneration has been required. Propargylamines, such as N-propargyl-1(R)aminoindan (rasagiline) and (-)deprenyl, have been probed to protect neurons against cell death, as shown by in vitro and in vivo experiments. The mechanism underlying the neuroprotection was studied using human neuroblastoma SH-SY5Y cells. Rasagiline, (-)deprenyl and their derivatives increased neuroprotective proteins, such as neurotrophic factors and bcl-2 family proteins. In addition, the induction was mediated by activation of transcription factor NFkappaB and MAPK. The effects of propargylamines were confirmed also in vivo using the primate model. Propargylamines are promising candidate of neuroprotective drugs applicable for PD and Alzheimer disease

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SY28-02 Hydrophobic dipeptide, Leu-Ile, protects against neuronal death by inducing BDNF and GDNF synthesis

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We have found hydrophobic dipeptides, Leu-Ile could stimulate glial cell-lined neurotrophic factor (GDNF) and brain derived neurotrophic factor (BDNF) synthesis in vivo and in vitro. Leu-Ile protected against the death of mesencephalic neurons from wild-type mice, but not from mice lacking the BDNF or GDNF gene. Peripheral administration of Leu-Ile inhibited dopaminergic denervation caused by unilateral injection of 6-hydroxydopamine into the striatum of mice. The number of rotations following a methamphetamine challenge was lower in the Leu-Ile-treated group than in the non-treated group. Leu-Ile did not suppress concanavalin A -induced secretion of interleukin-2 by cultured spleen cells, suggesting that the immunosuppressant activity of Leu-Ile may be negligible when used as a therapeutic tool for neurodegenerative diseases.

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SY28-03 Molecular mechanism underlying the neuroprotective and neurorescue activities of green tea polyphenol epigallocatechin-3-gallate

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[1]Res. Dept. Pharmacol. Faculty of Med, Technion

A number of in vivo and in vitro studies have shown that dietary antioxidant polyphenol flavonoids, possess a protective role in neurodegeneration. However, intense investigation on the mechanism of action of the major polyphenol component of green tea extract (-)-epigallocatechin-3-gallate (ECGG), has revealed the involvement of novel molecular targets. The list include a) control of calcium homeostasis; b) inhibition of the DAT and COMT activity; c)activation of Erk, PKC, antioxidant and phase II detoxifying enzymes and survival genes; d) processing of the APP to preferentially secrete the non-toxic soluble APP-alpha and inhibition of beta-amyloid fibrils formation and destabilization of pre-formed beta-amyloid fibrils. Overall, green tea extract and EGCG may constitute a source of neuroprotectant bioenergizers, with particular clinical relevance to neurodegenerative diseases such as PD and AD.

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SY28-04 Intracellular mechanisms involved in the neuroprotective effects of IGF-1and Gingko biloba in cultured hippocampal neurons

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The neuroprotective effects of IGF-1 and natural products against toxicity induced by various insults are well established. However, the underlying molecular mechanisms remain to be fully elucidated. Consistent with the role of Akt in cell survival, we have shown that IGF-1 primarily stimulates the activation of the PI3/Akt pathway in hippocampal neurons. IGF-1 promotes the survival of hippocampal neurons mostly by acting via the PI3-Akt pathway. This has been demonstrated using survival assays with pharmacological inhibitors, and transfections of various forms of Akt and its downstream targets like Foxo3a. In contrast, natural products such as the gingko biloba extract, EGb 761 and the red wine constituent, resveratrol, promote survival through their antioxidant properties or by inhibiting PKC. These findings suggest that various molecular mechanisms are involved in the survival effects of growth factors and natural products.

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SY28-05 Neuropeptide PACAP: neuromodulatory effects on the actions of classical neurotrophins

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Pituitary adenylate cyclase-activating polypeptide (PACAP), a member of VIP/secretin/glucagon family, acts as a pleiotropic neuropeptide in the nervous system. Here we focus on the mechanisms of neurotrophic and neuroprotective actions of PACAP. PACAP activates Rac1, a molecule involved in neuritegenesis, and synergizes with NGF to stimulate neurite outgrowth and PACAP gene transcription, differentially dependent on both the ERK1/2 and p38 MAP kinase pathways in PC12 cells. PACAP is up-regulated after a variety of brain injuries, such as cerebral ischemia. In an in vitro model, PACAP antagonist completely blocked glutamate-induced BDNF expression in neurons and exaggerated their death. In addition, PACAP can stimulates astrocytes to release neuroprotective factors. These results suggest that PACAP exerts neurotrophic and neuroprotective activities in part through the functional modulation of classical neurotrophins.

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SY30-01 Essential roles of heparan sulfate proteoglycans in postsynaptic development

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Heparan sulfate proteoglycans (HSPGs) are a group of proteins carrying heparan sulfate (HS) chains, which are sulfated polysaccharides consisting of alternating GlcNAc and GlcA residues. HSPGs are expressed mainly on the cell surface and in the extracellular matrix and play critical roles in growth factor/cytokine signaling, morphogen gradients, cell adhesion, and extracellular matrix assembly. In the nervous system, syndecan-2, a member of the syndecan HSPG family, is enriched in postsynapses and is involved in formation of dendritic spines, the principal targets for excitatory synapses. Spine formation is mediated by phosphorylation of syndecan-2 by EphB receptor tyrosine kinases. To determine the physiological role of the HS moiety of HSPGs, we created conditional knockout of EXT1, the gene encoding a glycosyltransferase crucial for HS synthesis. EXT1-null cortical neurons exhibit defects in the organization of postsynaptic site. Our study has revealed that HSPGs are involved in postsynaptic development through their extracellular HS chain and intracellular signaling.

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SY30-02 Glycosaminoglycans regulate neural network formation

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Glycosaminoglycans (GAGs), such as chondroitin sulfate and heparan sulfate, are richly expressed in the developing brain. GAGs as components of proteoglycans interact with many growth factors and extracellular matrix molecules, and regulate various biological processes. Recent evidences suggested that sepecific sulfations on GAGs by distinct GAG sulfotransferases lead to the formation of active domains which selectively bind with specific target proteins. To reveal the functional importance of structural diversity of GAGs in the brain development, we performed a series of immunohistochemical and in situ hybridization analyses. These investigations indicated that various developmental processes such as neuronal migration, neurite extension and neuronal maturation are associated with the expression of specific GAG structures and sulfotransferases. We will also report the importance of oversulfation in chondroitin sulfate for the regulation of growth factor signaling and neuritogenesis.

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SY30-03 Defective glycosylation in congenital muscular dystrophy

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O-Mannosylation is uncommon protein modification and is present in a limited number of glycoproteins of brain, nerve, and skeletal. One of the best known *O*-mannosyl-modified glycoproteins is α-dystroglycan, which is a central component of the dystrophin-glycoprotein complex isolated from skeletal muscle membranes. Muscle-eye-brain disease (MEB) and Walker-Warburg syndrome (WWS) are autosomal recessive disorders characterized by congenital muscular dystrophy, ocular abnormalities and lissencephaly. Both are caused by defects of *O*-mannosylation. The *POMGnT1* gene is responsible for MEB and the *POMT1* gene is for WWS. POMGnT1 is responsible for the formation of GlcNAcβ1-2Man. POMT1 has protein *O*-mannosyltransferase activity, but only when it is co-expressed with another homologue POMT2. Moreover, some muscular dystrophies are suggested to be caused by hypoglycosylation of α-dystroglycan. These findings suggest that defective glycosylation of α-dystroglycan is the common trait of muscle cell degeneration and abnormal brain structure.

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SY30-04 Molecular pathology of and development of enzyme replacement therapy for Fabry disease

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Fabry disease resulting from deficiency of alpha-galactosidase (a-Gal) causes the systemic deposition of ceramide trihexoside (CTH). We tried to clarify the molecular pathology of Fabry disease and develop an effective and economic enzyme replacement therapy for this disease. We characterized mutant enzyme proteins causing Fabry disease. C142Y mutant prevents the substrate from entering into the active site pocket, and it results in a complete deficiency of the enzyme activity. M72V mutant affects van der Waals contacts around the residue and it results in destabilization of the enzyme. Then, we produced a recombinant a-Gal having mannose 6-phosphate residues on a non-reduced end of the oligosaccharide chains by means of yeast expression system, and estimated the effect. Administrated a-Gal hydrolyzed CTH accumulated in Fabry fibroblasts and organs from Fabry mice.

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SY30-05 Metabolic Labeling of Sialic Acid in Neurons

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Metabolic "sialic acid engineering" techniques are based on the ability of certain non-natural analogs of *N*-acetylmannosamine (ManNAc) to intercept the sialic acid biosynthetic pathway and be displayed on the cell surface in the form of the corresponding non-natural sialic acids. By careful design of the analog, changes in sialic acid structure can directly modulate the behavior of the host cell; alternately, novel chemical groups can be presented on the cell surface where they function as tags for the delivery of a second agent thereby greatly increasing the repertoire of possible surface modifications. Because sialic acid is highly expressed on neurons and has been implicated in many aspects of the biology of these cells, ManNAc analog-based labeling strategies provide a novel, and potentially powerful, method to both study and manipulate nerve cells. Several specific examples will be discussed where nascent sialic acid engineering efforts, currently pursued at the cell level, hold promise in the development of "real-world" *in vivo* applications.

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SY30-06 Involvement of the HNK-1 carbohydrate in synaptic plasticity and spatial learning

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The HNK-1 carbohydrate, which originally gained notice through its expression on migrating cells during development, is associated with certain immunoglobulin superfamily cell adhesion molecules and on some glycolipids of the nervous system. To examine the physiological role of the HNK-1 carbohydrate, we generated the mice with a targeted deletion of a glucuronyltransferase, GlcAT-P, which is a key enzyme of the carbohydrate biosynthesis. GlcAT-P deficient mice reveal normal development of gross anatomical features in spite of almost complete lack of the glucuronyltransferase activity and the HNK-1 carbohydrate. However, the loss of HNK-1 carbohydrate resulted in reduced LTP at the Schaffer colatteral-CA1 synapses, and caused defects in spatial memory formation and the anxiety-related behavior. This is the first evidence demonstrating that loss of a single non-reducing terminal carbohydrate residue attenuates the synaptic plasticity in the hippocampus.

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SY31-01 Reconstruction of periaxonal environment that supports axonal regeneration by CNS glial cells after transection of the spinal cord in adult rats

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The failure of injured axons to regenerate long distances in the adult mammalian CNS leads to permanent paralysis and severe functional deficits. The failure can be ascribed not to the neurons but to their environment because they can regenerate axons if provided with an appropriate environment. The periaxonal environment that is mainly formed by CNS glial cells is destructed by tissue injury and must be reconstructed after injury. The repair process that reconstructs a new periaxonal environment appears crucial for axonal regeneration. CNS glial cells have been thought to reconstruct nonpermissive periaxonal environment although they form permissive environment during development. So, we have examined if CNS glial cells can reconstruct permissive periaxonal environment in adult rats. CNS glial cells could reconstruct the periaxonal environment similar to the glial framework seen during development, which supported axonal regeneration even in adult.

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SY31-02 A novel method for cell transplantation (neurosphere cells, bone marrow stromal cells) into damaged spinal cord via cerebrospinal fluid in rats

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We examined whether the cultured cells (neurosphere cells, bone marrow stromal cells) injected into CSF could reach the lesion and migrate into the damaged tissue of the spial cord or not. Neurospheres were cultured from the hippocampal tissues of the rat fetus. Bone marrow stromal cells were cultured from the bone marrow of the femur of the rats. Either neurosheres or bone marrow stromal cells were injected into the 4th ventricle of the rats with contusion injury of the spinal cord. Several weeks after surgery, histological evaluation was performed. In both groups, injected cells attached to the pia matter and invaded extensively into injured spinal cord tissue. It is possible that some signals released from injured tissues might be delivered to the grafted cells, making them invade into the lesion.

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SY31-03 Axon growth inhibition signals from p75NTR

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Injury to the adult CNS is devastating because of the inability of central neurons to regenerate correct axonal and dendritic connections. It is now well established that axons of the adult central nervous system are capable of only a limited amount of regrowth after injury, and that an unfavorable environment plays major roles in the lack of regeneration. Much of the axon growth inhibitory effects is associated with myelin. Identification of the myelin-derived inhibitors leads to a spurt in our knowledge about the molecular mechanisms of the biological activities. The neurotrophin receptor p75NTR transduces the signal from all of the myelin-derived inhibitors found to date. We now know that p75NTR has the ability to elicit bi-directional signals, that result in the inhibition as well as the promotion of the neurite outgrowth. The key intracellular signal for the regulation of neurite outgrowth has been shown to be small GTPase Rho. Elucidation of the signaling mechanism will prompt us to explore strategies to overcome the inhibitory signals.

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SY31-04 Stem cell therapy of injured spinal cord

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For successful functional recovery of the damaged CNS, the availability of signals to induce the appropriate differentiation of the transplanted and endogenous neural stem or progenitor cells (NSPCs) as well as the timing of the transplantation are important Because the immediately post traumatic microenvironment of the spinal cord is in an acute inflammatory stage, it is not favorable for the survival and differentiation of NSPCs transplants. On the other hand, in the chronic stage after injury, glial scars form in the injured site that inhibit the regeneration of neuronal axons. Thus, we believe that the optimal timing of transplantation is 1 to 2 weeks after injury. In this symposium, I will talk about our trials of NSPCs transplantation in rats and non human primate models.

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SY32-01 Roles of the growth factor midkine in neural development and mental functions

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Midkine is a heparin-binding growth factor, expression of which is developmentally regulated. The Xenopus midkine is exclusively expressed in the neural anlagen during early development, inhibits mesoderm induction, and enhances anterior neural tissue formation in collaboration with Smad2 signaling. Midkine-deficient mice exhibit a deficit in working memory and an increased anxiety 4 weeks after birth. These phenomena are correlated with a transient retardation of development of the dentate gyrus in midkine-deficient mice. Furthermore, midkine is expressed in periventricular neural stem cells, is induced in reactive astrocytes, and exerts neuroprotective activity against brain ischemia and neurodegenerative insults, such as constant light exposure to photoreceptors. Taking into account that midkine is also implicated in pathogenesis of Alzheimer's disease, multiple system atrophy and schizophrenia, these data suggest that midkine plays fundamental roles in neural development and neurological disorders.

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SY32-02 Virus-induced neurobehavioral disorders: mechanisms and applications for developmental defect model

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Neurobehavioral disorders, including most psychiatric diseases, can be multifactorial in origin, suggesting that both genetic and environmental factors play important roles in the manifestation of symptoms. Several working hypotheses propose that viral infection contributes to the induction of neural cell dysfunction, resulting in a wide range of behavioral abnormalities. Borna disease virus (BDV) is a highly neurotropic RNA virus and readily establishes persistent infection in infected brain cells. Epidemiological studies have suggested that BDV infection may link to human mental illnesses. We have recently demonstrated that a BDV product induces behavioral disorders in association with neurobiological abnormalities, and that impairment of glial cell function might exist as a common cause for the induction of neurobehavioral disorders. We present here possible mechanisms of virus-induced neurological disorder and application of viral products for developmental defect model.

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SY32-03 A potential link between impaired neurogenesis and behavior abnormalities

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Pax6 transcription factor is expressed in brain regions where postnatal neurogenesis occurs such as the SVZ and the SGZ of the hippocampal dentate gyrus. We have shown that Pax6-expressing cells in the hippocampus have a stem cell-like character and that BrdU-incorporation is 30% reduced in the Pax6 deficient (rSey2/+) rat. Thus, Pax6 gene dosage is critical for hippocampal neurogenesis. We also found that rSey2/+ rats exhibited abnormalities in social interaction, low performance in fear-conditioned memory, and deficit in the sensorimotor gating. Interestingly, the impairment of the sensorimotor gating was not clear in juvenile rats, but obvious after puberty, more severe in aged rats, and restored by treatment with clozapine, an anti-psychiatric drug. Together with previous reports, our data suggest that impairment in adult neurogenesis may be relevant to vulnerability in various mental illnesses.

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SY32-04 Behavioral and psychopharmacological abnormalities of Fyn-deficient mice and their clinical implications

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Fyn tyrosine kinase-deficient mice show behavioral abnormalities such as increased fearfulness and defective maternal behavior. Amygdaloid dysfunction may underlie these emotional disorders. This mutant also shows reduced response to anti-psychotic drugs. Haloperidol-induced cataleptic behavior and tyrosine phosphorylation of NMDA receptor through Fyn activation in the striatum as observed in the wild type were significantly attenuated. On the other hand, this mutant shows defective neocortical development restricted to the upper layers and reduced tyrosine phosphorylation of cell migration-related proteins. Thus, Fyn may be involved in the signal transduction both in the neural development and higher brain function. Clinical studies concomitant with above findings strongly suggest the involvement of Fyn in the pathogenesis of psychiatric disorders such as schizophrenia.

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SY32-05 Molecular and neurobiological study on attention deficit hyperactivity disorder (ADHD)

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Attention deficit hyperactivity disorder (ADHD) is one of the most common psychiatric disorders for children. Methylphenidate, a psychostimulant, has calming effects in 70 % of ADHD patients, whereas it increases motivation level in both ADHD patients and healthy people. Dopamine transporter (DAT) is a target of methylphenidate, and DAT knockout (KO) mice show ADHD-like behaviors. In DAT-KO mice, methylphenidate decreases locomotor activity but increases motility in a tail-suspension test, a test for motivation level. In contrast, weaver mutant mice and EL mice show hyperactivity which can not be treated by methylphenidate. Investigations using these model animals would further our understanding of molecular and neurobiological mechanisms underlying ADHD.

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SY33-01 Cortical neurons at different developmental stages show distinct migratory behaviors

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This study's objective is to know whether the migratory behavior of cortical neurons is stage-dependent. Cortical ventricular zone (VZ) cells of E12.5 and E15.5 mouse were labeled with *gfp*-encoding plasmid by in utero electroporation and GFP-labeled cells were examined after appropriate periods. Both early-born neurons (the earliest emigrants from E12.5 VZ) and late-born neurons (those derived from E15.5 VZ) first transformed into stellate shape, then gradually changed into radially oriented bipolar cells as they moved upwards. Thereafter, however, only the late-born neurons showed clear radial fiber-associated migration. Impairment of activity of cyclin-dependent kinase 5 only affected the migration of the late-born neurons. These results suggest that cortical neurons at different developmental stages show distinct migratory behaviors, each regulated by distinct molecular mechanisms.

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SY33-02 The role of Pax6 in tangential migration of cortical neurons

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In early stages of cortical development neurons born at the dorsal part of the telencephalon migrate ventrally, but turn their migratory direction caudally at the pallium-subpallium boundary (PSB). The migration pattern of these neurons are altered in Pax6 mutant telencephalon, i.e., the neurons migrated ventrally toward the ventral part of the telencephalon by crossing the PSB without turning caudally. We identified that blockade of ephrin-A/Eph-A interactions mimicked the migratory defect of the Pax6 mutant. ephrin-A5, one of ephrin-A ligands, was expressed at the ventral part of the telencephalon, and its expression level was decreased in the Pax6 mutant telencephalon. Furthermore, overexpression of ephrin-A5 restored the migration pattern of the neurons in the Pax6 mutant. These lines of evidence suggest that Pax6 plays an crucial role for guidance of neuronal migration by regulating ephrinA/Eph-A interactions in the developing telencephalon.

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SY33-03 The role of Notch signal in bone marrow-derived stem cells

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The Notch regulation is important for stem cells except. One of the big differences of Notch protein from other types of receptor is that it can interact with its ligands in a cell-autonomous manner and can modulate its receptivity. From our findings, this mechanism supports the specific trait of Notch signal known as Lateral Inhibition. Notch protein receives the modifications from the intra/extra-cellular factors and changes its property. In vertebrate, a novel gene family, called CCN was evolved and associates with Notch receptor, which stimulates its signal and suppresses the cell differentiation. Several members of the CCN gene family are expressed in the mesenchymal stem cells and seem to play a specific role. We investigate a bone marrow-derived stem cells, Kusa as a model system. This cell line basically exhibits strong osteogenesis but also differentiates to adipocytes or neurons. We constructed several Notch signal mutants of Kusa and analyze their differentiation ability.

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SY33-04 A novel cell surface protein of Ig superfamily member in developing CNS

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Cell surface proteins of the immunoglobulin (Ig) superfamily play crucial roles during neural development, such as cell adhension and axon guidance. Using cDNA subtraction strategy, we have identified a novel gene of this family, whose expression is in the neuroepithelium cell layer of whole CNS before neurogenic stages, and subsequently confined to the dorsal parts of CNS in chick embryos. Deduced protein is characterized by four Ig domains and five fibronectin type III repeats in the extracellular domain, structurally related to DCC and Neogenin, the cell surface receptors for Netrin-1. Loss-of-function of this gene by siRNA in vivo resulted in the augmentation of the number of *delta*-positive cells in the midbrain region. On the other hand, over expression of this gene reduced the number of *delta*-positive cells in the same region. These results suggest that this cell surface protein may be involved in the process of early neurogenesis.

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SY33-05 The role of Slitrk transmembrane protein family in neural development

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The Slitrk family consists of six structurally related transmembrane proteins (Slitrk1-6) in the human and mouse. In the extracellular region, they share two conserved leucine-rich repeat domains that have a significant homology to a secreted axonal growth-controlling protein, slit. These proteins also have a homology to trk neurotrophin receptors in their intracellular domains, sharing a conserved tyrosine residue. Expression of Slitrk is highly restricted to neural tissues, but varies within the family. Slitrk1 expression is in the mature neurons, whereas Slitrk2 is strongly expressed in the ventricular layer, and Slitrk6 shows compartmentalized expression in diencephalon. Slitrk1 induced unipolar neurites in cultured neuronal cells, whereas Slitrk2 and other Slitrk proteins inhibited neurite outgrowth. When the Slitrk2 protein was overexpressed in dorsal spinal cord in transgenic mice, the commissural fibers and other neuronal processes were abnormal. Further characterization of the family members is now in progress.

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SY34-01 Dynamism of stress relevant receptors in the hippocampus

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Stress induces high level of corticosteroid hormones. Hippocampus is one of the main target areas for the corticosteroid hormone action. Hippocampal neurons actually possess receptors for corticosteroids, glucocorticoid receptor (GR) and mineralocorticoid receptor (MR). By using green fluorescent protein (GFP), dynamic movements of these receptors were monitored at the cellular level. Particular emphasis was paid on the analysis of the translocation mechanism between cytoplasm and nucleus, and the dimerization and mobility of GR and MR in the nucleus with the use of FRET and FRAP. The results showed that importin alpha was involved in trafficking of GR and MR, GR/MR heterodimerization took place in the nucleus, and GR/MR proteins are much more mobile within the nucleus than having been thought. Ref) Nishi, M. et al.: Visualization of glucocorticoid receptor and mineralocorticoid receptor interactions in living cells with GFP-based fluorescence resonance energy transfer (FRET). J. Neurosci. (in press)

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SY34-02 Functional genomics of hypothalamic homeostatic plasticity

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In response to the challenge of dehydration, hypothalamic neurons secrete the antidiuretic hormone vasopressin (VP) into the circulation. VP release is accompanied by a functional remodelling of hypothalamic activity, the molecular basis of which is not understood. We have adopted a global approach to understanding this plasticity. Using expression-profiling technologies, we have catalogued genes that are expressed in hypothalamic neurons, and identified genes that are turned on or off following dehydration. For example, cDNA microarray analysis identified Interleukin 6 as a candidate up-regulated gene (1). Using somatic gene transfer methods that have already suggested a role for protein kinase A in the physiological regulation of VP gene expression (2), we are now studying the function of our novel genes in hypothalamic homeostatic plasticity. 1. Ghorbel et al., (2003) J Biol Chem 278: 19280-192852. Wong et al., (2003) Mol Cell Neurosci 24: 82-89

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SY34-03 Analysis of crucial roles of the neurosteroidal environment in organization processes of reproductive behaviors by embryonic engineering

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Estrogen in the brain plays important roles as a multifunctional neurosteroid. To assess functional siginificance of brain estrogens in organization processes of reproductive behaviors, we prepared aromatase (estrogen synthase)-knockout (ArKO) mice. ArKO mice were deep affected on reproductive behaviors. They showed deficits in sexual behaviors and abnormal behaviors in aggression, maternal instincts, and partner preference. Human aromatase cDNA with the brain-specific promoter of mouse aromatase gene was introduced into ArKO mice. The mice expressed human aromatase only in specific neurons with a transient peak at a neonal critical period, and recovered from disorders in the reproductive behaviors. These results suggest that according to a genetic program, brain aromatase neonatally produces neurosteroidal estrogen environment which consequently determines the organization processes of reproductive behaviors.

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SY34-04 A novel neurosteroid stimulating locomotor activity and acute stress response

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The neuronal mechanism in response to stress is not fully understood. A novel active neurosteroid stimulating locomotor behavior was identified as 7α-hydroxypregnenolone in an amphibian brain. The production of this novel active neurosteroid was high in the brain stem unlike other brain regions and peripheral steroidogenic glands. This neurosteroid specifically increased, in a dose-related way, locomotor activity and dopamine release and the effect on locomotion was abolished by dopamine D_2 receptor antagonists. Several stress conditions, such as fast, osmotic and heat stresses, enhanced acutely this neurosteroid formation and locomotor activity. These results indicate that the identified 7α-hydroxypregnenolone acts as a novel neuronal activator to stimulate locomotor activity through the dopaminergic system. This novel mechanism may induce hyperlocomotion in response to stress.

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SY34-05 Neural correlates of reward anticipation and impulsive choice.

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Newly-hatched domestic chicks have been widely studied toward understanding the neural basis of behaviors such as imprinting, avoidance and reinforcement. In a Go-NoGo task, neurons in the medial striatum (mSt) have been shown to code the chick's anticipation of forthcoming reward (Yanagihara et al. 2001). Accordingly, excitotoxic lesions to the posterior mSt caused an impulsive choice, so that the ablated chicks shifted their choice toward an immediate-small reward and avoided a late-large reward without any mnemonic deficiencies (Izawa et al. 2002, 2003), except a delayed extinction of learned operant (Ichikawa et al. 2004). Further characterization of the mSt neurons revealed specific coding of the temporal proximity and the quantity of the anticipated reward (Izawa et al. 2003 SfN abstract). These results, together with the findings in limbic arcopallium (Aoki et al 2003), suggest that the neural representatives of the future events play critical roles in the impulsive decision-making.

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SY35-01 Spontaneous D-aspartic acid formation in protein from elderly human lenses

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Proteins consist exclusively of L-αamino acids, however, we found that the configuration of the Asp-58 and Asp-151 residues in αA-crystallin from elderly human lenses was inverted and isomerized to the D-β-asp -isomer (D/L ratio: 3.1 for Asp-58, 5.7 for Asp-151). A D/L ratio higher than 1.0 is not considered to be due to simple racemization, but rather results from stereoconfiguration inversion. The simultaneous inversion and isomerization of Asp residues in protein indicate that the reaction occurs via a succinimide intermediate. The formation of succinimide in protein proceeds rapidly when the residue at the carboxyl side of the Asp residue is small. The Asp-58 and Asp-151 residues in αA-crystallin are followed by Ser and Ala, an arrangement conducive to the formation of succinimide. Another important factor for D-β-asp formation in protein is the conformation surrounding these Asp residues. In this presentation, we will discuss that αA-crystallin has a chiral reaction field, which induces the inversion of the Asp-58 and Asp-151residues.

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SY35-02 Isoaspartate Formation and Neurodegeneration in Alzheimer's Disease

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Protein isomerization is enhanced in amyloid-beta peptides (Abeta) and paired helical filaments (PHFs) purified from Alzheimer's disease (AD) brains. Biochemical analyses revealed that Abeta purified from senile plaques and vascular amyloid are isomerized at Asp-1 and Asp-7. Biochemical analyses of purified PHFs also revealed that tau contains L-isoaspartate at Asp-193, Asn-381, and Asp-387. Since protein isomerization as L-isoaspartate enhances the aggregation process, this modification is proposed as one of the progression factors in AD. Protein L-isoaspartyl methyltransferase (PIMT) is suggested to play a role in the repair of isomerized proteins containing L-isoaspartate. Given the enhanced protein isomerization in AD brains, it is implicated that the upregulated PIMT may associate with increased protein isomerization in AD. PIMT-deficient mice showed the neurodegenerative changes concomitant with accumulation of L-isoaspartate. We propose the pathological implications of protein isomerization in the neurodegeneration in model mice and AD.

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SY35-03 Biological function of D-aspartate in mammals

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D-Aspartate (D-Asp) in the mammalian body is predominantly present in the neuroendocrine and endocrine tissues. In the adult rats, D% (proportion of D-Asp in the total Asp) is as high as 30-40% in the pineal gland and the testis, and 10% in the embryonic cerebrum, respectively. We have carried out immunohistochemical analysis with anti-D-Asp antibody and studies on the biological activities of D-Asp with primarily cultured cells. In the neuronal cells, D-Asp first emerges in their cell bodies and thereafter it appears in the processes of neuroblasts and is localized in axons once the distinct axon layer had been established. In the pineal gland, D-Asp is localized in the pinealocytes and suppresses melatonin secretion. In the testis, D-Asp is predominantly present in the matured germ cells and is presumed to act on Leydig cells in a paracrine fashion to increase testosterone production. In Leydig cells, D-Asp appears to stimulate the gene expression of StAR, which is an essential factor for steroid synthesis, by modulating intracellular signal transduction.

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SY35-04 D-Serine and schizophrenia

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We have demonstrated that a co-agonist of the N-methyl-D-aspartate (NMDA) glutamate receptor D-serine is an endogenous substance that shows a brain-selective and NMDA receptor-related distribution throughout the life in mammals. Furthermore, extracellular release and uptake of D-serine, glycine- or L-serine-induced increase and cysteamine-induced decrease in the D-amino acid contents have been observed in the brain tissues. Interestingly, it has been reported that potent NMDA antagonists including phencyclidine cause schizophrenia-like psychosis and D-serine inhibits phencyclidine-induced abnormal behavior in the rodents and positive and negative symptoms of schizophrenic patients. These data indicate that D-serine may play a substantial role in the control of mental functions by acting as an intrinsic modulator of the NMDA receptor and disturbed brain D-serine signaling could be involved in the pathophysiology of schizophrenia.

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SY35-05 Localization of free D-serine in the brain

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Dr. Solomon H. Snyder and his coworkers reported that D-serine and serine racemase, the enzyme producing D-serine from L-serine, were localized exclusively in astrocytes. They hypothesized that D-serine produced in the astrocytes will be released from them in response to glutamatergic stimulation, and D-serine thus released from astrocytes and glutamate released from axon terminals bind to NMDA receptors co-activating the NMDA receptors. On the other hand, we produced highly purified antibody against D-serine and found that D-serine immunoreactivity was localized mainly in neurons, while it was also found in glias in a few areas of the brain, for example in the corpus callosum. Because the result was contradictory to that of Snyder's, we tried to demonstrate localization of mRNA for serine racemase by an in situ hybridization technique. Using oligo DNA probes, we found mRNA for serine racemase mainly in neurons. Then, we believe D-serine is produced mainly in neurons and will be released from axon terminals in response to neural activity.

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SY35-06 Functional roles and pathophysiology of brain D-amino acid oxidase

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D-amino acid oxidase (DAO) is a flavoenzyme that catalyzes the oxidative deaminaiton of wide range of D-amino acids. D-serine acts as allosteric modulator of NMDA receptor. Recently, DAO and G72 genes have been reported to be linked to susceptibility to schizophrenia. pG72 protein interacts with DAO and activates enzyme activity. In search of cell type specific expression of DAO in the brain, we examined the DAO gene expression in cultured rat astrocytes. We detected highest expression in type-1 astrocyte cultures from cerebellum. Higher expression was detected in type-1 astrocyte cultures than that in type-2. In order to investigate the function of DAO in the brain, we examined the metabolism of D-serine in glial cells. The results indicated that astroglial DAO is involved in the regulation of extracellular levels of D-serine. Future studies are necessary to understand how the interaction of two proteins (DAO and pG72) and the modulation of enzyme activity contribute to the molecular mechanisms leading to the pathology of schizophrenia.

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SY36-01 Glutamate receptor δ2 as a key molecule of cerebellar wiring and learning

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We found the δ subfamily of glutamate receptor (GluR) by molecular cloning. From the amino acid sequence identity, the GluRδ subfamily positions in between the classical AMPA/kainate and NMDA subtypes of GluR. GluRδ2, the second member of this subfamily, is selectively expressed in cerebellar Purkinje cells (PCs) and GluRδ2 proteins are exclusively localized at parallel fiber-PC synapses. Our molecular genetic studies showed that GluRδ2 plays a key role in cerebellar LTD, motor learning and PC synapse formation. Recent advances including conditional gene targeting of GluRδ2 provide new insights into PC wiring and hippocampus-cerebellum interaction for motor learning. Thus, GluRδ2 research will be a key issue to understand the cerebellar wiring and motor learning.

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SY36-02 Discoordination of motor control in a GluRδ2 knockout mouse

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Previous studies on mutant mice revealed that deficits in synaptic regulations in the cerebellum are accompanied with motor discoordination and motor learning failures. However, how synaptic regulation failures cause behavioral abnormality has been elusive. We addressed this issue working on the mutant mouse deficient in glutamate receptor δ2 subunit (GluRδ2), which is selectively expressed in Purkinje neurons (PNs). The mutant mouse deficient in GluRδ2 (δ2-/-) lacks the LTD and shows abnormal synaptic organization, motor discoordination and motor learning deficits. We found that a δ2-/- showed involuntary spontaneous eye movement with characteristic 10 Hz oscillation. Our results suggest that GluRδ2 deficiency produces the oscillating PN activity by enhancing climbing fiber inputs, causing surplus movement and affecting motor control worse than no signal at all. A δ2-/- also showed abnormal dynamic properties in reflex eye movements, which were dependent on activity of inferior olivary nuclei.

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SY36-03 Synaptic mismatching and disconnection by inducible and Purkinje cell-specific ablation of glutamate receptor δ2 in mature cerebellum

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We examined the role of glutamate receptor (GluR) δ2 in the adult brain by inducible and cerebellar Purkinje cell (PC) -specific gene targeting under the C57BL/6 genetic background. Activation of Cre recombinase-progesterone receptor fusion protein by antiprogestin treatment diminished the expression of GluRδ2 proteins selectively in cerebellar PCs. Concomitant with the decrease of GluRδ2 proteins, mismatching between pre- and postsynaptic specializations appeared in a significant fraction of parallel fiber (PF) -PC synapses. Furthermore, many PC spines lost their synaptic contacts with PF terminals and GluRδ2 proteins were absent in these free spines. These results suggest that GluRδ2 is a key molecule for matching and connection of PF-PC synapses in the adult brain.

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SY36-04 Molecular mechanisms for competitive synaptic wiring in cerebellar Purkinje: GluRδ2 and P/Q-type calcium channel.

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In mice lacking GluRδ2, PC spines on distal dendrites become free from parallel fiber (PF) innervation. Toward distal dendrites, climbing fibers (CFs) expand their territory to take over free spines. The distal expansion further extends to adjacent PCs, causing multiple CF innervation. Thus, GluRδ2 is essential for PF synaptogenesis and restricting CFs to proximal dendrites. By contrast, in mutant mice defective in P/Q-type calcium channel α1A subunit, PF territory expands down to the soma, whereas CF territory is diminished to the basal portion of proximal dendrites and soma, at which multiple CFs as well as ectorpic PFs are co-wired. Thus, P/Q-type calcium channel is essential for strengthening the innervation of proximal dendrites by a main single CF and expelling other CFs and PFs from there. Therefore, GluRδ2 and P/Q-type calcium channel play as a molecular stabilizer selective to PFs or to a main CF, respectively. Armed with both mechanisms, mono-innervation by the CF and territorized innervation by the PF and CF are established.

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SY36-05 A novel activation mechanism and function of δ2 glutamate receptors

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The glutamate receptor δ2 (GluRδ2) plays a critical role in the cerebellum: GluRδ2$^{-/-}$ mice show ataxia, impaired motor learning, and loss of long-term depression (LTD). However, the mechanisms by which GluRδ2 participates in these functions have been elusive. It is also unclear whether GluRδ2 is functional in the mature brain. We recently found that application of an antibody against the putative ligand-binding region of GluRδ2 to cultured Purkinje cells induced AMPA receptor endocytosis and abrogated LTD. Moreover, injection of this antibody into the supracerebellar space of adult mice caused transient ataxia. These results indicate that GluRδ2 plays a novel metabotropic receptor-like role to controls AMPA receptor endocytosis in adult Purkinje cells. In addition, we found that a GluRδ2 transgene, which had a mutation in the putative ligand-binding motif conserved in iGluRs, could rescue most phenotypes of GluRδ2$^{-/-}$ mice. Therefore, although GluRδ2 is classified as iGluR, it may not require glutamate-like ligands to function in the cerebellum.

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一般演題（口演発表）

	1st		2nd		3rd	
	Morning	Afternoon	Morning	Afternoon	Morning	Afternoon
神経疾患 I／Neurological Diseases I	Room B OB1-01~10					
神経疾患 II／Neurological Diseases II		Room B OB1-11~22				
再生、移植、神経細胞死／Regeneration, Transplantation and Neuronal Death			Room B OB2-01~11			
自律神経系、行動、睡眠、生体リズム／Autonomic Nervous System, Behavior, Sleep and Biological Rhythms				Room B OB2-12~23		
運動、視覚／Motor System, Visual System					Room B OB3-01~10	
学習、記憶、睡眠、生体リズム／ Learning and Memory, Sleep and Biological Rhythms						Room B OB3-11~23
視覚／Visual System	Room G OG1-01~04	Room G OJ1-11~16		Room H OH2-11~22		
疾患モデル／Animal Models	Room G OG1-05~10					
老化、痴呆／Aging, Dementia		Room G OG1-11~21				
突起伸展、回路網形成／Axonal Outgrowth and Network Formation			Room G OG2-01~10			
受容体、輸送体／Receptors and Transporters				Room G OG2-11~16		
グリア、ミエリン、血液脳関門／Glia, Myelin and Blood-Brain Barrier				Room G OG2-17~22		
シナプス、シナプス可塑性／Synapse, Synaptic Plasticity					Room G OG3-01~10	
シナプス可塑性／Synaptic Plasticity						Room G OG3-11~19
情報伝達、伝達物質、受容体／Signal Transduction, Neurotransmitters and Receptors	Room H OH1-01~10					
栄養因子、サイトカイン／Trophic Factors and Cytokines		Room H OH1-11~16				
再生、移植／Regeneration, Transplantation		Room H OH1-17~22				
情報伝達、神経伝達物質／Signal Transduction and Neurotransmitters			Room H OH2-01~05			
軸索輸送、細胞運動、細胞骨格／Axonal Transport, Cell Motility and Cytoskeleton			Room H OH2-06~10			
イオンチャネル、興奮性膜／Ion Channels and Excitable Membranes					Room H OH3-01~07	
シナプス／Synapse						Room H OH3-08~12
パターン形成／Pattern Formation						Room H OH3-13~18
認知、コンピュータ科学／Cognition and Computer Science	Room J OJ1-01~10					
情報伝達、受容体、嗅覚、痛覚／Signal Transduction, Receptors, Olfaction and Pain		Room J OJ1-17~21				
イメージング、分子生物学的、細胞生物学的方法／Imaging, Molecular and Cellular Biological Techniques			Room J OJ2-01~10			
聴覚、嗅覚、味覚、化学感覚／Auditory System, Olfaction, Taste and Chemical Senses				Room J OJ2-11~16		
痛覚／Pain				Room J OJ2-17~22		
細胞分化／Cellular Differentiation					Room J OJ3-01~12	

OB1-01 Molecular and pharmacological analysis of PACAP in CNS functions

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PACAP is a highly conserved neuropeptide which is widely expressed in the brain. To address the roles of the endogenous PACAP, we have recently produced three genetically engineered mice (two knockouts and a transgenic), examining the phenotypic consequences in mutants with the aim to investigate the potential of PACAP-signaling pathways as drug targets.
Mice lacking PACAP ($PACAP^{-/-}$) were born normally but revealed high postnatal mortality. The surviving adult $PACAP^{-/-}$ mice displayed 1) altered psychomotor behaviors, including increased exploratory locomotor activity and abnormal jumping in a novel environment, 2) defects in sensorimotor gating as measured by prepulse inhibition of the startle response, 3) impairment of the maintenance of late LTP and the expression of long-term memory.
The observed phenotypes have not only led to a better understanding of the physiological roles of endogenous PACAP, but have also revealed unexpected roles for PACAP.

OB1-02 Evidence of novel neuronal functions of dysbindin, a susceptibility gene for schizophrenia

Ryota Hashimoto[1], Tadahiro Numakawa[1], Yuki Yagasaki[1], Tetsuya Ishimoto[1], Tatsuyo Suzuki[1], Nakao Iwata[1], Norio Ozaki[1], Takahisa Taguchi[1], Masahiko Tatsumi[1], Kunitoshi Kamijima[1], Richard Straub[1], Daniel Weinberger[1], Hiroshi Kunugi[1]
[1]Dept of Mental Disorder Research, NIN, NCNP, Kodaira, Japan

Genetic variation in dysbindin (DTNBP1: dystrobrevin binding protein 1) has recently been shown to be associated with schizophrenia. We found a nominally significant association with schizophrenia in a multi-marker haplotype analysis (p=0.0004) in a Japanese sample. We then explored functions of dysbindin protein in primary cortical culture. Overexpression of dysbindin induced the expression of two pre-synaptic proteins, SNAP-25 and synapsin I, and increased extracellular basal and released glutamate levels. The overexpression of dysbindin increased phosphorylation of Akt and protected cortical neurons against neuronal death due to serum deprivation through PI3-kinase-Akt signaling pathway. Genetic variants associated with impairments of these functions of dysbindin could play an important role in the pathogenesis of schizophrenia.

OB1-03 Novel zincfinger protein that interacts with Disrupted-In-Schizophrenia 1 (DISC1)

Tsuyoshi Hattori[1], Atsushi Yamaguchi[1], Kiyoshi Inoue[1], Shinsuke Matsuzaki[1], Akiko Honda[1], Kousuke Baba[1], Masaya Tohyama[1]
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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 in a Scottish family. Previously we have shown that fasciculation and elongation protein zeta-1 (FEZ1) is one of interacting partners of DISC1 by a yeast two-hybrid system and involved in neurite outgrowth through interacting with DISC1 and F-actin. In this study, we report a novel DISC1 interacting partner with one zinc finger domain and coiled-coil region. By northern blotting and in situ hybridization study, we detected its expression exclusively in the brain, especially high in hippocampus, olfactory bulb and cereberal cortex. We further demonstrate the functional interaction between this novel protein and some neuronal growth factors like NGF and PACAP (Pituitary adenlyate cyclase activating polypeptide).

OB1-04 DISC1 regulates the transport of NUDEL/LIS1/14-3-3 via Kinesin-I

Shinichiro Taya[1], Kumiko Nagai[1], Tomoyasu Shinoda[1], Setsuko Kuroda[1], Kozo Kaibuchi[1]
[1]Dept. of Cell Pharmacology, Nagoya Univ., Nagoya, Japan

Disrupted-In-Schizophrenia 1 (DISC1) is a candidate gene for schizophrenia as a susceptibility factor. DISC1 is reported to interact with NudE-like (NUDEL), which forms a complex with lissencephaly-1 (LIS1). Here, we identified 14-3-3 as a DISC1-interacting molecule. 14-3-3 is thought to be necessary for the transport of NUDEL/LIS1 to the axon from the cell body. DISC1 formed a complex with NUDEL/LIS1/14-3-3. Knockdown of DISC1 in cultured hippocampal neurons by RNAi resulted in shorter axons and caused the delocalization of NUDEL/LIS1/14-3-3 from the axons. We also found that DISC1 formed a complex with conventional kinesin-I. Knockdown or dominant negative form of kinesin-I induced the delocalization of DISC1 and 14-3-3 from the axons. The proper localization of the NUDEL/LIS1 complex is thought to be required for axon growth. Taken together, these results indicate that DISC1 links kinesin-I to NUDEL/LIS1/14-3-3, and regulate its transport for axon elongation and neuronal morphogenesis.

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OB1-05 Roles of Disrupted-in-Schizophrenia-1 (DISC1) in neuronal morphologies

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High risk of developing schizophrenia is genetic and several chromosomal regions are implicated candidate genes as susceptibility factors. Disrupted-in-Schizophrenia 1 (DISC1) is a novel gene associated with schizophrenia. The disease mutant form of DISC1 inhibits neurite outgrowth. However, the function of DISC1 remains to be clarified. To understand the role of DISC1, we here identified Purα as a DISC1-interacting molecule. Purα links RNA-protein complexes (RNPs), which include translation-component, to microtubules in dendrites. Recent studies indicate that specific mRNAs are transported into dendrites and are translated for synaptic plasticity. We showed that DISC1 formed a complex with Purα. DISC1 co-localized with Purα and RNPs in dendrites. DISC1 also localized with RNPs at dendritic spines. These results indicate that DISC1 forms a complex with Purα and RNPs in dendrites. The role of DISC1 in neuronal morphologies will be discussed.

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OB1-06 Reversed astrocytic glutamate transporter GLT-1 crucial to the development of neuronal ischemic tolerance in astrocyte/neuron co-cultures.

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In the brain, prior sublethal ischemia (preconditioning, PC) is known to produce ischemic tolerance of neurons to subsequent lethal ischemia (PC-IT). We have found that the activation of NMDA receptors during PC was crucial to the development of PC-IT. This study aims at elucidating whether glutamate (Glu) released via reversed Glu transporters during PC is responsible for the development of PC-IT in astrocyte/neuron co-cultures. When the function of astrocytic GLT-1 during PC was suppressed by treatment with DHK, the elevation of extracellular Glu during PC was significantly suppressed, and PC-IT was also significantly depressed. These results suggested that the marked elevation of extracellular Glu resulting from the reversed uptake of Glu mainly by astrocytic GLT-1 was crucially involved in the development of neuronal PC-IT.

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OB1-07 Stimulating the medial septum can protect against ischemia-induced delayed neuronal death in rat hippocampus

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The present study aimed to clarify whether an increase in hippocampal blood flow (Hpc-BF) during activating the medial septum (MS) could prevent delayed death of the hippocampal neurons following transient ischemia in rats. During intermittent (every 5 s) occlusions for 60 min of unilateral carotid artery in addition to permanent ligation of bilateral vertebral arteries, Hpc-BF ipsilateral to the occluded carotid artery decreased by about 25% of the control level, and 5d later, delayed death occurred in about 24% of the hippocampal CA1 neurons. Repetitive electrical stimulation of the MS for 65 min, starting 5 min before the occlusions, increased Hpc-BF and prevented the occlusion-induced decrease in Hpc-BF. The delayed death of the CA1 neurons occurring after occlusions was attenuated by MS stimulation to about 5% of the CA1 neurons. The results suggest that MS-originating vasodilative activation can protect ischemia-induced delayed death of hippocampal neurons.

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OB1-08 Brain serotonergic nervous system modulates spreading depression: A pathophysiology of migraine

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The vascular serotonergic system has been implicated in pathophysiology of migraine, however, involvement of the serotonergic nervous system of the brain parenchyma in the pathophysiology has remained unknown. We prepared an experimental model of migraine by generation of spreading depression (SD), characterized by spreading of neuronal/glial membrane depolarization accompanied by temporal elevation of the cerebral blood flow (CBF) throughout the cerebral cortical hemisphere in the rats anesthetized with 0.8% halothane. SD could be induced in non-treated animals and also in animals which underwent degeneration of serotonergic neurons in the dorsal raphe nucleus by pharmacological treatment. Velocity of the spreading was significantly increased in the treated animals. These results indicate that the brain serotonergic nervous system modulates spreading depression in migraine.

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OB1-09 The Role of Synaptic Plasticity in the Bicuculline-Induced Epileptic Discharges in Guinea-Pig Hippocampal Slices.

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NMDA receptor (NMDAR) and Ca^{2+}/calmodulin dependent protein kinase II (CaMKII) can be related to the induction process of synaptic plasticity. We studied the relationship between the bicuculline-induced epileptic discharges (BIED) and synaptic plasticity. BIED were recorded extracellularly from the transverse hippocampal slices of guinea pig, when 20 μM bicuculline was applied. NMDAR antagonist, AP5 and the inhibitor of CaMKII, KN-93 or KN-62, decreased the frequency of BIED, while the negative control of KN-93, KN-92, did not. In addition, the evoked pEPSP at CA3-CA3 synapses was potentiated after the disappearance of BIED when bicuculline was rinsed. This potentiation was suppressed by the application of AP5 or KN-93. Moreover, above 5 μM CNQX, AMPA receptor antagonist, also decreased the frequency of BIED on the concentration-dependent manner. Theses results suggest that the frequency of BIED can be modulated by the excitatory synaptic potentiation, which is dependent on NMDAR and CaMKII.

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OB1-10 The role of prostaglandin in neuronal loss induced by kainate

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Prostaglandin (PG) have been shown to have important roles in the central nervous system. After convulsions, PG levels in the brain were increased and cyclooxygenase-2 (COX-2), an enzyme involved in synthesis of PGs, was induced in hippocampal neurons. In our previous study, the result suggests that seizure-induced COX-2 facilitates the recurrence of hippocampal seizure. In this study, we investigated the roles of COX-2 in hippocampal neuronal loss using COX-2 knockout mice and COX-2 selective inhibitor. Injection of kainate (KA) into the hippocampal CA3 subfield increased the PGE_2 concentration and induced neuron death in this region. PGE_2 production and CA3 cell loss were significantly reduced under COX-2 deficient conditions. These results suggest that seizure-induced COX-2 accelerates KA-induced neuronal loss in the hippocampus by increasing PGE_2 production.

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OB1-11 Analysis of spontaneously hyperactive Wig rats

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We studied the mechanisms of motor hyperactivity in Wiggling (Wig) rats. Spontaneous motor activities of Wig rats were significantly higher than controls at 4-6 weeks of age. This hyperactivity was not diminished by methamphetamine injection. Tyrosine hydroxylase immunoreactivities were low in the midbrain and striatum. However, DNA macroarray analysis revealed that gene expression profiles in these regions differed from those in hyperactive rats with neonatal 6-hydroxydopamine lesions. Wig rats may represent an animal model for attention-deficit hyperactivity disorder that does not respond to psychostimulants. It further prompts investigation of the mechanisms of this clinically pervasive disorder.

OB1-12 Defective neocortical development in Fyn-tyrosine-kinase-deficient mice

Shigeki Yuasa[1], Kotaro Hattori[2,3], Eiko Nakahira[1], Takeshi Yagi[2,3]
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Fyn tyrosine kinase is involved in the tyrosine-phosphorylation of Disabled-1 in the Reelin signaling pathway, and absence of Fyn is expected to result in a reeler-like phenotype. Thus, this study investigated neocortical development in Fyn-deficient mice. BrdU labeling revealed the under-migration of later-generated neurons despite the normal placement of earlier-generated neurons. Calbindin- and CaMKII-immunostaining showed that layer II-III neurons were aberrantly stratified but the neurons in the deeper layers showed little evidence of abnormality. Fyn was intensely expressed in the migratory cortical neurons generated in the later stage. These findings strongly suggest that Fyn is required for the cortical arrangement of later-generated neurons at the final stage of settlement, but that it is dispensable for the Reelin-dependent inside-out layer formation during radial migration.

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OB1-13 Vulnerability of Complexin2 knockout mouse for maternal deprivation

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Complexin2(CPLX2), a protein expressed at presynapse, has been reported being altered in the brain of schizophrenic patient by postmortem study. We utilized CPLX2 knockout mouse to investigate its physiological role. The knockout mouse shows the disability of spatial memory by Morris water maze test compared to wild-type mouse. We administered methamphetamine and mK-801 to test the effect on the spontaneous locomotor activity. The knockout mouse shows increased locomotor activity statistically significant than wild-type mouse. The knockout mouse was treated by the maternal deprivation (MD) stress from PN 1 to 14 day to investigate their vulnerability to the stress. By the water maze test, the knockout mouse applied MD stress showed remarkable disability of spatial memory, however, that of wild-type mouse was not affected by the stress. Taken together, we concluded that CPLX2 is protective against MD stress and that the knockout mouse is possibly a model for schizophrenia.

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OB1-14 Impaired neurite regeneration in the retina of a murine model of GM2 gangliosidosis

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We examined the morphological features and neurite regeneration capability of retina in a mouse model of GM2 gangliosidosis (Sandhoff disease). Light and electron microscopic studies revealed extensive storage of GM2 ganglioside in the neurons, glial cells, and pigment epithelial cells of Sandhoof mouse retina, but no apparent cell death was detected. The number of neurites from the retinal explants after 3, 6, and 10 days in culture were significantly lower in the Sandhoff mice than in the age-matched wild type mice. The application of BDNF partially improved neurite regeneration from Sandhoff retina. These results suggest that lysosomal storage of GM2 ganglioside impairs the capability of retinal neurons to regenerate neurites, and that BDNF might be effective for this impairment.

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OB1-15 Modification of LC3, a novel autophagosomal marker, in brain and peripheral tissues of mice deficient in cathepsin D and those doubly-deficient in cathepsins B and L

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We show here how autophagy contributes to the accumulation of lysosomes in neurons deficient in cathepsin D or those deficient in cathepsins B and L by demonstrating the conversion of molecular forms of LC3, a mammalian homolog of yeast Atg8p essential for autophagosome formation, from LC3-I (18 kD), a cytosolic form, to LC3-II, a membrane-bound form (16 kD).

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OB1-16 Effect of sialylation on acquiring neural plasticity.

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We have used kindling to determine the progress of acquiring neural plasticity. In the Japan Neuroscience Society in 2002, we showed gradual elevation of α2,3-sialyltransferase, ST3Gal IV, during kindling epileptogenesis. In the present, we show that sialylation levels of glycoproteins and glycolipids increase during kindling epileptogenesis. Concerning glycoproteins, mouse brain homogenate is applied to affinity purification with MAA (Macckia amurensis) lectin, recognizing α2,3-Sia-Galβ1,4-GlcNac, in which Galβ1,4-GlcNac is sialylated by ST3Gal IV. 2D-SDS-PAGE evaluated at least 2 spots showing significant increases following kindled-seizures. Alternatively, GQ1b among neutral and acidic lipid fractions in mouse hippocampus was significantly up-regulated following kindled-seizures. Finally, we propose important roles on sialylations of both glycoproteins and glycolipids for acquiring neural plasticity.

OB1-17 Bax-inhibitory peptide (BIP) prevents caspase-independent cell death in Machado-Joseph disease

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Machado-Joseph disease is an autosomal dominant hereditary ataxia caused by toxicity of expanded polyglutamine (Q79) in ataxin-3. Bax-inhibitory peptide (BIP), small five amino acids, is designed from KU70 which prevents bax to translocate from cytosol to nucleus by biding to Bax. Q79 induced-cell death is not prevented by the caspase inhibitors, but is almost completely inhibited by BIP or the siRNA for bax. DU145, bax-deficient cell, is resistent to Q79 toxicity. Q79 induced-cell death shows the cytoplasmic vacuolation and nuclear fragmentation. Caspase inhibitors block the nuclear change, but not the vacuolation, whereas BIP prevents both. Therefore, Q79 induced-cell is caspase independent, but exclusively dependent on bax (not bak). SInce BIP can easily enter neuron, BIP is expected to be a new drug to prevent the disease progression in Machado-Joseph disease.

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OB1-18 Neuron type-specific Hsp70 induction by huntingtin suppresses cell death in cerebellar neurons

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Molecular mechanisms for selective neuronal death in polyglutamine diseases remain to be clarified. By microarray analysis, we investigated changes of gene expression profiles in different neurons under expression of normal and mutant htt. Hsp70 gene is upregulated in response to mutant htt in cerebellar neurons. Western blot shows that Hsp70 ptotein level is also upregulated in cerebellar neurons. Hsp70 protein is increased in the cerebellar tissues of Huntington's disease (HD) patients but not in the cerebrum. Suppression of the Hsp70 response by siRNA changes character of cerebellar neurons from resistant to vulnerable to mutant htt. Our results show that endogenous response of Hsp70 is a key element regulating selectivity in the HD pathology.

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OB1-19 Establishing a molecular therapy for the polyglutamine diseases using the cell permeable peptide inhibitor PTD-QBP1

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The polyglutamine (polyQ) diseases are a class of inherited neurodegenerative diseases including Huntington's disease, in which the expanded polyQ stretch in each disease protein causes aggregation and cytotoxicity. We previously identified QBP1 (PolyQ Binding Peptide 1) and showed that QBP1 co-expression inhibits polyQ protein aggregation and cytotoxicity in cellular and *Drosophila* models. Here we show that QBP1 fused with a protein transduction domain (PTD) is efficiently delivered into cultured cells, and inhibits polyQ protein aggregation and cytotoxicity. PolyQ disease *Drosophila* fed PTD-QBP1 showed rescue of premature death due to neurodegeneration. Furthermore, continuous administration of PTD-QBP1 into the lateral ventricle of polyQ disease mice inhibited polyQ protein aggregation in neurons around the administration site. These results indicate the potential of PTD-QBP1 for treatment of the currently untreatable polyQ diseases.

OB1-20 Alsin, the product of ALS2 gene, suppresses SOD1 mutant neurotoxicity via RhoGEF domain by interactiong with SOD1 mutants

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Mutation of the ALS2 gene encoding alsin is linked to the onset of autosomal recessive motor neuron diseases (MNDs). Alsin has two splicing variants, and alsin long form (LF) is thought to be a guanine nucleotide exchanging factor (GEF) for small GTPases. Expression of alsin LF, but not alsin short form, abolished neurotoxicity by Cu/Zn superoxide dismutase (SOD1) mutants, which cause dominant ALS. In contrast, alsin LF did not show any protective effects on other neurodegenerative insults. Deletion analysis of alsin LF has indicated that alsin RhoGEF domain was the responsible domain for the neuroprotection. Furthermore, we found SOD1 mutants, but not wild type SOD1, bound to alsin RhoGEF domain. The functional and physical interactions between alsin and SOD1 will be a promising clue to clarify the intricate pathomechanism of ALS and other MNDs.

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OB1-21 Functional Analysis of Novel Ubiquitin Ligase, Potential Target Antigen of Paraneoplastic Neurologic Disorder

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We have cloned a novel gene, Kspot, from mouse brain by expression screening using the autoimmune antiserum from a patient with stomach cancer associated with psychiatric symptoms. Kspot encoded a 286-amino-acid protein with an F-box domain and a SPRY domain that appeared to be a novel F-box protein. Kspot was localized to the cytoplasm and its expression was restricted to the brain. We also showed that Kspot formed an SCF complex with Skp1 and Cul1 and ubiquitinated unidentified substrates. Recently many neurodegenerative disorders have been thought to be caused by accumulation of harmful proteins in CNS because of malfunction of ubiquitin-proteasome system. We suggest that identification of substrate of Kspot may explain the pathophysiology of the psychiatric disorders associated with malignancy.

OB1-22 Autoantibodies against MuSK ectodomain inhibited multiple pathways of AChR clustering.

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We defined the MuSK autoantibodies also in generalized myasthenia gravis (MG) with detectable AChR autoantibodies. MuSK is critical to the clustering of AChR and plays multiple roles at neuromuscular junctions. We investigated the effect of anti-MuSK antibodies on clustering of AChR. Rabbits immunized with MuSK ectodomain protein manifested a MG-like muscle weakness. Unexpectedly, the antibodies blocked AChR clustering induced not only by agrin but also clustering induced by agrin-independent mediators that do not activate MuSK. That is, the MuSK antibodies rigorously inhibited AChR clustering mediated by multiple pathways, an outcome that broadens general comprehension of the pathogenesis.

OB2-01 Selective Neuronal Differentiation and Evaluation 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 developed new way of large scale culture maintaining totipotency without contacting feeder cells indispensable for primate ES cells. Also, our induction method for selective neuronal differentiation without co-culturing has become so sophisticated as to differentiate super selectively into dopaminergic neurons, although immunostaining on differentiated cells at beginning suggested that dopaminergic neurons had been induced only about 50% or more. In vivo transplantation and evaluation have started, but differentiation of other types of neurons, induction of neural network formation and fully electrophysiological functioning must be established before therapeutic application.

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OB2-02 Grafted neuroepithelial stem cells in the rat striatum differentiate to multiple neurons and receive synaptic inputs from the host area

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Morphological and slice-patch-clamp analyses were carried out to investigate the fate of neuroepithelial stem cells, obtained from GFP-carrying rat embryos and grafted in the striatum. Four to six weeks after the implantation, tyrosine hydroxylase-positive neurons were observed among donor-derived cells. In addition, 6 months after the grafting, DARPP-32-positive or ChAT-positive neurons also appeared in these cells. The donor-derived cells generated action potentials 4-6 weeks after the implantation, and both glutamatergic and GABAergic postsynaptic currents were evoked in these cells by the electrical stimulation applied to the host area. These results suggest that grafted neuroepithelial stem cells differentiate to functionally active neurons in the host brain and make synaptic contacts to the environment.

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OB2-03 Vascular Adventitia Generates Neuronal Progenitors in the Monkey Hippocampus after Ischemia

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Recently, we demonstrated in monkeys that transient brain ischemia induces an increase of the neuronal progenitor cells in subgranular zone (SGZ) but not in CA1. Although adult neurogenesis in SGZ occurs within an angiogenic niche, light microscopic observation can hardly confirm the exact origin of in vivo primary precursors. Ultrastructural observation appears to be indispensable for the precise identification in situ. Here, after confirmation of BrdU-, and neuronal- or glial maker- double-positive proliferating cells in SGZ of the postischemic monkeys by laser confocal microscopy, we investigated the same specimen by immuno and conventional electron microscopy. While some BrdU-positive cells were endothelial cells, we found in primates that many BrdU-positive cells originated from or were closely related to the vascular adventitia. The adventitia in SGZ showed high levels of brain-derived neurotrophic factor and polysialylated neural cell adhesion molecule, in marked contrast to that of CA1.

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OB2-04 Diphohoinositol pentakisphosphate (IP7) is a mediator as cell death

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Inositol phosphates occur in many animal and plant tissues. Higher inositol phosphates containing energetic pyrophosphate bonds have been characterized, especially diphohoinositol pentakisphosphate (IP7). IP7 is formed by a family of inositol hexakisphosphate kinases which have three distinct subtypes (IP6K1, K2, and K3). However, physiological functions of inositol pyrophosphates such as IP7 have not been extensively explored. We examined the effect of IP7 on cellular functions employing overexpression systems of IP6K1, K2, and K3. Under stress conditions, many cells overexpressing IP6K1, K2, K3 exhibited reduced viability compared to the cells overexpressing a control vector. Notably, IP6K2 overexpressing cells displayed the largest number of dead cells. These findings suggest that IP7 is a mediator of cell death.

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OB2-05 Glutamate-induced caspase activation in acute and delayed neuronal death

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Objective Since the mechanisms underlying the delayed neuronal death involve apoptotic process, glutamate may activate caspase-3. We examined the effects of brief (20 min) and continuous exposure to glutamate on the activation of caspase-3 in rat cultured hippocampal neurons. **Methods** Morphological changes in the neurons were observed with a video-enhanced contrast-differential interference contrast microscope. Activations of caspase-3 were analyzed using a fluorescent cell-permeable caspase-3 substrate FAM-DEVD-FMK. **Results** 20-min exposure to glutamate induced delayed neuronal death and significantly activated caspase-3 in 6 h in a dose-dependent manner. Interestingly, the continuous exposure for 1 h, which induced necrosis, also significantly activated caspase-3. **Conclusion** Caspase pathway is activated during acute as well as delayed neuronal death induced by glutamate. Even in the necrosis, apoptotic signal is already activated but covered with the acute cell death.

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OB2-06 Bax overexpression reveals two distinct modes of programmed neuronal death in the chicken embryo

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Bax is a pro-apoptotic protein that is required for programmed cell death (PCD) of many neuronal populations. To further clarify the role of Bax on developing neurons, we have investigated the action of Bax in several different neuronal populations using a viral delivery model. In the neural retina and the dorsal root ganglia (DRG), Bax overexpression enhances PCD of neuronal populations selectively undergoing PCD. In contrast, Bax overexpression does not enhance an early phase of motor neuron (MN) death in the cervical spinal cord. However, overexpressed Bax appears to be activated in dying MNs which exhibit a punctate Bax distribution with caspase-3 activation as observed in the retina and the DRG. Thus, Bax appears to act as a proapoptotic molecule selectively in neurons exposed to death signals. Furthermore, although Bax appears to mediate the execution pathway for PCD, the effect of Bax overexpression on susceptibility to death is distinct among different neuronal populations.

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OB2-07 Anti-apoptotic effect of the NAIP up-regulating dopamine receptor ligands

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Neuronal apoptosis inhibitory protein (NAIP) is an anti-apoptotic protein with therapeutic potential in neurodegenerative disorders. Exploring the compounds that up-regulate endogenous NAIP is one of the attractive steps to develop therapeutic agents for the intervention or even cure of such diseases. Here, using the NAIP-ELISA-based drug screening system, we identified several candidate compounds, and characterized one of the compounds, a dopamine receptor ligands, exhibiting most prominent effects in vitro. This compound specifically up-regulated the NAIP level and prevented the cultured cells from an oxidative stress-induced apoptosis. Moreover, administration of the compound attenuated the ischemic damage with concomitant increase in NAIP in vivo. These results suggest that NAIP upregulating dopamine receptor ligands is a potential drug in preventing disorders associating with oxidative stress -induced neuronal cell death.

OB2-08 Nipradilol inhibits glutamate-induced apoptosis in cultured cortical neurons via nitric oxide-dependent mechanism

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Nipradilol has α_1, β-adrenoceptor antagonistic properties with a nitric oxide (NO) donative action from its nitroxy moiety and is used clinically as an antiglaucoma ophthalmic agent. It is also known that nipradilol prevents *N*-methyl-D-aspartate-induced retinal cell damage, but underlying mechanism is unknown. In this study, we investigated the effect of nipradilol on glutamate neurotoxicity using primary cultures of rat cortical neurons. When cultures were exposed to 100 μM glutamate for 24 hr, the cell viability was significantly reduced and this neuronal death had apoptotic features. Nipradilol inhibited glutamate-induced apoptosis in a concentration-dependent manner, whereas de-nitroxy-nipradilol did not show the neuroprotective effect. *S*-nitroso-L-cysteine and 8-bromo-cGMP also inhibited neuronal death. These results indicate that nipradilol protects cortical neurons from glutamate-induced apoptosis by release of NO and subsequent formation of cGMP.

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OB2-09 Apoptosis-Associated Tyrosine Kinase (AATYK) undergoes phosphorylation during neuronal apoptosis.

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We previously reported that AATYK expression was up-regulated in the LK-induced apoptosis of mature CGN and that the overexpression of exogenous AATYK increased the susceptibility to this apoptosis. To clarify AATYK signaling in this process, we analyzed phosphorylation states of AATYK. We found that changing the culture media from HK to LK induced AATYK phosphorylation that was blocked by LiCl. Interestingly, even at HK the treatment with nimodipine, an inhibiter for L-type voltage-dependent calcium channels, not only induced AATYK phosphorylation but also caused the apoptosis. These results indicate that AATYK has two phosphorylation states, dephosphorylation state at non-apoptotic HK and phosphorylation state at apoptotic LK. The dephosphorylation state at HK is likely calcium-dependent. In addition, the LK-induced phosphorylation state may be correlated with CGN apoptosis, which could be cancelled with lithium.

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OB2-10 Calpain-dependent proteolysis of p35 Cdk5 activator to p25 in ER stress-induced neuronal cell death

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Deregulation of Cdk5 by calpain-dependent limited proteolysis of neuron-specific Cdk5 activator p35 to p25 has been suggested to be a cause for neuronal cell death in neurodegenerative diseases. ER stress is also shown to induce neuronal cell death. However, it is unknown whether Cdk5 takes part in ER stress-induced neuronal cell 5death. Here, we investigated the deregulation of Cdk5 during neuronal cell death induced by ER stress. ER stress was loaded to cultured cortical neurons with Tunicamycin or Thapsigargin. The proteolysis of p35 to p25 was observed 3 days after treatment when neuronal cell death was induced. p25/Cdk5 relocated to nucleus in Tunicamycin or Thapsigargin-treated neurons. Olomoucine, a Cdk5 inhibitor, suppressed ER stress-induced neuronal cell death. These results suggest that Cdk5 may be involved in ER stress induced-neuronal cell death through the limited proteolysis of p35 to p25 accompanied by translocation of p25 to nucleus.

OB2-11 Humanin antagonists

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Humanin (HN) protects neuronal cells from insults of various Alzheimer's disease (AD) genes and Aβ by forming a homodimer. We previously showed that P3A, S7A, C8A, L9A, L12A, T13A, S14A, and P19A mutations nullify the neuroprotective function of HN. We here reported that HN neuroprotection was blocked by P3A, L12A, S14A, and P19A mutants, but not by S7A or L9A mutants, which are the homodimerization-defective mutants. P3A-, L12A-, S14A-, and P19A-HN could not inhibit neuroprotection by EFLIVIKS (a homodimerization tag)-fused HN. Furthermore, insertion of S7A, the mutation that inhibits the homodimerization of HN, but not of G5A abolished the antagonizing function of L12A-HN. While L12A-HN and G5A/L12A-HN actually inhibited HN homodimerization, S7A/L12A HN had no effect. These data indicate that P3A-, L12A-, S14A-, and P19A-HN function as HN antagonists by forming an inactive dimer with HN. This study will provide a novel insight into the understanding of the in vivo function of HN.

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OB2-12 Identification of sympathetic premotor neurons in medullary raphe regions mediating fever and other thermoregulatory functions

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Medullary raphe regions are thought to contain sympathetic premotor neurons for thermoregulatory functions. Here we found that pyrogenic stimulus or cold-exposure induced Fos expression in vesicular glutamate transporter (VGLUT) 3-expressing neurons in rat medullary raphe regions, and most of these neurons were non-serotonergic. These neurons were transneuronally labeled with pseudorabies virus from thermoregulatory organs. Furthermore, these neurons directly projected onto sympathetic preganglionic neurons in the intermediolateral cell column (IML), and VGLUT3-positive terminals in the IML formed asymmetric excitatory synapses. These results suggest that these VGLUT3-expressing neurons constitute a novel group of sympathetic premotor neurons for fever and other thermoregulatory functions.

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OB2-13 Spatio-temporal pattern of spontaneous burst activity in the limbic system and its relation to respiratory activity in the isolated brain preparation from newborn rat

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We analysed spontaneous neuronal activity in the limbic system and its relation to respiratory activity in a diencephalons-brainstem-spinal cord preparation from 0-1day-old rats. For optical recordings, the preparation was stained with a voltage-sensitive dye. We optically detected spontaneous burst activity that initially appeared in the piriform cortex and then propagated to the amygdala. We also detected spontaneous hyperpolarization in these area with lower frequency of the occurrence. These burst and hyperpolarizing activity were also confirmed by electrophysiological recordings of population activity of the neurons in these area. The optical recording analysis suggested that the rhythmic burst activity tended to synchronize with spinal inspiratory activity. The detailed condition for this synchronization between limbic system activity and respiration could be determined by combining electrophysiology and optical recording.

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OB2-14 Substance P responsive neurons in the ventral medulla regulate nauseous autonomic reactions in rats.

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We examined the response of neurons near the nucleus ambiguus (NA) to the administration of apomorphine, emetic agent, and the electrical stimulation of the efferent fibers from the nucleus tractus solitarii (NTS). Intravenous administration of apomorphine (0.1mg/kg) induced excitatory and/or inhibitory responses of neurons adjacent to the NA in anaesthetized rats. We further determined the effects of the electrical stimulation of the efferent fibers from the NTS and the bath-application of substance P (SP) on neurons adjacent to the NA by using whole-cell recording in a slice preparation of young rats. Some of these neurons during pulse-train stimulation (50Hz, 1s) or SP application (5μM) showed long lasting responses. These results suggest that nausea induced by the apomorphine administration are mediated by SP responsive neurons near the NA.

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OB2-15 Odor restores hair cycle delay caused by immobilization stress in mice.

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Stress has long been suspected to cause human hair loss despite the definitive clinical evidence was absent. In this study we have found that chronic immobilization stress for 2weeks (2hours/day) delayed hair cycle, and prolonged resting (telogen) phase in mice. This delay was monitored even by 3days stress period at the shortest. We have explored the possibility to restore this stress-induced response by odor exposure. Among various odorants, valerian (plant root) oil odor has been reported to have sedative effect on both human beings and mice. When the mice were exposed to this valerian oil odor during immobilization stress, their active (anagenII) hair ratio significantly increased compared to that of mice exposed to the vehicle (mean±SE ; 73±11% vs. 40±10%, n=16, $p<0.05$). These results suggest that odor may controls the hair cycle which is subjected to the emotional stress.

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OB2-16 Localized peripheral inflammation augments brain prostaglandin E_2 formation through interleukin-6 signaling

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We examined if localized inflammation activates brain prostaglandin E_2 biosynthesis through interleukin-6 (IL-6) signaling. Rats were injected with lipopolysaccharide (LPS) or saline into a sterile, subcutaneous air-pouch. They were killed 3 h after the injection, and cerebrospinal fluid (CSF), plasma, subarachnoidal blood vessels (SABV), and brain were sampled. LPS injected rats showed elevated plasma levels of IL-6, PGE_2 levels in CSF, PGE_2 secretion from SABV when incubated *ex vivo*, and expression of cyclooxygenase-2 and microsomal PGE synthase-1 (enzymes for PGE_2 biosynthesis) in brain endothelial cells and SABV. Intraperitoneal injection of a neutralising antiserum raised against rat IL-6, 2 h before injection of LPS, significantly reduced all of the above responses. These results indicate localized inflammation augments brain PGE_2 formation through IL-6 signaling.

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OB2-17 Effects of spontaneous and forced running on induction of Fos expression in hypothalamic paraventricular nucleus of rats.

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We examined the effects of acute spontaneous and forced running on activation of the paraventricular nucleus of hypothalamus (PVN) in rats. To determine neuronal activation we assessed the expression of Fos immunoreactivity in the PVN. Rats were randomly assigned to two groups, i.e., spontaneous running group (SP) and forced running group (FO), and performed an 1-h free wheel running either spontaneously or compulsively. Running distance was not significantly different between groups. Forced running induced a significant increase in the number of Fos positive cells in the PVN. On the other hand, spontaneous running slightly activate Fos expression in the PVN. The mean number of Fos positive cells of the PVN in SP was significantly lower than that in FO ($p<0.05$). The results indicate that neuronal activation of the PVN by running occurred specifically with forced running, suggesting that forced running could be more mentally stressful than spontaneous running.

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OB2-18 Neural Activity Associated With Risk Anticipation: An fMRI Study

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Risk anticipation is an important cognitive/emotional component of decision making. The Iowa Gambling Task (Bechara et al., 1994) is the most widely used "risk-anticipation task" in clinical studies. However, the neural circuitry involved in this complex task has not yet been fully clarified. Using a 3T scanner, we performed an event-related fMRI study in 14 healthy subjects performing the task. The statistical parametric mapping showed that the risk anticipation component exclusively activated the medial frontal gyrus. Furthermore, we found a significant inter-individual correlation between the task performance and the brain activity during risky decisions. These results indicate that the Iowa Gambling Task does recruit the neural circuitry that is critical in decision making under uncertainty, particularly when subjects perceive the risk of their decision.

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OB2-19 The role of the central nucleus of the amygdala in arousal response evoked by hypertension and hypotension in rats

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We examined the effects of hypertension and hypotension on activation of the amygdala and brain stem in rats. To determine the neuronal activation in response to changes in blood pressure (BP) we assessed the expression of Fos immunoreactivity in the central nucleus of the amygdala (CeA) and brain stem regions including locus coeruleus (LC), dorsal raphe (DR), and laterodorsal tegmental nucleus (LDTg). Hypertension or hypotension was produced by midodrine (5mg/kg, ip) or hydralazine (10mg/kg, ip). Hypotension caused intensive activation of cortical electroencephalogram (EEG) compared to hypertension. Hypotension induced a significant increase in the expression of Fos immunoreactivity in CeA, LC and LDTg. On the other hand, although hypertension induced a small increase in Fos expression in CeA, we found few of the Fos positive cells in LC, LDTg and DR. These results suggest that hypotension could cause cortical activation through the neural pathway containing CeA, LC and LDTg.

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OB2-20 The role of the activation of NMDA receptor in two types carbachol-induced βoscillation

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Hippocampus receives the cholinergic and GABAergic fibers from the medial septum (MS). These fibers contribute to generate hippocampal β rhythm. We have already reported that the application of carbachol (30 μM), a cholinergic agent, could induce β oscillation (osc) whose frequency was 15.1 ± 1.3 Hz (carb β; mean ± s.e.m.) in rat hippocampal slices. The application of 30 μM carbachol plus 5 μM bicuculline also induced β osc. Its frequency was 14.3 ± 2.1 Hz (carb-bic β). Both β osc appeared intermittently with the interval of 15-60 sec. In the present paper, the results are as follows. 1) Pre-treatment of 100 μM AP5, an NMDA receptor antagonist, suppressed the induction of both carb β, and carb-bic β. 2) Once carb β was induced, AP5 did not affect the osc, while in the case with carb-bic β, AP5 increased the frequency. These results suggest that the activation of NMDA receptor is necessary to induce hippocampal β osc. It is also suggested that GABAergic fibers from MS to hippocampus will modulate the activation of NMDA receptor in hippocampal β osc.

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OB2-21 Method for Continuous measurement of renal sympathetic nerve activity in conscious mice

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In the present study, we have succeeded in measuring renal sympathetic nerve activity (RSNA) in freely moving mice (C57BL/6J). The left kidney was exposed retroperitoneally and the sympathetic nerves running on or beside the renal artery were identified. Then the two tips of the stainless steel electrodes were attached to the nerve and embedded in a two-component silicone gel. Thereafter the incision was closed. The RSNA was recorded successfully without contamination by external noise, such as that from the electrocardiogram and/or electromyogram, over 3-5 days after the surgery. The RSNA activity increased by ~ 240% during movement relative to the quiet awake state and was suppressed completely by the administration of hexamethonium. Thus, this novel method for measuring the RSNA would be useful to elucidate the role of sympathetic nerve activity in regulating cardiovascular function in genetically modified mice.

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OB2-22 Regulation of the firing of PS-on neurons by GABAergic inhibition

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Paradoxical sleep (PS) is regulated by the cholinergic neurons in the brainstem. Some of them are specifically active during PS and are completely or almost silent during waking (PS-on neuron). The monoaminergic neurons in the brainstem, which are active during waking, suppress or stop the firing of the PS-on neurons. $GABA_A$ antagonist, bicuculline, when injected into the brainstem, induces PS, suggesting that GABAergic inhibition have some roles in regulation of PS. Here, we compared the effects of noradrenaline (NA) and GABA on the cholinergic PS-on neurons. NA and GABA, when applied iontophoretically adjacent to the neurons, suppressed the firing of PS-on neurons during PS. The inhibitory effect of NA was cancelled by pre-application of α_2 antagonist, idazoxan, however the cessation of firing of PS-on neurons during waking was recovered by bicuculline but not by idazoxan. GABAergic inhibition has more crucial influences than noradrenergic one on suppression of the firing of PS-on neurons during waking.

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OB2-23 Brain-state-dependent synchronized oscillation in prefrontal cortex

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Various oscillations are observed depending on brain-states. In this study, we propose that spike-wave complex (SW), 7-11Hz rhythmic activities with harmonics, is one of the brain-state-specific oscillations. We recorded local field potentials and multi-unit activities (MUAs) in the medial prefrontal cortex and electroencephalograms in the bilateral frontal regions of rats. Occurrence probabilities of SW significantly increased after the rats performed cognitive tasks and they frequently fell asleep following the SW. These results indicate that SW occurs depending on vigilance levels. We also observed that each epoch of the SW lasted only several seconds and MUAs were organized into phase-locked patterns in oscillatory cycles of the SW. These results suggest that active networks during the oscillation function as attractor networks in a specific brain-state.

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OB3-01 Brain activations during motor imagery of standing and assuming of standing postures

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Positron emission tomography (PET) was used to study brain activations during motor imagery of standing and during performance of standing postures. Twelve healthy human subjects performed the following tasks during lying (1-3) and standing (4-5): 1) staring at a marker, 2) staring at a standing human object, 3) imagery of standing, 4) standing with eyes open, 5) standing with eyes closed. Regional CBF data were analyzed by SPM99. Compared with task 2, the right orbitofrontal cortex, left foot region and precuneus were significantly activated during imagination of standing. Comparison of assuming standing posture with its imagery yielded increases in CBF in the cerebellar vermis (flocculus) and precuneus. The superior parietal-precuneus CBF correlated with the Romberg ratio measured by gravicorder in eye-closed standing. The cerebellar vermis engages in postural balance and the precuneus may subserve as a higher brain center of standing in humans.

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OB3-02 Bilateral activation of movement-related neurons in the ventral premotor cortex reflecting coordinate transformation in reaching

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It has been controversial whether cortical motor areas, such as the primary motor cortex (MI) and the ventral premotor cortex (PMv), of primates contribute to bilateral, rather than contralateral, forelimb movements. Three monkeys were trained to perform a reaching task by either left or right hand, that was instructed by one of two tones in each trial. While recording neuronal activity, shift prisms were attached to their eyes to dissociate visual from motor space. Quantitative and statistical analyses revealed that the movement-related activity reflecting the visual space (V neuron) was recorded in the PMv, but seldom in the MI. The V neurons were often active before both left and right hand movements, whereas the PMv and MI activities reflecting the motor space were active almost only before the contralateral hand movements. The results suggest that the V neurons represent an early stage of motor command which are further transformed to the final motor commands.

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OB3-03 Processing of cortical inputs in the Striatal local circuts

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The striatum, an input stage of the basal ganglia, is thought to process the convergent cortical inputs. The purposes of this study are the quantitative investigation of intrastriatal connections and depict the local circuitry. We made the paired whole-cell recordings (n = 346) from corticostriatal slices. We found that the feed-forward inhibition of medium spiny neurons by a network of fast-spiking interneurons dominates the local striatal circuitry, and that medium spiny neurons within a sphere of ~150 μm in diameter are under inhibitory control. The inhibition mechanism detects synchrony of the cortical inputs and facilitates the synchronous firing of medium spiny neurons, which significantly improves the signal-to-noise ratio of the striatal outputs.

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OB3-04 Dexterous Finger Movements in Primate after Lesion of the Direct Corticomotoneuronal Connection

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Based on the previous studies on pyramidotomy, it is believed that independent finger movements (IFMs) are controlled by the direct (monosynaptic) cortico-motoneuronal (CM) pathway in primates. However, pyramidotomy additionally interrupts several corticofugal connections to the brain stem and spinal cord. To clarify the role of indirect (oligosynaptic) CM pathways, complete transection of the direct CM pathway was made at C4/C5 in three adult macaque monkeys. Electrophysiological recording revealed lack of monosynaptic CM excitation, but intact disynaptic CM excitation. IFMs were observed already after 1-28 days postoperatively. These results suggest that indirect CM pathways are important for neuro-rehabilitation.

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OB3-05 BILATERAL MST LESIONS SEVERELY IMPAIRED SHORT-LATENCY VISUAL TRACKING EYE MOVEMENTS

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Recent studies revealed three distinct visual tracking eye movements with ultra-short latencies (~60ms in monkeys), which are thought to help reduce the visual disturbances that we experience as we move around the environment. One of these eye movements is 'ocular following', which deals with the visual stabilization problems confronting the observer who looks off to one side. Two other eye movements, 'disparity vergence'; and 'radial-flow vergence', deal with the binocular fusion problems of the observer who looks in the direction of heading. To understand the role of the MST in eliciting these eye movements, we examined the effects of injecting ibotenic acid into the MST bilaterally in five monkeys. When ibotenic acid (15 mg/ml) was injected for a total of 16-19 injections for each monkey (32-38 µl total), we observed significant attenuation in the initial responses. The data support the hypothesis that MST neurons have some role in producing all three visual tracking eye movements at ultra-short latencies.

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OB3-06 The oscillatory synchronization in retinal ganglion cells plays a key role in visually evoked escape behavior

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An expanding dark spot presented on LCD triggered the escape behavior in frogs. This behavior was significantly suppressed by injection of bicuculline, a $GABA_A$ receptor antagonist, into both eyes, whereas facilitated by injection of TPMPA, a GABAc receptor antagonist. However, either antagonist did not affect the optokinetic responses (pursuit head movement) to moving stripes. OFF-sustained ganglion cells (dimming detectors) in the frog retina are assumed to be related to the escape behavior. Thus, spike discharges to the expanding dark spot were recorded from multiple dimming detectors in the isolated retina with a planar multi-electrode array. The strength of oscillatory synchronization was reduced by bicuculline but enhanced by TPMPA. Either antagonist increased the number of spikes and occurrence of synchronization. We conclude that the oscillatory synchronization is essential for the escape behavior.

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OB3-07 Neurons in the area MST represent visual movements in world-centered coordinates.

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We are usually unaware of retinal image motions resulting from our own eye movements. For instance, during smooth pursuit, the world around us remains perceptually stable, although the eye movements produce slips of the visual scene on our retinae. To clarify the role of the parietal cortex in this stability, we recorded neuronal activity in the areas MT and MST of monkey cortex, when the background was moved at various velocities either during fixation or pursuit. During pursuit, the retinal motion of the background is the difference between the eye and background motion. We found that most of the MT neurons responded in relation to the velocity of the retinal image motion, on the other hand, most of the MST neurons responded in relation to the velocity of the background motion in world-centered (or head-centered) coordinates. Our data suggest that when information is processed from the MT to MST, a transformation from the retinocentric to the non-retinocentric (world-centered) coordinate takes place.

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OB3-08 Contribution of Pedunculopontine tegmental nucleus neurons to motivational state during visually guided saccade tasks

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The cholinergic pedunculopontine tegmental nucleus (PPTN) is one of the major ascending arousal systems in the brain stem, and is linked to motor and limbic systems. In order to investigate contribution of PPTN neuron activity to motivation, we changed the volume of juice reward for success trials of visually guided saccades in monkeys, and compared the reward volume in each trial with task performance and the PPTN neuronal activity in the following trial. Increase in the reward volume enhanced the tonic PPTN neuron activity, decreased the error rate in the next trials. Thus, PPTN neurons are suggested to be involved in the process by which motivation for reward drives the animal to perform the task more efficiently.

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OB3-09 Oculomotor vermis hypothesis applied to the acceleration and erasure of saccadic adaptation

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The core of the oculomotor vermis hypothesis (OVH) is a concept of "associative motor learning (Fujita, 2003)". Primary motor commands will gradually progress, taking in by association part of the subsequent motor commands for corrective movements in training. The cerebellum will form the association whose strength will be adjusted by LTD of the transmission efficacy from parallel fibers to Purkinje cells. Here this theory is challenged by new phenomena. One is the selective acceleration of gain-decrease in a horizontal but not a vertical component of saccades during gain-decrease adaptation of oblique saccades. Subjects were processed beforehand by a series of gain-decrease, -increase adaptive procedures (Kojima et al. 2003). Another is the erasure of adapted gain-change through repetitive trials in which the second target was presented at a perturbed location within one degree of the post-saccadic eye position to induce small corrective saccades. These data suggests the extension of LTD and de-LTD process on the OMV, and are fully accounted by OVH.

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OB3-10 Functional significance of aldolase C expression pattern in the cerebellar cortex in its relationship to the olivocerebellar projection

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Aldolase C (=zebrin) expression in Purkinje cells delineates stripe-shaped compartments in the cerebellar cortex. We identified olivocerebellar projections to every aldolase C compartment by labeling with biotinylated dextran injected into various small areas within the inferior olive in rats. Based on the localization of olivary origins of the projection to similar compartments, the aldolase C compartments and olivary projections could be sorted into a small number of groups. Parts of the inferior olive belonging to each group receive specific afferent inputs, indicating functional cerebellar partitions linked with aldolase C compartments. In short, aldolase C negative compartments receive somatosensory inputs and aldolase C positive compartments receive vestibular, superior collicular, accessory optic, and mesodiencephalic inputs. The results indicate the integration of molecular, topographic, and functional organizations of the cerebellum.

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OB3-11 Changes of Sleep EEG Dynamics in Rat Barrel Cortex Associated with Long-term Sensory Deprivation

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Sensory deprivation is known to alter the neural organizations involved for processing the deprived neural information especially in developmental process. However, long-term effects of the sensory deprivation have not yet been well-understood. In order to investigate this effect on the post-mature neural organizations and possible contribution of sleep to them, cortical EEG dynamics during sleep in adult rats were measured during up to a month, while somatosensory inputs were constantly deprived by cutting whiskers unilaterally. The spectral powers of EEG in the sense-deprived barrel cortex decreased in terms of their ratio to the intact during initial 10 days. Subsequently, the ratio was gradually restored to the initial level. The resulting change in EEG dynamics is supported to be caused by a certain change of neural circuits associated with the long-term sensory deprivation even after maturation.

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OB3-12 Over-expression of BMAL1 suppressed circadian cytosolic Ca^{2+} rhythms in cultured suprachiasmatic nucleus neurons

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The core circadian oscillation is proposed to be generated by transcription-translation feedback loops of "clock genes" in the suprachiasmatic nucleus (SCN) in mammals. We have demonstrated that TTX-independent circadian oscillations in cytosolic Ca^{2+} levels may link the core movements to physiological activities of SCN neurons, yet genes controlling the Ca^{2+} rhythms are unknown. Here, we transfected bmal1 (a candidate core movement) and YC2.1 (a fluorescent Ca^{2+} indicator) genes into the identical SCN neurons by a "Gene-Gun", and observed over-expression of BMAL1 in the nucleus of YC2.1-positive SCN neurons. Since the over-expression of BMAL1 suppressed circadian cytosolic Ca^{2+} rhythms in SCN neurons, we concluded that endogenous BMAL1 oscillations are essential for the formation of circadian Ca^{2+} rhythms and thus for diverse physiological rhythms in SCN neurons.

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OB3-13 Modulation of the circadian change of *Bmal1* promoter activity by ROR response element binding factors

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To investigate the molecular mechanism of the circadian system, we attempted to establish a screening method to identify clock-controlling factors.The transient reporter assays using mouse *Bmal1* promoter constructs showed that RORαis the most potent activator of *Bmal1* transcription. Using a real-time monitoring system for *Bmal1* promoter activity, we examined whether the factors that regulate *Bmal1* transcription affected the circadian changes in the reporter activity of the *Bmal1* promoter. The real-time monitoring system showed that the transient co-expression of RORα, RORβ, and Rev-Erbβ with the *Bmal1* reporter construct in NIH3T3 cells abolished the circadian changes of the reporter activity. These results indicate that ROR α, ROR β, and Rev-Erbβ affect not only the level of *Bmal1* transcription, but also its circadian expression.

OB3-14 Disruption of synaptic transmission through *period*-expressing cells blocks retrieval of long-term memory in *Drosophila*

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We have measured LTM by courtship conditioning and reported that a circadian clock gene, *period* (*per*) mutations inhibit formation of LTM, which is rescued by induction of a *per* transgene in a *per* null mutant (24TH, 25TH and 26TH Annual meeting of the Japan neuroscience society). We next used the GAL4/UAS system to demonstrate whether disruption of synaptic transmission through *per*-expressing cells affects LTM formation. The yeast transcriptional activator, GAL4, activates UAS-linked target genes. The *Drosophila shibire* (*shi*) gene encodes DYNAMIN, which is essential for synaptic vesicle recycling. A temperature-sensitive allele, shi^{ts1}, is defective in synaptic vesicle recycling at restrictive temperatures (30°C). In F1 males between *per*-Gal4 and UAS-shi^{ts1}, shi^{ts1} is expressed in *per*-expressing cells. In these males, we found that synaptic transmission through *per*-expressing cells is required during retrieval of LTM.

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OB3-15 Neural correlates of recognition memory for pictures - an fMRI study-

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A number of neuroimaging studies have indicated a role of hippocampus (HIP), and prefrontal cortex (PFC) in memory retrieval. In the present event-related fMRI study of picture recognition, BOLD signal changes in these regions were examined in seven normal healthy subjects. The subjects encoded pictures under deep (D) or shallow (S) condition, and were scanned using 3T MRI system while making a recognition judgment for new, old and repeated items. The imaging data were analyzed using SPM2 with a random effects model. The subtraction between old and new items (successful retrieval) showed activation in left PFC, posterior cingulate gyrus and parietal lobe. Anterior HIP was significantly associated with successful retrieval under D condition, while posterior HIP predominantly responded to new items. The results indicate a differential role of anterior and posterior HIP in episodic retrieval and perceptual priming, and an involvement of left PFC in memory success.

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OB3-16 Morphometric analysis of hippocampal mossy fiber synaptogenesis in spatially learning mice

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In the mouse hippocampal CA3 region, dentate granule cells project mossy fibers (MFs) mainly to apical dendrites of pyramidal cells. However, in spatially learning mice, MFs forms new synapses in the stratum oriens on basal dendrites of CA3 pyramidal cells. Here, morphometric analyses were carried out to reveal where new projections are induced along septo-temporal axis of the hippocampus. Mice strain C57BL/6 were trained by Morris water maze between P21 and 27. ether-anesthetized, and fixed for the brain. The MF trajectories were examined on septo-temporal serial sections of hippocampus by Timm staining method. Enlargement of MF projection area were observed in septal hippocampus of mice with spatial memory whereas not in middle to temporal hippocampus. MF synaptogenesis appears to be accompanied with spatial memory establishment.

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OB3-17 Lesion effects of inferotemporal subdivisions on associative memory in monkeys

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Associative memory was assessed by a mixture of object-delayed matching and nonmatching-to-sample with increasing delays (10-600sec) and lengthened lists (3-10 objects): in the test phase, monkeys had to select either matching or nonmatching rule, depending on whether the sample objects were baited or not in the sample phase. Four groups were tested: normal controls, monkeys with lesions of either gyral TEO, new TEO, or anterior TE (TEa) cortex. The dorsal and ventral borders of gyral TEO were defined as the lip of the superior temporal sulcus and the lateral bank of the occipitotemporal sulcus (ots), respectively, whereas new TEO was defined as expanding ventromedially beyond the ots. New TEO lesions produced significant impairments which were comparable to those following TEa lesions. Gyral TEO lesions produced no significant change. The present findings suggest the involvement of the posterior inferotemporal lobe, in particular, its ventromedial portion, in mediating associative memory.

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OB3-18 Real-time sorting for sub-millisecond neuronal interactions *in vivo*

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Recent advancement of *in vitro* electrophysiological technique allows us to investigate electrically coupled cells via gap junctions and dendritic backpropagations of spikes. However, such sub-millisecond activity interactions among neurons have not been reported in *in vivo* studies. Here, we introduce an *in vivo* electrophysiological system that can detect sub-millisecond interactions among closely neighboring single-neurons in real-time, using a combination of independent component analysis (ICA) and newly developed multi-electrodes. The system could record up to 10 interacting single-neurons simultaneously with multi-electrodes implanted in the hippocampal formation and the somatosensory cortex in each rat. The result suggests that the system detect dendritic backpropagations of spikes and electrically coupled cells via gap junctions. These results demonstrate that the system can be employed to investigate precise neuronal interactions, as well as the functions of dendritic compartments in behaving animals.

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OB3-19 Neuronal correlation in the monkey prefrontal cortex in working memory for stimulus duration

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Neuronal representation of stimulus duration in memory remains completely unknown. This study recorded activities of multi-neurons in the prefrontal cortex of monkeys while they were performing a behavioral task in which working memory for stimulus duration was needed. We have already described specific neurons whose discharge rates reflect encoding or retention processes of the duration of stimuli to be remembered (Sakurai et al., 2003). Here we describe correlation of the neuronal activities in the process of working memory. Most correlations among neurons detected by a single tetrode showed no change throughout the processes of working memory but some correlations among neurons detected by different tetrodes showed dynamic changes in the encoding, retention and comparison processes. These results suggest that almost neighboring neurons behave together but somewhat distant neurons have process-dependent functional connections with each other.

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OB3-20 Relationships between the hippocampus and the supramammillary nucleus for spatial encoding of a novel place in rats

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Since neurons in the supramammillary nucleus (SuM) are known to terminate on the principle cells of the dentate gyrus and of the CA2/CA3a region directly, we focus on the roles of SuM on the hippocampal excitability. In the present study, in order to elucidate relationships between the hippocampus and SuM in freely-moving rats, neuronal expression of c-Fos protein was quantified in the hippocampus of rats with lesion of SuM when they are placed in a novel place. The number of c-Fos positive neurons was significantly decreased in the dentate gyrus and CA3. In CA1, the number of the positive neurons showed a tendency toward decrease. In our previous study on behaviors of rats placed in an open field, we have demonstrated that there was a significant increase in ambulation in rats with SuM lesion. Together, lesion of SuM can lead to deficit of spatial encoding for a novel place through disruption of signal flow in the hippocampus.

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OB3-21 Hippocampal CA3 NMDA receptors are crucial for adaptive timing of trace eyblink conditioned response

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To examine the involvement of hippocampal CA3 NMDA receptors (NRs) in nonspatial associative memory, CA3-specific NR1 KO mice were subjected to eyeblink conditioning paradigms. We first confirmed that wild-type mice require an intact hippocampus in a 500 ms-trace conditioning. Under the same condition, CA3-NR1 KO mice were impaired in the formation of an adaptively-timed CR within the first few trials in the daily acquisition sessions, while the control mice could express adaptive CR from the beginning of these sessions. This impairment in the mutant was rapidly overcome by the following repetitive CS-US association. These results indicate that CA3-NR1 KO mice are unable to rapidly retrieve adaptive CR timing, suggesting that CA3 NRs play a crucial role in the memory of adaptive CR timing in trace conditioning.

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OB3-22 Rat hippocampal neuronal activity during a delayed stimulus-response association task on a mobile robot.

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Rats were placed on a treadmill to induce locomotion (LM), which was attached to a motion stage. The stage was translocated (TRANS) on an 8-shaped pathway, and stopped at 2 corners where a stimulus-response association task (TK) was imposed. Hippocampal (HF) neurons were recorded in the following 4 phases; 1) TRANS + LM + TK, 2) TRANS + LM, 3) TRANS, 4) TRANS + LM + TK. Of the 70 HF neurons analyzed, 32 displayed place specificity. Of these, activity of the 28 neurons increased in the same places in the phases 1 and 2 regardless of the TASK. Of these neurons, 11 showed the same place fields in the phases 2 and 3 regardless of LM, while 17 showed different place fields. The remaining 4 neurons showed a difference in phases 1 and 2. These results suggest multiple spatial and non-spatial representation in the HF, which might be the neural bases of human episodic memory.

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OB3-23 Glucose increase in CSF by food intake is necessary to learning and memory

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The glucose (G) concentration in CSF is 2-3 mM and increases twice by food intake. When G was injected in the rat hippocampus to turn to 7 mM in concentratuion, spatial learning and memory were facilitateed. Using hippocampal slices, G in perfusate was changed from 3.5 to 7 for 15 min and returned to 3.5 mM. Augmentation of CA1 synaptic potentials occurred by the change in G and continued after returning to 3.5 mM. LTPs produced by tetanic stimulations given the time just changing from 3.5 to 7 mM, were more facilitated than those produced in 3.5 mM. Presynaptic transmitter releases were also facilitated by 7mM. Postsynaptic responses to NMDA etc. applied to apical dendrites were slightly increased. Membrane potentials and input resistances were changed a little. The CaMII α and β and PKC were not changed by 7 mM. We are now measuring these changes during LTP. These indicate that food intake is necessary not only for keeping homeostatis of body machinery but also reinforcing the higher brain function.

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OG1-01 Three-dimensional curvature tuning in area CIP (caudal intraparietal) of macaque monkey

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The mechanisms underlying the reconstruction of 3D objects from 2D retinal images have been a central issue in neuroscience. It has revealed that neurons in area CIP selectively respond to axis orientation and surface orientation in 3D space, suggesting that the parietal cortex may play a crucial role in 3D object recognition. Here we report that CIP neurons are selective to 3D curvatures as well. A set of 3D curvatures (ellipsoids, cylinders and saddles), defined by equations of J. J. Koenderink, was presented in random dot stereogram to a monkey performing a fixation task. Some CIP neuron showed selective response to curvatures that contain convexity or concavity. Most of them had significant modulation to one of the shapes. A few neurons were highly selective to a certain shape. In addition, some shape selective neurons had modulation to maximum disparity and orientation of the stimulus. These results suggest that specific shapes of curvature are represented in CIP.

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OG1-02 Histochemical confirmation of binocular and monocular zones identified by intrinsic optical imaging in rat visual cortex

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We investigated the parcellation of visual areas in rats using intrinsic optical imaging and a histochemical analysis. First, we identified the binocular (Oc1B) and monocular zones (Oc1M) in the primary visual cortex with our previously reported method. Briefly, we visualized the neural activities evoked by the separate whole visual field stimulation to the left and right eyes with white light-emitting diodes using the intrinsic optical imaging technique. Then, the Nissl and cytochrome oxidase staining methods were applied to the recorded cortex. The border between Oc1B and Oc1M identified by optical imaging agreed with the boundary between Oc1B and Oc1M determined by the histochemical stainings. This good agreement justifies the reliability of intrinsic optical imaging for the identification of Oc1B and Oc1M.

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OG1-03 Functional Segregation of the Cortical Region Representing Cardinal Contours

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Our previous data suggested that, in cat area 17, the representation of cardinal orientations overlaps less to the representation of their nearby angles. To investigate the underlying single cell property, optical imaging was performed first to map the cortical regions corresponding to the four principal contours. The cortical region activated by a principal orientation but not by the + or -10-degree neighboring angles (RIOS) was mapped together with the regions that overlapped with the +10-degree and/or -10-degree neighboring angles (non-RIOS). A comparison between the RIOS and non-RIOS regions documented a significantly higher percentage of responsive cells in the cardinal RIOS region versus that seen in the other regions. Additionally, the cells in the cardinal RIOS region tuned sharper and the cells in cardinal non-RIOS region tuned broader than the oblique RIOS and/or the non-RIOS region. This data strongly suggests the existence of functional segregation in the region corresponding to the cardinal contours.

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OG1-04 Molecular organization of the ferret visual thalamus

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The visual system separates different types of information into parallel, anatomically distinct processing streams. In carnivores the three physiological retino-geniculo-cortical streams are known as X, Y and W. Despite their significance for visual processing, the molecular organization of these physiological streams is largely unknown. To identify molecular correlates of functionally distinct streams, we fabricated a custom cDNA microarray of the ferret, a highly visual mammal. By combining with fluorescent labeling and microdissection, we compared mRNA expression profiles between distinct layers in the LGN. We found molecules whose unique distribution and developmental profiles define the LGN itself, its constituent layers, or identify cells comprising one of the physiological streams in the LGN. These results suggest that a rich molecular repertoire exists which distinguishes functional structures in the LGN.

OG1-05

OG1-06 Generation and analysis of transgenic mice that express human α-synuclein in dopamine neurons

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We generated Tg mice expressing human α-synuclein (or NACP) containing an A53T mutation under the control of the rat tyrosine hydroxylase promoter. Human α-synuclein was expressed in dopamine neurons in the substantia nigra pars compacta. About 1/3 of α-synuclein-positive neurons were reactive to an antibody which specifically recognizes α-synuclein phosphorylated at Ser129. Phosphorylation at the residue was detected even at 8 weeks of age. However, age dependent increase in the number of antibody-positive neurons was not observed, suggesting that this modification is maintained to a certain level until the aged stages.

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OG1-07 Generation of transgenic mice that show a part of pathological features of Parkinson's disease

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We generated Tg mice expressing a truncated form (residue number: 1 to 130) of human α-synuclein (or NACP) under the control of the rat tyrosine hydroxylase promoter. The transgene product was localized in dopamine (DA) neurons in the substantia nigra pars compacta (SNc). The Tg mice showed significant loss of DA neurons in the SNc. Consequently, striatal DA level and spontaneous locomotor activity of the Tg mice were reduced. Thus, the Tg mice of this study show some of typical symptoms of Parkinson's disease, and may provide a useful tool to explore therapeutic possibilities for this disorder.

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OG1-08 Nigrostriatal dopaminergic degeneration causes motor dysfunction as detected by the tail suspension test in MPTP-treated C57BL/6 mice

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The systemic application of the neurotoxin MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) can lead to severe damage to the nigrostriatal dopaminergic system and cause concomitant loss of striatal dopamine contents in C57BL/6 mice. We have already presented the increase in duration of immobility in the tail suspension test in MPTP-treated mice. Here we showed the stimulation of dopaminergic activity induced by L-dopa or a dopamine D_2 receptor agonist ameliorated the increase in immobility time. At the effective dose of each drug, the locomotor activity was also increased. Threshold level of striatal dopamine that produced the increase in immobility time was estimated to be between 11 and 27% of control level. We concluded that the increase in immobility time in the tail suspension test was thought to be a motor dysfunction in this mouse model of Parkinson's disease.

OG1-09 Involvement of Neuromelanin in Selective Vulnerability of Dopamine Neurons in Parkinson disease

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In Parkinson disease selective degeneration of dopamine neurons in the substantia nigra and existence of specified inclusion bodies, Lewy bodies, are the characteristic feature. However, factors determining vulnerability of dopamine neurons have not been well elucidated. Neuromelanin (NM) is produced by dopamine oxidation and accumulated in the cells in an age-dependent manner. The effects on cellular function were studied using NM prepared from human brain and SH-SY5Y cells. NM reduced in vitro and in situ activities of 26S proteasome activity, as shown with the cell transfected with a proteasome sensor vector. These results may be relevant recent findings that accumulation and aggregation of abnormal proteins and impairment of ubiquitin-proteasome system may play a major role in the pathogenesis of PD. The selective death of dopamine neurons in aging and PD may be attributed to existence of neuromelanin.

OG1-10 Role of quinone formation in protective effects of parkin against manganese-induced dopaminergic cytotoxicity

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We have previously reported that transfection of parkin gene inhibited manganese (Mn)- or Mn plus dopamine (DA)-induced cell death of DAergic cells, but not of non-DAergic cells, suggesting involvement of some intrinsic molecules in the DAergic cells. In the present study, we revealed that Mn exposure showed same levels of increase in reactive oxygen species between DAergic or non-DAergic cells, and showed elevation of proteasome activities in neither cell. Mn-induced increase in PDI ER chaperones in DAergic cells were not changed by transfection of parkin gene. Pretreatment of DA reducer could prevent Mn-induced cell death of DAergic cells. Furthermore, transfection with antisense parkin cDNA increased formation of protein-bound quinones in DAergic cells. These results suggest that DAergic cell-specific attenuating effects of parkin against Mn-induced cell death are associated with DA itself and formation of quinones but not with free radicals, ER stress or proteasome system.

OG1-11 Axonal Regeneration and Functional Recovery after Administration of Strong and Selective Semaphorin3A Inhibitor into the Injured Spinal Cord

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Semaphorin 3A (Sema3A), a member of class 3 semaphorins, is one of the best characterized axonal guidance molecules, that exhibits inhibitory actions against axonal growth of certain subsets of neurons. The Sema3A expression is induced in fibroblast scar within the injured spinal cord, which is likely to inhibit the regeneration of the damaged axons. In the present study, we examined the blockade of Sema3A's action within the injured rat spinal cord, by developing its selective inhibitor, to induce the axonal regeneration and functional recovery.

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OG1-12 Ameliorative effects of withanolide derivatives on spatial memory impairment by neurite outgrowth and synaptic construction

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We aimed to clarify that withanolide A, withanoside IV and withanoside VI could induce neurite outgrowth and synaptic construction *in vivo*, and could improve memory deficit. I.c.v. injection of Aβ(25-35) (25 nmol) induced spatial memory impairment in mice. At 7 days after the Aβ(25-35) injection, treatment with each 3 compound (10 μmol/kg/day, for 13 days, p.o.) was started. All the compounds recovered the memory disorder to almost the control level. In brain slices of Aβ(25-35)-injected mice, length of axons and presynaptic density were decreased in the hippocampus and the cerebral cortex, compared with control mice. In each 3 compound-treated mice, however, axons and presynapses were almost equal to ones in control mice. These results suggest that these 3 compounds may be useful and powerful anti-dementia drugs which can construct the neuronal network.

OG1-13 Potent neuroprotective effect of CsJ, a novel cyclosporin A-analogue in ischemic brain damage

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Previous study demonstrated that immunosuppressant cyclosporin A (CsA) had potent neuroprotective effect in rat forebrain ischemia by inhibiting calcineurin and cyclophilin D. The inhibition of both enzymes leads to decrease intracellular calcium and inactivate calcium-dependent cell death effectors, and stabilized MPT (mitochondrial permeability transition). We discovered a novel natural peptides CsJ with three substitutions of amino acids of CsA, which showed potent neuroprotection and lower side effect than CsA in rat ischemic model. This compound would be a promising drug for treatment of not only ischemic tissue damages such as ischemic brain and heart diseases, but also degenerative diseases from the view of the molecular mechanisms.

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OG1-14 Clozapine but no haloperidol reverses the effect of MK-801 on neuropeptide-processing enzymes in rat brain regions

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Alzheimer's disease (AD) is a neurodegenerative disorder that is characterized by progressive cognitive impairment and neurodegeneration as well as decreased neuropeptide levels. However, the correlation between neurodegeneration & neuropeptide levels and their regulation by neuropeptide metabolizing enzymes is not clearly known. In the present study, we investigated the change of neuronal and glial cells as well as neuropeptide-degrading enzymes in different rat brain regions after systemic administration of MK-801 and protective effect of typical vs atypical antipsychotic drug has been tested against MK-801 toxicity. The present biochemical and immunohistochemical results suggest that glia activation might play an important role in AD pathology and corrlate with the level of neuropeptide and neuropeptide-processing enzymes that are recovered with clozapine.

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OG1-15 Distinct expression of calsyntenin family in β-treated neurons and its role in neuronal death

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β-amyloid (Aβ) may play an important role in pathogenesis of AD, because Aβ has toxic effect to cultured neuronal cells. To understand the molecular mechanisms of Aβ actions, we have applied cDNA subtraction between Aβ1-42 treated and non-treated cerebral cortical neurons to generate Aβ-induced cDNA pools. Up regulation of 3 genes, including calsyntenin-3, was confirmed by Northern blot and RTQ-PCR. The calsyntenin (Cst) protein family, Cst-1, Cst-2 and Cst-3, is the postsynaptic membrane protein in excitatory synapses. Northern blot analysis revealed up-regulation of Cst-3, down-regulation of Cst-2 and no alteration of Cst-1 in Aβ 1-42 treated neurons. Then we examined the effects of Cst-3 overexpression in cultured cortical neurons on cell death by serum withdrawal. Neurons overexpressing Cst-3 were more susceptible to apoptosis. These results indicate that Aβ differentially regulates the calsyntenin genes in cortical neurons and induces neuronal death via, in part, Cst-3 up-regulation.

OG1-16 Subcellular localization of γ-cleavage of βAPP.

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β-amyloid precursor protein (βAPP) is cleaved by presenilin (PS)-γsecretase at two or more different site to generate amyloid-βpeptide (Aβ) and AICD (dual-cleavage). It has not been concluded whether intracellular or extracellular Aβ cause Alzheimer's disease related toxicity. Indeed, even the subcellular localization of γ-cut of βAPP is still controversial. Recent studies on PS/γ-secretase complex suggest that γ-cut occur at or near the plasma membrane. We established cell-free γ-secretase assay of βAPP, by which we can determine the level and cleavage site of both *de novo* Aβ and AICD simultaneously. Using this assay, we separated the γ-cut on the plasma membrane from that on the intracellular membrane. Moreover we tested if γ-cleavage occur on the plasma membrane by inhibiting endocytosis through expressing dynamin 1 K44A mutant.

OG1-17 Characteristics of $A\beta_{1-40}$ efflux transport mechanism at the blood-brain barrier

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Amyloid β peptide (Aβ) efflux transport mechanism(s) is present at the blood-brain barrier (BBB), whereas its mechanism(s) remains unclear. The purpose of present study is to characterize the $A\beta_{1-40}$ efflux transport mechanism(s) from brain across the BBB by using intracerebral injection of $[^{125}I]A\beta_{1-40}$ (Brain Efflux Index method). $[^{125}I]A\beta_{1-40}$ was eliminated with a halftime of 44.8 min from brain. The efflux of $[^{125}I]A\beta_{1-40}$ was significantly inhibited by unlabeled $A\beta_{1-40}$, but not by $A\beta_{1-42}$, $A\beta_{40-1}$. To evaluate the possibility that complex formed Aβ was excreted, the effect of midkine (MK), an Aβ binding protein in senile plaques, was examined. The efflux of $[^{125}I]A\beta_{1-40}$ in the presence of MK was decreased than that in control. These results suggest that $A\beta_{1-40}$ efflux transport from the brain is mediated by the sequence and conformation sensitive transport system(s) and the efflux transport of $A\beta_{1-40}$ is suppressed by its complex formation with MK.

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OG1-18 Phosphorylation of tau by CaM kinase II in Alzheimer's disease brain

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The paired helical filaments (PHF) found in Alzheimer's disease (AD) brain are composed mainly of the hyperphosphorylated form of tau (PHF-tau). It was reported that CaM kinase II phosphorylates serine 416, numbered according to the longest human tau isoform. However, hyperphosphorylation of this site in PHF-tau has not been reported. In the present study, we produced a specific antibody against tau phosphorylated at serine 416. Immunoblot analysis revealed that the antibody reacted with purified tau only after phosphorylation by CaM kinase II. When the cytosol fraction from rat brain was prepared in the presence of protein phosphatases inhibitors, the antibody reacted with a fetal form of tau more strongly than adult forms of tau. Interestingly, PHF-tau was strongly phosphorylated at serine 416. These results suggest that CaM kinase II is involved in hyperphosphorylation of tau in AD brain.

OG1-19 C. elegans model of tauopathy

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We have constructed a C.elegans model of tauopathy. These lines of C.elegans expressed wild-type or FTDP-17 mutant tau (P301L and R406W) in the mechanosensory neuron. In comparison with wild-type tau transgenic worms, the worms expressing FTDP-17 mutant (P301L and R406W) tau showed severe dysfunction in touch response in an age-dependent manner. Morphological studies also showed severe degeneration of those mechanosensory neurons in mutant transgenic worms. These results indicate that the worm lines are useful for the investigation of the molecular mechanisms and pathways involved in the neurodegeneration associated with tauopathy. Breeding with ced 3-/- or ced 4-/- worms did not prevent the degeneration, strongly suggesting that the neurodegeneration is not through apoptotic process. Heat shock stress aggravated the touch sensitivity of tau transgenic worms, and overexpression of hsp 70 improved the responses.

OG1-20 Immature Nicastrin Stabilizes APH-1 independent of PEN-2 and Presenilin

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γ-Secretase, an aspartyl protease implicated in Alzheimer's disease, has recently been identified as a complex composed of presenilin (PS), nicastrin (NCT), APH-1 and PEN-2. To dissect functionally important domains of NCT, we mutagenized conserved amino acids. The mutant NCT variants were expressed in NCT knockdown cells and investigated for their ability to restore PS, APH-1 and PEN-2 expression and γ-secretase activity. We identified mutations in the ectodomain of NCT, which rescued expression of APH-1 but not of PEN-2 or PS and thus failed to restore γ-secretase activity. These immature NCT variants selectively bound to APH-1. Thus, immature NCT can stably interact with and stabilize APH-1 independent of PS and PEN-2. Apparently, binding of the latter two complex partners critically depends on the integrity of the NCT ectodomain. Further results on the functional characterization of NCT and APH-1 will be presented.

OG1-21 Potent anti-amyloidogenic and fibril-destabilizing effects of red wine-related polyphenols in vitro

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A consistent protective effect of wine consumption on AD has been documented by epidemiological studies. In the present study, we used fluorescence spectroscopy with thioflavin T and electron microscopy to examine the effects of wine-related polyphenols on the formation, extension, and destabilization of beta-amyloid fibrils (fAbeta) in vitro. All examined polyphenols dose-dependently inhibited formation of fAbeta from fresh Abeta, as well as their extension. Moreover, these polyphenols dose-dependently destabilized preformed fAbetas. In cell culture experiments, polyphenol-treated fAbeta were suggested to be less toxic than intact fAbeta. Wine-related polyphenols could be a key molecule for the development of preventives and therapeutics for AD.

OG2-01 A novel intercellular communication system, the CD47-SHPS-1 system, in neuronal network

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SHPS-1 and CD47 are receptor-type membrane proteins, which are highly expressed in the brain. In cultured neurons, SHPS-1 and CD47 seem to constitute a directional cell-cell communication system (the CD47-SHPS-1 system) between dendrite and axon. We also found that exogenously expressed SHPS-1 and CD47 were markedly accumulated at the contact sites of neurites, which were derived from neurons transfected separately, indicating that the interaction of these molecules affects their localization. Furthermore, we found that CD47 can promote neurite extension and filopodium formation through the interaction with SHPS-1 in neuroblastoma cells, N1E-115. Our results suggest that the CD47-SHPS-1 system can regulate neuronal morphology at the interaction site between axon and dendrite through their polarized localization.

OG2-02 Arx homeobox gene is essential for development of mouse olfactory system

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In this study, we elucidated roles of Arx homeobox gene in the developing olfactory system by analyzing its mutant phenotypes. Arx protein was expressed in interneurons and radial glia of the olfactory bulb, but neither in bulbar projection neurons nor olfactory sensory neurons. Arx-deficient mice showed severe abnormalities in the olfactory system: (1) impaired rostral migration of bulbar interneuron progenitors, (2) disorganization of layer structure in the olfactory bulb, and (3) abnormal axonal termination of most of olfactory sensory neurons without reaching the olfactory bulb. These results demonstrate that Arx is required not only for proper migration of the Arx-expressing interneurons, but also for establishment of olfactory neural circuitry from the epithelium to the bulb by affecting development of Arx-non-expressing projection neurons and olfactory sensory neurons.

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OG2-03 Regulation of neurite extension of neural retina by Wnt-LEF-1 signalling pathway

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To delineate the role of Wnt signal in retinogenesis, we miss-expressed various mutants forms of Wnt signaling members into E17.5 mouse retinal explant by using retrovirus vector. Expression of constitutive active (ca) β-catenin or caLef-1 in retinal cells resulted in failure of extension of neurite. Conversely, it was enhanced by dominant negative Lef-1. On the other hand, proliferation and differentiation into mature retinal cells were not affected by perturbation of Wnt-Lef-1 pathway, suggesting importance of shut-off of Wnt pathway for proper neurite extension. Using drugs specifically suppress MEK1, we found that MAPK pathway plays enhancing role for neurite extension. In PC12 cells, the activation of β-catenin-Lef-1 signaling inhibited the NGF induced neurite extension without affecting MAPK activation. Taken together, it is surmised that switching of signals from Wnt-Lef1 to MAPK play a role for proper neurite extension of retina.

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OG2-04 GSK-3β regulates phosphorylation of CRMP-2 and neuronal polarity

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Neurons are one of the most highly polarized cells known and are comprised of two structurally and functionally distinct parts, an axon and dendrites. We previously showed that CRMP-2 is critical for specifying axon/dendrite fate, possibly by promoting neurite elongation via microtubule assembly. Here, we showed that GSK-3β phosphorylated CRMP-2 at Thr-514 and inactivated it. The expression of constitutively active GSK-3β impaired neuronal polarization, possibly by suppressing neurite elongation, whereas the nonphosphorylated form of CRMP-2 counteracted the inhibitory effects of GSK-3β, indicating that GSK-3β regulates neuronal polarity through the phosphorylation of CRMP-2. Treatment of hippocampal neurons with NT-3 induced inactivation of GSK-3β and dephosphorylation of CRMP-2. Knockdown of CRMP-2 by siRNA inhibited the NT-3-induced axon outgrowth. These results suggest that NT-3 decreases phosphorylated CRMP-2 at Thr-514 and increases nonphosphorylated active CRMP-2, thereby promoting axonal outgrowth.

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OG2-05 T-cadherin expression in cortical circuit formation

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To understand the molecular basis of cortical circuit formation, we generated monoclonal antibodies raised against brain homogenate prepared from perinatal rats. One of the clones, 6C9 was selected by immunohistochemical screening. Amino acid sequence analysis revealed that the affinity-purified antigen was T-cadherin (T-cad). Immunohistochemistry showed fiber-like staining in the intermediate zone, the anterior commissure and the cerebral peduncle of embryonic and early postnatal cortex, but not in the corpus callosum. *In situ* hybridization farther showed strong signals in the cortical plate at embryonic stages and in the deep layers of the postnatal cortex. These expression patterns demonstrate that T-cad is expressed in a subpopulation of cortical neurons and tends to localize in axons, suggesting that T-cad may be involved in axonal differentiation or elongation in specific pathways.

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OG2-06 Axonal plasticity in neurite extention

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Rat sciatic nerves removed from their cell bodies can extend neurites from peripheral transected-nerve sites after in collagen gel culture. The number of neurites increased about 10 times more than in control at 14 days after application of freeze/crush injury. On the other hand, the number decreased with shortening length of removed sciatic nerve. Cycloheximide scarcely affected the number. These results show that the injury promotes neurons to synthesize essential substances for neurite extension and the transported substances in nerve fibers enable the process extension. This indicates that axons are plastic in neurite extension to reflect signals sent to neurons.

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OG2-07 Severe hydrocephalus in *fyn*$^{-/-}$ mice

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Fyn, a member of Src-family tyrosine kinases, is highly expressed in the brain. *fyn*$^{-/-}$ mice have several developmental defects that include uncoordinated structure of hippocampal CA3 region, however, little is known about how Fyn contributes to brain development. Here we report that *fyn*$^{-/-}$ mice with C57BL/6 genetic background develop severe hydrocephalus. At the age of 4-week, about 20% of *fyn*$^{-/-}$ mice had a dome-shaped head and massively enlarged lateral ventricles. Enlargement of lateral ventricles in *fyn*$^{-/-}$ mice was already significant at E18.5. In addition, aberrant neocortical structures were evident in neonatal *fyn*$^{-/-}$ mice, further demonstrating essential roles of Fyn in embryonic brain development. We also found that Fyn phosphorylated L1 cell adhesion molecule at two tyrosine residues, which are crucial for L1-mediated signaling. Given that L1-deficient mice show similar hydrocephalus to that of *fyn*$^{-/-}$ mice, regulation of L1 by Fyn may participate in brain development.

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OG2-08 An *in vitro* model of critical period for corticospinal synapse elimination during development

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There has been no *in vitro* model of critical period for developmental plasticity. We have shown *in vitro* slice co-culture of the cortex and spinal cord that corticospinal synapses are formed diffusely over the spinal cord, but later the ventral synapses are eliminated. This elimination was blocked when APV was present from 0 to 14 DIV, suggesting that this process is NMDA-dependent. Using this *in vitro* system, we made a detailed quantitative analysis on the time course of the sensitivity to APV. This process is irreversible because the ventral synapses were no longer eliminated until 21 DIV when APV was removed at 14 DIV. APV application of 0-6 or 11-14 DIV did not affect the elimination. When application started at 7, 8, 9, or 10 DIV, the effect decreased in this sequence. Furthermore, 6-11 DIV application had full effect, i.e. the same effect as the 0-14 DIV application. Thus we have determined the critical period in our system; 6-11 DIV application of APV is necessary and sufficient for a full and irreversible block of the synapse elimination.

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OG2-09 Unilateral and bilateral origins of precerebellar neurons as revealed by exo utero gene transfer.

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The precerebellar neurons (PCNs) derived from the lower rhombic lip (lRL) migrate tangentially and form bilaterally symmetrical pairs of nuclei. Due to technical limitations, whether they have unilateral or bilateral origin has remained unknown. To address this issue, we developed a gene transfer method that utilizes exo utero electroporation. This has allowed us to unilaterally label the lRL and to follow their fate in vivo. After electroporation of YFP gene, labeled neurons clearly emigrated from one side of the lRL and formed the external cuneate and lateral reticular nuclei contralaterally, as reported previously. Contradictory to previous views, however, cells forming a more anteriorly situated stream gave rise to bilateral cell clusters corresponding to the pontine gray and reticulotegmental nuclei. Thus, our method has uncovered migratory streams of and the laterality of the origin of lRL-derived PCNs.

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OG2-10 Time course of corticospinal synapse regression during development

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We showed in slice coculture of the cortex and spinal cord (SC) that corticospinal (CS) synapses are distributed in the whole SC at 7 DIV and eliminated from the ventral side until 14 DIV. We examined if this type of elimination occurrs in vivo. Stimulation of the CS tract at the medullary pyramid elicited negative field potentials (NFPs) in the SC, which were blocked by CNQX and attenuated in amplitude by frequency stimulation. The NFPs were widely recorded in the SC at P7, obscured in ventrolateral (VL) side at P8-9 and replaced by the positive field potentials (PFPs) at P10. The PFP amplitudes increased at P14 and P21. The PFPs were not affected by strychnine or bicuculline. Moreover the peak of PFP was coincident with the NFP peak. These results suggest that the NFP reflects active sink of EPSC and the PFP, its passive source and that the CS synapses were widely distributed at P7, eliminated from VL-SC at P8-9 and the distribution nearly matured at P10. Those findings were also suppeorted by anterograde labeling of CS terminals.

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OG2-11 Characterization of rat melanin-concentrating hormone receptor 1 internalization

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Melanin-concentrating hormone (MCH) is an orexigenic neuropeptide that binds two G protein-coupled receptors, MCH1R and MCH2R. MCH1R has been linked to the regulation of food intake. By using HEK293T cells stably transfected with Flag-tagged rat MCH1R, we investigated the mechanism underlying the MCH-induced internalization pathway, which is important for the desensitization or regulation of the receptor response. Quantitative analysis by flow cytometry indicated that the rate of MCH1R internalization progressed in a rapid and time-dependent manner during the first 30 min, and was partly inhibited by pretreatment with the selective protein kinase C inhibitor Go6850. Overexpression of dominant-negative b-arrestin 2 or dynamin I-K44A significantly prevented MCH-induced internalization of MCH1R. Mutational study further suggested the crucial importance of a distal portion of the C-terminal tail in the optimal internalization process.

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OG2-12 Preferred assembly of AMPA receptor subunit

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Initial models of AMPA receptor assembly postulated the unrestricted stochastic association of individual subunits. The low Ca^{2+} permeability and non-rectified current voltage relationship of most native AMPA receptors were ascribed to dominant effects of the GluR2 subunit. However, we propose instead the preferred assembly of GluR1 and GluR2 subunits into tetrameric complexes as pairs of identical heteromeric dimers. To compare unrestricted versus selective models of GluR1 and GluR2 assembly, these subunits, in both flip and flop isoforms, were expressed in varying ratios in HEK293 cells. The data from this study support the selective expression of heteromeric receptors, and are compatible with an equilibrium model of assembly of tetramers as pairs of identical heteromeric dimers. Expression of co-assemblies of the flip and flop isoforms, like that of the GluR1 and GluR2 subunits, is strongly favored over that of homomeric assemblies.

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OG2-13 Identification of agonists for a putative G-protein-coupled receptor, C06H5.7

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We have previously isolated *Caenorhabditis elegans* C06H5.7 as a receptor candidate for L-3,4-dihydroxyphenylalanine (DOPA). The conserved aspartate residue involved in binding the amine group of the catecholamine side chain, however, is substituted by glycine in the transmembrane 3 of C06H5.7. We confirmed that DOPA metabolites, but not DOPA itself, induced inward current in the C06H5.7-expressed Xenopus oocyte. No electrophysiological activity was detected in DOPA metabolites prepared in the presence of ascorbic acid (AA). DOPA metabolites were separated to six fractions by HPLC. The 5th and 6th fractions were reduced by AA, and exhibited electrophysiological activity. The two fractions were positive for the NMR signal of phenyl group, but negative for ninhydrine reaction. Thus, agonists for C06H5.7 are predicted to be non-amine L-DOPA metabolites containing phenyl group.

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OG2-14 Na, K-ATPase α2 subunit is indispensable for functional maturation of respiratory neural network

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The sodium pump is a plasma membrane protein essential for maintaining the sodium and potassium ion gradient across the cell membrane. The pump consists of α and β subunits. α2 isoform mainly expresses brain, skeletal muscle, and cardiac muscle. We have developed mice that are defective of this gene and examined specific roles of α2 isoform in the developing nervous system. The homozygotes survived until birth with no evident phenotypic abnormalities, but had severe motor deficits and died immediately after birth due to the failure of respiration. Here we show that the lack of α2 isoform caused the imbalance of intracellular ion homeostasis critical for generation of the respiratory neuron activities in perinatal period. The homozygous mice showed disconnection between rhythm generator neurons and pattern generator neurons in the respiratory neural circuit of the brainstem.

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OG2-15 A novel method to assess the function of a K^+-Cl^- cotransporter by using electrophysiological technique

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GABA, an inhibitory neurotransmitter within the adult CNS, is also excitatory at early developmental stages due to the elevated $[Cl^-]_i$, which is primarily attributable to a K^+-Cl^- cotransporter (KCC), KCC2. To analyze the function of KCC2 electrophysiologically, HEK293 cells were transfected with KCC2 and a glycine (Gly) receptor, and gramicidin-perforated patch-clamp recordings were performed to measure E_{Gly} with Gly application. The results showed E_{Gly} of KCC2-expressing cells was -63.9±12.9 mV, while that of the controls was -19.5±6.7 mV. When $[K^+]_o$ was raised or lowered, E_{Gly} of KCC2-expressing cells shifted positively or negatively, respectively. In addition, DIOA, an inhibitor of KCCs, shifted E_{Gly} positively in KCC2-expressing cells. These data suggest this electrophysiological method is reliable for the functional evaluation of KCCs.

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OG2-16 Effect of GLUT1 deficient in developmental expression of mouse GLUT1 mRNA

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Patients with glucose transporter 1 mutation (GLUT1 deficient syndrome) exhibit infantile seizures and delayed development. The purpose of this study was to investigate the brain expression of GLUT1 in the GLUT1 knockout mouse to clarify the effect of GLUT1 deficiency on the energy supply pathway to the brain. At postnatal day 0 (P0), the brain mRNA expression of GLUT1 in the female GLUT1+/- was about half that in female GLUT1+/+, while that in male GLUT1+/- was almost the same as that in male GLUT1+/+. At P14, in both males and females, the GLUT1 mRNA in the brain in GLUT1+/- was about half that in GLUT1+/+. In adults, no significant difference in the brain GLUT1 mRNA was detected when GLUT1+/- and GLUT1+/+ were compared. These results suggest that GLUT1 gene deficient influences its gene expression in neonate brain with gender difference but not in the adult brain. These effects of GLUT1 gene deficient may relate to the symptoms of GLUT1 deficient syndrome.

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OG2-17 Astrocytic BMP Receptor IA signal maintains the cerebral blood vessel

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Bone Morphogenetic Proteins (BMPs) are known to contribute gliogenesis in the mouse telencephalon. Here we show that BMP signaling through the type IA receptor (BMPRIA) is critical for maturation of astrocytes to establish the blood brain barrier. To study the role of BMP signaling in the astrocyte in vivo, we disrupted *Bmpr1a* in a cortical stem cell-specific manner in mice. We found that the end-feet of the astrocytes in the mutant mice failed to attach to the outside of cerebral blood vessels. To know how BMPRIA signaling leads changes of cell adhesion mechanism, we applied DNA microarray technique to detect alternation of gene expressions in astrocytes that express a constitutive active form of BMPRIA. We will discuss how BMPRIA signaling contributes the cell-cell interaction between astrocytes and endothelial cells.

OG2-18 A novel, noncompact myelin-pecific, transmembrane glycoprotein in CNS

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The noncompact myelin membrane, found in the paranodal loop, and inner and outer mesaxon, contains junctional specializations including tight, gap, and adherens junctions. Through the mouse cerebellar development transcriptome project, we found a brain-specific, developmentally upregulated gene. Its spatiotemporal expression profiles were closely similar to those of the MOG, a marker for the mature oligodendrocyte. Immunoelectronmicroscopic analyses showed that The protein (143 amino acids with a transmembrane helix and N-glycosilation sites) was specifically localized at the paranodal loop and inner spiral loop membrane. Heterologous expression in MDCK cells showed that it localized at plasma membrane and co-stained with β-catenin suggesting it functions at adherens junction.

OG2-19 The expression of Myelin Basic Protein 21.5KDa isoform with aging

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We investigate an age-related alternating myelin structure, and of its molecular character from 3-month-old to 30-month-old mice brain. The major glycolipids were enriched in myelin from both young and old mice, and the component distribution of these glycolipids showed the same pattern. Myelin Basic Protein (MBP) compositions in the young mice were different from those of the 21.5 KDa isoform in the old, which has peptide sequence encoded by all seven exons. The band of exon 2-containing 21.5 MBP in the demyelinated regions of an aged mouse was disappeared, contrary to the young brain and to the remyelinated regions in the Kanpou Yaku-treated mouse brain. Our results suggest that the age-related myelin loss might be due to exon 2-containing 21.5 MBP and be an early, sensitive index of remyelination.

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OG2-20 Immunohistochemical analysis of creatine biosynthetic enzyme, guanidinoacetate N-methyltransferase (GAMT), and creatine kinases (CKs) in the mouse brain

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The creatine/phosphocreatine shuttle system is known to be essential for the storing and buffering of high phosphate-bound energy in the brain. In the present study, we aimed to clarify the cellular expression profiles of creatine biosynthetic enzyme, GAMT, and its metabolic enzymes, ubiquitous mitochondrial CK (uCK-Mi) and brain-type cytoplasmic CK (CK-B) in the mouse brain by immunohistochemistry. GAMT was preferentially expressed in glial cells, whereas uCK-Mi was expressed selectively in neuronal mitochondria. CK-B was exclusive to astrocytes and inhibitory neurons, both of which are known to be resistant to acute energy loss. These results suggest that the creatine/phosphocreatine shuttle system stands on a novel neuron-glial relationship to maintain and ensure the energy homeostasis in the brain.

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OG2-21 Localization of ABCG2 in mouse brain, retina and peripheral tissues

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The purpose of the study was to clarify the complex formation and the localization of ABCG2 in mouse brain, retina, kidney, liver and small intestine. Western blotting indicated that ABCG2 was detected as a glycosylated and S-S complex in mouse brain capillaries, retina, kidney, liver and small intestine. Double-immunostaining showed that mouse ABCG2 was located on the luminal side of endothelium in brain and retinal capillaries, the hepatic bile canalicular membranes, the luminal membranes of the renal proximal tubules and the apical membranes of the epithelial cells of the small intestinal villi. Such characteristic immunostaining was not seen in ABCG2 knockout mice. It is suggested that functional ABCG2 is expressed in luminal side in brain and retinal capillaries, kidney, liver and small intestine, and performs a protective role as efflux transporter.

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OG2-22 LXR-mediated regulation of sterol transporters in rat choroid plexus

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The cholesterol metabolite levels in cerebrospinal fluid (CSF) was changed in certain neurodegenerative diseases, and sterol transport across the blood-CSF barrier (BCSFB) may plays a role in these changes. The purpose of this study was to investigate the expression and regulation of sterol transporters at the BCSFB. Isolated rat choroid plexus and a conditionally immortalized rat choroid plexus epithelial cell line (TR-CSFB3) was used as *in vitro* BCSFB models. RT-PCR analysis indicated that sterol transporters (ABCG1, 4, 5) were expressed in rat choroid plexus. The expression of ABCG1 in TR-CSFB3 cells was induced by the treatment with LXR ligands. These results suggest that sterol transporters are expressed at the BCSFB and ABCG1 expression is regulated by LXR-mediated pathway.

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OG3-01 Protein phosphorylation induces retrieval of synaptic vesicles at nerve terminals of Drosophila

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To maintain synaptic transmission exocytosis of synaptic vesicles (SVs) is followed by endocytosis at nerve terminals. We have previously shown that high K+ stimulation of Drosophila nerve terminals in the presence of La3+ causes exocytosis of SVs but blocks endocytosis. In the present study, we determined endocytosis of SVs by measuring the uptake of FM1-43, a fluorescence dye, into nerve terminals. After high K+ stimulation in the presence of La3+, nerve terminals were exposed to FM1-43. No FM1-43 uptake into boutons was observed in non-treated nerve terminals. Treatments of nerve terminals with protein kinase activators or protein phosphatase inhibitors induced FM1-43 uptake into nerve terminals while protein kinase inhibitors did not. This result suggests that SVs are retrieved from plasma membrane by protein phosphorylation after incorporated into plasma membrane of nerve terminals.

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OG3-02 Polarized dendritic targeting of a cell adhesion molecule telencephalin

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Neurons sort out a variety of functional molecules to their appropriate destinations. Telencephalin (TLCN) is a cell adhesion molecule specifically localized to somatodendritic membranes in the telencephalic neurons. Here, we identified a dendritic targeting determinant of TLCN by ectopic expression in the cerebellar Purkinje cells of transgenic mice. A full-length TLCN was localized to dendrites of Purkinje cells, but not to axons. In contrast, a deletion of cytoplasmic carboxyl-terminal 12 amino acids (residues 901-912) or a point mutation of Phe905 to Ala abrogated the dendritic targeting. Furthermore, an addition of the carboxyl-terminal 17 amino acids (896-912) of TLCN to an unrelated molecule (CD8) was sufficient for its specific targeting to dendrites in several types of neurons. Since the carboxyl-terminal region of TLCN does not contain any canonical dendritic targeting sequences, this study suggests a novel mechanism of protein trafficking to the dendrites.

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OG3-03 GTP binding protein-dependent vesicle endocytosis at a fast central synapse

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GTP-binding (G) proteins are widely involved in vesicle recycling. Synaptic vesicles are recruited via fast and slow endocytosis, but whether these are G protein-dependent is unknown. To address this issue, we loaded guanine nucleotide analogs or a dynamin fragment into the calyx of Held presynaptic terminal in rat brainstem slices, and tested their effects using capacitance measurements. GDPβS slowed endocytosis, whereas GTPγS or dynamin1 proline-rich domain peptide blocked endocytosis, resulting in a use-dependent rundown of exocytosis. These effects of GTPγS were not resulted secondarily from a reduction of Ca^{2+} currents by heterotrimeric G proteins. Prolonged repetitive stimulation in the presence of GTPγS eventually abolished evoked exocytosis. We conclude that endocytosis is entirely dependent upon G proteins including dynamin at this fast synapse.

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OG3-04 Endocannabinoid-mediated retrograde suppression of GABAergic inhibitory transmission in the striatum

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Endocannabinoids are shown to act as retrograde messengers at synapses in several brain regions. The cannabinoid CB1 receptor, a major target of enddocannabinoids in the brain, is highly expressed at nerve terminals in the striatum of the basal ganglia. To elucidate roles of endocannabinoids in the striatum, we examined GABAergic inhibitory synapses onto medium-sized spiny (MS) neurons. Coronal slices were prepared from C57BL/6 mice at P15-21 and whole-cell recording was made from MS neurons. Postsynaptic depolarization induced a transient suppression of inhibitory postsynaptic currents. This depolarization-induced suppression of inhibition (DSI) accompanied a change in the paired-pulse ratio, and was totally blocked by a cannabinoid antagonist, AM281. Activation of postsynaptic muscarinic receptors significantly enhanced DSI. These results indicate that endocannabinoids mediate retrograde signal for DSI in MS neurons of the striatum, which is positively regulated by the muscarinic system.

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OG3-05 Differential control of synaptic versus ectopic release of synaptic vesicles

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Exocytosis of synaptic vesicles is generally assumed to occur exclusively at presynaptic active zones. Here we report that AMPA receptor-mediated quantal events resulting from climbing fiber (CF) release are observed in Bergmann glial cells (BGs) in the cerebellar cortex. These quantal events are not coincident with quanta recorded in adjacent Purkinje cells sharing the same CF input. We propose that exocytosis can occur from ectopic release sites located directly across from BG membranes. We also find that vesicular release at synaptic and ectopic sites differ in the magnitude of their short-term plasticity as well as their sensitivity to Ca influx. Interestingly, high affinity glutamate transporter-mediated currents in BG follow the rules of synaptic release more closely than the rules of ectopic release. Ectopic release may be necessary to activate low affinity AMPA receptors on BGs, which may provide a geographical cue to guide BG membrane to surround active synapses and ensure efficient glutamate uptake.

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OG3-06 Reduction of AMPA receptor content in parallel fiber synapses by cerebellar motor learning

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AMPA receptor (AMPAR) is an essential component in long-term depression of parallel fiber (PF)-Purkinje cell synaptic transmission underlying cerebellar motor learning. Using SDS-digested freeze-fracture replica labeling, we quantitatively analyzed AMPAR density in PF synapses after adaptation of the horizontal optokinetic response (HOKR) eye movement. In the middle one third of the flocculus, which is involved in this motor learning, we found reduction of AMPAR content at PF-Purkinje cell synapses after one hour of HOKR training accompanied with a significant gain increase. No such difference was detected in untrained animals. These results suggest that short-term HOKR adaptation is caused by reduced synaptic AMPAR content in the flocculus.

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OG3-07 A truncated TrkB receptor, T1, regulates glial cell morphology via Rho GDI1

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BDNF has many biological functions, such as neural survival, differentiation, and plasticity. These actions may be exerted by TrkB subtypes of BDNF receptors. One of the TrkB subtypes, T1, predominates in the adult mammalian central nervous system, yet its role remains controversial. Here, we show that Rho GDI1, a GDP dissociation inhibitor of Rho small G proteins, bound to and was released from a C-terminal tail of T1 by BDNF. Furthermore, T1 acted negatively in the Rho signaling cascade in a BDNF-dependent manner and rapidly regulated the astrocytic morphology in primary cultures. Therefore, these findings indicate that T1 has a specific signaling cascade that can regulate the astrocytic morphology.

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OG3-08 Shrinkage of Dendritic Spines Associated with Induction of Long-Term Depression

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We found that, by using time lapse fluorescence imaging in acute rat hippocampal slices, dendritic spine undergo significant shrinkage after induction of NMDA receptor-mediated LTD at Shaffer collateral synapses on CA1 pyramidal neurons. This reduction in spine size was specific to low-frequency stimulation and restricted to a local area near the stimulation site. The morphological change required activation of NMDA receptors, activity of calcineurin, but not activity of PP-1. Furthermore, activity of cofilin seemed to play a crucial role in this change. This report is the first demonstration, using time lapse fluorescence imaging in acute brain slices of postnatal rats, of morphological changes in dendritic spines after induction of LTD of synaptic transmission. These findings will also shred light on further investigations on the interaction between the protein phosphatase and the cofilin pathways.

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OG3-09 Depolarization-induced LTD and LTP at inhibitory synapses depend on the activation of distinct voltage-gated Ca channel subtypes in rat visual cortex.

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Previously, we reported that long-term depression (LTD) and potentiation (LTP) occurred in IPSCs of layer 5 pyramidal cells after the application of depolarizing pulses to these cells in rat visual cortical slices. We also showed that the responses to GABA applied iontophoretically to the soma underwent only LTD while those to the dendrite underwent LTP as well as LTD. In this study, we tested the involvement of voltage-gated Ca channels (VGCC) in these LTD and LTP. In both soma and dendrite, LTD and LTP were selectively inhibited by the L-type VGCC blocker nifedipine and the P/Q-type VGCC blocker ω-agatoxin IVA, respectively. Because L- and P/Q-type VGCCs are localized predominantly in the soma and dendrite of pyramidal cells, respectively, this differential VGCC localization may contribute to the difference in these modifications in the soma and dendrite.

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OG3-10 Enhancement of optical signals recorded from CA1 of CaMKII-transgenic mouse during long term potentiation.

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Hippocampus system contributes to its information processing. However, the conventional techniques of electrical recording are restricted to the analysis from a few electrodes placed on a tissue. In order to overcome this limitation, optical recording from multi-sites of CA1 region has been carried out by using a photodiode array system. The optical signals of pre- and post-synaptic action potentials and synaptic responses by stimulation of Schaffer collaterals (SC) from CaMKII-transgenic mouse were pharmacologically identified and compared with electrical signals. Upon stimulation of SC, fluorescent optical signals of Hippocampal slices stained with Di-4-Anepps were recorded from WT and CaMKII-transgenic mouse, respectively. It is suggested that the responses were larger in amplitude and extended to wider region in transgenic than wt mouse and that optical recording could be evaluated to transgenic animals.

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OG3-11 Protein kinase G regulates entry of Vesl-1S-EGFP fusion protein into dendritic spines

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Synaptic tag hypothesis is proposed to explain synapse-specificity in the late phase of long-term potentiation (L-LTP). According to the hypothesis a molecular tag, which decays within 4 h, is established in the activated spines independent of protein synthesis. A set of proteins induced in L-LTP is specifically assimilated into the tagged synapse after carried along common route of dendritic transport. Although the hypothesis is critically important, molecular nature of the synaptic tag is totally unknown. L-LTP induces expression of Vesl-1S protein (VS), and enhances synaptic localization of VS in the activated regions. We hypothesized that the synaptic tag mechanism should regulate entry of VS into spines. Time-lapse imaging of VS-EGFP fusion protein (VSE) in rat hippocampal neuronal culture revealed that the NMDA receptor-NO-cGMP-PKG pathway specifically incorporated VSE into spines. Activation of PKG or its downstream signals persisted at least 2 but less than 4 h, a similar decay kinetics with the physiologically postulated synaptic tag.

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OG3-12 AMPA and NMDA receptor activities bidirectionally regulate postsynaptic actin organization in dendritic spine formation during development

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We previously demonstrated that spine morphogenesis in neuronal development requires synaptic clustering of actin components in dendritic filopodia, which are precursors of spines (J. Neurosci.23(16):6586-95. Takahashi et. al.). However, it is unknown how synaptic activity regulates their synaptic clustering in neuronal development. In this study, we investigate how AMPA receptor (AMPAR) and NMDA receptor (NMDAR) activities regulate synaptic clustering of drebrin with F-actin during development. Chronic blockage of AMPAR activity inhibits their synaptic clustering, and also inhibits the morphological spine maturation from filopodia. In contrast, chronic blockage of NMDAR activity promotes their clustering and the spine maturation. Our data suggest that AMPAR and NMDAR activities bidirectionally regulate the postsynaptic actin organization in developing dendritic spines.

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OG3-13 NMDAR2B tyrosine-phosphorylation modulates synaptic plasticity and memory formation in amygdala

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NR2B is the most prominent tyrosine-phosphorylated protein in PSD. The biological significance of its tyrosine-phosphorylation, however, remains elusive. Here we show that phosphorylation of Tyr-1472, a major NR2B-phosphorylation site, is critically relevant to neural functions in amygdala. Mice with a knockin mutation at the phosphorylation site, Tyr1472Phe, showed impaired amygdaloid LTP and fear-related learning. Electron microscopic analyses revealed that the Y1472F mutant of NR2B localized improperly at synapses. Moreover, the Y1472F mutant of NR2B showed limited endocytosis. These results indicate that Tyr-1472 phosphorylation modulates synaptic plasticity and fear-related learning through its ability to regulate the NMDAR localization at synapses.

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OG3-14 Dynamics of rabaptin-5/ neurocrescin are involved in LTD expression in cultured hippocampal neurons.

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A constant rate of receptors trafficking and removal at postsynaptic membrane provides a constant synaptic transmission, whereas unbalance of the receptors trafficking is leading to change the efficiency of synaptic transmission. However, little is known about contribution of endosome components to synaptic transmission. We investigated the dynamics of endosome components, Neurocrescin, in dendritic spines of cultured hippocampal neurons. Imaging of GFP-tagged Neurocrescin in living neurons revealed that rapid accumulations of GFP signals in dendritic shafts and spine heads were only induced by NMDA stimulation, which normally induce long-term depression (LTD). These results indicated that accumulation of neurocrescin are not involved in constitutive endocytosis but in enhanced internalization of AMPA receptors. This result suggested that molecular mechanisms of LTD expression involved not only receptor anchoring proteins but endosome fusion protein rabaptin5/Neurocrescin.

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OG3-15 Developmental redistribution of corticospinal synapses: An optical recording study with fast voltage-sensitive dyes

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We showed previously *in vitro* co-culture of the rat sensori-moter cortex and spinal cord <SC> that corticospinal synapses were formed throughout SC at 7 DIV, but the ventral synapses were eliminated later and restricted to the dorsal part at 14 DIV. This conclusion, however, was based primarily on field EPSP recordings, rather than direct membrane potential measurements. Here we imaged fast responses to cortical electric stimulation using voltage sensitive dyes <Di-4-ANEPPS or RH795> and high speed & resolving camera system <MiCAM02, Brainvision>. In some experiments, whole cell recordings were made from the SC cells. We focused on monosynaptic responses by raising extracellular Mg^{2+} to suppress polysynaptic ones. The response spread to the SC diffusely at 6, 7, 8 DIV, but the response concentrated in the dorsal area after 14 DIV. Imaging and whole cell recording data are in good accordance with those in the previous field EPSP study.

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OG3-16 Frequency-Specific Spike Timing-Dependent Modifications of Excitatory Synapses on GABAergic Multipolar Interneurons

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We have examined activity-induced plasticity of excitatory synapses between layer 2/3 pyramidal neurons and multipolar GABAergic interneurons in freshly isolated slices of P13-16 rat somatosensory cortex. Following repetitive correlated pre- and postsynaptic spiking, the amplitude of excitatory postsynaptic potentials was persistently increased or decreased, depending on the frequency and the relative timing of spiking in the pre- and postsynaptic neurons. We observed LTD when postsynaptic stimulation precedes presynaptic stimulation with different frequencies, whereas a reverse order of stimulation results in LTP or LTD, depending on the frequency of repetitive stimulation. Futhurmore, the postsynaptic induction mechanisms differ considerably from for those of excitatory synapses between glutamatergic neurons.

OG3-17 Long-term potentiation by activation of silent presynaptic terminals in the rat spinal dorsal horn

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Conversion of presynaptic silent terminals into functional ones is a likely mechanism of long-term potentiation (LTP). We here report that the novel form of presynaptic LTP by visualizing excitation of gross neuronal excitation, pre- and postsynaptic excitation in superficial dorsal horn by optical methods with a voltage-sensitive dye. Conditioning stimulation to the dorsal roots induced not only facilitation of presynaptic excitation, but also new excitation in presynaptic terminals that were silent before conditioning. Nitric oxide liberated from glial cells by the activation of glial metabotropic glutamate receptors mediated such facilitation of presynaptic excitation and conversion of silent terminals to active ones. Furthermore, the same conditioning converted silent postsynaptic projection neurons in lamina I to active ones. The presynaptic control of excitability would thus explain some events associated with hyperalgesia. Supported by JSPS and Yazaki Foundation.

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OG3-18 Quantitative analysis of AMPA receptors after LTP induction in the dentate gyrus *in vivo*

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Molecular mechanisms of the hippocampal LTP have been investigated mainly in acute brain slices and primary cultured neurons, but not *in vivo*. We, here, employed the unilateral dentate gyrus (DG) -LTP in the hippocampus of freely moving rats, and distribution of AMPA receptors (AMPAR) in single synapses was quantitatively analyzed by SDS-digested freeze fracture replica labeling method. Analysis was done 45 min after the LTP induction in the middle molecular layer of DG. Immunogold labeling densities for synaptic AMPAR did not differ significantly between control (450 ± 42.2 / μm^2, Mean ± SE, n = 25) and LTP side (478 ± 22.8, n = 34). Distribution of AMPAR labeling densities, however, was significantly different between LTP and control side ($P < 0.05$, K-S test) reflecting fewer synapses with low labeling density (< 300 / μm^2) in LTP side. These results indicate that synapses with low AMPA receptor densities were potentiated upon LTP induction *in vivo*.

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OG3-19 Gene expression profile of late LTP-induced rat hippocampal CA1

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In hippocampal area CA1, recurrent high-frequency stimulation (HFS) produces late long-term potentiation (L-LTP) that requires *de-novo* protein synthesis, but the precise mechanism is still poorly understood. To investigate the authentic, site- and time-specific mechanisms of L-LTP, we performed expression profiling of rat CA1 acute brain slices around the site of stimulation, 10, 30 and 60 minutes after recurrent HFS using a GeneChip microarray. To select genes significantly involved in L-LTP among 31099 genes tested, we utilized our original statistical analysis method. Our results showed a significantly different expression tendency between 10 minutes, and 30 and 60 minutes after recurrent HFS. Genes with a significant difference included the upreguration of *interleukin 1α* and *noggin*. Genes selected at 10 minutes may contribute to the initiation of L-LTP, and genes selected at 30 and 60 minutes to the maintenance of L-LTP.

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OH1-01 Cloning of a D-serine-regulated transcript *dsr-2* from the rat cortex

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D-Serine is an endogenuous neuromodulator of NMDA receptor in the mammalian brain. To obtain insight into the molecular pathways for D-serine metabolism and their physiological functions, we have explored transcripts that are responsive to D-serine in the rat cerebral cortex by using RAP-PCR, a differential cloning technique. The *dsr-2* (D-serine-responsive transcript-2) gene was located on the reverse strand within an intron of neurexin-3α(*nrxn3*) gene mapped in the rat chromosome 6q24-31. The ontogenic changes in the distribution pattern of *dsr-2* transcript in the brain were correlated closely with those of free D-serine content and of NMDA receptor R2B subunit, but different from those of *nrxn3*. These data suggest that *dsr-2* gene may be involved in D-serine metabolism and/or function in the mammalian brain.

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OH1-02 Expression cloning of a D-serine modulator gene *dsm-1*

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An antipsychotic substance D-serine is present in the mammalian brain and is suggested to be an endogenous modulator of the NMDA receptor. To understand the molecular mechanism regulating the uptake and release of D-serine in the brain, we have screened the rat cerebral neocortex cDNA library by using a *Xenopus* oocyte expression system. We have isolated a cDNA clone designated as *dsm-1* (D-serine modulator-1) encoding a protein that reduces a sodium-dependent or -independent accumulation of [^{3}H]D-serine to an oocyte by approximately 30%. The deduced protein, Dsm-1, was indicated to consist of 431 amino acids with 10 transmembrane domains. The *dsm-1* mRNA was predominantly expressed in the brain, spleen, heart, and liver. These findings suggest that *dsm-1* may be involved in the regulation of uptake and/or release of endogenous D-serine.

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OH1-03 SRF-mediated transcription through nuclear translocation of MAL coactivator by TECH, a neuronal RhoGEF

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As Rho GTPases play a critical role in regulating the actin cytoskeleton and activating transcription via the serum response factor (SRF). Since guanine nucleotide exchange factor (GEF) switch Rho proteins into their active, GTP-bound form, attention has been paid characterizing RhoGEFs expressed in neurons. Recently, we have identified a RhoA GEF that is a Transcript Enriched in Cortex and Hippocampus, called TECH. In cortical neurons, a truncated, constitutively active form of TECH, strongly stimulates SRF activation which is blocked by C3 transferase. Interestingly, when constitutively active TECH is expressed, MAL, known as an SRF coactivator, translocates from cytoplasm to nucleus and dominant negative MAL blocked the SRF-mediated transcription. Taken together, TECH-RhoA signaling pathway mediates the transcriptional activation of SRF through nuclear translocation of MAL in neurons.

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OH1-04 Ras-MEK pathway is required for NGF-induced expression of the GTP cyclohydrolase I gene

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Neurotrophins are essential for the development and survival of the catecholaminergic neurons. Previously, we reported that tyrosine hydrocylase (TH) requires the Ras-MEK pathway for its induction by NGF. The critical pathway for gene expression of GTP cyclohydrolase I (GCH), which is the first and rate-limiting enzyme in the biosynthesis of 5,6,7,8 - tetrahydrobiopterin (BH4), the required cofactor for tyrosine hydroxylase (TH), is still unclear. Here we examined the intracellular signals required for NGF-induced expression of the GCH gene in PC12D cells. The activity of GCH was increased to 3-fold by NGF stimulation, and the increase was repressed by pretreatment with U0126, a MEK1/2 inhibitor, but not with PKA, PI3K, and p38 inhibitors. Consistent with this observation, induction of GCH mRNA by NGF was abolished by pretreatment with U0126. These data suggest that the Ras-MEK pathway is also required for expression of the GCH gene induced by neurotrophins as well as the TH gene.

OH1-05 cDNA array analysis of gene expression profiles in the amygdaloid subdivisions of Fyn- deficient mice following NMDA administration

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Genetic disruption of Fyn tyrosine kinase represents increased fearfulness in mice. Fyn-deficient mice display enhanced excitability of the system for the emotional output from the amygdala. To gain insight into the molecular changes associated with the enhanced excitability of the amygdala, we used laser micro-dissection system and cDNA array analysis to detect differential changes in the gene expression among the amygdaloid subdivisions between the homozygote and heterozygote Fyn-deficient mice that were administered with NMDA. Among 805 cDNA tested, we have detected a gene that shows altered expression in the central nucleus between the mutant and control mice. The expression pattern of this gene was further confirmed by in situ hybridization analysis.

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OH1-06 Glutamate regulates the frequency of spontaneous synchronized Ca^{2+} spikes through group II metabotropic glutamate receptor in cultured mouse cortical networks

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Glutamate-induced signals through the iGluRs are profoundly involved in the generation of spontaneous intracellular synchronized Ca^{2+} spikes. In the present study, we revealed that glutamate-induced signals through mGluRs decreased the frequency of Ca^{2+} spikes in cultured mouse cortical neurons, mainly due to the signal through group II mGluR, inactivation of adenylate cyclase-cAMP-PKA signaling pathway. Taken together, glutamate generates the synchronized Ca^{2+} spikes through iGluRs and modulates simultaneously their frequency through group II mGluR signaling pathway in the present *in vitro* neural network.

OH1-07 The spontaneous Ca^{2+} elevation in the striatum is affected by motor cortex

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The striatum plays a major role in linking cortical activity to basal ganglia outputs. However, the effects of cortical activity on striatal cells are not understood well.
10-25 days old Sprague Dawley rat cortico-striatal slice was stained with Fura-PE3-AM to measure intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$) using a cooled-CCD imaging system. We previously reported long lasting spontaneous $[Ca^{2+}]_i$ elevations of striatal cells (10 s to about 300 s) in cortico-striatal slices. In these slices, 3/16 cells showed regular oscillatory $[Ca^{2+}]_i$ transients. However, in the striatal slices without cortices, 9/12 cells showed regular oscillatory $[Ca^{2+}]_i$ transients. The duration of the $[Ca^{2+}]_i$ transients in the cortico-striatal slice tended to be longer than that in the striatal slice. These results suggested that the cortico-striatal connection affected the activity pattern of the spontaneous $[Ca^{2+}]_i$ transients of the striatal cells.

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OH1-08 Enhancement of receptor-mediated phospholipase C activation by the preceding depolarization in hippocampal neurons

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Recent studies have revealed that endocannabinoids are released from central neurons in response to depolarization or activation of certain receptors (mGluR1/5, muscarinic M1/M3). These two types of stimuli work in a synergistic manner. The released endocannabinoids act retrogradely on presynaptic terminals and suppress transmitter release. Biosynthesis of a candidate endocannabinoid, 2-arachidonyl glycerol (2-AG), involves phospholipase C (PLC) and DAG lipase. In this study, we tried to estimate 2-AG production by monitoring the amount of its precursor DAG. We over-expressed a DAG-sensitive cation channel, TRPC6, into cultured hippocampal neurons. We found that depolarization strongly augmented the TRPC6-mediated currents in response to M1/M3 or mGluR1/5 activation. This suggests that the enhanced receptor-mediated PLC activation by the preceding depolarization underlies the synergism between the two types of stimuli for endocannabinoid release.

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OH1-09 Cross talk between metabotropic glutamate receptor and AMPA receptor in parallel fiber-induced inositol 1,4,5-trisphosphate production in cerebellar Purkinje cells

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Inositol 1,4,5-trisphosphate (IP_3) is a key second messenger that regulates many important cellular functions. In this study, we examined the functional interaction between ionotropic glutamate receptor and metabotropic glutamate receptor (*mGluR*) *in the generation* of IP_3 at parallel fiber (PF)-Purkinje cell synapses. We visualized local IP_3 dynamics upon PF stimulation in Purkinje cells using the GFP-tagged pleckstrin homology domain (GFP-PHD). Surprisingly, this PF-induced IP_3 production depended not only on group I mGluR but also on the AMPA receptor (AMPAR). The PF-induced IP_3 production was blocked by either the inhibition of G-protein activation by GDP-βS or intracellular Ca^{2+} buffering by BAPTA. These results show that the PF-induced IP_3 production is mediated cooperatively by group I mGluR and AMPAR through G-protein activation and Ca^{2+} influx.

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OH1-10 Modulation of spatiotemporal pattern in neuronal activities by synaptic enhancement.

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Using the rat hippocampal dissociated culture system on multi-electrode arrays, we investigate the spatiotemporal pattern of spontaneous action potentials. We calculated cross-correlation between all the pair of recorded neurons. We estimated "connectivity" and generate "*connection maps*". That connection map analysis revealed the functional connectivity between neurons was rearranged dynamically when the synaptic enhancement was induced in a cultured neuronal network.

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OH1-11 Necdin interacts with TrkA and p75 neurotrophin receptors

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Necdin is a growth suppressor expressed predominantly in postmitotic neurons. Necdin interacts with the p75 neurotrophin receptor (p75NTR) which mediates nerve growth factor (NGF)-dependent neuronal differentiation and apoptosis. We investigated the modulatory roles of necdin in neurotrophin signal transduction through its interactions with p75NTR and the receptor tyrosine kinase TrkA. Co-immunoprecipitation analyses revealed that necdin bound to both p75NTR and TrkA. Necdin interacted with TrkA in a kinase activity-dependent manner and facilitated the interaction between TrkA and p75NTR. Moreover, ectopic expression of necdin in PC12 cells, in which both TrkA and p75NTR are endogenously expressed, accelerated NGF-induced neurite extension. Since p75NTR facilitates the NGF-binding to TrkA and enhances TrkA-mediated neurotrophic signals, *necdin may serve as an adaptor molecule that links* TrkA and p75NTR to promote neuronal differentiation and survival.

OH1-12 Endothelin Increases the Expressions of Exon III- and IV-Containing BDNF Transcripts in Cultured Astrocytes

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Effects of endothelins (ETs) on BDNF production in astrocytes were examined. ET-1 (100 nM) increased mRNA levels and extracellular releases of BDNF in astrocytic cultures. RT-PCR analysis showed that exon III- and exon IV-containing BDNF transcripts existed in cultured astrocytes. On the other hand, exon I- and exon II-containing BDNF transcripts were not detected. ET-1 increased the expressions of astrocytic exon III and exon IV transcripts. Ca^{2+} chelating, W-7 and KN93 inhibited the increases in exon IV BDNF mRNA and C/EBPβ levels by ET-1. The ET-induced increases in exon III BDNF mRNA expression and phosphorylation of CREB were reduced by Ca^{2+} chelating, W-7, KN93, PD98059 and wortmannin. These results suggest that ETs stimulate transcription of astrocytic exon III and exon IV BDNF genes through CREB and C/EBPβ-mediated mechanisms, respectively.

OH1-13 SNPs in human BDNF gene; Decreased trophic effects of BDNF SNPs variants to CNS neurons

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Neurotrophins (NTs) regulate neuronal survival and synaptic plasticity. To understand the biological function of the precursor form of brain-derived neurotrophic factor (proBDNF), we focused on two single nucleotide polymorphisms (SNPs) in human BDNF gene; R125M and R127L, which cause amino acid substitutions near the cleavage site of proBDNF. We first examined the influence on the processing of proBDNF in cultured CNS neurons. Studies with Western blot and ELISA demonstrated that these substitutions influenced the production of mature form of BDNF. To examine whether these mutations altered biological activity of BDNF, CNS neurons expressing each mutant BDNFs were subjected to cell death assay in culture. This study indicated that mutant BDNFs showed decreased trophic effects to CNS neurons. Taken together, we suggest that these SNPs are implicated in neuronal death in CNS.

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OH1-14 Brain-derived neurotrophic factor induced recruitment of receptor tyrosine kinase TrkB into lipid rafts in primary neurons

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BDNF promotes neuronal survival and differentiation, and modulates synaptic plasticity. In this study we investigate whether cholesterol- and glycolipid-rich microdomains, lipid rafts, exist as a functional platform for BDNF-exerting signal transduction. We found that BDNF stimulation induced a translocation of the receptor TrkB tyrosine kinase into lipid rafts. This translocation was blocked by TrkB kinase inhibitors, indicating that the activation of TrkB is required for the translocation into lipid rafts. Disruption of lipid rafts by depleting cholesterol from neurons blocked the ligand-induced translocation. In addition, disruption of lipid rafts prevented potentiating effects of BDNF on transmitter release. These results indicate that ligand-induced TrkB translocation into lipid rafts may represent a signaling mechanism for synaptic modulation by BDNF.

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OH1-15 BDNF influences the decision of the laminar fate of cortical progenitors of mice

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Laminar formation of the cerebral cortex during development requires precisely regulated migration of the cortical cells. We examined a possibility that brain-derived neurotrophic factor (BDNF) is involved in the laminar formation. Green fluorescent protein (GFP) expression vector and BDNF protein were injected into the ventricular space of the E13.5 mouse embryos in utero, and the vector was electroporated into the ventricular cells. We found that the cells with GFP fluorescence were located in layer IV-VI of the cerebral cortex in 3 weeks. However, the GFP signal was limitedly found in the layer IV when vehicle was injected. These results demonstrated that BDNF altered the position of layer IV neurons. Other experiments conducted by electroporation of BDNF gene added a possibility that a timing of the withdrawal of cortical neurons from the proliferating pool, accelerated by BDNF, is important for alteration of laminar fate.

OH1-16 Type IV collagen of the novel structure "fractone" in mouse central nervous system

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Type IV collagen is a major component of basal lamina (BL), which influences the proliferation, motility, and differentiation of many cell types. Mercier et al. have been reported "Fractone", the extravascular BL, an extension from capillary BL to subependymal region where neuronal stem cells exist. To analyze functions of fractone, we examined whether type IV collagen was a component of the fractone and the distribution by immunohistochemistry using α1-6(IV) chains antibodies. Fractone bulbs were organized by at least type IV collagen and laminin, and distributed underneath the selected ependyma. The results suggested that the fractones were organized based on specialized microenvironment and have specific roles in CNS.

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OH1-17 Induction of neuronal cells from bone-marrow stromal cells and application for Parkinson's disease model rats

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Bone marrow stromal cells (MSCs) have the capability under specific conditions to differentiate into other cell types. Here we demonstrate a highly efficient and specific induction of cells with neuronal characteristics, without glial differentiation, from both rat and human MSCs using gene-transfection with Notch intracellular domain (NICD), and subsequent treatment with trophic factors. Treatment of the induced neuronal cells with GDNF increased the proportion of TH-positive and dopamine-producing cells. Transplantation of these GDNF-treated cells showed improvement in apomorphine-induced rotational behavior following intrastriatal implantation in a 6-hydroxy dopamine rat model of Parkinson's disease. This study shows that a population of induced neuronal cells may allow for a neuroreconstructive approach.

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OH1-18 The periodic regulation of the neural stem cell proliferation by clock-related genes

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We examined the role of clock-related genes in the regulation of the neural stem cells (NSCs) proliferation in vitro. The NSCs were expanded from the striatum of E15.5 mice. After the treatment with EGF, a growth factor for NSCs, cells were harvested at 4 h interval for 72 h and the expression of clock-related genes (*Per2* and *BMAL1*) was measured by RT-PCR. Likewise, the change of proliferative activity was determined by WST-8 and BrdU incorporation assay. We found that EGF treatment elicited the circadian expression of *Per2* and *BMAL1* mRNA in the NSCs with an antiphase relationship. Furthermore, the proliferative activity was also changed rhythmically with a period 24 h and correlated negatively with *Per2* and positively with *BMAL1* rhythm. These results suggest that clock-related genes, such as Per2 and BMAL1, periodically modulate the proliferation of NSCs.

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OH1-19 Retinal fate specification of mouse embryonic stem cells by ectopic expression of Rx/rax, a homeobox gene

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We show that subpopulations of retinal neurons can be generated in retinal explant cultures from grafted mouse embryonic stem (ES) cells expressing ectopically transcription factor Rx, which is known to be expressed in immature retinal cells. Dissociated embryo bodies were treated with retinoic acid for use as donor cells and co-cultured with retina explants for 2 weeks. In contrast to the parental ES cells, which could not migrate into host retinal cultures, ES cells expressing RX migrated into the host retina and extended their process-like structures between the host retinal cells. The retinal neurons generated by these ES cells expressed ganglion and horizontal cell markers and showed electrophysiological properties expected of ganglion cells.

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OH1-20 Identification of SSEA-1 as a novel surface antigen for retinal progenitor cells

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Neuronal retina is an important target organ for regenerative medicine, and isolation and expansion of retinal progenitor cells are critical issues. Here, we identify the SSEA-1(CD15) as a novel surface antigen, which can be used to enrich retinal progenitor cells from mouse embryo. SSEA-1 is known to be expressed in ES cells and neural stem cells. In neural retina, IHC and FACS analysis revealed temporal, spatial and quantitative transition of SSEA-1(+) sub-population during development. Furthermore, multi-color analysis of the expression of other markers suggested that SSEA-1(+) cells represented retinal immature cells.
Findings that SSEA-1(+) cells had prolonged proliferation activities leading to larger clonal size than that of SSEA-1(-) cells in reaggregation culture, supported the idea. Moreover, their normal development into late born retinal subtypes was demonstrated. This work provides a novel approach for the purification of retinal progenitor cells by surface antigen expression.

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OH1-21 MR tracking of neural stem cells, astrocytes and PC 12 cell transplanted into the rat striatum

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We investigated the migration patterns of PC12 cells, astrocytes and neural stem cells transplanted into the rat striatum using MR microscopy. All types of cells were labeled with Fe3+ using the hemagglutinating virus Japan-envelope vector. On one day after transplantation, all types of cells were clearly visualized in the injected area of the stratum. Three days to thirty days after transplantation, some neural stem cells and PC12 cells were migrated to the surface of the cerebral cortex through the needle route. However, astrocytes were remained in the same area as injected, where the intensity of signals was gradually decreased. The present result indicates that neural stem cells and PC12 cells have a tendency to migrate and the migration can be non-invasively monitored by MR microscopy.

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OH1-22 Administration of CD34+ cells post-stroke enhances angiogenesis and neurogenesis in a murine model

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It has been difficult to devise effective neuroprotective therapies in cerebral infarction. In this study we demonstrate that systemic administration of human cord blood-derived CD34-positive cells to immunocompromised mice subjected to stroke 48 hrs earlier induces neovascularization in the ischemic zone and provides a favorable environment for neuronal regeneration. Endogenous neurogenesis, suppressed by an anti-angiogenic agent, is accelerated due to enhanced migration of neuronal progenitor cells to the damaged area. Our data suggest an essential role for CD34-positive cells in promoting directly/indirectly an environment conducive to neovascularization of ischemic brain in order for neuronal regeneration to proceed.

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OH2-01 Characterization of the activity of CaM kinase kinase in the hippocampal neurons.

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We have been characterizing Ca/CaM-dependent protein kinase (CaMK) cascade (consisted from CaMKI, CaMKIV and CaMKK) in the hippocampal neurons. When the subcellular localization of CaMKK (the activator of CaMKI and CaMKIV) α and β were examined, both isoforms were found in the cytosol but only α was also found in the nuclei. We further examined to define the activities of CaMKK α and β, using the recombinant CaMKI protein as a substrate. We prepared the cytosolic and the nuclear extracts from the cultured rat hippocampal neurons, and CaMKK activities were examined for each extract. The Ca-independent activity of CaMKK was detected only from the cytosolic extract, not from the nuclear extract, although the Ca-dependent activity was detected both from the nuclear and the cytosolic extracts. Because only CaMKKβ shows Ca-independent activity, it was suggested that CaMKKα, not β, is the activator of nuclear CaMKIV in the nuclei of hippocampal neurons.

OH2-02 Role of CaMKII on neurite outgrowth of CAD cells

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A central nervous system cell line, CAD, is known to differentiate in the absence of serum. In the present study, we studied the relation between expression of Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII) and neurite extension in CAD cells during neural differentiation. Expression of CaMKII was induced on day 1, and was continued at a high level for 5 days after the exposure to differentiating conditions. Neurite extension was stimulated from day 1 and continued for 5 days, suggesting that CaMKII activity is correlated with neurite outgrowth. The importance of CaMKII for neurite formation was confirmed with CaMKII knockdown CAD cells stably expressing siRNA for CaMKII (KII(-) cells). The mRNA of endogenous CaMKII was not detected by RT-PCR in KII(-) cells, and these cells were round and had no neurites. When CAD cells were treated with KN93, a selective inhibitor of CaMKII, neurite extension was reduced. These results indicated that CaM kinase II is involved in the neurite outgrowth in CAD cells.

OH2-03 Synaptotagmin IX, a major Ca^{2+} sensor for dense-core vesicle exocytosis in PC12 cells

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Although PC12 cells express three synaptotagmin isoforms (Syts I, IV, and IX), all of which have been proposed to regulate dense-core vesicle exocytosis, it remains unknown which of the Syt isoforms acts as the major Ca^{2+} sensor for dense-core vesicle exocytosis (*J. Biol. Chem.* (2002) 277:4601-4604; *J. Biol. Chem.* (2003) 278:3220-3226). In this study I showed by immunoaffinity purification and immunocytochemistry that Syts I and IX, but not Syt IV, are present on the same secretory vesicles in PC12 cells. Silencing of Syt IX with specific siRNA (small interfering RNA) dramatically reduced high-KCl-dependent neuropeptide Y (NPY) secretion from PC12 cells, whereas silencing of Syt I with specific siRNA had no significant effect at all (*Biochem. J.* (2004) 380, in press). The results indicate that Syts I and IX are not functionally equivalent and that Syt IX, and not Syt I, is indispensable for regulation of Ca^{2+}-dependent dense-core vesicle exocytosis in PC12 cells.

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OH2-04 Ca^{2+} microdomains and fusion sites of synaptic vesicles in goldfish retinal bipolar cells

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Applying evanescence microscopy to the axon terminal of ON-type bipolar cells isolated from the goldfish retina, we visualized the Ca^{2+} microdomains and the fusion sites of synaptic vesicles with ms-order time resolution. A fluorescent Ca^{2+}-sensitive dye, Fluo-5F, was introduced into the bipolar cell via a whole-cell recording pipette. Upon activation of the Ca^{2+} current by a depolarizing pulse, several bright fluorescent spots appeared and spread out concentrically in the axon terminal region. Staining of synaptic vesicles with a FM dye enabled us to monitor the movement and fusion of synaptic vesicles. Exocytosis could be defined as the decay of FM-dye fluorescence followed by an annular increase of the fluorescence. By monitoring fluorescence of the Ca^{2+}-sensitive dye and the FM dye in the same cell, we will address the relationship between the sites of Ca^{2+} entry and vesicle fusion.

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OH2-05 GABA signaling couples to cellular redox activity by mediation of Src

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GABA, an inhibitory neurotransmitter, plays a vital role in controlling neuronal activities in CNS. We examined interplay between GABA-mediated signal and intracellular redox activity that might play important roles such as regulating gene expressions and channel activities in neurons. Redox activity of mouse cortical neuronal cells in culture was examined using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) as a substrate. After GABA stimulation (3 min at 50 μM), the redox activity was decreased to 93% at 2 min and to 86% at 30 min of the control. A src-tyrosine kinase inhibitor, PP2 (20 min at 4 μM), prevented the reduction to 94% at 30 min although PP2 alone had no effects on the activity. These results indicate that GABA signaling might link to cellular oxidation-reduction potential through a src-family tyrosine kinase (s) in neuronal cells.

OH2-06 CRMP-2 is involved in kinesin I-dependent transport of Sra-1/WAVE1 complex to growth cone of axon

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During neuronal development, various actin-binding proteins regulate actin dynamics and instability in growth cone of axon, thereby inducing axon outgrowth. However, the transport systems of these proteins to growth cone of axon are largely unknown. Here, we identified Sra-1, as a novel CRMP-2-interacting molecule. The expression of CRMP-2 D71N mutant, which reduced the binding to Sra-1, caused the inhibition of axon outgrowth in hippocampal neuron. In addition, kinesin I physiologically formed the complex with Sra-1/WAVE1 complex via CRMP-2. The CRMP-2-induced axon outgrowth was canceled by the knockdown of Sra-1 and WAVE1 by RNAi. The expression of CRMP-2 D71N or the knockdown of CRMP-2 and kinesin I evoked the delocalization of Sra-1 and WAVE1 from growth cone of axon. These results suggest that CRMP-2 is involved in the kinesin I-dependent transport of Sra-1/WAVE1 complex to growth cone, and thereby regulates axon outgrowth.

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OH2-07 Expression of some microtubule-associated protein 1B as a transmembrane protein during synaptogenesis in vitro.

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MAP1B is expressed mainly during early neuronal development and is exceptional in that it contains a transmembrane-like region and may exist as a membrane-spanning protein. In this study, we examined the relationship between MAP1B and the plasma membrane in cultured neurons by two different methods utilizing surface biotinylation; one combined with subcellular fractionation and the other with immunoprecipitation. Subcellular fractionation showed that the amount of MAP1B associated with plasma membrane increased during synaptogenesis, indicating the increased association of MAP1B with the plasma membrane and the membrane skeleton. The immunoprecipitation with surface biotinylation indicated that at least part of MAP1B associated with plasma membrane was actually present as a transmembrane protein. The amount of this transmembrane MAP1B also increased during synaptogenesis, suggesting that MAP1B may have an additional function besides MT stabilization.

OH2-08 Kinesin transports RNA: Isolation and characterization of the RNA-transporting complex

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RNA transport is an important and fundamental event for local protein synthesis, especially in neurons. RNA is transported as large granules; however, little is known about them. We isolated a large RNA-protein complex (size: 1000S~), composed of many kinds of proteins, as the binding partner of conventional kinesin. We completely identified 42 major proteins in the complex; three of which together with staufen, FMRPs and EF-1α were investigated here. The complex was RNase-sensitive but detergent resistant, and contained the mRNAs for CaMKIIα and Arc. These proteins together with the RNAs being transported were colocalized in large granules, which were transported exclusively in the dendrites at 0.034 +/- 0.025 μm / sec. This transport was enhanced by the overexpression of kinesin, but was reduced by its functional blockage. Thus, it was determined that kinesin transports RNA via this complex in dendrites.

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OH2-09 The analysis of regulation mechanism of NUDEL phosphorylation/dephosphorylation.

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We have been investigated for the regulation of neuronal cell migration, especially for the mechanism of Lis1, Nudel and cytoplasmic dynein signaling pathway. I found recently that 14-3-3ε regulated the phosphorylation/dephosphorylation of Nudel and this regulation was important for the correct migration of neurons. Here we tried to demonstrate the phosphorylation/dephosphorylation status of Nudel in detail, by using anti-phosho-Nudel specific monoclonal antibodies. The expression of Nudel was upregulated in M-phase and Nudel was specifically phosphorylated in M-phase. Phosphorylated Nudel was localized at spindle poles through prophase to telophase.In migrating cells in vitro, phosphorylated Nudel was localized at centrosome. Immunohistochemical analysis revealed that phosphorylated Nudel localized around nucleus and at centrosome in vivo.These data suggest that Nudel phosphorylation was regulated through cell cycle and cell migration, and might have a crucial role for nuclear movement.

OH2-10 Effect of dephosphorylation on neurofilament gel formation - real-time observation and biophysical analysis

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Highly cross-linked arrays of newrofilaments (NFs) observed in the mature axon are thought to determine the mechanical properties of the axoplasm as well as NF transport rates and NF aggregate formation. To investigate mechanisms regulating such higher order structures, we developed a sensitive time course observation method based on the analyses of Brownian movements of Latex microbeads. Our method can detect change at protein conc. of 0.3 to 1.5 mg/ml, and less than 10 microliter of volume. At RT, native NFs from rat spinal cords showed time and concentration dependent decrease of bead movement. This viscosity increase is due to NF association. Such effect was enhanced when Mg^{++} was added. When NFs were enzymatically dephosphorylated, bead movement decreased more rapidly, but with no sensitivity towards Mg^{++}. These results are discussed by local viscosity fluctuations.

OH2-11 Expression and function of receptor-type protein tyrosine phosphataseζ/β in mammalian retina

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Receptor-type protein tyrosine phosphataseζ/β (RPTPζ/β) is, a member of large RPTP family, predominantly expressed in the central nervous system. Despite abundant expression of RPTPζ/β in mammalian retina, its contribution to visual functions has not been elucidated. We found RPTPζ/β is expressed on the postsynaptic sites of rod bipolar cell, as well as the processes of retinal ganglion cell and Muller glia. The dephosphorylation of protein kinase Cα is impaired in rod bipolar cells of the RPTPζ/β-deficient mouse, suggesting a disorder in dark-adaptation. Expectedly, the behavioral test showed very poor entrainment to photic pulse in the well dark-adapted and free-running mice with RPTPζ/β-gene deficiency. The RPTPζ/β signal transduction relevant to this phenotype will be discussed on the day of meeting.

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OH2-12 Expression levels of opsin kinases in the zebrafish eye

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G protein-coupled receptor kinase (GRK) 1 and 7 have been assumed to phosphorylate light-activated opsins for terminating the visual excitation. To understand the comparative aspect of the deactivation processes of the light signaling in rods and cones, we preformed cDNA cloning and expression analyses of GRKs in the zebrafish eye. Two orthologs of GRK1 (GRK1A and 1B) and GRK7 (GRK7-1 and 7-2) were found in the zebrafish genome. In immunohistochemical and *in situ* hybridization analyses, GRK1A was localized in the rod outer segment, whereas both GRK1B and 7-1 were in the cone outer segment. Quantitative analyses of GRK proteins and mRNAs suggested that the ocular protein levels of cone-type GRKs (1B and 7-1) are higher than that of GRK1A (rod-type) in the zebrafish. These results are consistent with the observation that the recovery kinetics of the light response from cones was faster than that from the rods.

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OH2-13 *twilight*, a zebrafish mutant showing defects in the outer segment formation of photoreceptors

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The photoreceptors of the vertebrate retina are highly polarized cells and elaborate a specialized apical cilium called an outer segment (OS). The OS is filled with a stack of flattened membrane sacs, the disc membrane. *twilight* (*tli*) is a zebrafish visual mutant that was isolated by our OKR assay. In *tli* mutants, photoreceptors initially differentiate but rapidly degenerate. By electron microscopic analysis, we found that the OS fails to form properly in *tli* mutant at the initial stage of photoreceptor development, suggesting that Tli is required in the formation of the OS disc membrane. Such phenotypes resemble the retinopathy caused by mutations in ROM/peripherin. We identified seven zebrafish ROM homologues. They were mapped to gentic loci different from the *tli* locus, suggesting that Tli may be a novel regulator of the OS formation in photoreceptors.

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OH2-14 Properties of spikes and localization of voltage-gated Na+ channels in retinal AII amacrine cells

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AII amacrine cells (AII cells) play a crucial role in the rod pathway of the retina and they receive glutamatergic input from rod bipolar cells (RBCs). AII cells generate TTX-sensitive spikes. To investigate functional role of the spike, we performed whole-cell patch clamping of AII cells in the mouse retinal slice. Spontaneous spikes were suppressed by local application of glutamate to the outer plexiform layer, indicating that glutamate hyperpolarized RBCs. Frequency of the spike was dependent on intensity of the injected current through the recording pipette. Application of glutamate to the dendrites of AII cell increased spike frequency in dose-dependent manner. To examine the localization of voltage-gated Na^+ channels within AII cell, 1 μM TTX was applied. Application of TTX to proximal dendrite and soma blocked Na^+ current more effectively than that to distal dendrite. Our results suggest that the soma and surrounding region of AII cell code intensity of glutamate input from RBCs into spike frequency.

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OH2-15 Neural mechanisms of light-evoked oscillations in the frog retina

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Oscillatory spike discharges observed in off-sustained ganglion cells (dimming detectors) in the frog retina are assumed to be related to the escape behavior. We have already reported that dimming stimulation evoked rhythmical fluctuations superimposed on a plateau depolarization, which trigger oscillatory spike discharges in dimming detectors. In the present study, applying the whole-cell current clamp technique to the isolated retina, we investigated what kind of synaptic input generated the rhythmical fluctuations in dimming detectors. Dimming detectors responded to exogenously applied GABA, ACh or glutamate. Even when $[Cl^-]_{pipette}$ was drastically changed, oscillations were not affected. On the other hand, bath-application of curare totally abolished the rhythmical fluctuations without affecting the light-evoked plateau depolarization. These results suggest that the rhythmical fluctuations may be evoked by excitatory cholinergic synaptic inputs to dimming detectors.

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OH2-16 Retinal Adaptation to Space-Time Correlations

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The receptive field (RF) of retinal ganglion cells has a center-surround shape in space, and is bi-phasic in time. These antagonistic shapes are regarded as evolutionary adaptation to the natural visual environment. In average natural scenes, light intensity at each point tends to be very similar to, and well predictable from, intensity at the surrounding points. The antagonistic RF serves to subtract this predicted intensity from the actual intensity, thereby efficiently encoding the wide-range input using the narrow-range spikes. However, daily scenes can be very different from the average: e.g. a forest scene is correlated vertically, but not horizontally. An efficient encoder might adapt its prediction rule accordingly. By recording retinal responses after exposure to various conditions, we found that the RF shape indeed changes according to space-time correlations in the input. The adapted RF invariably subtracted the predicted intensity, indicating that the retina adapts the prediction rule. We propose a mechanism based on our results suggesting the involvement of amacrine cells.

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OH2-17 Contrast adaptation in human early visual cortex: an event related fMRI study

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We have used event-related fMRI to ask how adaptation to the stimulus contrast changes contrast-response functions of early human visual areas. Changes in BOLD signal to brief changes in stimulus contrast were measured for different contrast adaptation levels. From these measurements we constructed contrast-response curves for the BOLD signal. We found that adaptation resulted in primarily horizontal shifts of contrast-response curves, which are thought to underlie an adaptive mechanism allowing the brain to efficiently encode and process stimuli over a wide range of average contrast levels. In addition, we unexpectedly found that while early visual areas show transient decrease in BOLD signal to decreases in stimulus contrast, the ventral portion of area V4 (V4v) predominantly shows increases in BOLD response to both contrast increases and decreases. This observation suggests that V4v may encode stimulus salience rather than faithfully represent contrast.

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OH2-18 Temporal dynamics of neural adaptation effect in the human visual ventral stream

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A repetitive presentation of the same visual stimulus attenuates the brain responses to that stimulus (neural adaptation, NA). Although this effect has been widely observed in various regions of primate brain, its temporal dynamics as a neuronal population is mostly unclear. In the present study using magnetoencephalography, we dissociated three dimensions of the NA occurring in the human visual ventral stream: activation strength, peak latency, and temporal duration of neural response. The results revealed that visual responses to repeated stimulus showed a significant reduction in both activation strength and peak latency, but not in duration of neural processing. These results indicate that (i) NA induces the neural response changes in the temporal domain as well as the response attenuation reported previously, and (ii) this temporal change is mainly observed as a rapid rising of visual responses, rather than a temporal shortening of neural response curves within the visual ventral stream.

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OH2-19 Temporal frequency dominance domains in human primary visual cortex as revealed by high resolution fMRI study

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We previously found that the BOLD signal change depends little, if any, on the reversal frequency of checkerboards in Human primary visual cortex (V1) at coarse spatial resolution. This finding provided us with a unique opportunity to examine if there is any spatial heterogeneity within V1 that process visual information of different temporal frequencies. To explore this possibility, we used full-screen contrast reversing checkerboards and a differential mapping method similar to that used for mapping ocular dominance columns. We observed a highly reproducible patchy pattern in anatomically well defined section of V1, where different patches were preferentially activated by low (0.75 Hz) and high (15 Hz) reversal frequencies, respectively. These patches, which we term temporal frequency dominance domains (TFDDs), resembled ODCs in size, measuring ~1.5 mm in the narrow dimension, but displayed distinct spatial arrangements.

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OH2-20 Differential Effect by Distractors on Delay-Period Activity in Monkey Inferotemporal Cortex during Pair Association Task

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In our previous study, we recorded neural responses of the macaque inferotemporal cortex during pair association task and found that the delay-period activity reflected the cue stimulus itself or its paired associate. In this study, we investigated the effect of distractors on the delay-period activity. Out of 84 neurons that showed selective cue-period activity, 25 neurons showed selective delay-period activity. We found that, as population, delay-period activity for the optimal cue stimulus was larger than mean delay-period activity for stimuli from any other pairs. This delay-period activity decreased after appearance of distractors. As for the delay-period activity for the paired associate, it was also larger than mean delay-period activity for stimuli from other pairs. However, this delay-period activity did not decrease after appearance of distractors. These results suggest that these delay-period activities may arise from different neural mechanism for mnemonic processing.

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OH2-21 Effect of reward contingency on discharges of cells in macaque inferior temporal areas

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To study the role of the perirhinal cortex and area TE in stimulus-reward associations, we recorded spike activities of cells in these areas in two monkeys that had been trained to perform a visually cued go-no go task. To a visual cue presentation, the monkey either had to release and repress a lever (go) within 800ms, or widthhold it (no go), depending on the cue. At 1300 ms after the cue onset, a red circle and beep sound, with or without a liquid reward, were provided for correct responses. A fixed set of 80 object images was divided into 4 quarters for go-reward, no go-reward, go-no reward, and no go-no reward conditions, and it was used as the cues. Although the task didn't require the monkeys to anticipate the reward condition, the latency of go responses and the frequency and strength of suckling indicate that they correctly anticipated the reward condition. Two fifths of perirhinal cells and a quarter of TEad cells showed significant reward dependency in firing at some time during the period between the cue onset and the reward time ($p < 0.05$, t-test, corrected).

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OH2-22 An event-related potential associated with human action observation.

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In line with the research of "understanding the action of others", we have examined event-related potentials (ERPs) from healthy adults with tasks of action observation and also imitative reaction. In this report, we show an unexpected ERP pattern elicited by one of our experiments, where pairs of photographs showing human left hand were used as stimulus. In each photo, a white object (cross-like figure) was overlaid on the position of index finger. Stimulus was a successive presentation of the photos produced apparent motion of either the index finger or the object. Direct comparison between the stimulus-locked ERPs showed that only the object stimulus induced a negative deflection around the scalp areas over the vertex to the right precentral sites with a latency of ~ 300 ms. We interpret this pattern reflected a conflict in a kind of matching process, presumably the violation of expected finger motion induced by the visual presentation of human hand, as an analogous of "mismatch" negative components such as MMN, ERN, or N400.

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OH3-01 The ryanodine receptor in striated muscle of chaetognaths does not function as an intracellular Ca^{2+}-release channel

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Chaetognaths are a small, mainly planktonic marine phylum, which is entirely isolated from other phyla. Muscle fibres are cross-striated with an abundant sarcoplasmic reticulum (SR) coupled with regular transverse tubules via end-feet. Confocal microscopy revealed an array of micro-network structures inside the fibres stained with fluorescent ryanodine (BODIPY FL-X ryanodine), indicating the presence of ryanodine receptors. This pattern and dimensions coincide with the morphological configuration of the SR in chaetognath fibres. However, both ryanodine and caffeine had no effects on twitches of single muscle fibres in response to electrical stimuli, or intracellular Ca^{2+} transients evoked by externally applied ACh. These observations reveal that the ryanodine receptor in the chaetognath fibres does not function as an intracellular Ca^{2+} release channel, hence the SR is not involved in excitation-contraction coupling.

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OH3-02 Novel mechanism underlying marked hyperpolarization by activation of purinoceptor in t-BBEC 117

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Functional coupling between ion channels and receptors were examined in t-BBEC 117, an immortalized cell lines derived from bovine brain endothelial cell (BBEC). In t-BBEC 117, the stimulation of purinoceptors ($P2Y_2$ and $P2Y_{12}$) by ATP hyperpolarized cells to around -40 mV by activation of small conductance Ca^{2+}-activated K^+ channel (SK2). This often resulted in further activation of inward rectifier K^+ channels ($K_{ir}2.1$ and $K_{ir}2.2$), marked hyperpolarization to -80 mV and excessive increase in $[Ca^{2+}]_i$. Presumably due to Ca^{2+} influx through transient receptor potential (TRPC1). This switching of membrane potential regulation from SK2 to $K_{ir}2.X$ was blocked by preceded application of apamin. However, once membrane was hyperpolarized to -80 mV, it was reversed only by Ba^{2+}. These channels and receptors are involved in positive feedback mechanism in the regulation of $[Ca^{2+}]_i$.

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OH3-03 Functional role of ATP P2X receptors in deep dorsal horn neurons of the spinal cord

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Extracellular ATP has been demonstrated to act on P2X receptors to modulate synaptic transmission in CNS. In this study, we examined the functional role of P2X receptors in modulating excitatory synaptic transmission in deep dorsal horn (DDH) neurons by using patch-clamp recordings from spinal cord slice preparations. Bath application of ATPγS induced inward currents in about 30% of DDH neurons examined and largely increased mEPSC frequency in almost all DDH neurons. On the other hand, bath application of αβmeATP did not induce any inward current, but largely increased mEPSC frequency in almost all DDH neurons tested. These results indicate that distinct subtypes of P2X receptors are expressed in presynaptic and postsynaptic sites and that their activation enhances the excitability of spinal DDH neurons in different manners.

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OH3-04 Protons activate the δ-subunit of the epithelial Na^+ channel in humans

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The amiloride-sensitive epithelial Na^+ channels (ENaCα, β, γ, and δ) control Na^+ transport into cells and across epithelia. ENaCδ can associate with β and γ subunits and generate a larger current than that of homomeric ENaCδ. However, the distribution pattern of ENaCδ is not consistent with that of the β and γ subunits. To explain this discrepancy, we searched for novel functional properties of homomeric ENaCδ and investigated the detailed tissue distribution in humans. When hENaCδ was expressed in Xenopus oocytes and CHO cells, a pH reduction of activated this channel, and the acid-induced current was abolished by amiloride. ENaCδ mRNA was widely distributed throughout the brain. In conclusion, hENaCδ activity is regulated by protons, indicating that it may contribute to the pH-sensation and/or pH-regulation in the human brain.

OH3-05 Voltage-gated Ca^{2+} channels of Terminal Nerve (TN) Gonadotropin-Releasing Hormone (GnRH) Neurons and its modulation by GnRH.

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TN-GnRH neurons are functionally distinct from the hypophysiotropic hypothalamic GnRH neurons and have been reported to function as a neuromodulatory system. To reveal the major pathway for Ca^{2+} influx and regulation for them, we examined the expression profile of voltage-gated Ca^{2+} channels and its modulation by GnRH peptide. Whole-cell patch clamp recordings were made from TN-GnRH neurons in the dwarf gourami brain slice preparations. TN-GnRH neurons showed low-voltage activated (LVA) currents (T-type) and three types of pharmacologically distinct high-voltage activated (HVA) currents (N-, L- and R-type). Only HVA currents, but not LVA currents, were inhibited by GnRH in a dose-dependent manner. Here, we report the evidence for the paracrine/autocrine GnRH regulation of Ca^{2+} influx in TN-GnRH neurons.

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OH3-06 Transgenic mice that express GluR2 by glial cells specific promoter

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We constructed mice that express GluR2 under the control of the glial fibrillary acid protein (GFAP) promoter. GluR2 is a subunit of AMPA type ionotropic glutamate receptors that determine Ca^{2+} permeability and rectification properties. Recently, it is known that receptor is widely distributed not only in neurons but also in glial cells. To elucidate the role of AMPA receptors and their Ca^{2+} permeability in glial cell, we constructed mice overexpress GluR2 subunit in glial cells. The cDNA of GluR2 was placed between GFAP promoter and SV40 polyadenylation signal of a plasmid vector. After removal of the vector sequences, the expression unit was injected into fertilized eggs. The transgenic mice have smaller brains compared with wild type mice. Colocalization of GluR2 and GFAP was observed in Bergmann glial cells, which normally expressed AMPA receptors lacking GluR2.

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OH3-07 Spatiotemporal specificity in modification of dendritic integration by correlated activity

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Whether and how the dendritic integration is regulated by neuronal activity is largely unknown. Here we show that correlated pre- and postsynaptic activity, which is known to induce long-term synaptic modification, leads to persistent change in the linearity of spatial and temporal summation of excitatory postsynaptic potentials (EPSPs). Such change is spatiotemporal specific, in that it depends on the temporal interval and spatial order of the summated inputs. Furthermore, corresponding changes in EPSP- spike coupling was observed, indicating the physiological relevance of the changes in summation linearity. The modification of summation linearity depends on activation of NMDA receptor and a local modulation of Ih channel activity. These results unraveled an intriguingly novel rule in activity dependent plasticity beyond synapses.

OH3-08 Experience-driven regulation of synaptic size in *Drosophila* Neuromuscular Junctions

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How do neural cells form the appropriate size of synapses matched with their target cells? To solve this fundamental question of neural development, we studied the relationship between the synaptic size and the postsynaptic muscle size in *Drosophila* larval neuromuscular junctions. In abdominal muscle fibers 6 and 7 (M6 and M7) which are innervated by the same neurons, the volume of M6 is 1.5 times larger than that of M7 and accordingly the size of synapses is larger in M6 than M7. We refer to this phenomenon as "matching". We found that matching appeared several hours, but not just after hatching. When sensory inputs were suppressed, matching was not observed, while total synaptic growth was normal. These results suggest that experience-driven patterned neural activity is required for matching, but not for synaptic growth. Finally, matching was observed in a mutant of BMP type II receptor which is defective in synaptic growth. Possible involvement of other mechanisms including CaMKII will be discussed.

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OH3-09 Up-regulation of AMPA-receptor surface-expression by blocking of constitutive endocytosis in parallel fiber synapse of rat cerebellar Purkinje cell.

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Internalization of AMPA-type glutamate receptor (AMPA-R) from parallel fiber (PF) synapse is supposed to be underlying mechanism for long-term depression (LTD) in cerebellar Purkinje cell. This internalization is mediated through clathrin-dynamin dependent endocytosis. Using rat cerebellar slice preparation and whole-cell patch-clamp technique, we have found that blocking of dynamin by intracellular application of GTPγS, or dynamin proline-rich domain peptide enhanced PF-EPSC amplitude (160%). These results imply that there is constitutive insertion of GluR2 into subsynaptic membrane at PF synapse in Purkinje cell in slice preparation. Peptide of NSF/AP-2 binding site of GluR2 C-terminus also up-regulated PF-EPSC amplitude, suggesting that this site was also involved in internalization of GluR2 through constitutive endocytosis in cerebellar Pukinje cell.

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OH3-10 Signal modulation by glutamate uptake in the mouse outer retina

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We investigated how excitatory amino acid transporters (EAATs) contribute to the synaptic transmission from rods to a rod bipolar cell (RBC) in the mouse retina. The slice preparation was illuminated by a constant light to suppress the glutamate release from rods. When rods were stimulated by a current pulse, the voltage clamped RBC induced a transient outward current (a reduction of the L-AP4 sensitive inward current). As the pulse intensity was increased, the amplitude of the evoked current became large whereas the half-decay time did not change. However, application of TBOA, an antagonist of EAATs, increased the half-decay time for strong current pulses. Neither deletion of EAAT1 or EAAT3 gene nor pharmacological blockade of EAAT2 affected the half-decay time of the evoked current. These results suggest that the remaining EAAT, namely EAAT5, in the mouse outer retina may contribute to prevent confusion between the intensity and duration of light signals.

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OH3-11 Physical and Functional Interaction of the Active Zone Proteins, CAST, RIM1, and Bassoon, in Neurotransmitter Release

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CAST is a novel cytomatrix at the active zone (CAZ)-associated protein that directly binds another CAZ protein RIM1 and indirectly binds Munc13-1. We show here that all the CAZ proteins thus far known form a large molecular complex in the brain, including CAST, RIM1, Munc13-1, Bassoon, and Piccolo. RIM1 and Bassoon directly bind to the COOH-terminus and central region of CAST, respectively, forming a ternary complex. Piccolo also binds to the Bassoon-binding region of CAST. Moreover, disruption of these CAST-mediated bindings impair synaptic transmission in cultured neurons. These results indicate that CAST serves as a key component of the CAZ structure and is involved in neurotransmitter release by binding these CAZ proteins.

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OH3-12 The tonic inhibition mediated by γ Aminobutyric Acid$_A$ receptor in substantia gelatinosa neurons

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The persistent tonic form of GABAergic inhibition in addition to the phasic transmission has been observed in several brain region such as the hippocampus and cerebellum. Here we first report the GABAergic tonic current in substantia gelatinosa (SG) neurons of the spinal cord, which is important in transmitting nociceptive information. Whole-cell patch clamp recording from SG neurons in the spinal cord slice preparations from mice revealed two distinct types of GABAergic miniature IPSCs; the fast and slow decay kinetic currents. The neurons with the fast decay kinetic generated a persistent tonic or spontaneous inhibitory current that is revealed by the application of GABA$_A$ receptor blocker, picrotoxin. Further pharmacological profiles and RT-PCR experiment suggest that this tonic inhibition may be mediated by the GABA$_A$ receptor containing the ε subunit.

OH3-13 Developmental and regional expression of heparan sulfate sulfotransferases in the mouse brain

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Heparan sulfate (HS) glycosaminoglycans, polysaccharide chains expressed on cell surface and in extracellular matrixes as a part of proteoglycans, are known to be important for regulating various biological functions. We examined the expression of HS glucosaminyl 3-*O*-sulfotransferase (3-OST) isoforms, enzymes that transfer sulfate to C-3 position of glucosamine units in HS at Golgi, in the developing mouse brain. Structures of HS determined by a series of biosynthetic steps, and 3-OSTs are key enzymes to construct the final HS structure. As results of *in situ* hybridization for 3-OST mRNAs, each gene showed regionally different expression patterns in the developing mouse brain. Since different 3-OST isoforms are considered to generate distinct functional HS structures, our results suggest that the alteration of HS structure with 3-*O* modification correlates with various developmental processes in mammal brain.

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OH3-14 Generation of Cajal-Retzius Cells from the Caudal-Medial Wall of Telencephalic Vesicles

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An early and fundamental step of the laminar organization of developing neocortex is controlled by the developmental programs that critically depend on the activities of Cajal-Retzius (CR) cells in the marginal zone. However, the ontogeny of CR cells remained elusive. We show here the generation of CR cells from the caudal-medial wall of telencephalic vesicles, including the cortical hem, where the prominent expression of GFP is initially detected. These neurons tangentially migrate at the cortical marginal zone and are distributed throughout the entire neocortex in a caudomedial-high to rostrolateral-low gradient during the dynamic developmental period of corticogenesis. Therefore, our findings on CR cells, in addition to the cortical interneurons, add to the emerging view that the neocortex consists of neuronal subtypes that originate from a focal source extrinsic to the neocortex, migrate tangentially into the neocortex, and thereby underlie neural organization of the neocortex.

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OH3-15 A novel feedback signal through FGF18-ETS transcription factors is critical for the radial migration of cerebral cortical neurons

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Cerebral cortex is composed of six layers with distinct characteristics. Cortical neurons in layer 2-6 arise from progenitors located in the ventricular zone (VZ) in a temporally precise manner. Newly differentiated cortical neurons radially migrate toward the cortical plate (CP) and reside in their final layers. We show here that FGF18-dependent expression of ETS transcription factors (ETS-TFs) is required for the radial migration. Dominant-negative mutants of ETS-TFs and FGF receptors, normally expressed in the VZ, inhibited radial migration of newly differentiated cortical neurons. Forced expression of FGF18, normally expressed in the CP, induced an overexpression of ETS-TFs in the VZ. Contrary, the expression of ETS-TFs was reduced in FGF18-null mice. These results suggest a novel feedback signaling through FGF18-ETS TFs underlies the laminar formation in the cerebral cortex.

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OH3-16 Attempt of computational approach toward understanding the neural cell segregation during the cortical layer formation

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We recently found that cortical neurons born at embryonic day (E) 13 in mice settled mainly in layer V and those born at E14 came to be aligned around layer IV. Moreover, in cell aggregation assay of dissociated cells, E13-born and E14-born neurons were sorted out from each other. These results suggest that the neurons might acquire some birth-data-specific behavior, which contributes to the birth-date-dependent layer formation. To understand the mechanism of this layer formation, we have constructed a model developing cortex *in silico* by defining the various features of cell behavior including migration, cell signaling, differentiation, and proliferation. In this report, we will present our recent attempt of the computer simulation of corticogenesis, and would like to discuss how the cortical neurons are segregated into the distinct layers.

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OH3-17 Mediolateral compartmentalization of the cerebellum is determined on the "birth date" of Purkinje cells

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The adult cerebellum is functionally compartmentalized into clusters along the mediolateral (M-L) axis (M-L clusters). These M-L clusters appear to be the basic structure in which cerebellar functions are carried out, but the mechanisms by which cerebellar M-L compartmentalization is established are still unclear. To address these questions, I examined the development of M-L clusters using adenoviral vectors. The adenoviral vectors effectively introduced foreign genes into the neuronal progenitor cells of the cerebellum in a birthdate-specific manner. When the adenoviral vectors were injected into the midbrain ventricle of mouse embryos on embryonic days (E)10.5, E11.5, and E12.5, the virally infected cerebellar progenitor cells developed into Purkinje cells. Notably, the Purkinje cells that shared the same birthdate formed specific subsets of M-L clusters in the cerebellum. The results suggested that Purkinje cell progenitors are fated to form specific subsets of M-L clusters upon their birth between E10.5 and E12.5.

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OH3-18 Large-scale morphogenesis and neural network formation in a vertebrate, analyzed by using a photo-convertible coral fluorescent protein

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Large-scale cell rearrangement is a feature of the vertebrate development and a prerequeste for proper position-dependent cell differentiation, induction, and shaping of organs and the body. However, it is difficult to analyze because of its complex nature involving a number of cells whose individual behaviors are not strictly stereotyped. Here we focus on dynamic neighbor relations among cell populations labeled at a strategic time and space with green-to-red photo-conversion of a coral fluorescent protein, Kaede. This method critically visualizes a variety of morphogenesis in living zebrafish, such as gastrulation, crest migration, and neural network formation at global and individual cell levels. Moreover, it allows systematic surveys of dynamic processes: eg. coordinate transformation of a field, sliding vertical relations between germ layers during segmentation, and waves of cell exchange between left-right halves of a brain.

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OJ1-01 Dissociable activity in posterior parietal cortex associated with spatial and nonspatial attentional shifts

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The human posterior parietal cortex (PPC) has been implicated in both spatial and nonspatial functions. In the present functional MRI study, the functional organization of the PPC in relation to the spatial and nonspatial processes was investigated. The Wisconsin Card Sorting Test was modified such that neither eye movement nor spatial attention was required. A complementary oculomotor/spatial attention task was also performed. A double dissociation pattern between the adjacent regions was revealed in the IPS: the activity related to set shifting was found in the lateral parts, whereas the activity related to spatial processing was found in the medial parts. These results suggest the composite systems for attentional shifts in the IPS, the lateral and medial parts of which implement feature-based and location-based attentional shifts, respectively. These results also suggest the difference of the functional organization within the IPS between humans and monkeys.

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OJ1-02 ROLE OF THE PREMOTOR CORTEX IN COUNTING OF LARGE NUMBERS

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While it has been noticed that many animals have the ability to use small numbers, proficiency in using large numbers is unique to humans. In this study we applied fMRI to elucidate neural correlates of large number counting in the human brain. Sequential stimuli were delivered in three different sensory modalities: visual (white squares), auditory (440Hz beeps), and somatosensory (air puffs). Each stimulus lasted 200ms with an interstimulus interval of 300ms. Brain regions commonly activated irrespective of the input sensory modality were evaluated. During small number counting (1 to 4), such brain regions were detected in bilateral premotor cortices, pre-supplementary motor areas, posterior superior/middle temporal cortices, and the right posterior parietal cortex. During counting of large numbers (10 to 22), the left premotor cortex (an upper part of the ventral premotor cortex) was additionally activated. The results suggest that the left premotor cortex has a role in large number counting.

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OJ1-03 REPRESENTATION OF ACTION BOUNDARIES IN THE PREFRONTAL CORTEX OF THE MACAQUE MONKEY

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Complex behaviors are segmented into action subsets referred to as chunks by analogy to mnemonic chunking. We looked for evidence for such segmentation in the prefrontal cortex (PFC) while monkeys performing sequential saccade tasks. The monkey was required to capture the targets by making saccades to receive reward. Of the 658 task-related neurons in the PFC, many responded at each saccade. Nearly half of them showed an "extra" peak activity (EP) after the last target offset. EP occurred with nearly the same amplitude and timing in trial blocks in which sequence length, target-presentation intervals or reward parameters were varied. To test the possibility that EP coded task-end, we introduced an explicit end cue (making last target yellow). We found that EP was sharply diminished, and at the same time, the response to the end cue was sharply augmented, as was response at task start. We suggest that PFC EP cells can mark the boundaries of ongoing action sequences to aid network processing of sequential behavior states.

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OJ1-04 Interaction between bottom-up and top-down signals in area V4 during a multi-dimensional visual search task

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Natural scenes contain multiple objects that are unique in different dimensions, so that an object with feature contrast to surrounding objects pops out. If we know about the dimension in which a target object differs from surrounding objects, we can detect it by attending to that dimension. Previously, we examined V4 activity during a multidimensional search task in which two odd stimuli (shape and color-odd) were presented among other stimuli. Monkeys had to make a saccade to one of odd stimuli depending on the instructed search dimension. Our results showed that V4 activity encoded both stimulus-oddity and behavioral-significance of the receptive-field stimulus. Here, we now examined neural mechanisms that induced these modulations and found that significant response increments for the target occurred only when one particular odd stimulus became the target. This suggests that interactions between bottom-up (stimulus oddity) and top-down signals (search dimension) occur in V4.

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OJ1-05 Prefrontal-inferior temporal activation in the effort for the memory load; PET study of macaque monkeys with learning-set.

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To identify the regions which are critically involved in the retention and retrieval of the memory of stimulus representation, we carried out the PET study while the monkeys *(macaca mulatta)* who acquired the learning-set formation were performing the simple visual discrimination tasks (VDT) that consists of plural number of problems learned previously. SPM analysis revealed that the lateral prefrontal cortex and the area TE in the inferior temporal cortex were significantly activated during the execution of VDT depending on the number of items which the monkeys had to retrieve concurrently. These results suggest that the activation of the prefrontal-inferior temporal circuit is tightly associated with the effort for the memory load.

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OJ1-06 Medial prefrontal dopamine release during a visual search task

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Modulation of neural function by dopamine (DA) is essential for cognitive behavior. The aim of the present study was to investigate neuroanatomy of DA release during a memory guided visual search behavior. By using PET with ^{11}C-raclopride, we indirectly monitored changes in regional synaptic levels of DA. In the visual search task, red boxes were presented. Subjects searched through these boxes by touching each one. At any time, a single token was hidden, and the goal was to collect the tokens. Once a token was found, then that box would not be used again to hide a token. Therefore, the subjects had to remember the previous locations of the tokens, and developed a search strategy. In the control task, the subjects simply touched highlighted boxes. We found decreases in ^{11}C-raclopride binding in the right medial prefrontal cortex during the visual search task compared to the control task. The findings suggest DA release in the medial prefrontal cortex is involved in the visual search behavior.

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OJ1-07 Task-selective activation in Broca's area is differentially modulated during first and second language acquisition

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It remains to be elucidated whether cortical activations are modulated by factors of age and proficiency level (PL) during first language (L1) and second language (L2) acquisition. Using fMRI, we tested subjects aged 13 or 19, and compared the cortical activations involved in past-tense verb *identification* with those involved in verb matching. We found past-tense task-selective modulation of cortical activations in both L1 and L2. For L1 alone, the left F3O activation was observed to be greater in the 13-year-old subjects than in those aged 19, whereas both groups' PLs were matched. In contrast, the left anterior F3t activation decreased as PL improved without showing any significant effects of age. On the other hand, left F3op/F3t activation was enhanced for intermediate PL groups, which was observed only in L2. These results demonstrate that the task-selective activations in Broca's area are differentially modulated in the course of L1 and L2 acquisition.

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OJ1-08 The Effect of Word Familiarity on Visually Evoked Magnetic Fields

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Word familiarity is an index of the relative erase of word perception, and is characterized by facilitation and accuracy on word recognition. We studied the effect of word familiarity using visual stimuli of "Hiragana" (phonetic characters in Japanese orthography) characters on the elicitation of visually evoked magnetic fields with a word-naming task.The three components were observed in the averaged waveforms of the root mean square (RMS) value on the latencies about 100ms, 150ms and 220ms. The root mean square (RMS) value of the 220ms component showed a significant positive correlation ($F=(3/36)$; 5.501; $p=0.035$) with the value of familiarity. Increments in the RMS value of the 220ms component, which might reflect ideographical word recognition, retrieving "as a whole", and reflect a "pop-out" function with the escaping character inhibiting other character, were enhanced with increments of the value of familiarity.

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OJ1-09 NEURAL CORRELATES TO SEQUENCE AND CONTEXT IN THE RAT HIPPOCAMPAL FORMATION

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To investigate a hippocampal (HF) involvement in sequence- and context-dependent encoding and retrieval, CA1 neurons were recorded from the rat HF during a conditional delayed stimulus-response association task. A trial was initiated by one of 3 pure tones (CTs) with different tone frequencies which were presented from one of 3 directions, followed by one of the 3 different serial reinforcements [i.e., air puff (AP)-tube protrusion (TP) near a rat's mouth to evoke licking sucrose solution, TP-TP, and TP-AP] after a delay. Of the 111 HF neurons, 42 and 45 responded to the AP and TP. Activity of the most AP- and TP-responsive neurons was dependent on both presentation sequences of the AP and TP in the task and the CT frequencies and directions. The results demonstrated sequence- and context-dependent HF neuronal responses, suggesting a crucial role of the HF in episodic memory.

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OJ1-10 An orientation selective VLSI system emulating the simple cell response of the primary visual cortex

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We have designed an analog VLSI (Very Large Scale Integrated) systemto emulate the simple cell responses of the primary visual cortex to natural scenes. The system consists of two silicon retinas and two orientation selection chips. This structure mimics the fundamental hierarchical architecture of the visual system that leads to V1 from the retina. Namely, image is filtered by a Laplacian-Gaussian-like receptive field of the silicon retina and the orientation selection chip selectively aggregates multiple pixels of the silicon retina as proposed by Hubel and Wiesel. Using the system, we could reproduce the response of disparity selective cells predicted by the energy model (Ohzawa, DeAngelis & Freeman, 1990) in real-time. The developed system is useful to analyze the function of simple cells in early vision under real-time visualization of the neuronal image.

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OJ1-11 Monocular features define stereoscopic depth of binocularly unpaired component

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Simple geometrical features, such as relative size, have been thought to never affect stereoscopic depth perception in the distance dimension. So, as far as we know, an essentially important approach has never been attempted, that is, creating stereograms that the monocular depth information would define the stereoscopic depth. We found new stereograms, in which leading a subjective occluding edge enables a binocularly unpaired component to have the stereoscopic depth that depends on the relative size or relative luminance. The stereograms revealed that the relative size was rather strong source of monocular depth information for all our subjects. By using our stereograms, we could evaluate the monocular depth information quantitatively with the disparity measure. Measuring the perceived depth revealed that the monocular depth information is derived from the geometrical relationships in the visual space in subject-dependent manners.

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OJ1-12 Task specific effects of microstimulation in area MT during depth discrimination

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We have recently reported, using single unit recording techniques, that disparity-selective neurons in area MT contributes to "coarse", but not "fine" depth discrimination. Here we confirm this task specificity using electrical microstimulation during "fine" depth discrimination.
Visual stimuli were bi-partite (center/surround) random-dot stereograms tailored to match the receptive field properties of each stimulation site. Monkeys discriminated the relative depth between the two patches. Microstimulation was applied for half the trials and were randomly interleaved with non-stimulated trials.
Across 78 microstimulation sites, we found no overall effect of microstimulation. To confirm a task-specific effect of microstimulation, for a subset of 46 sites, we trained monkeys to switch between the "coarse" and "fine" tasks. We observed a significant effect of microstimulation for the "coarse"task, but not for the "fine" task. Results support the idea that MT contributes to "coarse", but not "fine" depth discrimination.

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OJ1-13 Selectivity for relative disparity in V4

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Relative, rather than absolute, disparity is a major cue contributing to depth perception. A previous study shows that coding of relative disparity begins, albeit incomplete, in V2. Here we tested responses of V4 neurons to various combinations of two depth planes in two alert monkeys. A circular patch of a dynamic random-dot stereogram, consisting of a center disk and a surround annulus, was presented over the receptive field of each neuron. If a neuron codes relative disparity, the disparity-tuning curves of the center disk should shift along the disparity axis by an equal amount and the same direction of the shift in surround disparity. We quantified such shifts by calculating a shift ratio (the shift in the tuning curve / the shift in the surround disparity). The distribution of shift ratios was unimodal and biased towards the expected direction (median=0.38, n=142). A comparison with V2 demonstrates that V4 neurons as a population exhibit substantially higher sensitivity to relative disparity.

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OJ1-14 Disparity tuning of area V4 neurons differs when tested with dynamic random-dot stereograms or solid-figure stereograms

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Neurons in the macaque visual area V4 are sensitive to binocular disparity in dynamic random-dot stereograms (RDSs) or solid-figure stereograms (SFSs). Because disparity change in an RDS does not accompany positional shifts in monocular images, subjects must use the stereo vision system to detect the change. Thus an RDS is useful for investigating the neural mechanism of stereo vision. However, RDS-like images are rare in natural scenes, while SFS-like images are common. Here we quantitatively analyzed disparity tuning of V4 cells, each tested with RDSs and solid bars. No correlation existed in the modulation amplitude, tuning sharpness, and preferred disparity between the tuning curves obtained with the two stimulus types. Disparity tuning curves tested with RDSs were sharper than those tested with SFSs. The results suggest that stereoscopic depth representations of RDSs and SFSs considerably differ in V4.

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OJ1-15 Area TE neurons have invariant color selectivity for both categorization and discrimination tasks

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To study the effects of the behavioral demands on the color processing in the inferior temporal cortex, we recorded neuronal responses in area TE of two Japanese monkeys during both a categorization task and a discrimination task. In categorization task, monkeys were trained to keep fixating or saccade after reddish or greenish test color was presented, respectively. In discrimination task, two colors were shown after presentation of a test color. Monkeys were trained to make saccade toward the one of two colors that corresponds to the test. In both tasks, we used eleven test colors that were arranged at equal intervals from red to green. We found complete agreement of color selectivity of neurons across different tasks including categorization, discrimination and simple fixation. These findings suggest that TE neurons code exact color information independent of the task demand.

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OJ1-16 A peripheral motion illusion and fixational eye movements

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In peripheral viewing, a static pattern with asymmetrical luminance gradients appears to move in one direction. To examine the hypothesis that the source signal of the illusion originates in image motions due to small eye movements, we tested subjects with two experiments, one for rating of illusion saliency and the other for recording of small eye movements. The stimulus was rated as moving more clearly when free eye movements were allowed than when steady fixation was required. Under steady fixation, a positive correlation in the inter-subject scattergram was found: subjects with larger fixation instability saw more salient illusory motion. Under free eye movements, correlation was insignificant. It is suggested that small eye movements are related to this illusion.

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OJ1-17 Chemically Specific Circuit Composed of VGLUT3- and PPTB-Producing Interneurons in Rat Neocortex

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The third vesicular glutamate transporter, VGLUT3, immunoreactivity was observed not only in axon terminals but in some cell bodies of neocortical neurons. Since the vast majority of VGLUT3-immunoreactive neurons showed immunoreactivities for GABA, preprotachykinin B (PPTB), VGLUT3-immunoreactive neocortical neurons were considered to constitute a subgroup of GABAergic interneurons. The neocortex contains two kinds of VGLUT3-laden axon terminals: one is serotonergic and derived from the raphe nuclei, and the other is GABAergic and intrinsic in the neocortex. Furethermore, VGLUT3-laden GABAergic terminals made basket-like structures and the axon baskets terminated mostly on cell bodies of PPTB-expressing interneurons, some of which themselves expressed VGLUT3. Thus, VGLUT3-expressing GABAergic interneurons were considered to form a chemically specific circuit within the PPTB-producing interneuron group in the rat neocortex.

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OJ1-18 Differential developmental expression of three DGK subtypes, α, β and γ.

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Diacylglycerol kinase (DGK) phospholyrates diacylglycerol, and indirectly terminates signal transduction mediated by protein kinase C. Among 9 subtypes of mammalian DGK, type I DGK which are Ca^{2+}-dependent and brain-specific, are suggested to play functional roles in the central nervous system. To elucidate which subtype is involved in postnatal neuronal development, we studied the developmental changes of DGKα, β, γ in the postnatal rat brain. α and γ subtypes were present at birth and then gradually increased, while DGKβ was not present at birth or postnatal day 3, then increased rapidly from day 14 to reach maximum at day 28. Immunohistochemically, DGKγ was expressed in the thalamus on day 3, but not found there at day 28. In primary cultured cortical neurons, endogenous DGKγ localized in the cytosol while DGKβ localized along the plasma membrane. These findings suggest that each subtyps has a spatio-temporally different function in the developmental neurons.

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OJ1-19 Endothelin-stimulated DNA synthesis in neural progenitor cells: Involvement of protein kinase C and integrin

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We have previously shown that administration of pertussis toxin (PTX) into the lateral ventricle of embryonic brain reduced total cell number and BrdU-positive cells in the cerebral cortex, suggesting impaired proliferation of neuroepithelial cells. Endothelin (ET) effectively stimulated the incorporation of [^{3}H]thymidine into cultured neural progenitor cells via the ET-B receptor-Gi2. In the present study, we found that DNA synthesis was stimulated by the phorbol ester, while protein kinase C (PKC) inhibitors inhibited ET- stimulated DNA synthesis. Fibronectin enhanced ET-stimulated DNA synthesis. ET stimulated tyrosine phosphorylation of paxillin, and an antagonist of ET-B receptor, PKC inhibitors, and RGD peptide inhibited this effect. These findings indicate that ET-B receptor signaling to maintain mitogenic activity involves PKC and integrin.

OJ1-20 Bcl-6 expression in olfactory sensory neurons

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Despite their neuronal identity, olfactory sensory neurons undergo continuous apoptosis and regeneration even in adults. Taking a speculative analogy between the olfactory and immune systems, we studied one of the key proto-oncogenes in lymphocyte terminal differentiation, Bcl-6, a transcriptional repressor required for the germinal center formation, in the mouse olfactory sensory neurons. Immunohistochemistry using anti-Bcl-6 antibodies showed its presence only in a limited population of olfactory sensory neurons as characteristic staining signals in nuclei. RT-PCR and Western blot analyses confirmed its high expression at the mRNA and protein levels in the olfactory epithelium, compared with other neuronal tissues. Artificial induction of neuronal apoptosis in the olfactory epithelium appeared to increase Bcl-6-positive cells. These data are consistent with other studies on non-olfactory cells and our hypothesis that Bcl-6 plays a role in apoptosis and differentiation of olfactory sensory neurons through a transcriptional control of its downstream genes.

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OJ1-21 Effects of extracellular ATP on the neuronal network function of the rat spinal cord dorsal horn

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We aimed to elucidate the role of extracellular ATP in spinal cord nociception at the neuronal network level of the dorsal horn. Transverse spinal cord slices prepared from juvenile rats were stained with a voltage-sensitive dye (di-4-ANEPPS) and maintained in vitro. Neural activity induced by electrical stimulation of dorsal roots was recorded in the dorsal horn with high-speed optical recording system. Extracellular ATP attenuated the depolarizing neural activity, which was antagonized by an adenosine receptor antagonist. After ATP was washed out, neural excitability was restored and became even stronger than before applying ATP. This augmentation was more prominent at the deep layers (lamina III-IV) than at the superficial layers (lamina I-II) of the dorsal horn. It is suggested that ATP has longer-lasting excitatory effect than inhibitory effect of adenosine on the dorsal horn neuronal network.

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OJ2-01 Neural mechanism of dual task: A human PET study

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The aim of this study is to clarify the neural mechanism of interference when several things were processed simultaneously. Reaction time (RT) and brain activity measured by positron emission tomography (PET) when subjects performed two tasks simultaneously (dual task condition: DT) were compared to those in the single task condition (ST). RT for each stimulus in DT was significantly prolonged as compared to that in ST. Image analysis with SPM99 indicated that the prolonged RT was negatively correlated to the right BA 6 activated only in DT. We did not find any activation in the prefrontal cortex thought to play an important role for the interference in DT. These findings suggested that BA6 would be also associated with the interference when we performed several things simultaneously.

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OJ2-02 Different fusion sites between 1st- and 2nd-phase insulin release from rat pancreatic β cells analyzed by TIRF

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Total internal reflection (TIRF) microscopy imaging is a powerful tool to determine the site of exocytosis of secretory granules in neuronal and endocrine cells. We analyzed the interaction in primary β cells between the docking/fusion of GFP-tagged insulin granules and syntaxin1 (Syx1) labeled by TAT-conjugated Cy3-tagged antibody using TIRF. TAT-conjugated Cy3-tagged anti-Syx1 antibody was transduced rapidly into the cells and specifically labeled the endogenous Syx1. TIRF revealed that Syx1 was distributed in numerous separate clusters where granules were preferentially docked. The fusion from previously docked granules during 1st-phase release occurred exclusively at the site of the Syx1 clusters, but the fusion from newly recruited granules during 2nd-phase occurred other than the clusters. Thus our data demonstrated that there were different fusion sites between 1st- and 2nd-phase insulin release from β cells.

OJ2-03 Cortical activation during apple peeling as studied by NIRS and fMRI

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As an application of neuroimaging to directly measure human brain activities during daily living, we investigated cortical activation patterns during apple peeling. A pilot NIRS study assessing the whole lateral cortical surface suggested substantial activation in the prefrontal cortex (PFC) in addition to expected activations in the sensorimotor areas during apple peeling.We next examined cortical activation during mock apple peeling by simultaneous measurement using NIRS and fMRI. We detected activations in the sensorimotor areas but not in the PFC. Finally, we conducted NIRS study focusing on the PFC, and found the significant increase in the regional concentrations of oxygenated hemoglobin during apple peeling. Thus, we demonstrated that apple peeling as practiced in daily life recruited the PFC but that such activation might not be detected for less laborious mock apple peeling.

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OJ2-04 Probabilistic anatomical cranio-cerebral correlation via the international 10-20 system

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Transcranial brain-mapping methods, NIRS and TMS, involve a technical drawback of measuring cortical activities from the head surface without anatomical information. To overcome this problem, we examined cranio-cerebral correlation using MRI via the guidance of the international 10-20 system for EEG. We manually projected the 10-20 standard cranial positions over the cortical surface and examined the cranio-cerebral correspondence for 17 healthy adults. We normalized the 10-20 cortical projection points to the standard coordinate systems and obtained their probabilistic distributions. The locations of 10-20 cortical projection points in the standard coordinate systems could be estimated with an average standard deviation of 8 mm. This study contributes to establishing a 3-D probabilistic anatomical platform that enables intra- and inter-modal comparisons of NIRS and TMS brain imaging data.

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OJ2-05 Imaging of Stem Cell Recruitment to Ischemic Infarcts in a Murine Model

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Neural progenitor cells (NPC) have been reported to aid in the functional recovery from neurological diseases and injuries. However, the kinetics of transplanted NSC were based on histological analyses and imaging tools have been needed to study the in vivo migratory behavior. We show that bioluminescence imaging (BLI) can be extremely favorable for detecting transplanted NSC in a living animal with ischemia. C17.2 NPC stably expressing luciferase were imaged by BLI over 3 weeks in nude mice with ischemia, followed by contralateral injection of NPC. NPC migrated to the site of infarct from the contralateral parenchyma. These BLI images correlate well with histological cell distributions. We conclude that NPC recruitment to infarct can be assessed noninvasively by serial *in vivo* imaging.

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OJ2-06 Imaging regenerative peripheral axons in live mice by fibered confocal fluorescence microscopy

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Introduction: An in vivo imaging procedure which would allow to monitor on line axonal behavior within injured nerves, would be of high interest to evaluate the effects of neurotoxic and neurotrophic treatments. Vizualisation of the peripheral nervous system with a single axon resolution, usually implies to monitor the degeneration-regeneration sequence in different samples. The optical technology we present allows high resolution imaging at a very same site. Materials and Methods: Adult transgenic Thy1-YFP mice were used to monitor axonal degeneration and regeneration after a unilateral crush injury or transection of the saphenous nerve. A hand-held optical probe (0.65 mm diameter) instrument (Cell-viZio, Mauna Kea Technologies) was used. Conclusion: FCFM allows for the sequential, in vivo imaging of nerve regeneration with limited invasivity. Thus the Cell-viZio approach provides a unique tool for medium throughput screening of molecules promoting or limiting neuronal recovery.

OJ2-07 RNAi-induced gene silencing by local electroporation (RISLE):Method to knockdown a target protein at the defined brain region in vivo

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Genetic manipulation for 'knockout (KO)' is a useful tool for characterizing a target gene. However, it has some shortcomings. Here we describe a novel knockdown technique, RNAi-induce gene silencing by local electroporation. Small interfering RNA (siRNA) introduction by electroporation at a specific brain region results in a marked reduction of the expression of both mRNA and protein of the target gene without affecting the expression of proteins other than the target or causing pathological changes in target tissues. The effective electric pulses consist of a strong short pulse and a weak long pulse in tandem. Moreover, the knockdown models constructed using this technique have physiological functions consistent with previous findings. These results suggest that this technique is applicable with spatial flexibility, temporal accessability, and easy establishment of knockdown models, and is expected to be applicable to disease therapy in the future.

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OJ2-08 Rapid microadministration of neurotransmitter toward neuron by electrochemical pump using blakened platinum electrode

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An electrochemical micropump powered by bubbling generated by water electrolysis at blackened platinum electrodes was designed for rapid chemical stimulation of neurons by a neurotransmitter. This pump can stop the jet of internal fluid very quickly by controlling voltage for disappearing bubble on the electrodes. Voltage of 3.0 V was applied to the electrodes to jet out the solution toward the neuron at the distance of 100 μm while monitoring its membrane potential. Acetylcholine administration to a resting neuron generated neural firing only when voltage was applied for 0.5 s without time lag. During administration of GABA to active neurons, the spontaneous firing disappeared after application of 3.0 V with a time lag of 1 s. We conclude that neurotransmitter administration using the electrochemical micropump using blackened platinum electrodes enables real-time control of excitation and inhibition of neurons as designed.

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OJ2-09 Safety Assessment of Neuronal Drugs using Protein Expression Profiles by SELDI-TOFMS

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To evaluate the safety and efficacy of neuronal drugs, protein expression database of neural cells treated with several known drugs can be used as a comparative reference. To compare the protein expression profiles in control and drug treated neural cells, we utilized surface enhanced laser deposition/ionization time of flight mass spectrometry (SELDI-TOFMS) as a high-throughput and sensitive screening tool for the analysis of complex protein samples.
We prepared cell lysates from mouse neural stem/progenitor cells (NSPCs) which were treated with various neuronal drugs, and analyzed their protein expression profiles using SELDI-TOFMS. We found that some commonly expressed proteins in NSPCs were dramatically changed when NSPCs were treated with the drugs. These findings suggested that protein expression profiles obtained by SELDI-TOFMS would provide useful information for evaluation of drug safety and efficacy.

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OJ2-10 Cerebellar development transcriptome database

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The mouse cerebellum develops through a series of cellular developmental events on a schedule that unfolds within the first three weeks of life. To decipher this genetic blueprint, we are exploring the developmental gene expression profiles by utilizing the genome-wide approaches such as differential display, DNA microarray, and GeneChip. The spatio-temporal expression patterns are further analyzed by RT-PCR (timetabling) and in situ hybridization (cellular mapping). We systematized these lines of spatio-temporal gene expression information and have generated an integrative CDT database that will be on-line for the public. The CDT database has various search functions and easy accessibility to relevant public databases (e.g., NCBI-Nucleotide/PubMed, JAX-MGI, EBI-Ensembl), and thus will be a resource for the CDT informatics. In this presentation, we will introduce the basic design of the CDT database and how to use it.

OJ2-11 Propagation of Electrically Evoked Activity in Guinea Pig Primary Auditory Cortex

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In primary auditory cortex (AI), a pure tone activates a row of neurons, called an isofrequency strip (IS). A fundamental issue concerns the role of thalamocortical input and intracortical connectivity in the formation of ISs. Here we studied the contribution of intracortical circuits to the formation of ISs in guinea pigs. We found that a pure tone evoked propagating activity along a strip in AI, and that a focally applied electrical pulse triggered similar activities. The propagation velocity of electrically evoked activity, however, was significantly slower than that of tone-evoked activity, but comparable to the velocity of horizontal activity propagation reported in cortical slices, suggesting that the electrically evoked activity propagation is mediated by intracortical circuits. Our results suggest that cortical circuits play a role in defining the pattern of activity propagation in AI.

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OJ2-12 The insulin-like signaling pathway regulates the plasticity of salt chemotaxis in *C. elegans*

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The nematode *C. elegans* shows an attractive behavior to a variety of salts such as NaCl. This behavior is dramatically switched to an avoidant behavior by prolonged exposure to salts under stressful conditions. We found that mutants affected in the insulin-like signaling pathway have defects in this form of learning. These include mutants of *ins-1, daf-2, age-1* and *akt-1*, which encode homologues of insulin peptide, insulin/IGF-I receptor, PI 3 kinase and Akt/PKB, respectively. We determined the time and space in which this signaling pathway acts. Expression of *ins-1* is required at the adult stage. INS-1 protein is expressed in a pair of interneurons and the expression of *ins-1* is required in these neurons. Expression of *age-1* is required in the main salt-sensing chemosensory neuron. These data suggest a model in which the PI 3 kinase pathway in the salt-sensing chemosensory neuron is activated by the insulin-like molecules secreted from interneurons, and regulates the plasticity of salt chemotaxis.

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OJ2-13 The Ras-MAPK pathway controls neural circuit-dependent odor adaptation by regulating localization of glutamate receptors in *C. elegans*

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C. elegans shows a decrease in chemotaxis to odorants after exposure to the odorant for 5 min. This plasticity, called early adaptation, requires AIY interneurons, which receive synaptic inputs from olfactory neurons. Mutants of the Ras-MAPK pathway showed defects in this adaptation. AIY-specific expression of *let-60Ras* rescued the defects in the *let-60(lf)* mutants. Continued exposure to an odorant caused activation of MAPK in AIY. These results suggest that the functions of the Ras-MAPK pathway in AIY are important for adaptation.The function of GLR-1, a non-NMDA type glutamate receptor, is also required in AIY for adaptation. Odorant stimuli caused gathering of the GLR-1 protein at postsynaptic sites in AIY. Mutation of the Ras-MAPK pathway impaired localization of GLR-1. These results suggest that the Ras-MAPK pathway controls odor adaptation by regulating GLR-1 localization in AIY.

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OJ2-14 Na_x channel in the subfornical organ is essential for the control of salt-intake behavior

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Na concentration in body fluids is strictly maintained in mammals. Na_x channel is sensitive to an increase in the extracellular Na concentration and expressed in the subfornical organ (SFO) in the brain. We have proposed that Na_x is the Na-level sensor of body fluids. *Na_x*-deficient mice (KO) keep drinking salty water under dehydrated conditions, while wild-type mice (WT) avoid Na under such conditions.Here we tested the Na-level sensor hypothesis by direct infusion of hypertonic Na solution to the cerebral ventricle. The infusion induced prompt intake of water and aversion to salt in WT. In contrast, such aversive behavior was not observed in KO. When recombinant *Na_x* gene was successfully introduced to the SFO with Adeno-virus vector, KO began to avoid Na solution. Thus the defect of KO in sensing the systemic Na elevation was recovered by the expression of transfected *Na_x*. These results support the view that Na_x is the Na-level sensor in the brain.

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OJ2-15 Effects of β-caryophyllene on the depressive mood during the Menstrual Cycle

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In women depressive symptoms often occur during the premenstrual period. We examined the effect of β -caryophyllene and linalool on emotions of women during the menstrual cycle because we have recently demonstrated that odor substances modulate brain functions. Six healthy women smell vehicle (Dipropylene Glycol) followed by 3% β -caryophyllene or 8% linalool dissolved in vehicle in the follicular phase (F), ovulatory phase (OV), early luteal phase (EL) and late luteal phase (LL). Thirty min after exposure to these odors, emotional symptoms were estimated by questionnaires. β -caryophyllene increased the score of happiness ($p=0.06$) in the EL and decreased fatigue in the LL. linalool no improvement was found in emotional categories. These findings suggest that β -caryophyllene improve the symptoms of depressive disorder especially in the luteal phase.

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OJ2-16 Gustatory responses in the hippocampal CA1 area in freely behaving rats

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Although the hippocampus (HF) receives highly integrated information of all sensory modalities, there have been no reports indicating existence of HF taste neurons. In the present study multiple neural activities were recorded from the HF CA1 area of freely behaving rats. Rats were trained to ingest several sapid solutions from a small spout in an open field: 0.1M NaCl, 0.3 M sucrose, 0.1 M monosodium L-glutamate (MSG), 0.01 M citric acid, 0.0001 M quinine HCl, 0.5m M guannosine 5 monophotphate (GMP), mixture of MSG and GMP, and distilled water. Of 200 neurons analyzed, activity of 124 increased during licking. Among these 124 neurons, 24 differentially responded to the sapid stimuli. On the basis of response magnitudes, taste neurons were classified as follows; 1 NaCl-best, 2 sucrose-best, 2 citric acid-best, 7 QHCl-best, 6 MSG-best, and 6 miscellaneous types. These results demonstrated that the HF encoded not only spatial but also gustatory information.

OJ2-17 Effect of adenosine on hyperalgesia after spinal cord injury

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Sensory abnormality is one of the serious problems in the patients with injured spinal cord. We tested intrathecal application of adenosine receptor agonists and antagonists in rats with experimental spinal cord injury. Under general anesthesia, rat spinal cord was compressed by 20-g weight for 20 minutes. Rats showed hyperalgesia, which was evaluated thermal threshold using Hargreave's plantar test apparatus. Three days after the injury, Cl-adenosine, an unselective adenosine receptor agonist, was applied intrathecally. Hyperalgesia, induced by spinal cord injury, was inhibited by the application of Cl-adenosine. Simultaneous application of DPCPX, an inhibitor of A1 receptor, blocked the effect of adenosine on hyperalgesia. Intrathecal application of R-PIA, an agonist of A1 receptor, also inhibited spinal cord injury-induced hyperalgesia. These results suggest that activation of adenosine A1 receptor may be effective therapeutic method for the treatment of the neurogenic pain.

OJ2-18 The effect of Neuromedin U on excitatory synaptic transmission in the rat spinal cord

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Neuromedin U (NMU) is a brain-gut peptide firstly isolated from the spinal cord. We report on the localization of NMU and the actions of NMU on synaptic transmissions in adult rat spinal cord slices using whole patch-recording technique. NMU was confined in the medium- to large-size neurons in dorsal root ganglion and neurons in deep dorsal horn. Bath-applied NMU increased frequency of glutamatergic miniature excitatory postsynaptic currents (mEPSCs) but not amplitude both in superficial and deep layer of dorsal horn. These results indicate that NMU enhances not only Aγ -mediated nociceptive transmission, but also Aβ- mediated tactile transmission. NMU is a novel neuropeptide that facilitates excitatory transmission presynaptically in the rat spinal cord.

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OJ2-19 Anti-nociceptive action of dopamine in substantia gelatinosa neurons of the rat spinal cord.

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Compared with the enormous literatures devoted to anti-nociceptive action of NA and 5-HT, spinal actions of Dopamine (DA) have received scant attention. We investigated the DA effects on substantia gelatinosa (SG) neurons in the rat spinal cord by the use of whole-cell patch-clamp technique. Bath applied DA produced hyperpolarization or outward currents (under the current or voltage-clamp mode, respectively) in almost all SG neurons. The D1 like receptor agonist SKF 38393 did not make any actions, while the D2 like receptor agonist quinpirole induced outward currents as well as DA. In addition, the D2 like receptor antagonist sulpiride inhibited the DA-induced outward currents. These results suggest that the anti-nociceptive effect of DA can be mediated by the hyperpolarization of spinal dorsal horn neurons via D2 like receptors.

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OJ2-20 Roles of nerve growth factor in gastric hyperalgesia induced by experimental ulcer in the rat

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Aim of Investigation: To investigate the role of nerve growth factor (NGF) in gastric hyperalgesia induced by experimental ulcer in the rat.
Methods: Ulcer was made by acetic acid injection into the stomach wall. The NGF expression, stomach innervation, and expression of NGF receptors in gastric sensory neurons were examined by immunohistochemistry. The involvement of NGF in hyperalgesia was behaviorally evaluated.
Results: An increased NGF immunoreactivity with accumulation of polymorphonuclear leucocytes was observed in the ulceration. The 200-kDa neurofilament protein- and growth associated protein 43- immunoreactive nerve fibers increased in gastric mucosa. Both high and low affinity NGF receptors (TrkA, p75) in the nodose ganglia decreased, while TrkA in the spinal ganglia did not. Gastric hyperalgesia was reversed by neutralizing anti-NGF antibody.
Conclusion: These data suggest that the NGF contributes to the development of gastric hyperalgesia.

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OJ2-21 Activation of contralateral thalamus in deafferentation pain (DP) patients after successful motor cortex stimulation (MCS)

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MCS reduces DP, but the detailed mechanism has been unknown.To investigate the mechanism of MCS, we used PET to determine rCBF changes. Six right-handed patients with intractable pain of left hands were implanted with electrodes and studied. With 30 minutes MCS, their pain were remarkably reduced.The rCBF was studied using PET with ^{15}O-labeled water in the before MCS (painful) and after MCS (painless). The significant rCBF increase in contralateral thalamus ($p<0.05$, corrected, SPM99), and ipsilateral rectal gyrus, anterior cingulate, dorsal pons, and contralateral orbitofrontal gyrus and anterior insular cortex were revealed. This is the first report describing that the contralateral thalamus was significantly activated and pain relief was achieved by MCS.

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OJ2-22 Mechanisms of neuropathic pain in histamine H_1 and H_2 receptors gene knockout mice

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Histamine is released from damaged cells in response to tissue injury and excites polymodal nociceptors. In the present study, H_1 and H_2 receptor knockout mice were used to examine the contribution of histamine receptors to the development of neuropathic pain. To induce a neuropathic pain, we partially ligated the sciatic nerve and measured changes in sensitivity to thermal and mechanical stimuli. We observed significant difference in the development of allodynia between H_1, H_2RKO and wild-type mice. We also found that H_1, H_2RKO mice showed less neuropathic pain behaviors than wild-type mice. These results indicate that histamine receptors play an essential role in the development of neuropathic pain after peripheral nerve injury.

OJ3-01 Effects of histone deacetylase Sir2α on proliferation and differentiation of neural stem cells.

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Silent information regulator 2 (Sir2) is firstly identified as NAD-dependent histone deacetylase in yeast. The function of some homologues of Sir2 still remains to be elucidated in mammals. By immunohistochemistry and neurosphere assay, we found that Sir2α (SIRT1) was highly expressed in the neural stem cells and ependymal cells. To examine roles of Sir2α in the proliferation of neural stem cells, nicotinamide, splitomicin, and sirtinol, specific inhibitors of *Yeast* Sir2 and *Human* Sir2α, were administered to culture medium of neurosphere. Fewer and smaller neurospheres generated in the culture with nicotinamide or splitomicin. Next, we have examined functions of these drugs on the differentiation. We found that compounds strongly inhibited differentiation into neurons and oligodendrocytes. These results suggest that histone deaceylase Sir2α may play a critical role for proliferation and/or differentiation of neural stem cells.

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OJ3-02 HDAC inhibitor-mediated neuronal differentiation of adult neural progenitors

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Neural stem cells are defined as cells with the ability to self-renew and give rise to neurons and glial cells. Here, we show that an inhibitor of histone deacetylase, valproic acid (VPA), also known as an anti-epileptic, induces neuronal differentiation of adult hippocampal neural progenitors (AHPs). In addition, VPA inhibited glial cell differentiation of AHPs, even in conditions that favored lineage-specific differentiation. Among the VPA-upregulated neuron-specific genes, a neurogenic bHLH transcription factor, NeuroD was identified. Overexpression of NeuroD in AHPs resulted in the induction and suppression of neuronal and glial differentiation, respectively. Taken together, our results suggest that VPA promotes neuronal fate and inhibits glial fate simultaneously through the induction of neurogenic transcription factors including NeuroD.

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OJ3-03 Role of AINT in the developing brain

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Neuroepithelial cells remain undifferentiated when cultured in the presence of bFGF. The molecular mechanism is, however, still poorly understand. Thus, we sought to compare gene expression profiles in neuroepithelial cells cultured with or without bFGF using cDNA microarray. Among bFGF-upregulated genes, a transforming acidic coiled-coil family, AINT was identified. AINT was highly expressed in the subventicular zone of the fetal mouse brain where neural stem cells normally reside. Forced expression of AINT in telencephalic germinal layer cells by in utero electroporation at embryonic day 14.5, inhibited migration of the cells and retained them close to the germinal layer. Furthermore, neuroepithelial cells infected with retrovirus engineered to express AINT exhibited impared neuronal differentiation. These results suggest that AINT has a role in inhibiting migration and differentiation of neuroepthelial cells.

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OJ3-04 Roles of Musashi Family in mammalian nervous development

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Musashi(Msi) is an evolutionarily conserved RNA-binding protein. In mammalian CNS, we identified two members of Msi family, Msi1 and Msi2, which are co-expressed in neural precursor cells including CNS stem cells. We supposed Msi regulate CNS stem cell proliferation or maintenance of immaturity through post-transcriptional regulation. To reveal the roles of Msi *in vivo*, we generated Msi1 and Msi2 deficient mice.
Msi2 deficient mice survive to adult, however, show poor weight gain, low spontaneous movement and temperature insensitivity. Msi1 and Msi2 double deficient 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 the expression of several downstream targets, like m-numb and pleiotrophin, are regulated by Msi. These results suggest Msi bind to target mRNAs, control the expression post-transcriptionally and play important roles cooperatively in mammalian CNS development.

OJ3-05 Developmental stage dependent regulation of epigenetic modification of a premature astrocytic gene promoter

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Astrocytes are typically generated from neuroepithelial cells after neurons during brain development. However, the mechanism of this sequential generation is not fully understood. In this study, we show that a particular cytosine residue in the promoter of the gene for premature astrocyte marker, S100β become demethylated, correlating with the timing when the S100β expression commence at embryonic day (E) 14. In addition, a methyl DNA binding protein, MeCP2-binding to the S100β gene promoter in neuroepithelial cells was found to be reduced. Furthermore, astrocyte-inducing cytokine, BMP2 increased histone acetylation around the CpG site in neuroepithelial cells at E14 but not E11 when S100β expressing astrocytes are absent. These results suggest that the epigenetic modification plays a critical role as a cell intrinsic factor in concert with external cues, like cytokines, in the cell fate determination.

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OJ3-06 Dlx1 and Dlx2 homeobox genes determine competent state of retinal progenitor cells

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In this study, we examined the roles of Dlx1 and 2 homeobox genes in developing retina. Misexpression of Dlx1 or Dlx2 significantly increased the numbers of amacrine cells (AC) and retinal ganglion cells (RGC) and concurrently caused a reduction of rod photoreceptors. Math5 bHLH gene initiates generation of RGC, and activate Brn3b expression. The activation results in promoting terminal differentiation of RGC. In Math5 mutant mice the expressions of Dlx1 and 2 were significantly down-regulated, and the expressions of Dlx1 and 2 were significantly up-regulated in Brn-3b mutant mice. Therefore, we propose a new hypothesis that Math5 determines competence state of post-mitotic precursor cells to differentiate all retinal neurons as we reported, and then Dlx1 and 2 can determine more detail competent state to differentiate RGC or AC. Therefore Brn-3b expression must promote the terminal differentiation of RGC from the competent state precursors as a result of the suppression of the Dlx gene expressions.

OJ3-07 Neurogenesis beneath the temporal neocortex in the adult rat brain

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Persistent neuron production in the adult rodents brain has been recognized in two restricted regions; the subventricular zone (SVZ) lining the anterior lateral ventricle wall and the subgranular zone (SGZ) in the dentate gyrus of the hippocampus. This study provides evidence for unrecognized neurogenesis undergoing beneath the neocortex of the temporal lobe in the adult rat brain. In this area, dividing Pax6- and Olig2-positive neural progenitor cells continually give rise to doublecortin-positive immature neurons, however, the majority are lost within a week after their birth. This unique profile of neuron production in the temporal lobe would be a clue to understand functional significance of neurogenesis throughout the adult life.

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OJ3-08 Ventralizing effect of bFGF on cortical neuroepithelial cells

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Basic fibroblast growth factor (bFGF) is commonly used to enrich and maintain neural stem cells in vitro. Here we found that bFGF induced cortical neuroepithelial precursors to express Olig2 transcription factor, which is normally expressed in the ventral part of the embryonic forebrain. Cortical precursors freshly prepared from E14 mouse were initially Olig2(-). However, 70% of total and 85% of Nestin(+) cells became Olig2(+) after culture with 10 ng/ml bFGF for 4 days in vitro. There were no significant difference in BrdU incorporation (proliferation) or PI staining (cell death) between Olig2(+) and (-) cells in the culture containing bFGF. bFGF-treated cortical precursors were adapted for generating numerous O4(+) Oligodendrocytes and GABA neurons, which are believed to be generated only from the ventral forebrain in vivo. Taken together, these results suggested that bFGF changed the fate of cortical precursors to acquire a ventral phenotype.

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OJ3-09 Screening of the ventricular zone-specific secretory or cell surface molecules in the developing cerebral cortex

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Embryonic cerebral cortical neurons terminally differentiate beneath the MZ after migration from the VZ through the IMZ. Recently, we found that neurons born at E14 were segregated from neurons born at E13 or E15.5 before they reached beneath the MZ. These results and the previous transplantation studies using ferret suggest that the cell fate of the final positioning is determined by secretory or cell surface molecules that are expressed in the VZ. In this study, to reveal the molecular mechanisms of the cell fate determination in the VZ, we searched for the molecules that were expressed in the VZ but not in the CP by using DNA chip analysis. Combining with the screening using physicochemical algorithms that determine the secretory and cell surface molecules and in situ hybridization, we identified 20 clones that were expressed in the VZ or IMZ specifically. This method is useful for screening the secretory or cell surface molecules that are expressed in the specific region of interest.

OJ3-10 Novel transcription factor ZFH-5 expressed in the differentiating neurons is negatively regulated by the natural antisense RNA

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ZFH-5 is a novel transcription factor isolated from the mouse brain. *zfh-5* mRNA was expressed in the developing brain, especially in differentiating neurons. In addition to mRNA, an antisense RNA was also expressed. Neurons at the early phase of differentiation including migrating cells expressed the antisense RNA, while cells at the later phase expressed mRNA. In a gene-targeted mouse line, in which a putative promoter region of the antisense RNA was deleted, the antisense RNA was not detected and ectopic expression of *zfh-5* mRNA was observed. In another line, *EGFP* mRNA, which was expressed under the control of the *zfh-5* (sense) promoter but had no complementary RNA expressed, was expressed in wider areas than *zfh-5*. From these results, we propose that ZFH-5 is a factor functioning in a particular phase of neural differentiation, and at earlier stages, production and/or accumulation of mRNA is repressed by its own antisense RNA.

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OJ3-11 Engrailed-1 expression marks a primitive class of inhibitory spinal interneuron

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We used genetic, morphological, and physiological studies to test Engrailed-1 (En1) marks functional subtypes of spinal interneurons in larval zebrafish. We found that En1 expression uniquely marks a class of ipsilaterally projecting interneurons that express GLYT2, a marker of inhibitory neurons. Patch recordings show that the En1 neurons are rhythmically active during swimming and that En1 neurons produce monosynaptic, strychnine sensitive inhibition of dorsal sensory interneurons and also inhibit more ventral neurons, including motoneurons and descending interneurons. We conclude that En-1 expression marks a class of inhibitory interneuron that seems to provide all of the ipsilateral glycinergic inhibition in zebrafish. Individual En-1 cells are multifunctional, playing roles in both sensory gating and motor pattern generation. This primitive cell type may have given rise to several, more specialized glycinergic inhibitory interneurons in mammals.

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OJ3-12 ER81 is expressed in a subpopulation of layer 5 projection neurons in rodent and primate cerebral cortices

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Lamina-specific gene expression is a key issue to understand cortical cell differentiation. By screening cDNA libraries for the genes enriched in layer 5, we found that *ER81*, an Ets transcriptional factor, was expressed in a subset of layer 5 neurons throughout the cerebral cortex in mice and rats from embryonic days to young adult stages. *ER81* was also detected in layer 5 neurons in infant macaques, but undetectable in adults. Retrograde labeling combined with immunohistochemistry showed that nearly all neurons projecting to the spinal cord and the superior colliculus expressed ER81, while only one-third of the neurons projecting to the contralateral cortical areas expressed the molecule. These findings suggest that ER81 is involved in cellular differentiation of a subset of layer 5 neurons and that this mechanism is preserved among mammalian species.

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一般演題（ポスター発表）

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P1-001 The expression of INSL7/relaxin3 in the rat brain

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INSL7/relaxin3 (INSL7) was recently identified as a new member of insulin/relaxin family. Although it was reported to be dominantly expressed in the brain, its detailed distribution and its function in the central nervous system were still unknown. In the present study, we investigated the expression of INSL7in the rat brain. Immunocytochemistry and in situ hybridization revealed that the INSL7 expressing cells were mainly localized in neurons of the median dorsal tegmental pons, called nucleus incertus (NI). Other INSL7 expressing neurons were sparsely observed in the pontin raphe nucleus, in the periaqueductal gray and in the dorsal area of substantia nigra. The fibers of INSL7 neurons were densely distributed in the septum, diagonal band, hippocampus, lateral hypothalamus. The INSL7 containing neurons also coexpressed corticotropin releasing factor type 1 receptor in the NI, suggesting that these INSL neurons are involved in the stress response.

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P1-002 Distribution of cholinergic neurons in the common marmoset brain

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We determined the distribution pattern of cholinergic neurons in the common marmoset by an immunohistochemical method using anti-cholin acetyltransferase (ChAT) antibody. ChAT immunoreactive neurons (Ir) were observed in the following regions of the marmoset brain. Small to large oval, spindle and pyramidal neurons were distributed in the olfactory tubercle, caudate-putamen, globus pallidus, diagonal band, medial septal area, substantia innominata area, red nucleus, parabrachial nucleus, dorsal raphe nucleus, superior olive, locus coeruleus, nucleus ambiguus, nucleus of the solitary tract, lateral reticular nucleus, and cranial nerve motor nuclei. These findings are similar to those obtained for rats. The major features of these findings are that ChAT Ir neurons are mainly distributed in the locus coeruleus neucleus and that the cranial nerve motor nuclei are large.

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P1-003 Re-evaluation of cholinergic innervation of rat ocular tissues using choline acetyltransferase of a peripheral type as a marker

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We have recently discovered pChAT, a splice variant of choline acetyltransferase preferentially expressed in peripheral neurons. Using pChAT as an indicator of cholinergic structures, we re-evaluated cholinergic innervation of rat ocular tissues, by immunohistochemistry, Western blot, RT-PCR and enzyme assay. By immunohistochemistry combined with denervation or tract tracing, we found three types of pChAT-positive neuronal elements there. They were, 1) parasympathetic postganglionic fibers in the iris and cornea originating from the ciliary ganglion, 2) trigeminal sensory fibers in the iris and cornea, and 3) some retinal ganglion cells. The localization and origins of pChAT were verified by Western blotting, RT-PCR and ChAT assay, suggesting a role of pChAT not only in the parasympathetic regulation of eye tissues, but also in sensory and visual neurotransmission.

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P1-004 Differences in intracellular translocation between two types of choline acetyltransferase of the rat

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We investigated differences in the subcellular localization between choline acetyltransferase (ChAT) of a peripheral type (pChAT) and ChAT of the common type (cChAT), by using green fluorescent protein (GFP) tagged pChAT and cChAT expressed in HEK293 cells. pChAT was exclusively distributed in the cytoplasmic compartment, whereas cChAT was located in both the nucleus and cytoplasm. Inhibition of nuclear export pathway with leptomycin B (LMB) resulted in accumulation of pChAT-GFP in the nucleus, indicating that pChAT can pass through the nuclear envelopes. Protein kinase C inhibitor induced significant accumulation of pChAT-GFP, but not cChAT-GFP, into the nuclei, suggesting that phosphorylation may affect the subcellular localization of pChAT. The results suggest that pChAT and cChAT play different roles in neurons.

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P1-005 Effects of adenosine A3 receptor on N-type Ca^{2+} channels in cultured rat striatal neurons

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Adenosine has been known to be a neuromodulator in nervous systems and adenosine receptors have been classified into five types (A1, A2a, A2b, A3 and A4). In this study, the effects of N-type Ca channel currents (Nifedipine-insensitive channel currents) induced by adenosine A3 receptor agonist (IB-MECA) were examined in cultured rat striatal neurons, using a whole-cell clamp recording. Ca currents were reduced after application of IB-MECA (0.1-1000μM) gradually, and reduction ratios of currents were increased according to increase the concentration of IB-MECA. In I-V relation from -30 to +50mV of test potentials (holding potential was -40mV), reduction ratios were not different at any test potentials, indicating that the voltage dependency of reduction of IB-MECA was not observed. In addition, no reduction of currents was observed in records by GDPβS contained patch electrodes. These results indicate that adenosine A3 receptor agonist reduces N-type Ca channel currents through G-protein in rat neurons.

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P1-006 Regulation of extra-cellular monoamine by cognate monoamine transporter

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It is well documented with recombinant gene experiments that monoamines have relatively high affinity to cognate as well as relevant monoamine transporters. Since there is little evidence of monoamine re-uptake by cognate transporter, we investigated monoamine re-uptake in monoamine transporter knockout (KO) mice using *in vivo* microdialysis method. Systemic administration of cocaine enhanced extra-cellular serotonin (5-HT*ex*) in caudate putamen (CPu) and nucleus accumbens of 5-HT transporter (SERT)-KO mice. Extra-cellular dopamine (DA*ex*) was also enhanced in CPu and prefrontal cortex (PFc) of DA transporter (DAT)-KO mice as well as in PFc of DAT/SERT double-KO mice. Local administration of cocaine into CPu still enhanced the 5-HT*ex* in SERT-KO mice. Since the relevant monoamine transporter is absent in the KO mice, these results may suggest the regulation of extra-cellular monoamine by cognate monoamine transporter.

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P1-007 Deletion of N-terminus of human tyrosine hydroxylase type 1 dramatically enhances the stability in mammalian cells

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N-terminal region of tyrosine hydroxylase (TH) is known to play significant role in the determination of DA biosynthesis. To clarify how deeply the stability of TH in mammalian cells is involved in a more potent determinant of the efficient production of DA, AtT-20 mouse neuroendocrine cells being capable of fully converting L-DOPA into DA were transfected with wild-type and the mutants of human TH type 1 (hTH1) missing the first 52 and 157 amino acid residues (del-52 and del-157). The level of DA accumulation in AtT-20 cells expressing hTH1 gene was in the order: del-52 = del-157 > wild-type. We report that the high stability of the N-terminus-deleted hTH1 should be a more potent determinant of the efficient production of DA in mammalian cells and the N-terminus region upto Ala^{52} of hTH1 should determine the intracellular stability of hTH1 molecule.

P1-008 Serotonergic regulation of the orexin/hypocretin neurons through the 5-HT1A receptor

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We examined the effects of 5-HT on orexin neurons, using slices prepared from orexin/EGFP transgenic mice in which EGFP is expressed exclusively in orexin neurons. Patch-clamp recording from EGFP-expressing cells showed that 5-HT hyperpolarized all orexin neurons. The response was inhibited by the 5-HT1A receptor antagonist, WAY100635. A 5-HT1A receptor agonist, 8-OH-DPAT, also evoked hyperpolarization on orexin neurons with potency comparable to 5-HT. Intracerebroventricular injection of WAY100635, increased locomotor activity during the latter half of dark phase in wildtype mice, but not in orexin/ataxin-3 mice suggesting that activation of orexin neurons is necessary for the WAY100635-induced increase in locomotor activity. These results indicate that 5-HT hyperpolarizes orexin neurons through the 5-HT1A receptor.

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P1-009 Endogenously released 5-HT depress the spinal monosynaptic reflex via 5-HT_{1D} receptors

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In the spinal cord, various 5-HT receptor subtypes are involved in the modulation of motor output. Although 5-HT_2 receptors facilitate the spinal monosynaptic transmission (MSR), it is unknown which receptor subtypes mediate the inhibitory effect of 5-HT. Previously we have shown that 5-HT_{1B} receptors mediated the depression of MSR induced by exogenously applied 5-HT converted from the 5-HT precursor 5-HTP in spinalized rats (Eur. J. Pharmacol. 482, 2003). In the present study, we determined the effects of 5-HT, which was released by DL-p-chloroamphetamine (PCA), on MSR of spinalized rats. MSR was recorded from the L5 ventral root following stimulation of the L5 dorsal root. PCA depressed the MSR and it was abolished in 5-HT-depleted rats. The depression was antagonized by GR127935, a 5-$HT_{1B/1D}$ receptor antagonist and BRL15572, a 5-HT_{1D} receptor antagonist, but not by isamoltane, a 5-HT_{1B} receptor antagonist. These results suggest that endogenous 5-HT depresses the MSR via 5-HT_{1D} receptors.

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P1-010 Dopamine is present in raphe-striatal serotonergic neurons of rats with nigro-striatal dopaminergic denervation

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The aim of this study is to examine whether raphe-striatal serotonergic neurons of the rat brain are induced to contain dopamine by nigro-striatal dopaminergic denervation. In two groups of rats with nigro-striatal dopaminergic denervation who received intraperitoneally L-DOPA with a peripheral decarboxylase inhibitor or not, we showed with a double-labeling immunofluorescence method that dopamine was localized in serotonergic terminals of the lesioned striatum and cell bodies of the midbrain raphe nuclei. In rats without L-DOPA injection, no dopamine was detected in serotonergic terminals of the intact striatum. The findings suggest that a part of exogenously injected and endogenous L-DOPA may be carried into raphe-striatal serotonergic neurons and be converted to dopamine in rats with nigro-striatal dopaminergic denervation.

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P1-011 An effect of milnacipran on norepinephrine content in spontaneously hypertensive rat: AD/HD model rat.

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Attention deficit hyperactivity disorder (AD/HD) is characterized by inattentiveness, hyperactivity and impassivity in a childhood. Many studies suggest that AD/HD appears to involve dysfunction of the CNS dopaminergic and noradrenergic system. The nucleus locus coeruleus (LC), the most major source of noradrenergic tone in a CNS is explored to examine the effect of milnacipran (MIL), serotonin and norepinephrine transporter inhibitor, a potential candidate for the therapeutic agent of AD/HD. We previously reported that MIL produced a hyperpolarizing response associated with decrease in the membrane resistance by activation of the inward rectifier K^+ channels via α_2-adrenoceptor of LC neurons in the rat. In the present study, an effect of MIL on norepinephrine content in LC was explored using HPLC. Protein level of a norepinephrine transporter and a dopamine transporter in the CNS was also evaluated in Wistar and SHR rats.

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P1-012 Effect of Methylphenidate on Locus Coeruleus Neurons in Spontaneously Hypertensive Rat: an Animal Model of AD/HD.

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The spontaneously hypertensive rat (SHR) is often used as a model for childhood attention-deficit/hyperactivity disorder (AD/HD). The nucleus locus coeruleus (LC), the most major source of noradrenergic tone in a CNS is explored to examine the effect of methylphenidate (MPH), the most commonly used agent for a treatment of AD/HD. We already reported MPH produced a hyperpolarizing response associated with decrease in the membrane resistance by activation of the inward rectifier K^+ channels via α_2-adrenoceptor of LC neurons on the adult rat. In the present study, the effect of MPH on neural activity of the LC of juvenile SHR was examined by using a whole-cell patch clamp technique. MPH caused an outward current associated with an increase in inward rectifier K^+ channels. MPH significantly potentiated the amplitude and time course of slow IPSCs. We compared neuronal activity and effects of MPH of juvenile SHR and normal rats.

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P1-013 Tissue plasminogen activator-plasmin system modulates depolarization-evoked dopamine release

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Recently, we found that morphine-induced dopamine (DA) release is attenuated in the nucleus accumbens (NAcc) of tissue plasminogen activator (tPA) knockout mice. In this study, we investigated the role of tPA-plasmin system on depolarization-evoked DA release in the NAcc of mice, by using in vivo microdialysis. Microinjecton of tPA and plasmin into the NAcc enhanced high potassium-evoked DA release. In contrast, microinjection of plasminogen activator inhibitor-1 into the NAcc reduced high potassium-evoked DA release. Attenuated high potassium-evoked DA release in tPA knockout mice was reversed by microinjection of either exogenous tPA or plasmin into the NAcc. Our findings demonstrate that tPA-plasmin system is involved in the regulation of DA release in the NAcc.

P1-014 Behavioral effects in mice of the fragrant compounds which activate ionotropic GABA receptors

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We have found that many fragrant compounds potentiate the response of the $GABA_A$ receptors expressed in *Xenopus* oocytes by injecting cRNAs of the bovine receptors. These compounds may be absorbed into the blood, carried to the brain and potentiate the GABAA receptor response, which may result in the change of human mood and consciousness. To confirm this hypothesis, we examined the effect of these compounds on mouse behaviors, using an elevated plus maze and a tilting-type ambulometer. Terpenen-4-ol and 1-octen-3-ol increased the staying time in the open arm of an elevated plus maze as musimol and diazepam did, suggesting their anti-anxiety effect. However they unexpectedly enhanced the ambulation-promoting effect of caffeine measured by a tilting-type ambulometer, when they were administered together.

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P1-015 Reduced hypothermic and hypnotic responses to ethanol in PACAP-deficient mice

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Using pituitary adenylate cyclase-activating polypeptide (PACAP)-deficient mice, we investigated whether PACAP is involved in the intoxicating effects of ethanol. Sleep time (duration of the loss of righting reflex) was markedly shortened in PACAP-deficient mice compared with wild-type, although latency to the loss of righting reflex was not different between the two groups. Ethanol- induced hypothermia in wild-type control mice was significantly reduced in PACAP-deficient mice. Blood ethanol levels were not different between the two groups, excluding the possibility of increased ethanol metabolism. Thus, PACAP deficiency in mice caused a reduced sensitivity to ethanol. These results suggest that PACAP plays significant roles in modifying the intoxicating effects of ethanol.

P1-016 An intermediate type of feedback regulation of cortical excitability that is not attributable to synaptic inhibition or refractoriness

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For treatment of epilepsy, an efficiently run feedback regulation of neural excitability would be far more specific and beneficial than general inhibition with drugs currently used. There are indeed a number of feedback regulations implemented in CNS neurons or circuits. At the membrane level, the refractory period precludes the firing interval under one msec or two, but allows any interval thereafter. At the local circuit level, GABAergic inhibition may be concerned only with the firing interval over several msec, because of the transmission delay. Hence comes a time-window, in which these two mechanisms could not work (2-3 ms ~ 7-8 ms). By unit recording from anesthetized rat hippocampi, we revealed that no such window exists, suggesting an intermediate type of feedback regulation that must not be synaptic or membrane-delimited. We further showed that this putative feedback depends on muscarinic acetylcholine receptors. Pharmacological intensification of such feedback may be therapeutically useful.

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P1-017 GABA and/or glycine immunoreactive synapses in the trigeminal motor nucleus of rats during postnatal development

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Mammalian jaw movement changes drastically in the transition phase from suckling to chewing behaviors. GABA and glycine (Gly) are the principal inhibitory neurotransmitter. We found the change in GABAnergic terminal boutons in trigeminal motor nucleus (Vm) during masticatory transition phase. In this study we analyzed distribution of GABA-immunoreactive (GABA-IR) and Gly-IR synapses in the Vm of rats using immunohistochemical study during postnatal development From one to 3 weeks of age, a lot of GABA-IR and Gly-IR synapses localized around the soma and the dendrites of motoneurons in the Vm. Gold particles of Gly-IR and GABA-IR in the synapses shown high density. At 4 and 5 weeks of age, a small number of GABA-IR and Gly-IR synapses were observed in the Vm. These results suggest that the inhibitory inputs are regulated with GABAergic and/or glycinergic projection during the development of the masticatory apparatus.

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P1-018 Tonic $GABA_A$ receptor-mediated inhibition in rat neocortex

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We examined the pharmacological properties of tonic and phasic currents mediated by $GABA_A$ receptors in layer V pyramidal neurons of the rat neocortex. Pharmacological isolated GABAergic mIPSCs were recorded whole cell recording at voltage-clamp mode (*Vh=-60 mV*). Bath application of $GABA_A$ receptor antagonist BMI blocked mIPSCs and outwardly shifted baseline holding current (Ihold). SR95531, a $GABA_A$ receptor antagonist similarly abolished mIPSCs but had no significant effect on Ihold. Bath application of midazolam causes a concentration-dependent increase in the amplitude of the tonic inhibition. These tonic currents were also abolished by BMI but not by SR95531, indicating benzodiazepine augmentation of the tonic $GABA_A$ receptor mediate inhibition. Thus, this tonic inhibition is functionally and pharmacologically distinct from phasic inhibition.

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P1-019 GABA$_A$ receptors shape columnar activity in the monkey prefrontal cortex

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With optical recording in vitro, we have demonstrated that the dorsolateral prefrontal cortex (dlPFC) of monkey has functional columnar organization. In some sensory areas, GABA inhibition has been implicated in regulation of the columnar architecture. Therefore, it is likely that functional column in the dlPFC may be regulated by GABAergic inhibition. To examine this hypothesis, we investigated the effect of GABAergic compounds on columnar activity, using an optical recording with voltage-sensitive dye. Slices of the monkey dlPFC were stimulated electrically, and evoked columnar activity was recorded optically. Bath applications of GABA$_A$ agonist (muscimol:10μM) and GABA$_B$ agonist (SKF97541:10μM) inhibited the columnar activity, whereas the antagonist of GABA$_A$ (BMI:10μM), but not GABA$_B$, expanded neuronal activity beyond the boundary of columns. These findings suggest that GABA$_A$ inhibition plays a critical role in maintaining/shaping functional columns, whereas GABA$_B$ inhibition would modulate them.

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P1-020 Quantitative mRNA analysis of GABABR1 and R2 subunits in various regions of the rat brain by real-time RT-PCR

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Objective The object of this study was to evaluate expression level of GABABR1 and R2 subunit mRNAs in various regions of rat brain.
Methods Various regions of rat brains were dissected. GABABR1 and R2 subunit mRNA expression levels were analyzed in each region using LightCycler quantitative PCR assay.
Results GABABR1a, c, and d subunits mRNA expression level in cerebral cortex were significantly higher than those in other brain regions. GABABR1b subunit mRNA expression was the highest in ventrobasal nucleus of thalamus.
Conclusion Differences of each GABAB receptor subunit mRNA expression level may reflect distinct functions of respective brain regions.

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P1-021 Regional expression of GABA, glycine and related neuronal markers in rat auditory brainstem

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In rat auditory brainstem stations, both glycinergic neurons and GABAergic neurons play key roles in development and propagation of cochlea-derived information. GAD65 and GAD67, two isoforms of GABA-synthesizing enzyme, are present in most classes of GABA neurons. The two enzymes, however, are differentially regulated in some auditory nuclei in the rat brainstem. By using in situ hybridization and immunohistochemistry, we examined whether the regional distribution of chemically defined neurons is uniform. In the periolivary neuronal groups, GAD65 was expressed most heavily. In the lateral superior olive, GAD67 neurons were dominant. In lateral lemniscal nuclei, glycine transporter type 2, calbindin and parvalbumin immunoreactivities are accumulated in the intermediate subnucleus. In the other lemniscal divisions, GAD65 and GAD67 neurons were major components. The results showed that neurons labeled with these markers are distributed in a non-uniform fashion.

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P1-022 GABAergic signaling in the developing cerebellum

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GABAergic signaling in the developing cerebellumChitoshi Takayama, Yoshiro Inoue Department of Molecular Neuroanatomy, Hokkaido University School of Medicine In the adult brain, γ-amino butyric acid (GABA) is an inhibitory neurotransmitter, which regulates glutamatergic activity. Recent studies have revealed that GABA serves as an excitatory transmitter in the immature brain, and is involved in brain morphogenesis. To reveal the GABAergic signaling in the developing CNS, we investigated the developmental change in distribution of GABA, vesicular GABA transporter, and GABA$_A$ receptor α subunits in the cerebellum. In this study, we found that GABA could be extrasynaptically released by diacrine and exocytosis from all GABAergic neurons during cerebellar development. The GABA could activate GABA$_A$ receptors containing α2 and α3 subunits and be involved in neuronal maturation. After finishing maturation, GABA is synaptically released, activates GABA$_A$ receptors containing α1 and α6 subunits and mediate inhibitory transmission.

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P1-023 The observation of spatiotemporal GABA release patterns in cerebella culture

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GABA and glutamate are known to play important roles as modulators of survival and development of cerebella neurons. Recently we reported the spatial nature of glutamate release from organotypic cerebella cultures with an enzyme-linked assay system and CCD imaging technology. In developing cerebella cortex, glutamate was released transiently up to 600 μM. Whereas it was even high concentration, granule cell development should require transient glutamate release. To examine the mechanisms of this glutamate transient, we evolve the measurement system of spatiotemporal pattern of GABA release using of GABase and NADPH. In developing cerebella culture, transient and massive GABA release is observed nearby Purkinje cell layer just after glutamate transient. We suggest that GABA release would modify the timing of cerebella development by controlling of glutamate release.

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P1-024 Distinct re-filling profiles of inhibitory neurotransmitters into presynaptic terminals projecting to spinal neurons

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mIPSCs were recorded from synaptic bouton preparations of sacral dorsal commissural nucleus neurons by whole-cell recording. Three types of mIPSCs were identified: glycinergic, GABAergic and mixed mIPSCs. Focal stimulation of a synaptic bouton evoked three types of eIPSCs, indicating that three different quantal releases occur from a synaptic bouton. Prolonged treatment with bafilomycin A1 suppressed frequency and amplitude of the mIPSCs. Washing out of bafilomycin A1, complete recovery in the amplitude of glycinergic mIPSCs was observed, while GABAergic and mixed mIPSCs was incomplete. This indicates that the re-filling of glycine and GABA are different. This could be explained by the decrease in the cytosolic concentration of GABA, or by the presence of subtypes of vesicular inhibitory amino acid transporter in the vesicle membrane.

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P1-025 The role of glutamate signaling in development of *Dictyostelium discoideum*.

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The mold *Dictyostelium discoideum* is a simple model for evaluation of cellular development and differentiation of complex organisms. In this study, we starved *Dictyostelium* on agar plates together with different agonists for ionotropic (iGluR) and metabotropic (mGluR) receptors for the mammalian central neurotransmitter glutamate (Glu), followed by observation of morphological changes every 4 h on a microscope. The sorus was larger in fruiting bodies of *Dictyostelium* starved in the presence of iGluR agonists than that found in their absence, while starvation with mGluR agonists led to smaller sorus than control. Moreover, Northern blot analysis revealed marked facilitation of the expression of the prespore marker psA and the spore marker spiA in a dose dependent manner when starved in the presence of Glu. These results suggest that Glu signaling would be involved in mechanisms associated with regulation of the cellular differentiation and/or development after starvation in *Dictyostelium*.

P1-026 Role of glutamate on radiation-induced functional damage of the central nervous system in rats

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X-ray or g-ray irradiation impaired some functions of the central nervous system, which is one of radiation-resistant tissues in the body. However, the effects of radiation on the neuronal tissue have not been studied extensively. It is known that the glutamatergic system is involved in the development of neuronal damage of the brain. In this study, we investigated the effects of X-ray and heavy-ion irradiation on glutamate release measured by in vivo microdialysis in the hypothalamus, hippocampus and medulla oblongata of rat. Total body irradiation (4MV X-ray: 4Gy, carbon 290 MeV/u: 10 Gy) significantly increased glutamate release in the hypothalamus after 2 h of total body irradiation, but not in the hippocampus and medulla oblongata. These findings suggest that the neurochemical modification of hypothalamic glutamatergic system may contribute the development of radiation-induced functional damage of the central nervous system.

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P1-027 Effects of clozapine on extracellular glutamate in rat medial prefrontal cortex

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The atypical antispychotic clozapine has been thought to alleviate hypofrontality in schizophrenics. In line with this assumption, we previously reported the preferential ability of clozapine to attenuate phencyclidine-induced expression of the heat shock protein gene in rat medial prefrontal cortex (mPFC). The present study investigated the effects of clozapine on extracellular glutamate (Glu) in the mPFC, using in vivo microdialysis of awake, freely moving rats. Clozapine had no significant effect on basal Glu levels but attenuated high potassium-evoked Glu levels in the mPFC. The group II metabotropic Glu receptor antagonist increased basal Glu levels, and this effect was prevented by clozapine. Moreover, clozapine also attenuated an increase in extracellular Glu induced by the blockade of the Glu transporter GLT-1. These results suggest that, when extracelluar Glu elevates upon presynaptic or glial evoked conditions, clozapine may reduce it to exert a neuroprotective effect on mPFC neurons.

P1-028 Enhanced Excitability of Hippocampal Mossy Fibers under Dietary Zinc Deficiency

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On the basis of the evidence of the increased glutamate concentration in hippocampal extracellular fluid in zinc-deficient rars, excitability of mossy fibers was examined using hippocampal slices. The dynamics of zinc and calcium was monitored using their indicators, membrane-impermeable ZnAF-2 and membrane-permeable fura-2 AM, respectively. When dentate granule cells were stimulated with 100 mM KCl for 1 s, the increased percentages of extracellular zinc in mossy fibers were higher in zinc-deficient mice. The basal calcium concentration in mossy fibers detected with fura-2 AM was higher in zinc-deficient mice. The increased percentage of intracellular calcium in mossy fibers by stimulation with high K^+ was also higher and more sustained in zinc-deficient mice. These results suggest that glutamate release from mossy fibers is enhanced by zinc deficiency. Alteration of hippocampal calcium homeostasis seems to be involved in the enhanced excitability of dentate granular cells.

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P1-029 Localization of Estrogen Receptor α in the Rat Hippocampus

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Estrogens have profound effects on the structure and function of the hippocampus. However, the localization of estrogen receptor α (ERα) remains contradictory so far. Using newly prepared antibody, RC-19, we investigated ERα in the rat hippocampus by Western blot and immunohistochemistry. We confirmed by Western blot that column-purified antibody RC-19 recognizes the full length (67kDa) ERα in the hippocampus, while other widely used antisera including MC-20 failed to show the reaction. Next we performed immunohistochemistry with fresh frozen section (20μm). We found the ERα immunoreactivity in pyramidal neurons of CA3 and CA1, and granule neurons in the dentate gyrus. These results imply that estrogen effects is mediated through ERα in glutamatergic neurons.

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P1-030 Estrogen and nicotine interactions in terms of transcription factors in the brain

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Estrogen and nicotine exert influences on neurons in the same direction in several aspects, such as memory function and neuroprotective effects on cortical neurons. Both of them increase intracellular Ca ion concentration in cultured rat hippocampal neurons and both induce IGF-1 mRNA expression in the hippocampus. Nevertheless, nicotine inhibits estrogen's effects in some of these experimental systems. Previously, we showed that nicotine inhibited ERE binding immediately and markedly in the rat hippocampus. In relation with these results, male rats were injected with 17β-estradiol and/or nicotine prior to examine ERE, AP1- and CREB-DNA bindings in hippocampus and cerebral cortex by gel shift assay. Single injection of either estrogen or nicotine caused increase of both AP1- and CREB-DNA bindings, in less degree by nicotine than estrogen. Simultaneous injection of them caused more increase of these bindings, while nicotine inhibited ERE binding markedly.

P1-031 Kainic acid and nicotine have reverse effects on estrogen-induced ER and IGF-1 mRNA expressions in the hippocampus.

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In our previous studies, we confirmed that estrogen induced IGF-1 mRNA expression in the rat hippocampus which was intensified by kainic acid injuries, and concluded that this was one of the basic mechanisms of nerve protection by estrogen. Further, we noticed that nicotine also induced IGF-I mRNA expression in the hippocampus, while nicotine inhibited estrogen-induced IGF-1 mRNA expression when simultaneously injected.
In this study, we examined interactions among estrogen, kainic acid and nicotine in terms of estrogen receptor (ER) and IGF-1 mRNAs by means of real time PCR assay. When estrogen and kainic acid were simultaneously injected into rats, both ER and IGF-1 mRNAs were much more induced in the hippocampus when compared with the cases of single kainic acid injection. On the other hand, nicotine inhibited estrogen-induced these mRNA expressions.

P1-032 Regulation of AP1 and Tex261 gene expression in mouse hippocampus after intraperitoneal injection of NMDA.

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We have demonstrated transient induction of the transcription factor AP1 complex containing c-Fos and c-Jun in murine hippocampus following an injection of NMDA. In this study, we have searched for particular genes expressed in response to the administration of NMDA in murine hippocampus on differential display screening. An intraperitoneal injection of NMDA led to upregulation of a gene identified as Tex261 with an AP1 site at the promoter region 6 h after administration. Northern blot analysis revealed that NMDA induced marked and sustained expression of Tex261 gene for a period of 6 to 24 h after administration with concomitant transient expression of c-fos gene within 30 min. On in situ hybridization, Tex261 mRNA was highly localized to the CA1 to CA4 pyramidal layers and the dentate granular layers in hippocampus. These results suggest that Tex261 may be expressed downstream to AP1 complex induced in response to activation of NMDA receptors in murine hippocampus.

P1-033 Properties of $GABA_A$ receptor-mediated signaling in neocortical neurons with identified birth period by *in utero* electroporation of EGFP.

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GABA can exert trophic influences during early CNS development, and many of the developmental actions of GABA are mediated through activation of depolarizing $GABA_A$ receptors. Developmental changes in properties of GABAergic response of mice neocortical neurons were studied. To identify the newborn cells existing in ventricular zone (VZ) at E14, EGFP was transfected into VZ cells by means of *in utero* electroporation. $GABA_A$-receptor mediated current were recorded from GFP-expressing migrating and settled cortical plate neurons at 1, 3, 6, 9, and 12 days after the electroporation. Dose-response properties of GABA evoked currents and bicuculline induced tonic currents were compared in each developmental stage. After recordings, neurons were harvested for *single-cell RT-PCR to* determine subunit composition and Cl^- homeostasis underlying these GABAergic responses.

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P1-034 Regulation of exocytotic events by protein kinase C isoform in single adrenal chromaffin cells

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To clarify the implication of each protein kinase C (PKC) isoform in the regulation of exocytosis, we examined the effects of overexpression of PKC α and β on the kinetics of exocytosis of single vesicles using transfected cells. Expression of PKC isoformes was confirmed by co-expression of EGFP. Similar expression levels of both PKC isoforms were detected. In the PKCα-overexpressed cells, the frequency of exocytotic events evoked by KCl (60 mM, 5 min) was significantly increased. This effect was apparently due to the increase in the later 4 min period, but not in the initial 1 min. Overexpression of inactive PKCα mutant had no effect on KCl-evoked events. In the PKCβ-overexpressed cells, the frequency was increased both the initial and the later 4 min periods, but the effects was much evident in the initial 1 min in contrast to PKCα. This might suggest that target molecules of phosphorylation by PKCα and PKCβ are different, resulting distinct regulation of exocytosis in adrenal chromaffin cells.

P1-035 Effect of neurotrophins on neural acitivities in dissociated neurons

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Neurotrophins have been well known as promoting neural outgrowth and protecting from neural death. However, it has also been reported for regulating neural activities and neurotransmitter release. Here we investigated the effect of neurotrophins on electrical acitivies and intracellular calcium concentration in cultured rat cortical and hippocampal neurons. Multi-electrode array measurement indicated the enhancement of the acitivities after addition of neurotrophins, suggesting the synaptic activitiy increment. Responses of intracellular calcium concentrations were investigated using either confocal laser microscope or flow cytometry. Transient changes of intracelular calcium concentration were measured. Addition of tyrosine kinase inhibitors indicated that relatively fast and slow responses were detected. Its mechanism and the delay of the responses will be discussed.

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P1-036 Tempo-spatial regulation of the vesicle dynamics by PKC in adrenal chromaffin cells.

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In adrenal chromaffin cells, activation of PKC was shown to enhance catecholamine release, potentiate the priming process, and increase the size of the readily releasable pool of secretory granules. Our amperometric studies on the exocytotic events suggested that PKC promote granule recruitment for exocytosis. To clarify the implication of PKC in the recruitment of secretory granules, we studied the effects of TPA on the movements of granules in living chromaffin cells by fluorescence microscope-cooled CCD system, and dynamics of the granules were analysed. About 60% of the chromaffin granules showed a rapid movement, about 30% showed a moderate movement and the rest showed a slow movement in resting and after stimulation. The vesicles with a slow movement increased to 50~60% upon a depolarizing stimulation, and TPA increased this population. TPA-treatment increased the number of visible chromaffin granules beneath the plasma membrane, suggesting the potentiation of the recruitment.

P1-037 Presynaptic Localization of AMPA-type Glutamate Receptor in Corticostriatal and Thalamostriatal Axon Terminals

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The striatum receives glutamatergic inputs from cortex and thalamus. Vesicular glutamate transporters 1 and 2 (VGluT1 and VGluT2) are located on cortico- and thalamo-striatal inputs, respectively, whereas VGluT3 is found in cholinergic interneurons. In this study, quantitative analysis using postembedding method revealed about 1/4 and 2/5 of GluR2/3-positive particles were found in VGluT1- and 2- presynaptic terminals, respectively. In contrast, almost no GluR2/3 particles were observed on presynaptic profiles of VGluT3-terminals in the neostriatum, or on those of VGluT1- or 2-presynaptic terminals in the neocortex. The present results suggest glutamate released from cortico- and thalamo-striatal terminals controls the activity of the axon terminals through presynaptic AMPA-type glutamate autoreceptors.

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P1-038 Concentrated localization of AMPA-type glutamate receptors in Bergmann glial plasmalemma facing synaptic cleft at Purkinje cell synapses

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Ca^{2+}-permeable AMPA-type glutamate receptors (AMPARs) in Bergmann glia mediate enwrapping of glutamatergic synapses by glial processes. To elucidate the subcellular localization of AMPARs in Bergmann glia, we performed immunohistochemical analyses using antibodies for GluRα1 and GluRα4, two subunits expressed in Bergmann glia. Immunofluorescence revealed that immunoreactivities for GluRα1 and GluRα4 subunits in the cerebellar molecular layer were punctate pattern without showing overlap with presynaptic or postsynaptic molecules. By pre-embedding immunoelectron microscopy, immunoparticles for GluRα1 and GluRα4 subunits were dominantly observed on the Bergmann glial plasma membrane facing the synaptic cleft of parallel fiber-Purkinje cell synapses. These results suggest that AMPARs in Bergmann glia are regulated to accumulate around the glutamatergic synaptic cleft.

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P1-039 Internalization of NMDA receptors

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We have previously shown that in vitro cellular maturation may accelerate the mechanism underlying desensitization of NMDA receptor channels in cultured neurons. Rat cortical neurons were cultured in DMEM/F12 for 3 to 15 days in vitro (DIV), followed by homogenization and subsequent SDS-PAGE for immunoblotting using an antibody against α-adaptin (AP-2), clathrin or dynamin I. Similarly with altered sensitivity to NMDA, expression of these proteins all increased in proportion to the duration of culturing up to 15 DIV. Cortical neurons cultured for 9 DIV were briefly exposed to NMDA for 5 min, followed by biotinylation of cell surface and subsequent affinity purification using avidin beads. Biotinylated proteins immunoreactive to NR1 subunit significantly decreased in a manner dependent on the time after stimulation by NMDA from 5 to 45 min. These results suggest that transient stimulation by NMDA may lead to desensitization of NMDA receptor channels through the internalization of NR1 subunit in cultured cortical neurons.

P1-040 NMDA receptor GluRε/NR2 subunits are essential for postsynaptic localization and protein stability of GluRζ1/NR1 subunit

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We investigated GluRζ1 subunit in cerebellar granule cells lacking GluRε1/NR2A and GluRε3/NR2C subunits. In the mutant cerebellum, synaptic labeling for GluRζ1 subunit was reduced to the extrasynaptic level at mossy fiber-granule cell synapses. Quantitative immunoblot revealed substantial reductions of GluRζ1 subunit. The severe reductions in the PSD fraction suggest that any C-terminal forms of GluRζ1 subunit can not virtually accumulate at postsynaptic sites without GluRε subunits. By turnover analysis, the degradation of GluRζ1 protein was accelerated in the mutant cerebellum. Therefore, accompanying expression of GluRε subunits is essential for postsynaptic localization and protein stability of GluRζ1 subunit.

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P1-041 Altered sensitivity to NMDA during maturation in cultured rat brain neurons

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In this study, we have attempted to elucidate mechanisms underlying signaling processes of the neurotransmitter glutamate (Glu) in cultured neurons of discrete blain structures. Rat cortical, striatal, hippocampal and cerebellar neurons were cultured for 3, 9 or 15 days *in vitro* (DIV), followed by determination of intracellular free Ca^{2+} on fluorescence image analysis. The transient exposure to NMDA rapidly increased the number of neurons with high fluorescence intensity in a concentration-dependent manner at a concentration range of 1 to 200 μM, which occurred irrespective of brain structures examined. In proportion to the culture period from 3 to 15 DIV, EC_{50} values decreased in cortical, striatal and hippocampal neurons, but increased in cerebellar neurons, with no changes in maximal responses. These results suggest that cerebellar neurons may undergo developmental changes different from those in telencephalic neurons with regard to responsiveness of NMDA receptor channels.

P1-042 NMDA signaling to adult neurogenesis in murine hippocampus

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Neural progenitor cells were isolated from adult murine hippocampus according to the percoll density gradient centrifugation method, followed by cultivation in DMEM/F12 medium containing bFGF for the formation of round neurospheres up to 12 days in vitro (DIV) and subsequent harvest for detection of NMDA receptor subunits by quantitative RT-PCR. Culturing of cells led to a marked increase in expression of mRNA for NR1, NR2A, NR2B and NR2C subunits throughout culture periods between 2 and 10 DIV, while NR2D subunit mRNA was expressed in cells before cultivation with a gradual decline and abolition before 10 DIV. Brief exposure to NMDA resulted in marked expression of particular Fos and Jun family member proteins in neurospheres cultured for 4 DIV. These results suggest that NMDA receptors would be constitutively expressed in immature neural progenitor cells before differentiation to play a role in mechanisms associated with subsequent commitment and differentiation in hippocampal adult neurogenesis.

P1-043 GluR4c, an alternative splicing isoform of GluR4 is abundantly expressed in adult human brain

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We report the cloning of human GluR4c, an alternative splicing isoform of GluR4. Similar to rodent and chick GluR4c mRNA, human GluR4c had a 113-bp insert containing a stop codon prior to the exon encoding the long C terminus, resulting in a short C terminus. The expression of human GluR4c was widespread in the brain, but mainly found in the cerebellum (Cbl) and cerebral cortex (Cx) where the level of expression was upregulated with development and expressed at a level of about 30% of total GluR4 mRNA even in adult stage, whereas it decreased significantly in the cerebral white matter. The conversion from flip isoform to flop isoform of both GluR4 and GluR4c with development was most extensive in Cx and to a lesser extent in Cbl. These results suggest that the GluR4 subunit plays a pivotal role in both regulating channel properties and trafficking of AMPA receptors in the adult human brain.

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P1-044 Ligand-induced rearrangement of the dimeric conformation of metabotropic glutamate receptor1α

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The G protein-coupled metabotropic glutamate receptor 1α (mGluR1) is crucial for some forms of synaptic plasticity. Crystallographic analysis of the extracellular domain of the receptor has demonstrated that glutamate stabilizes the active states of the dimeric conformation. However, the conformational change of the cytoplasmic domain, which plays an important role in the signaling, remains unknown. Here we investigated the ligand-induced conformational change of the cytoplasmic domains by analyzing fluorescence resonance energy transfer (FRET) under total internal reflection field. Agonistic ligands altered intermolecular FRET efficiency, while did not intramolecular FRET efficiency. These results indicate that ligand binding induces dimeric rearrangement of the cytoplasmic regions.

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P1-045 Direct interaction of GluRδ2 with Shank scaffold proteins in cerebellar Purkinje cells

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Glutamate receptor (GluR) δ2 selectively expressed in cerebellar Purkinje cells plays a central role in cerebellar long-term depression (LTD), motor learning and formation of parallel fiber synapses. By yeast two-hybrid screening, we identified members of the Shank family of scaffold proteins as major GluRδ2-interacting molecules. We found that GluRδ2 bound directly to the PDZ domain of Shank proteins through an internal motif in the carboxyl-terminal putative cytoplasmic domain, designated as S segment. S segment peptide bound to the PDZ domain of Shank2 with a high affinity by surface plasmon resonance analysis. We also present evidence suggesting that GluRδ2, Shank1, Shank2, Homer and mGluR1α form a postsynaptic signaling complex and that GRIP1 interacts with Shank2 in the cerebellum. These results suggest that through Shank1 and Shank2, GluRδ2 interacts with the mGluR1α, the AMPAR and the IP_3R that are essential for cerebellar LTD.

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P1-046 C-terminal domain of δ2 glutamate receptor controls its intracellular localization

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The ionotropic glutamate receptor (iGluR) plays a major role in fast excitatory synaptic transmission, neurodevelopment, and synaptic plasticity. It has become increasingly clear that functions of iGluRs are mainly modulated by trafficking processes that control the expression of receptors on the cell surface. Unlike other iGluRs, the δ2 glutamate receptor (GluRδ2) is efficiently expressed on the surface of spines at distal dendrites of Purkinje cells. To obtain clues to the trafficking mechanism of iGluRs, we deleted various portions of the C-terminal intracellular domain of GluRδ2 and analysed the localization of the mutant proteins. We found that GluRδ2 lacking the juxtamembrane region of 13 amino acids was retained in the endoplasmic reticulum (ER) in heterologous cells and cultured neurons, a result indicating that this region is necessary for GluRδ2's exit from the ER. We are currently analyzing other regions controlling intracellular trafficking of GluRδ2.

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P1-047 Zebrafish glutamate receptor δ2 is expressed in selective neurons with wiring patterns similar to cerebellar Purkinje cells

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In mice, we have shown that cerebellar Purkinje cell-specific glutamate receptor (GluR) δ2 plays a key role in cerebellar plasticity, motor learning and neural wiring. Here, we cloned the zebrafish ortholog of GluRδ2. Zebrafish GluRδ2 mRNA was expressed not only in cerebellar Purkinje cells, but also in the crest cells of the medial octavolateral nucleus (MON) and type I pyramidal neurons of the optic tectum. By immunohistochemical analysis, we detected zebrafish GluRδ2 proteins at distal dendrites in these neurons. Interestingly, the crest cells and type I pyramidal neurons have wiring patterns similar to cerebellar Purkinje cells that receive a large number of parallel fiber inputs at their distal dendrites and a single strong climbing fiber input at the proximal dendrites. These findings raise an intriguing possibility that GluRδ2 is a key molecular organizer of cerebellar Purkinje cell-like connection pattern.

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P1-048 Association of group III metabotropic glutamate receptor-equipped axon terminals with the dendrites of cholinergic neurons in rat striatum

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Group III metabotropic glutamate receptors (mGluR4, 7 and 8) are localized to presynaptic membranes in the brain. In the present study, mGluR4 and mGluR8 immunoreactivities were observed not only as fine boutons, but also as larger boutons in the rat striatum. Larger mGluR4-positive boutons seemed aligned linearly, whereas fine boutons were distributed randomly. We examined, by the double immunofluorescence method, which population of striatal neurons received the larger boutons. Many dendrites of cholinergic and a very few dendrites of parvalbumin-positive neurons were aligned with the larger boutons. Furthermore, almost all larger boutons were associated with NK1 receptor (NK1R)-positive dendrites, and about a half of NK1R-positive dendrites were surrounded by mGluR4-positive boutons. These results suggest that mGluR4 is selectively used by a specific connection in the striatum.

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P1-049 Expressional changes in the $GABA_A$ and glycine receptor subunit mRNAs in the rat freeze-lesion model of cortical malformations.

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A focal freeze-lesion of the cerebral cortex in newborn rat produces a cortical malformation that resembles human polymicrogyria. Here, alterations in regional expression of $GABA_A$ and glycine receptor subunits were investigated in situ hybridization histochemistry using oligonucleotide probes in developmental rat cortex (P1-P21) with focal cortical malformations attributable to neonatal freeze-lesions. Within the cortical malformation, glycine receptor alpha2 subunit was upregulated, but several $GABA_A$ receptor subunits were down regulated at P4 in "bridge" structure as compared to the exofocal cortex. These results suggest that immature cortical plate neurons make up "bridge" structure during formation of microgyrus, and that altered neuronal Cl^- homeostasis is involved in neuronal migration disorder that ultimately results in cortical malformations.

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P1-050 Functional expression of metabotropic $GABA_B$ receptor in rat cerebrocortical astrocytes

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Among GABA receptors, the $GABA_B$ receptor couples to adenylylcyclase (AC) through trimeric G-proteins to regulate intracellular 2nd messenger system. However, there is considerably less evidence for the presence of $GABA_B$ receptor on astrocytes. In the present study, we investigated the functional expression of $GABA_B$ receptor in astrocytes from rat cerebral cortex. In the presence of 10 μM GTP, forskolin increased adenylylcyclase (AC) activity in membranes prepared from rat astrocytes in a concentration-dependent manner. Baclofen reduced forskolin (1 μM)-stimulated AC activity in the presence of 10 μM GTP in a concentration-dependent manner. This effect was completely abolished by treatment of membranes of astrocytes with pertussis toxin. RT-PCR showed that metabotropic $GABA_B$ receptor 1 and 2, and AC II and IV mRNAs are expressed in rat cerebrocortical astrocytes. These results demonstrate that functional metabotropic $GABA_B$ receptor are expressed in rat cerebrocortical astrocytes.

P1-051 Dopamine-D1 and D2 receptor agonists differently modulate gene expression of Homer 1a.

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To elucidate the molecular mechanism for dopamine-glutamate interactions, we investigated the effects of the selective agonists for dopamine receptor subtypes on regional gene expression of rat *Homer1a*, an immediate-early gene related to the group I metabotropic glutamate receptors, using RT-PCR. The D1 agonist SKF38393 significantly increased the mRNA levels of *Homer1a* in the striatum and nucleus accumbens, but not the medial prefrontal cortex, hippocampus or substantia nigra, compared with controls, whereas the D2 agonist quipirole had no significant effect on *Homer1a* mRNA levels in any brain region examined. Combination of SKF38393 and quinpirole increased *Homer1a* mRNA levels in the nucleus accumbens, striatum and hippocampus. The effect of combination of both agonists appeared more remarkable than that of SKF38393 alone. Stimulation of D1 receptors may thus induce gene expression of *Homer1a*. Moreover, synergic action of both D1 and D2 receptor stimulation may also exist.

P1-052 Effects of several 5-HT receptor antagonists on 5-HT-stimulated [^{35}S]GTPγS binding in rat brain membranes

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The effects of several 5-HT receptor antagonists on 5-HT-stimulated [^{35}S]GTPγS binding in rat brain membranes were determined. WAY-100,635 inhibited 5-HT (1 μM)-stimulated [^{35}S]GTPγS binding in hippocampal and cerebral cortical membranes with a mean IC_{50} value of 7.1 nM, but not in striatal membranes. On the other hand, both GR-127,935 and SB-224,289 were potent inhibitors only in rat striatal membranes with a mean IC_{50} value of 19 nM, but not in the other two brain regions. It was demonstrated that all other compounds tested (ketanserin, GR-55, 562, and BRL-15, 572) were less potent inhibitors against the 5-HT-stimulated [^{35}S]GTPγS binding in any brain regions. These results, together with our previous experimental data with a series of 5-HT receptor agonists, strongly suggest that 5-HT-stimulated [^{35}S]GTPγS binding is mediated through $5\text{-}HT_{1A}$ receptors in rat hippocampal and cerebral cortical membranes and through $5\text{-}HT_{1B}$ receptors in rat striatal membranes.

P1-053 Change of Glutamate transporter and glutamine sunthetase in rat cultured astrocytes by adenosine exposure: Role for neuroprotection via EAA scavenging

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Elevation of excitatory amino acids, such as glutamate, has been believed to be one of the key mechanisms in the neurodegenerative processes. Using rat cultured astrocytes, we tested the effect of adenosine on glutamate scavenging activities. Exposure of Cl-adenosine, an unselective adenosine receptor agonist, increased glutamate uptake activity. The exposure of Cl-adenosine also increased the contents of glutamate transporter (GLT-1) and glutamine synthetase. The adenosine-induced potentiation of glutamate uptake activity seemed to be mediated by both mechanisms, i.e. increase of glutamate transporter and activation of glutamate metabolism in the cells by increased glutamine synthetase. These results indicate that adenosine receptor activation may be a possible therapeutic method for several neurodegenerating conditions such as trauma and ischemia of CNS.

P1-054 Regulated expression of human norepinephrine transporter splice variants

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The norepinephrine transporter (NET) acts to terminate noradrenergic neurotransmission by reuptake of released norepinephrine (NE). We have reported the splice variants of human NET at COOH terminal region produced by exon(E)15 (hNET1) and E16 (hNET2), which showed different function and expression at cell surface. To elucidate the importance of this region for NET functional expression, we prepared the various deletion mutants, and transiently expressed in COS-7 cells for their functional characterization by uptake assay and immunoblot analysis with cell surface biotinylation. The results demonstrated the regulated expression of NET through COOH terminal region, which participates differently for hNET1 and hNET2.

P1-055 Regulation by extracellular glucose of GABA transporter activity in rat calvarial osteoblasts.

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We have previously demonstrated constitutive expression of particular machineries required for central γ-aminobutyric acid (GABA) signaling with marked suppression by GABA of the maturation in cultured osteoblasts. By contrast, osteopenia is frequently seen in patients suffering from insulin-dependent and -independent diabetes mellitus. In this study, therefore, rat calvarial osteoblasts were cultured in α-MEM containing either 5.5 mM or 25.5 mM glucose for 7 to 14 days in vitro (DIV). In cultured osteoblasts, In cultured osteoblasts, the accumulation of [^{3}H]GABA was significantly increased in osteoblasts cultured for 14 DIV under hyperglycemic conditions, compared to that found in the presence of equimolar mannitol. These results suggest that the GABA transport system may play an unidentified role in mechanisms associated with pathology and etiology of hyperglycemic osteopenia in cultured rat calvarial osteoblasts.

P1-056 Inhibitory regulation of plasma dopamine transporter activity by complete knockout of vesicular monoamine transporter-2 (VMAT-2)

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Vesicular monoamine transporter 2 (VMAT2) heterozygous knockout mice are more vulnerable to neurotoxin. Using E14 mesencephalic dopaminergic cultures from VMAT2 (-/-), (+/-) or wild-type mice, the number of tyrosine hydroxylase (TH)-positive cells and activities of dopamine transporter (DAT) were measured. The number of TH-positive cells remained unchanged among these genotypes. Of interest, DAT activity in the homozygous cells was significantly decreased, but not in the heterozygous cells, suggesting that deletion of VMAT-2 induced compensatory modulation of DAT function.

P1-057 Ultrastructural localization of high-affinity choline transporter on synaptic vesicles.

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In cholinergic neurons, Na^+- and Cl^--dependent, high-affinity choline uptake system is thought to be the rate-limiting step in acetylcholine (ACh) synthesis. The system is highly regulated by neuronal activity; the choline uptake is increased by a condition in which ACh release is favored. Here we analyzed the ultrastructural localization of the high-affinity choline transporter (CHT) in the rat neuromuscular junctions with two separate antibodies. The majority (>90%) of immunogold labeling of CHT was observed on synaptic vesicles rather than the presynaptic plasma membrane. Less than 5% of the gold-silver particles were associated with the plasma membrane, and more than 70% of such particles were localized within or in close vicinity to presynaptic active zones. Our data suggest that trafficking of CHT from synaptic vesicles to the plasma membrane couples neuronal activity and choline uptake.

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P1-058 Regional differences on glycosylation of neutral amino acid transporter ASCT1 in rat brain

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The present study addresses the difference of N-linked glycosylation of the neutral amino acid transporter, ASCT1 in rat brain regions. The rat ASCT1 was found to be highly glycosylated in the rat primary neurons and astrocytes, with molecular weight, 66-70 and 73-78 kDa, respectively. We also found that molecular weight forms of ASCT1 were different among brain regions in the rat. This study is the first molecular identification of glycosylated ASCT1 and may provide important insights into the multiple molecular forms and functions of the ASCT1.

P1-059 Neuronal localization of a system asc neutral amino acid transporter (Asc-1) in the rat cerebral and cerebellar cortices.

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D-serine is an endogenous modulator of NMDA receptors. L-serine is essential for survival and development of neurons. We have recently identified a system asc amino acid transporter Asc-1 which transports D- and L-serine with high affinity. To determine the localization of Asc-1 in rat cerebral and cerebellar cortices, we generated two kinds of polyclonal antibodies against rat Asc-1 and performed immunohistochemical analysis. Asc-1 immunoreactivity was detected in neurons, including cerebellar Purkinje neurons and pyramidal neurons in the hippocampus and the neocortex. It was clearly localized in somata and dendrites. The localization of Asc-1 in brain suggests the significant contribution of Asc-1 to the synaptic clearance of D-serine and neuronal uptake of L-serine that is essential for dendrite growth.

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P1-060 Developmental expression pattern of sodium bicarbonate cotransporter NBC in mouse brain

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NBC exists in 3 different isoforms (kNBC, pNBC, bNBC), different splicing variants of the same gene. Ionic substitution studies and inhibition of injury suggest that NBC is involved in astrocyte vulnerability to acidic injury. But their distribution and physiological role in brain are not known. Here we examined the expression and localization of each NBC isoform in developmental mouse brain by using their specific antibodies. Expression in brain was first seen at approximately P7, increased at P13, and persisted in the adult brain. Expression pattern in cerebellum was different between isoforms: bNBC in morecular layer, pNBC in medullary layer, and kNBC in granular layer. It suggested that the functional difference is exist between each isoforms in brain.

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P1-061 Contribution of System Xc⁻ to Al Citrate Uptake by SH-SY5Y Cells

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Aluminum (Al) is an abundant metal, of which accumulation in the brain has potential to cause neuronal death. We previously reported that system Xc^-, an L-glutamate/L-cystine exchanger, played an important role in transport of Al citrate, the predominant species in the brain, at the blood-brain barrier. Here, we studied whether Al citrate was taken up by SH-SY5Y cells, which were differentiated to neuron-like cells by treating with retinoic acid, *via* system Xc^-. The mRNAs for xCT and 4F2hc, the components of system Xc^-, were expressed in SH-SY5Y cells. Al citrate uptake showed temperature- and concentration-dependency, and did not require a Na^+-gradient as a driving force. L-Glutamate, L-aspartate and L-cystine, ligands for system Xc^-, had *cis*-inhibitory and *trans*-stimulatory effects on the Al citrate uptake. These results demonstrated that system Xc^- handled the Al citrate uptake in SH-SY5Y cells.

P1-062 Functional expression of the organic cation/carnitine transporter OCTN2 in rat astrocytes

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L-Carnitine plays an important role in lipid metabolism by facilitating the transport of long-chain fatty acids across the mitochondrial inner membrane followed by fatty acid β-oxidation. It is taken up into the cells by the recently identified organic cation/carnitine transporter OCTN2. Here we assessed the functional expression of OCTN2 in cultured rat astrocytes. The cells were found to take up L-carnitine by a Na^+-dependent process. This uptake was saturable, and the K_m for the process was 103.6 μM and the V_{max} was 5149 pmol/mg protein/h. L-Carnitine uptake was inhibited by D-isomer, acetyl-L-carnitine, palmitoyl-L-carnitine and organic cations (e.g. TEA, quinine, quinidine or cimetidine). RT-PCR with primers specific for the rat OCTN2 revealed the existence of OCTN2 mRNA in rat astrocytes. We conclude that the organic cation/carnitine transporter OCTN2 is functionally expressed in rat astrocytes.

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P1-063 Roles of presynaptic TrkB receptors in the hippocampal CA1 region

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TrkB receptors, whose ligands are brain-derived neurotrophic factor (BDNF) and NT-4, are essential for neuronal differentiation, neuronal signaling and synaptic plasticity. Although TrkB receptors are expressed at both pre- and postsynaptic sites in the hippocampal CA1 region, their precise roles are poorly understood. Thus, we have generated conditionally gene-targeted mice in which the knockout of the trkB gene is restricted to the CA3 region from which presynaptic afferents in the CA1 region originate (E. Kato et al.). Adult mutant mice show impairment of short-term presynaptic plasticity induced by larger Ca2+ influx into presynaptic terminals. We discuss possible mechanisms by which presynaptic TrkB receptors regulate synaptic plasticity.

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P1-064 C57BL/6 mice lacking TrkB neurotrophin receptor in hippocampal CA3 region

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TrkB receptor plays important roles in synaptic plasticity and learning as well as the survival and differentiation of neurons. To dissect the functional roles of TrkB in brain functions, we generated the target mouse line carrying the *TrkB* gene flanked by two *loxP* sequence using ES cells derived from the C57BL/6 strain. The target mice were crossed with Cre mice carrying the *GluR*γ1 gene promoter-driven Cre recombinase gene. In situ hybridization analysis showed the successful ablation of the *TrkB* mRNA expression mainly in the hippocampal CA3 pyramidal neurons of the mutant mice. The mutant mice will provide a valuable tool to investigate the role of hippocampal TrkB in synaptic plasticity and learning under pure C57BL/6 genetic background.

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P1-065 Synergistic regulation of activity-dependent stabilization of trkB FL mRNA in cultured hippocampal neuron

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In hippocampal neurons, neuronal activities promote extrasomatic localization of trkB FL mRNA in dendrite. We obtained full-length sequence of rat trkB FL mRNA 3'UTR by 3'RACE and found that it contained two clusters of AU-rich element (termed AU-box 1 and AU-box 2). Both AU-boxes were required for rapid destabilization of trkB FL mRNA. KCl depolarization and BDNF stimulation enhance the stabilization of trkB FL mRNA in cultured hippocampal neuron. This activity-dependent stabilization of trkB FL mRNA was under synergistic regulation of receptor type protein tyrosine kinase and glutamate receptor which share PI3K signaling and was synchronized with mRNA trafficking along with microtubles. Furthermore, we identified 7 candidate genes which encode cis-acting factors of trkB FL mRNA 3'UTR by northwestern screening. These molecules might provide significant approach to study the localization of trkB FL mRNA.

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P1-066 Effect of leukotriene B_4 on neural stem cell proliferaiton

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Leukotriene B_4 (LTB_4) is a lipid mediator that plays an important role in inflammatory response. However, no studies have been reported about the role of LTB_4 in neural stem cell (NSC) functions. Here, we showed that LTB_4 stimulated the proliferation of murine NSCs. Physiological concentrations of LTB_4 and synthesized agonist stimulated cell growth of NSCs. In contrary, leukotriene (LT) synthetase inhibitors and LTB_4 receptor antagonist inhibited cell growth of NSCs in concentration-dependent manners. Cell death induced by LT synthetase inhibitors and LTB_4 receptor antagonist was apoptosis accompanied with nuclear condensations, a morphological marker of apoptosis. In Gene Chip data exhibited the alteration of genes that are related in cell cycle, apoptosis, proliferation and differentiation. These data indicate that LTB_4 may regulate the proliferation of NSCs.

P1-067 Effects of several intravenous anesthetics on sigma-1 receptors

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Sigma-1 receptors (Sig-1R), which are endoplasmic reticulum protein and constitute Sig-1R/ankyrinB/IP3 receptors (IP3R) complex, are related to various CNS effects. Sig-1R agonists dissociate Sig-1R/ankyrinB complex from IP3R, resulting in IP3R activation. The aim of this study was to investigate whether several intravenous anesthetics affected the Sig-1R by receptor binding assay and whether the anesthetics, which were once determine to affect the Sig-1R, caused IP3R activation by measuring the potentiation of bradykinin-induced increase in intracellular Ca^{2+} concentrations ($[Ca^{2+}]_i$). Clinically relevant doses of propofol, a commonly used intravenous anesthetic, affects Sig-1R, but did not affect the bradykinin-induced increase in $[Ca^{2+}]_i$, suggesting that propofol may be a Sig-1R antagonist, and psychoactive effects of propofol may be mediated, at least partly, by Sig-1R.

P1-068 Dynamics of corticosteroid receptors and importins in living neural cells and non-neural cells

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Glucocorticoid receptor (GR) and mineralocorticoid receptor (MR) are ligand-inducible transcriptional factors, both of which are translocated from the cytoplasm to the nucleus upon ligand binding. Molecules less than 20-40 kDa can passively diffuse through aqueous channels that span the nuclear pore complex (NPC) from the cytoplasm to the nucleus, whereas more than 40 kDa are transported through gated-channels of the NPC by active mechanisms. Importin α and importin β are docking proteins for karyopherin-mediated binding of substrate in a nuclear import pathway across the NPC. In the present study, we investigated the trafficking of GR and MR, and importins in living cultured hippocampal neurons and COS-1 cells with GFP color variants, and the interaction of these receptors and importin α using fluorescent resonance energy transfer (FRET) technique. We also examined dynamics of importin α subtypes, importin α 1, 2, and 3 in living cells with GFP.

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P1-069 Neural progenitor cells isolated from discrete brain regions of embryonic rats

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This study was aimed to isolate neural progenitor cells from discrete brain structures, including neocortex, hippocampus, striatum and other regions, in embryonic rats. Cells were collected from lower layer obtained after percoll density gradient centrifugation of individual homogenates, followed by culturing in DMEM/F12 medium containing bFGF for 12 DIV. Marked round spheres were formed on 2 to 4 DIV in cultures prepared from different brain structures, followed by continuation of growing of their size and subsequent proliferation to form large neurospheres in proportion to the duration of cultivation. High immunoreactivities were detected with nestin but not with either MAP2 or GFAP in neurospheres cultured for 12 DIV. However, removal of bFGF induced subsequent expression of both MAP2 and GFAP in cultured cells on 16 DIV. These results suggest that the percoll density gradient method is suitable for isolation of neural progenitor cells from different embryonic brain structures.

P1-070 Characterization of binding sites for adenine, a neurotrophic factor of cultured Purkinje cells

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Our recent study revealed that adenine acts as a neurotrophic factor in cultured Purkinje cells, suggesting the presence of specific receptors for adenine in the brain. In this study, we identified specific binding sites for [^{3}H]adenine in rat brain membranes. As the [^{3}H]adenine binding was specific, reversible and saturable, it fulfils an important criteria as the receptor for hormone or drug. [^{3}H]adenine binding sites were also found to consist of adenosine-sensitive and -insensitive because adenosine partially displaced the binding. Other adenosine receptor ligands or nucleoside transporter inhibitors were ineffective on [^{3}H]adenine binding. Among the tissues examined, the brain showed the highest [^{3}H]adenine binding activity. These results support the presence of a novel adenine receptor in rat brain membranes.

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P1-071 AFM image of inositol 1,4,5-trisphosphate receptor (IP3R) tetramer structure in its native environment

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The IP3R plays an integral role in Ca^{2+} signaling and neural plasticity. To analyze particular conformational change of intact IP3R is very important to recognize its function. According to the observation by electron microscopy, the structure of IP3R was morphologically changed between a pinwheel structure and a smaller square at different Ca^{2+} level.
Here we obtained the high-resolution image of the IP3R surface in its native environment by using atomic force microscopy (AFM). Contact mode AFM topographs were recorded at minimal loading forces. We observed the purified membrane with domain gathered IP3R proteins. Compared with the flat and featureless surface of lipid, the IP3R domain was assembled. At high magnification, the image showed the IP3R molecules that were arranged in the lattice on the membrane.

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P1-072 Neuropeptide Y Y1 receptor mRNA in the rodent brain: Distribution and coexpression with the melanocortin-4 receptor

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The central neuropeptide Y (NPY)/Y1 receptor (Y1-R) system has been implicated in feeding, endocrine, and autonomic regulation. By using radioisotopic in situ hybridization histochemistry with a novel sensitive probe, we observed Y1-R mRNA in a number of relevant hypothalamic and extra-hypothalamic sites in the rat. A similar pattern of Y1-R hybridization was evident in the mouse brain. The melanocortin-4 receptor (MC4-R) is another established regulator of energy homeostasis. By using a transgenic mouse line expressing green fluorescent protein under the control of the MC4-R promoter, we observed MC4-R-positive cells coexpressing Y1-R mRNA in several brain sites including the paraventricular hypothalamic and central amygdaloid nuclei. These observations agree with the proposed roles of the NPY/Y1-R system.

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P1-073 Feeding behavior of NPY-Y5 knock out mice

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NPY is the most potent agent for feeding. NPY receptors are involved in positive feeding regulation. To clarify the role of Y5 receptor on feeding, feeding behavior of Y5-KO mice and gene expression of feeding-related neuropeptides were studied. Body weight, food intake and effect of fasting were measured. After behavioral experiments, mice were sacrificed and whole brains were dissected or sliced to perform RT-PCR or ISH. From 4 to 40 weeks old, body weight of Y5-KO mice was twice as much as wild-type mice. Food intake was increased in male Y5-KO mice. Fasting did not change the amount of food or water intake in male Y5-KO but increased both in female. RT-PCR and ISH suggested the decreased gene expression of NPY and galanin but increased gene expression of CART. Increase in body weight in Y5-KO mice was produced partly due to augmented feeding behavior. Changes in gene expression of *NPY, galanin and CART in* hypothalamus might suppress the feeding behavior.

P1-074 Colocalization pattern of mineralocorticoid receptor (MR) and glucocorticoid receptor (GR) in the rat hippocampus and paraventricular nucleus

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We investigated the distribution and the colocalization pattern of the MR and the GR in the hippocampus and the paraventricular nucleus (PVN) of the hypothalamus in the rat under a confocal laser scanning microscopy. MR- and GR-immunoreactivity (ir) was colocalized in the CA1 and CA2 pyramidal neurons and granule cells of the dentate gyrus in the hippocampus and the parvocellular regions of PVN. Subcellular distribution of MR-ir was more cytoplasmic than that of GR-ir. After adrenalectomy (ADX), the distribution patterns of both MR and GR were changed to cytoplasmic. Administration of corticosterone to the ADX rats recovered the similar distribution pattern to the control. In addition, we performed the lesion experiment of fornix and the effect on the expression of mRNAs of GR, MR and *CRH in the PVN was also examined.*

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P1-075 Transgenic rats expressing green fluorescent protein under the control of estrogen receptor α promoter

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Transgenic rats expressing enhanced green fluorescent protein (EGFP) under control of the estrogen receptor α (ERα) promoter 0/B were generated to tag ERα-positive neurons in the brain. Fluorescence of EGFP was observed extensively in the brain, particularly in the cortex, hippocampus, preoptic area (POA), dorsal hypothalamic area, amygdala and midbrain central gray. A few, weakly fluorescent cells appeared in the ventromedial hypothalamic nucleus. ERα-immunoreactivity labeled approximately 70% of EGFP cells in the POA and amygdala, however, unexpectedly, a few ERα/EGFP double labeled cells were observed in the other areas of the brain. EGFP was down regulated by estrogen as ERα protein in the POA, showing functional relevance to estrogen-dependent regulation of ERα. These results suggest that the action of rat ERα promoter 0/B is regulated specifically in subregions of the brain. The transgenic rats would be useful for physiological studies on the role of estrogen signaling.

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P1-076 ER expression in the kainic acid-injured hippocampus

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ER expression in the kainic acid-injured hippocampus was mapped in terms of both mRNA and protein to see if the latter changes in parallel with the former by means of whole mount *in situ* hybridization combined with immunohistochemistry. Three week-old rats were injected with kainic acid. After 4 days, the hippocampus was excised, then cultured in the medium with/without 17β-estradiol. After various periods of incubation, 3 to 7 weeks, ER expression was examined by *in situ* hybridization and immunohistochemistry. In most of the hippocampal regions, including CA1 to 3 and the dentate gyrus, ER mRNA expressions changed in parallel with ER-like immunoreactivities. Treating by β-estradiol markedly increased ER expression, particularly in the CA3 region in the kainic acid-injured hippocampus.

P1-077 Interleukine-1β induces aquaporin 4 expression in rat astrocytes

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Aquaporin 4 (AQP4), a water transporter, is expressed in astrocyte foot processes near capillaries. It is suggested that AQP4 plays an important role in brain water balance and the accumulation of brain water inresponse to brain damages.On the other hand, Interleukine-1β (IL-1β) has been reported as one of factors, which facilitated brain edema.In this study, we examined whether IL-1β affects the AQP4 expression in astrocytes. AQP4 expression was significantly induced in response to IL-1β in a concentration- and time-dependent manner. Taken together, IL-1β may facilitate brain edema formation through the up-regulation of AQP4 expression.

P1-078 Hetero-oligomerization of A_1 Adenosine receptors and $P2Y_2$ purinergic receptors

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Recently, oligomerization of G protein-coupled receptors has attracted much attention. Here we show the evidence for hetero-oligomerization between purinergic receptor subtypes, i.e. A_1 adenosine receptor and $P2Y_2$ receptor, in cotransfected HEK293T cells by ligand binding, immunohistochemical and coimmunoprecipitation assays. A_1 adenosine receptor agonist binding was found to be inhibited by a $P2Y_2$ receptor agonist in the cotransfected cell membranes. Cotransfected HA-A_1 and Myc-$P2Y_2$ receptors in HEK293T cells were observed to co-localize by a confocal laser microscopy. Furthermore, Myc-$P2Y_2$ receptors were coimmunoprecipitated by anti-HA antibody along with HA-A_1 adenosine receptors from the cell lysate of HA-A_1/$P2Y_2$ receptor-cotransfected cells. These results indicate that A_1 adenosine receptors and $P2Y_2$ receptors form hetero-oligomers in cotransfected cells.

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P1-079 Regulation of P2X recepters by parkin

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Loss of parkin function causes an autosomal recessive juvenile-onset form of Parkinson's disease (AR-JP). Parkin was shown to function as an E3 ubiquitin protein ligase. However, the function of parkin in neuronal cells remains elusive. Here, we show that parkin modulates ATP receptors that are widely distributed in the brain and involved in various biological activities including neurosecretion. ATP-induced inward currents due to the activation of P2X receptors were measured in mock-, parkin or mutant parkin-transfected PC12 Tet-Off cells under the conventional whole-cell patch clamp configuration. The amplitude of ATP-induced currents was significantly greater in wild type parkin-transfected cells, which was attenuated by H-89. Our results suggest that parkin affects neurotransmission by regulating P2X receptors with activation of PKA.

P1-080 P2X receptor mediates vagal inspiration-promoting reflex in the rabbit

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The central respiratory rhythm is under strong influence of the pulmonary stretch receptor (PSR) afferents. Low-frequency vagal PSR activity or lung deflation promotes the central inspiratory activity (Takano and Kato, *J Physiol*, 1999, 2003). We examined whether ATP-gated receptor channels (P2X receptors), which regulate synaptic transmission in the nucleus of the solitary tract (NTS; Shigetomi and Kato, 2004), mediate this reflex. In the anesthetized rabbits, vagal afferents were stimulated at 5 Hz and 160 Hz to elicit inspiratory promotion (I-pro) and suppression (I-sup), respectively. A microinjection (20 nmole/side/rabbit) of PPADS into the caudal NTS, but not that of the vehicle, significantly suppressed the I-pro but not the I-sup. The I-pro was resistant to kynurenic acid (10-20 nmole/side/rabbit), which however significantly suppressed the I-sup. We conclude that the P2X receptors in the NTS mediate the pro-inspiratory responses to the lowered lung volume.

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P1-081 Changes in intracellular free Ca^{2+} by nicotine in cultured astrocytes

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This study was aimed at investigation of mechanisms underlying signaling mediated by nicotinic acetylcholine receptors (nAChRs) in cultured rat cortical astrocytes. Cortical astrocytes were incubated with fluo-3 AM for determination of intracellular free Ca^{2+} on fluorescence image analysis. Astrocytes were exposed to nicotine at different concentrations from 10 to 1,000 μM, followed by determination of the number of cells with high fluorescent intensity 2 min later. The exposure to nicotine increased the number of cells with high fluorescence in a concentration-dependent manner. This increase by nicotine was significantly prevented by the addition of the α4β2 heteromeric subtype specific antagonist dihydro-β-erythroidine and the α7 homomeric subtype specific antagonist methyllycaconitine. These results suggest that nicotine may increase intracellular free Ca^{2+} through the influx of extracellular Ca^{2+} across α4β2 and α7 channels functionally expressed in cultured rat cortical astrocytes.

P1-082 Syntaxin 5 affects organization of the Golgi apparatus

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Syntaxin 5 (Syx5) is ubiquitously expressed in many cell types and thought to be required for vesicle trafficking between the ER and the Golgi apparatus, as a *t*-SNARE. However, the precise role of Syx5 in cells is poorly understood. In cultured neurons, we found that the amount of Syx5 was reduced with ER stress or progression of maturation. In order to understand the physiological meanings of these phenomena, we examined the effects of overexpression and knockdown of Syx5 in cell lines. Downregulation of Syx5 using RNA inteference induced the change in the structure of the Golgi apparatus in HeLa and NG108-15 cells. The GM130 (marker for *cis*-Golgi) and β-COP (component of COP I vesicle) were dispersed in these cells. Similarly, the Membrin and GS28 that are SNAREs in the Golgi apparatus showed diffused pattern of localization. Overexpression of Syx5 also revealed abnormal structures in the Golgi apparatus. These results indicate that appropriate amount of Syx5 is necessary for vesicle trafficking and may have a role in the maintenance of the Golgi structure.

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P1-083 Identification of Aip-1 as an addicsin/GTRAP3-18 associated protein

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We identified addicsin as a highly up-regulated factor in amygdala nuclei in mice repeatedly given morphine. Its rat homologue GTRAP3-18 was independently reported to associate with the neural glutamate transporter EAAC1 and to modulate the glutamate uptake by EAAC1. However, its function remains largely unknown. To clarify the molecular mechanisms, we searched addicsin/GTRAP3-18 associated factors by the yeast two-hybrid screen using a cDNA library prepared from the amygdala in mice, and identified an uncharacterized molecule termed ADP-ribosylation-like factor 6 interacting protein (Aip-1). To confirm the result, we carried out co-immunoprecipitation assay, glycerol gradient assay, mammalian two-hybrid assay and immunohistochemical analysis with NG108-15 cells transiently co-expressed Aip-1 and addicsin/GTRAP3-18. These assays showed that Aip-1 associated with addicsin/GTRAP3-18 in NG108-15 cells.

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P1-084 3,4-Dihydroxybenzaldehyde, a novel agonist for *Caenorhabditis elegans* C06H5.7

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We have previously isolated C06H5.7 as a receptor candidate for L-3,4-dihydroxyphenylalanine (DOPA). It is a putative member of the seven transmembrane (TM) domain G-protein-coupled receptor family. The conserved aspartate residue involved in binding the amine group of the catecholamine side chain, however, is substituted by glycine in the TM3 of C06H5.7. We confirmed that L-DOPA and L-*threo*-DOPS, a DOPA-like agonist, did not induce inward current in the C06H5.7-expressed Xenopus oocyte. On the other hand, metabolites of L-*threo*-DOPS activated C06H5.7 more effectively than those of L-DOPA. There were at least two agonists for C06H5.7 among L-*threo*-DOPS metabolites. We identified 3,4-dihydroxybenzaldehyde (DHBAL) as one of agonists. The non-amine structure of DHBAL might be associated with the lost amine-recognition site in C06H5.7.

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P1-085 Identification of raft-localized ATPases of synaptic membranes

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In order to elucidate the physiological function of membrane microdomain(raft), raft-localized ATPases were characterized and found the localization of Cl^--pump activity. Further solubilization and immunoprecipitation analysis showed the enrichment of the activity in the immunoprecipitate obtained using anti-NAP-22, not anti-Go, GAP-43, or F3. The localization of V-ATPase was found in another attempt to fractionate raft component using Blue-Native-PAGE. V-ATPase was found to be a main component in the rafts from spm and synaptic vesicle fractions. Since the ATPase activity was reduced after the extraction of membrane cholesterol, this result showed the importance of cholesterol in membrane functions.

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P1-086 Identification and characterization of 4-5F6 antigen as the neural tissue-enriched synaptic protein in the lipid raft of rat brain

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A membrane microdomain enriched in cholesterol and glycosphingolipids, lipid raft, was found to contain many kinds of functional molecules. Production of monoclonal antibodies (mAbs) using rat brain-derived raft fraction as the antigen was attempted. And one mAb (4-5F6) detected a raft-localized protein (4-5F6 antigen). Biochemical assay showed high expression of 4-5F6 antigen in brain. Part of this antigen was not solubilized with SDS treatment, and this insolubility was increased during aging. This SDS resistance, however, diminished through a wash with propanol. Immunohistochemical studies demonstrated the synaptic localization of 4-5F6 antigen. These results suggest that 4-5F6 antigen is involved in synaptic functions during aging. Identification and further characterization of this antigen are under progress.

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P1-087 Identification of tyrosine phosphorylation sites of Git1, a substrate for Ptprz

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We previously identified G protein-coupled receptor kinase-interactor 1 (Git1) as one of the substrates for protein tyrosine phosphatase receptor type z (Ptprz) by using the yeast substrate-trapping system. Git1 is a multidomain protein that is known to function as an integrator of signaling pathways controlling vesicle trafficking, cell adhesion and cytoskeletal organization. In addition we demonstrated that VacA, a cytotoxin secreted by *H. pylori*, functions as a pseudo ligand for Ptprz, and induces gastric ulcer. However, functional regulation of Git1 by its tyrosine phosphorylation is unknown. To understand the physiological roles for Ptprz signaling, identification of tyrosine-phosphorylation sites of Git1 is essential. Here, we show that Git1 is phosphorylated on three tyrosine residues in COS7 cells treated with pervanadate, a PTP inhibitor, and one of the sites is selectively dephosphorylated by recombinant Ptprz *in vitro*.

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P1-088 MAPPING ESSENCIAL DOMAIN OF AKAP79/150 FOR MUSCARINIC SUPPRESSION OF THE M-CURRENT

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The M-channel is characterized by its slow gating and suppression induced by stimulation of muscarinic acetylcholine receptor. We had a special interest in a scaffold protein called AKAP79/150 because it can bind to most of the candidate mediators for muscarinic suppression of M-currents. We performed a series of experiments to test whether AKAP79/150 regulates the KCNQ2 channel and the M-current in superior cervical ganglion (SCG) neurons. We induced gene silencing of rat AKAP150 by expression of short hairpin RNA (shRNA) in SCG neurons. The gene silencing toward rat AKAP150 validated that this anchoring protein is required for the muscarinic suppression of the M-current. Finally, we determined the minimal requirement for regulation of the signal transduction by rescuing these AKAP150 silenced neurons with various deletion mutants of the human orthologue, AKAP79.

P1-089 Crosstalk between vitamin D and glutamate signaling in primary cultured rat cortical neurons.

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Vitamin D receptor (VDR) is a transcription factor of zinc finger domain with ability to specifically recognize vitamin D responsive element (VDRE) on inducible target genes after hetero-dimerization with retinoid X receptor. Oligonucleotide containing VDRE was radiolabeled with [α-^{32}P]dCTP using Klenow fragment as a probe for gel retardation electrophoresis analysis. In rat cortical neurons cultured for 9 days in vitro, no significant change was found in VDRE binding after the exposure for 2 h to the neurotransmitter glutamate at 100 μM. In the presence of the VDR agonist 1α, 25-$(OH)_2D_3$ at 100 nM, however, glutamate markedly decreased VDRE binding at 100 μM. By contrast, glutamate led to a transient increase in DNA binding of the other transcription factor activator protein-1, which was less apparent in the presence of 1α, 25-$(OH)_2D_3$. These results suggest that crosstalk could occur between vitamin D and glutamate signals in cultured rat cortical neurons.

P1-090 Action of noradrenaline on rat subforcal organ (SFO) neurons in slice preparation

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Injection of noradrenaline (NA) into SFO causes an increase of water intake. Hemorrhaging increases NA release into SFO area. NA may be a chemical substance to control body fluid balance and cardiovascular regulation, by acting on SFO neurons. In the present study, we examined the action of NA on rat SFO neurons using patch clamp methods. In current-clamp mode, NA produced depolarization in more than half of SFO neurons. In voltage-clamp mode, NA induced inward current at a holding potential of -70 mV, in a dose-dependent manner. In some neurons, NA-induced inward currents were coupled to an increase in membrane conductance. The estimated reversal potential was -29.4 mV, suggesting an involvement of non-selective cation channels. In other neurons, NA-induced inward current exhibited no change of membrane conductance. α1 and β adrenergic agonists produced inward currents on SFO neurons. These suggest that NA acts mainly via α1 and β adrenergic receptors postsynaptically on SFO neurons.

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P1-091 Investigation of Intracellular Magnesium Stores by Simultaneous Fluorescent Imagings in PC12 Cells

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Both the intracellular Mg and Ca concentration ([Mg] and [Ca]) in PC12 cells were increased by application of a mitochondrial uncoupler, FCCP. Simultaneous measurement of Mg and Ca mobilizations with a Mg indicator, KMG-104, and fura-2 revealed no Mg release from ER or divalent cation binding sites. This Mg was also not released from Mg-ATP because [ATP] did not reduce in the first 2 min following FCCP exposure. FCCP-induced [Mg] increase was observed at mitochondria localized area, and the initial [Mg] increase followed that of mitochondrial depolarization. We, therefore, conclude the sources of Mg in the FCCP-induced [Mg] increase are mitochondria, and mitochondrial depolarization triggers the Mg release.

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P1-092 Pseudosubstrate of 3-phosphoinositide-dependent protein kinase 1 (PDK1) regulates PKC functions

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PDK1 phosphorylates the activation loop of PKC, being required for its maturation and following activation. However, the roles of PDK1 in neuronal and cellular functions have remained unclear. In this study, we constructed pseudosubstrate of PDK1, consisting of the peptide around δ-PKC activation loop and PDK1 binding site of PRK2 (PIF). We designated it as δAL-PIF and examined the effects of δAL-PIF on PKC functions. δAL-PIF significantly inhibited the the activation loop phosphorylation of PKCs and reduced the amount of expressed γ-PKC-GFP but not δ-PKC-GFP. Furthermore, in case of receptor-mediated translocation of PKCs, δAL-PIF significantly prolonged their retention period at the plasma membrane. These results indicate that PDK1 regulates the stability and targeting of PKCs by phosphorylating their activation loops.

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P1-093 Identification and analysis of new interacting proteins with transcription factor CREB

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CREB has been known to play essential roles in memory consolidation, circadian rhythm and emotional behavior. CREB is activated through phosphorylation by PKA and CaMKs. Only phospho-CREB can interact with transcription coactivator CBP and activates transcription. To further understand the molecular mechanisms of transcription regulation by CREB, we performed yeast two hybrid screening using brain cDNA library to identify new interacting proteins with CREB. We have identified several interacting proteins with CREB and analyzed the roles of them in CREB-mediated transcription. One of interacting proteins is a C-terminus-truncated form of TORC2, which has been shown to act as a positive regulator for CREB, We found that this interacting protein (TORCΔ) is generated by alternative splicing of TORC2 gene and strongly enhances CREB-mediated transcription. Currently, we are comparing the function of TORC2 and TORC2Δ as a modulator of CREB activity.

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P1-094 Visualization and monitoring of αCaMKII activation *in vivo* using FRET

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αCaMKII plays an essential role in neuronal plasticity. In response to increase in Ca^{2+} concentration, αCaMKII is activated by the interaction with Ca^{2+}/CaM. Prolonged increase in Ca^{2+} concentration leads to further activation of αCaMKII through autophosphorylation at Thr286. Previous studied has predicted that interaction with CaM as well as autophosphorylation induce conformational change of αCaMKII. To understand the regulation of αCaMKII activity *in vivo*, we have tried to visualize and monitor the αCaMKII activity in living cells using FRET. In this report, we examined the roles of autophosphorylation in the interaction with CaM and the conformational change using various αCaMKII mutants at T286, T305 and T306. We analyzed the mutants including T286A, T286D, T305/306A, T305/306D and T286/305/306A, and observed evidences that autophosphorylation at T286 is required for "CaM trap" *in vivo* and that autophosphorylations of T305/306 strongly affect the interaction with CaM but not CaM-independent conformational change of αCaMKII.

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P1-095 Transgenic mice carrying inactivated α subunit of Ca^{2+}/calmodulin-dependent protein kinase II

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Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII) is highly enriched in the central nervous system, and is implicated in synaptic plasticity, as well as in the suppression of epileptiform activity and ischemia-induced neuronal damage. However, whether protein kinase activity, calmodulin-binding capacity or multimeric structure and/or interaction with other proteins are involved remains to be determined yet. To determine the specific role of its protein kinase activity, we generated mice carrying inactivated α subunit of CaMKII (CaMKIIα) by replacing Lys-42 with Arg-42 (R42) using knock-in method. Forebrain homogenate from homozygotes with CaMKIIα (R42) showed a 60% decrease in CaMKII activity compared to that from wild type littermates without apparent decrease in CaMKIIα protein level. On the other hand, cerebellar homogenate from homozygotes showed normal CaMKII activity. Further analyses of these mice are underway.

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P1-096 Transcriptional regulation of mPer1 promoter through CaM kinase II and MAP kinase pathways

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CaM kinase II activity is required for light-induced phase delays and induction of Per1 and Per2 genes in the hamster suprachiasmatic nucleus. We here focused on transcriptional regulation of mPer1 promoter by CaM kinase II and MAP kinase. We defined CaM kinase II- or MAP kinase-responsive regions on the mPer1 promoter using luciferase reporter gene assay. Transfection of active CaM kinase II delta and MEKK largely increased the mPer1 promoter activity in NG108-15 cells and NB2A cells, respectively. Deletion and mutation analyses of the mPer1 promoter revealed that 5'-GAGGGG-3' sequence motif near exon 1B, in which several zinc finger proteins seem to bind, was essential for the CaM kinase II-induced activation of the mPer1 promoter. Elimination of GC box near exon 1B reduced the MAP kinase-induced activation. Taken together, CaM kinase II delta and MAP kinase signaling pathways synergistically activate the mPer1 promoter in the suprachiasmatic nucleus.

P1-097 Phosphorylation of Tau to sites found in Alzheimer's disease brain is catalyzed by CaMKII

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Neuronal Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII) is one of the most abundant protein kinases in the brain, and phosphorylates a broad range of substrate proteins. One of its major substrates is cytoskeletal protein, including tubulin, MAP2 and tau. Hyperphosphorylation of tau causes an accumulation of paired helical filaments (PHFs) in Alzheimer's disease. The phosphorylation of tau by CaMKII was investigated using tandem mass spectrometry (MS/MS). Recombinant human tau441 was phosphorylated at Thr212, Ser 214, Ser262, and Ser356 by CaMKII. The phosphorylation of these sites is found in paired helical filament (PHF)-tau. In addition to these sites, Ser131 and Thr135 were phosphorylated by CaMKII. Phosphorylation at Ser131, Thr135, Thr212 and Ser214 by CaMKII has not been reported previously. These results suggested that CaMKII is involved in the phosphorylation of tau in Alzheimer's disease brain.

P1-098 THE EFFECT OF OPHIOBOLIN A, A NOVEL CALMODULIN ANTAGONIST, ON NEUROTRANSMITTER RELEASE FROM PC12 AND RAT CEREBELLAR GRANULE CELLS

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Neurotransmitter is released from synaptic nerve terminals by an exocytosis of synaptic vesicles. Neurotransmitter release is triggered by Ca^{2+} and the identification of Ca^{2+} sensor protein is quite important for molecular mechanisms of presynaptic function. Recent accumulating evidences suggest that calmodulin plays important roles in neurotransmitter release from adrenal chromaffin cells. In order to reveal whether calmodulin has characteristic of the Ca^{2+} sensor for neurotransmitter release, we studied the effect of ophiobolin A, novel calmodulin antagonist. Ophiobolin A prominently suppressed Ca^{2+}-dependent releases of dopamine and acetylcholine from PC12 cells, but not affected glutamate release from cultured cerebellar granule cells. These findings suggest that there exist a difference in Ca^{2+}-sensing mechanism for neurotransmitter release between secretory cells and neurons.

P1-099 Sterol regulatory element-binding protein-2 modulates human brain acyl-CoA hydrolase gene transcription

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Brain has high catalyzing activity of long-chain acyl-CoA hydrolysis. A brain long-chain acyl-CoA hydrolase (BACH) specifically distributes in neurons and is mainly responsible for the catalytic activity in the brain. We analyzed whether transcription of the human BACH gene is modulated by the sterol regulatory element-binding protein-2 (SREBP-2). SREBP-2 is a transcription factor that activates the genes involved in cholesterol biosynthesis. When SREBP-2 gene was transfected into human neuroblastoma cells, transcription of a BACH promoter-luciferase reporter gene was activated. Additional analyses using various BACH gene promoter mutants suggest that a sterol regulatory element (SRE) motif is critical for the SREBP-2 dependent BACH gene promoter activation. Further, a gel shift assay demonstrated that SREBP-2 specifically bound to the SRE motif. These results suggest that BACH gene is involved in both acyl-CoA and cholesterol metabolisms in neurons.

P1-100 Regulation of mitochondrial dynamics and morphology by calcium signaling

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Mitochondria exhibit dynamic movement and morphological changes. We have labeled mitochondria in cultured hippocampal neurons by expressing mitochondria-targeted fluorescent protein and investigated the intracellular signaling that regulates mitochondrial movement and morphology. Glutamate-induced NMDA-dependent Ca^{2+} elevation and calcineurin activation resulted in slow onset of cessation of movement and mitochondrial contraction, which is accompanied by cytochrome c release and neuronal death. High-K+-induced VDCC-dependent Ca^{2+} influx and CaMKI activation led to immediate freezing of movement and mitochondrial division, which protected neuron from excitatory toxicity. These results suggest that mitochondrial dynamics and morphology are regulated by channel specific intracellular signaling and these changes control neuronal survival and death.

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P1-101 Calpain-Mediated Cleavage and Down-Regulation of NMDA Receptors

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Elevation of intracellular Ca^{2+} can lead to the activation of calpain. Here, we examined the effect of calpain-mediated cleavage of NMDA receptors on the channel function in neurons. An application of prolonged NMDA or glutamate significantly reduced whole-cell NMDAR current in both acutely dissociated and cultured neurons. The down-regulation of NMDAR current was blocked by bath application of the membrane-permeable specific calpain inhibitory peptide 11R-CS, and by the selective protease inhibitor ALLN. Injection of the calpain inhibitory peptide CS, but not a control peptide, also eliminated the down-regulation of NMDAR current induced by prolonged NMDA or glutamate treatment. Immunoblotting analysis showed that NR2A subunits of NMDA receptor were markedly degraded in glutamate-treated cultured neurons and calpain inhibitors inhibited the degradation. The results suggest that prolonged activation of NMDA receptors in neurons activates calpain, and activation of calpain in turn down-regulates the function of NMDA receptors.

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P1-102 NYAN1: the tyrosine kinase substrate that connects the NMDA receptor to Akt activation.

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Non-receptor tyrosine kinases of the Src family are closely associated with the NMDA receptor; however the link between the Src family kinases and NMDA receptor-induced signaling events is not clear. In a study of characterizing the Src family-mediated signaling pathway, we have identified a novel substrate for tyrosine kinases: NYAN1.
NYAN1 is a novel protein and apparently does not have functional domains. Northern blot analysis revealed that NYAN1 is specifically expressed in neuron. Furthermore, expression of NYAN1 is high at perinatal period and gradually decreases after postnatal day 14, suggesting that NYAN1 might function in the process of late stage neuronal development such as myelination and synapse formation. NYAN1 is localized at the tip of dendritic spines, and is associated with the NMDA receptor complex. Src-family kinases phosphorylate NYAN1, which activates Akt kinase. Taken together, NYAN1 may regulate PI3K-Akt signaling pathways from NMDA receptor complex and, after all, neuronal survival and neuronal functions.

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P1-103 PI(4,5)P_2 microdomains and the sites of exocytosis

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We investigated the relationship between the spatial organization of PI(4,5)P_2 and exocytotic machineries in PC12 cells. Both PI(4,5)P_2 and syntaxin, a SNARE protein essential for exocytosis, exhibited punctate clusters in isolated plasma membranes, and the number of PI(4,5)P_2 clusters colocalized with LDCVs docked on syntaxin clusters was decreased after occurring exocytosis. Alternatively, exogenously expressed PIP5KI increased the number of PI(4,5)P_2 clusters at the LDCV/syntaxin clusters, and enhanced exocytotic activity by probably increasing in the release sites. Most of syntaxin clusters were not overlapped with thy-1, a specific marker of lipid raft, but there is a distinct population of PI(4,5)P_2 clusters colocalized with thy-1. These results suggest that the PI(4,5)P_2 clusters were formed at two distinct sites, and PI(4,5)P_2 accumulation at the syntaxin cluster is essential for the LDCVs to acquire exocytotic activity.

P1-104 Gamma oscillations enhance temporal precision in CA3 recurrent network

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During different behavioral states, different population activity is present in the brain region, including the hippocampus, producing periodic field potentials in electroencephalography. The oscillatory activity is often characterized by their frequencies, i.e., theta (~5 Hz) and gamma (~40 Hz) bands, and their coherent activities. Here we report that periodic electric fields per se can amplify CA3 recurrent excitation in a frequency-dependent manner, thereby enhancing spike precision. Coherent gamma oscillations are, therefore, not merely a result of synchronized neuron assemblies but can also play an active role in information processing.

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P1-105 Identification of a 32 kDa phosphoprotein in Drosophila brain

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To screen for phosphoproteins that may be involved in learning and memory in Drosophila, protein phosphorylation in brain was examined. Incubation of brain homogenate with [γ-32P]ATP in the presence of cAMP resulted in the phosphorylation of several proteins. The most heavily labeled protein was a 32 kDa protein (pp32). When brain was labeled 32Pi and treated with dopamine or forskolin, incorporation of radioactivity into the pp32 was increased. To determine the function of pp32, the protein was partially purified and characterized. Since the protein was soluble, cytosol fraction obtained from fly head was subjected to column chromatography. After proteins were separated on 2-dimensional gels, the spot corresponding to pp32 was excised and the protein was analyzed by matrix-assisted laser desorption ionization-time of flight (MALDI-TOF) mass spectrometry using peptide mass fingerprinting.

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P1-106 Molecular analyses for cathepsin Z

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Papain-like cysteine proteases, cathepsins have been reported to be associated with various diseases, such as cancer, muscular dystrophy and diabetes. In the nervous system, Cathepsins also participate in pathology of Alzheimer's disease, multiple sclerosis, and stroke. Recently, a new member of the cathepsin family, cathepsin Z, was reported. According to previous reports, cathepsin Z mRNA found to be widely expressed in many tissues and tumors. In another experiment, disruption of homolog of cathepsin Z in C. elegans, cpz-1, suggests that cpz-1 has a role during molting and morphogenesis. Although this report may suggest an essential role in the physiological processes, in vivo function of cathepsin Z in mammals is unclear. As a step in revealing the function of cathepsin Z, we conducted the gene expression analyses of the cathepsin Z mRNA such as in situ hybridization histochemistry and real time quantitative RT-PCR under various conditions. Here, we report the results of these analyses.

P1-107 HuD, a mRNA binding protein, is methylated by CARM1 in PC12cells and it is demethylated during neuronal differentiation by NGF.

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Hu proteins belong to a conserved family of RNA-binding factors that were originally identified in Drosophila as embryonic lethal abnormal vision proteins, and are essential for neuronal development. HuD,one of four Hu proteins, is expressed uniquely in the nervous system. It has been shown to bind to AU-rich elements located in the 3'UTR of short-lived mRNA including c-Fos, Tau, p21, and GAP43. Binding of HuD to these elements is identified to stabilize the mRNA, therefore leading to increase protein levels. Recently, it is indicated that HuR, one of the Hu RNA-binding proteins,is methylated by CARM1.Accordingly, we have initiated experiment of HuD methylation in PC12cells and,we demonstrate HuD is also methylated by CARM1 in PC12cells. Furthermore, we identify that it is demethylated during PC12cells neuronal differentiation by NGF. Here, we will show a role of this demethylation.

P1-108 A localization of protein arginine N-methyltransferase 3 (PRMT3) and its interacting proteins in the mouse development.

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Recent discoveries of protein arginine N-methyltransferase(PRMT)family member raise the possibility that protein methylation is highly regulated in response to external stimuli and plays roles in the physiological processes. Among the PRMT proteins, PRMT3 has some characteristic motifs in the amino terminal tract which follows its active methyltransferase site. One of them is a coiled-coil tract. We performed a two-hybrid screening, using the coiled-coil tract as a bait. We adopted mammalian cell two-hybrid system, because the bait containing the region was reported to cause false positive in yeast two-hybrid system. As a result, we detected two partial cDNA clone, HuD and FEZ1. Immunoprecipitation with anti-PRMT3 could detect both of the immunoreactivities. We further examine the colocalization of PRMT3 and the two interactants in mRNA and protein level.

P1-109 Signalling pathways for gicerin gene experession

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Gicerin is a cell adhesion that consists of five immunoglobulin-like loop structures in an extracellular domain, a single transmembrane region, and a short cytoplasmic tail. Gicerin participates in nuerite extension and structural organization of nervous system. Expression of gicerin is highly observed during development and dramatically decreased after birth. To elucidate the mechanisms of regulation of gicerin expression, we cloned a 14kbp mouse gicerin gene. It contained 8kbp gicerin gene composed of 16 exons and 6kbp upstream region. The gene has a TATA less promoter with several Sp1 and a CRE binding site. On the other hand we found that the expression of gicerin in PC12 cells was regulated by NGF. To characterize its promoter region we examined the promoter activity in PC12 cells by luciferase assay. It showed that NGF-induced transcriptional activity of the gicerin promoter was mediated by CRE. PD98059 significantly attenuated the increase of the promoter activity, suggesting the involvement of MAPK pathway.

P1-110 Effects of nicotine on stress stimulation: *in vitro* experiments

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Cells exposed to a variety of stresses such as heat or ethanol respond by inducing expression of heat shock proteins. Among heat shock proteins, hsp27 is deeply related with thermotolerance, while hsp70 blocks apoptosis by binding Apaf-1.
In this experiment, we studied nicotine's effects on stress-exposed cells using differentiated H19-7 hippocampal neural cells Hsp70 and 27 were assayed by Western blotting, cell survival rate by MTT method.
Hsp27 responded in parallel with hsp70 to various stress stimulation.
Nicotine itself did not cause stress responses in term of hsps, but decreased cell survival rate. When nicotine was applied together with heat stimulation, it reduced heat shock-induced stress responses and partly inhibited the decrease of cell survival rate. Ethanol stimulation also induced expression of hsp27 and 70 as stress responses, in less degree when compared with the cases of heat stimulation. Nevertheless, ethanol stimulation more decreased cell survival rate.

P1-111 Regulation of mixed lineage kinase (MLK) 3 activity by hippocalcin

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Hippocalcin is a member of the neuronal calcium sensors (NCS) family predominantly expressed in the hippocampal pyramidal cells. We searched for hippocalcin-binding proteins and identified one candidate molecule; MLK3. Immunoprecipitation study using anti-hippocalcin antibody displayed the co-precipitation of MLK3 in hippocampal lysate. Co-precipitation analysis using domain specific constructs of MLK3 revealed that hippocalcin bound with C-terminal region of MLK3. Immunocomplex kinase assay displayed that resting MLK3 activity in hippocalcin deficient mice was higher than that in wild type. Then we detected the phosphorylation state of MLK3 because phosphorylation events in MLK3 play crucial roles in its activation process. Immunoblot analysis using anti-phospho-Ser/Thr antibodies revealed that immunoprecipitated MLK3 in hippocalcin deficient mice was higher phosphorylated than that in wild type. These observations suggest that hippocalcin inhibits the MLK3 activation via regulating MLK3 phosphorylation state.

P1-112 Sympathetic adrenoreceptors and intracellular signal transduction molecules in the duct system of the submandibular gland

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Cellular activities of the duct system of the salivary gland are primarily regulated by sympathetic nerve. In rat submandibular glands (SMG), unique duct segments are differentiated from the striated duct; granular convoluted tubules (GCT). Adrenoreceptors and several pivotal molecules associated with the intracellular signal transduction were studied immunohistochemically in rat SMG. In GCT cells containing a large number of granules, immunoreactive signals of cAMP-synthetic enzymes (adenylyl cyclase 4 and 6; AC4, AC6) and cAMP cGMP-hydrolytic enzyme (phosphodiesterase 10A; PDE10A) were mostly co-localized in the same cells but not all cells. In search of the proper cascade activating adenylyl cyclases, β-1 adrenoreceptor showed relatively higher immunoreactivity over GCT segments but not always comparable to those of AC4, AC6 and PDE10A. On the other hand, α-2B adrenoreceptor (Gi-coupled receptor, cAMP modulation) exhibited intense immunoreactivity corresponding to those of AC4, AC6 and PDE10A.

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P1-113 Cloning and characterization of diacylglycerol kinase ι splice variants in rat brain

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Diacylglycerol kinase (DGK) catalyzes phosphorylation of a second messenger DG to phosphatidic acid in cellular signal transduction. In this study we isolated from rat brain cDNA library cDNA clones for a rat DGKι (rDGKι-1). In addition, we found novel splice variants, which contain either the insertion 1 (71 bp) or the insertion 2 (19 bp) or both in the C-terminus portion. Each of the insertions causes a frame shift and the resultant premature stop codons produce two truncated forms (termed rDGKι-2 and ι-3), the former lacking the ankyrin repeats and the latter lacking a part of the catalytic domain and the ankyrin repeats. Truncation of the C-terminus portion clearly exerts effects on the enzymatic activity. These results suggest that the C-terminus-truncated forms of rDGKι-2 and ι-3 might show a dominant negative effect against the intact rDGKι-1 in neurons.

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P1-114 Localization of diacylglycerol kinase β in the brain

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Diacylglycerol (DG) kinase (DGK) is thought to be a regulator of protein kinase C through DG matabolism. Among DGK isozymes so far cloned, DGKβ is shown to be distributed throughout the cytoplasm in a filamentous pattern in the transfected COS-7 cells. However, detailed subcellular localization of this isozyme remains unclear in neurons. In this study, we performed immunohistochemical examination on rat brain with a specific antibody against DGKβ, together with cDNA-transfection into primary cultured neurons. DGKβ-immunoreactivity was observed clearly in the perinuclear area and the processe. In the transfected hippocampal neurons, GFP-DGKβ was predominantly detected as button-like structures in the dendritic processes. These results suggest that DGKβ may be involved in synaptic transmission at postsynaptic site.

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P1-115 Characterization and partial purification of novel Purα binding proteins in mouse brain

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Purα is an abundant protein in the brain and shows a high affinity for a PUR element. We tried to search for Purα-binding proteins (PurBPs) in mouse brain by the overlay assay with GST-Purα as a ligand. Three PurBPs of 35, 38 and 40 kDa were found mostly in the nuclear extract (N. Ext.) of 4 week-old mouse brain and they were not detected by the pretreatment of N.Ext. with trypsin, but not with RNase or DNase. The three PurBPs disappeared by the addition of ssCRE (single stranded cAMP response element) containing the PUR element, but not by ΔGGN ssCRE (deletion of the PUR element from the ssCRE). The PurBPs were abundantly expressed in the brain as Purα. We also determined a region in Purα which is required for the association with the PurBPs by using deletion mutants of Purα. By the HiTrap Q ion-exchange column chromatography we found that the PurBPs behave as neutral proteins.

P1-116 Characterization of Rho guanine-nucleotide exchange factor implicated in peripheral nervous disease

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The Rho GTPases are likely involved in regulation of cytoskeleton in neuronal cells. The physiological functions of Rho GTPase are regulated by guanine nucleotide exchange factors (GEFs), which may play an important role in signal transduction but the detailed mechanisms are poorly understood. Recently, we have identified a new GEF for Rho GTPases from rat brain, which turned out to be the same as a human RhoGEF10. Interestingly, recent work reported that mutation in RhoGEF10 cause slowed nerve-conduction velocities with thinly myelinated fibers ofperipheral nerve. Furthermore, RhoGEF10 has a DH domain but lacks typical PH domain. These observations suggest that RhoGEF10 may play a role in myelin formation in a different way from the conventional RhoGEFs having DH/PH tandem module. In this study, we characterize the expression and biochemical function of RhoGEF10.

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P1-117 Expression of Foxp1 in the mouse striatum

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Developmental process of the central nervous system (CNS) depends on the expression of specific sets of genes at appropriate places and times during the development. The spatio-temporal expression pattern of the genes is regulated by transcription factors, including forkhead transcription factors. First, we examined the expression pattern of the forkhead transcription factor Foxp1 in the adult CNS. Foxp1 was highly expressed in the striatum and moderately in the cerebral cortex, CA1-2 subfields of the hippocampus, and several thalamic nuclei. In the striatum of adult mice, Foxp1 mRNA was detected in a subset of projection neurons, not in interneurons. In addition, the expression of Foxp1 mRNA was observed in the developing basal ganglia with the exception of the globus pallidus. The expression pattern of Foxp1 mRNA suggests that Foxp1 may play a role in the development and formation of a circuit in the basal ganglia, which is involving the matrix neurons.

P1-118 Protein kinase A contributes to the irreversible depolarization following *in vitro* ischemia in rat hippocampal CA1 neurons.

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Intracellular recordings were made from CA1 pyramidal neurons in rat hippocampal tissue slices. Administration of oxygen- and glucose-deprived medium (*in vitro* ischemia) produced a rapid depolarization. When oxygen and glucose were reintroduced immediately after the rapid depolarization, the membrane depolarized further and reached 0 mV (the irreversible depolarization) 5 min after the reintroduction. In the majority of CA1 neurons pretreated with a PKA inhibitor (H89) or adenylyl cyclase inhibitors (SQ22536 and Rp-cAMPS), the membrane was restored to the pre-exposure potential level with a concentration-dependent manner after the reintroduction. Pretreatment with a calmodulin inhibitor (W-7), but not a calmodulin kinase II inhibitor (KN62) or a myosin light chain kinase inhibitor (ML-7), restored the membrane to the pre-exposure level. These results suggest that the activation of PKA via calcium calmodulin complex produces the irreversible depolarization produced by *in vitro* ischemia.

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P1-119 Skin-to-sensory neurons interaction via ATP

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ATP acts as an intercellular messenger in a variety of cells. Here we characterized the Ca^{2+} wave mediated by ATP in cultured keratinocytes (NHEKs) also co-cultured with mouse DRG neurons. When a cell was gently stimulated, an increase in the $[Ca^{2+}]i$ was observed, followed by propagating Ca^{2+} waves in neighboring cells in an ATP- and P2Y2 receptor-dependent fashion. DRG neurons are known to terminate in the basal layer of keratinocytes. In the co-culture of NHEKs and DRG neurons, mechanical stimulation-evoked Ca^{2+} waves in NHEKs evoked a $[Ca^{2+}]i$ rise in adjacent DRG neurons, which was also dependent on extracellular ATP and P2Y2 receptors. Taken together, ATP is a dominant messenger that forms Ca^{2+} waves in NHEKs. In addition, Ca^{2+} waves in NHEKs could produce a $[Ca^{2+}]i$ elevation in DRG neurons, suggesting dynamic cross talk between skin and sensory neurons mediated by extracellular ATP.

P1-120 Facilitatory effect of insulin on adenosine-induced K^+ current response in the follicular cells of *Xenopus* oocyte

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Application of adenosine (Ade) induces K^+-current response in the follicular cells surrounding a *Xenopus* oocyte under voltage clamp. The Ade-induced K^+ current response is produced by activation of the cAMP-dependent protein kinase and by opening of the ATP-sensitive K^+ channel. Application of insulin significantly augmented the Ade-induced response. The effect of insulin on the K^+ current response was reversible during the washout with normal Tyrode. Prior application of Lavendustin A, an inhibitor of protein tyrosine kinase, markedly blocked the effect of insulin on the Ade-induced response. The results suggested that the effect of insulin on the Ade-induced K^+ current response might be regulated by activation of protein tyrosine kinase.

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P1-121 Rnd1 directly binds to FRS2β and provides control mechanism for FGF-induced neurite outgrowth

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Rho family GTPases are central regulators of neuronal morphology. Rnd1 dominantly expresses in the brain, exerts antagonistic effect on RhoA signaling, but molecular systems of this Rnd1 activity in neuronal functions have not yet been elucidated. Here we have demonstrated that Rnd1 stably associates with FRS2β, which is a docking protein of FGFRs and mediates FGF signaling. When FGFR1 is activated, the receptor phosphorylates FRS2β and releases Rnd1 from FRS2β, liberated Rnd1 inhibiting RhoA activity. Furthermore, knock-down of Rnd1 by Rnd1 specific siRNA suppresses the FGF-induced neurite outgrowth of PC12 cells, supporting the notion that FGF-induced PC12 cells differentiation requires reduction of RhoA activity by Rnd1 in a FRS2β phosphorylation-dependent manner. We are now further investigating the molecular mechanism of FRS2-Rnd1 signaling.

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P1-122 Desensitization by GRK4 of the $GABA_B$ receptor-mediated signaling.

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We investigated the involvement of the G protein-coupled receptor kinase 4 (GRK4) in the desensitization of $GABA_B$ receptors ($GABA_BR$) using *Xenopus* oocytes and baby hamster kidney (BHK) cells. Function of $GABA_BR$ was monitored by activation of G protein-activated inwardly rectifying K^+ channels (GIRKs). Distribution of $GABA_BR$ and GRK4 was studied with a confocal microscopy. In *Xenopus* oocytes expressing $GABA_BR$, no GIRK desensitization was observed by repetitive application of the $GABA_BR$ agonist baclofen, whereas significant desensitization was observed in oocytes coexpressing GRK4 with $GABA_BR$. In BHK cells, functional $GABA_BR$ was expressed on cell surface, while GRK4 was diffusely expressed in cytosol. However, once $GABA_BR$ is activated, GRK4 moved to cell surface. These results suggest that GRK4 is recruited to cell surface by activation of the receptor and desensitizes $GABA_BR$.

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P1-123 Receptor tyrosine kinase-related protein RYK regulates cell migration during cortical development

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Mammalian RYK is a tyrosine kinase-related receptor with unknown function. We have previously reported that rat RYK is dominantly expressed in neural progenitor cells and neurons in developing central nervous system. Independently, mouse RYK has shown to bind to EphB2/B3 receptors. Here we show that rat RYK bound to EphB3 *in vitro*, and extracellular leucine-rich motifs play a crucial role for the binding. Over-expression of RYK in MNS-70, a rat neural progenitor cell line, inhibited the cell migration. Furthermore, in the embryonic cortical slice culture, over-expression of RYK retarded the migration of neural progenitor cells out of the ventricular zone to the pial surface. Meanwhile, over-expression of the RYK deletion mutant without the leucine-rich motifs did not affect the cell migration. These results suggest that RYK regulates the migration of neural progenitor cells during the cortical development, possibly through the binding to Eph receptors.

P1-124 Tangential migration of cortical GABAergic neurons along rostrocaudal axis

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Cortical GABAergic interneurons derived from the subpallium migrate ventrodorsally in the cortex. These neurons show multidirectional tangential migration in the marginal zone, suggesting that they spread rostrocaudally within the cortex. We hypothesized that this sort of spread occurs more widely and tested this by examining migration of GABAergic neurons using time-lapse imaging of GAD67-GFP mice cortices and analysis of labeled cells a few days after in vivo DiI focal injection. In horizontal slices, GFP-positive cells migrated rostrally in the subventricular zone. In flat-mount preparations, they migrated tangentially in all directions in the marginal and ventricular zones. DiI-labeled cells spread in all directions. Our results indicate that multidirectional tangential migration of GABAergic neurons occurs in three zones of the cortex, which might contribute to rostrocaudal spread of the interneurons. (Supported by SORST, JST).

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P1-125 P-Rex1, a Rac specific activator - its expression during brain development and involvement in cell migration

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Rho-family GTPases play key roles in cytoskeletal reorganization, contributing to orchestrated changes in neuronal morphology. Here, we report intensive expression analyses of *P-Rex1* gene, which encodes a Rac-specific activator (Rac-GEF), in the developing CNS, showing that this gene is expressed in a variety of cells which include migrating neurons in the intermediate zone of the cerebral cortex. Exogenously expressed P-Rex1 was able to activate Rac in a NGF-dependent manner in PC12 cells, leading to increase of cell movement. Deletion analyses suggested roles of distinct domains of this molecule. Moreover, experiments using a dominant negative form for this molecule showed that endogenous P-Rex1 is involved not only in NGF-dependent cell movement of PC12 cells but also in neuronal migration in the developing cerebral cortex. These findings suggest a variety of roles of P-Rex1 in the nervous system development.

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P1-126 Ectopic expression of neuron-specific K^+-Cl^- cotransporter, KCC2, does not affect neuronal migration in developing cerebral cortex

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GABA is not only an inhibitory neurotransmitter in adult central nervous system, but also thought to be crucial for neuronal migration in developing cerebral cortex. In developing stages, GABA is excitatory by provoking Cl^- efflux through $GABA_A$-receptor channels due to high $[Cl^-]_i$. The functional change of GABA from excitatory to inhibitory is mostly attributable to KCC2, of which expression is developmentally up-regulated in neuronal cells. Here, to explore the effect of KCC2 and/or $[Cl^-]_i$ on neuronal migration, KCC2 was transfected by means of *in utero* electropolation into cells in ventricular zone at E14. The results show that KCC2-expressing cells migrate normally to the cortical plate during stages we examined. These data indicate that KCC2 expression and/or subsequent reduction of $[Cl^-]_i$ might not contribute to neuronal migration.

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P1-127 Immunocytochemical analysis of Drebrin A and PSD-95 in cultured hippocampal neurons

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Our previous study demonstrates that supression of drebrin A expression inhibits synaptic clustering of PSD-95, suggesting that the arrival of Drebrin A to spines precedes the arrival of PSD-95. In this study, a newly generated antibody that selectively recognizes Drebrin A was used to compare the distribution of Drebrin A and PSD-95 in cultured neuron from 7 days in vitro (DIV) to 21 DIV. Immunocytochemical analysis demonstrated that most dendritic spines have both Drebrin A and PSD-95. On the other hand, there were three types of dendritic filopodia: one has neither Drebrin A nor PSD-95, another has Drebrin A but not PSD-95, and the last has both Drebrin A and PSD-95. Our findings suggest that Drebrin A concentrated in dendritic filopodia might initiate spine formation.

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P1-128 Drebrin A accumulates F-actin and PSD-95 into dendritic filopodia of immature hippocampal neurons

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It has been shown that drebrin A expression is necessary for synaptic targeting of F-actin and PSD-95, and for dendritic spine morphogenesis. However, it has not been elucidated whether over-expression of drebrin A promotes spine morphogenesis in immature neurons. Here, we micro-injected GFP-drebrin A expression vectors into hippocampal neurons of low-density cultures at 7DIV. After 48 hours, we fixed the neurons, and analyzed the localization of GFP-drebrin A, F-actin and PSD-95 using fluorescent-microscopy. GFP-drebrin A was accumulated at terminal dendritic branches (megapodia), which lack MAP2 immunostainig. They showed leaf-like shapes, which differed from typical dendritic spines. Within megapodia, F-actin and PSD-95 were accumulated in parallel with the GFP-drebrin A level. These data suggests that drebrin A accumulates F-actin and PSD-95 into dendritic filopodia, and forms megapodia in immature neurons, but it does not promote spine morphogenesis.

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P1-129 Localization of drebrin-binding protein 1 in dendritic spine depends on its C-terminal region

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Drebrin-binding protein 1 (DBP1) is a novel molecule which is isolated in yeast two hybrid screen by using drebrin as bait. In rat primary cultured hippocampal neurons, DBP1 was localized primarily in nucleus, and lesser amounts in soma and dendrite. DBP1 represents dot like immunofluorescence pattern along dendrites, suggesting its spine localization. In this study, we examined the subcellular localization of several mutated DBP1 constructs, and identified a functional domain that regulates localization of DBP1 into dendritic spines. This region was named spine localization domain (SLD). When SLD itself was over-expressed in cultured neuron, it was localized in spines; in addition, it was not concentrated in nucleus although it was a short fragment of 25kDa. These results suggest that SLD governs localization of DBP1 at dendritic spine and extranuclear area.

P1-130 Identification of membrane binding domain of kinectin

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Background: Kinectin had been found as kinesin binding protein on endoplasmic reticulum (ER). Membrane insertion portion was identified in the end of N-terminus but specific localization domain was obscure and thought to be require the half of the molecule. Methods: We newly cloned full length kinectin from human brain. Four truncated constructs following N-terminus was made connecting with pEGFP to visualize in Cos7 cells. Distribution and movement of organelles were analyzed in the GFP positive cells. Results: 186 amino acids (1-186kinectin-GFP) were enough for localize in reticular pattern appearing ER, whereas 1-81 construct distributed evenly in the cytoplasm. The reticular pattern was identical DiOC6(3) as ER marker except 1-186kinectin-GFP did not show mitochondria. Distribution and movement of organelles were the same between GFP positive cells and negative cells. Conclusion: Membrane binding of kinectin requires amino acid number 87-186 to localize on ER.

P1-131 Tubulin and CRMP-2 complex is transported via conventional kinesin

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The transport of tubulin and microtubules in growing axon is essential for axonal growth and maintenance. However, the molecular mechanism of how tubulin and microtubules link to motor proteins is not yet clear. Collapsin response mediator protein-2 (CRMP-2) is localized at the distal part of growing axons in primary hippocampal neurons and plays a critical role in axon differentiation through interaction with the tubulin dimer. I here show that CRMP-2 regulates tubulin transport by linking tubulin and conventional kinesin. The C-terminal region of CRMP-2 directly binds to the tetratricopeptide repeat domain of kinesin light chain (KLC). Soluble tubulin binds to the middle of CRMP-2 and forms a trimeric complex with CRMP-2/KLC. Furthermore, the movement of GFP-tubulin in the photobleached area is significantly weakened by knockdown of KLC or CRMP-2. These results indicate that the CRMP-2/kinesin-I complex regulates soluble tubulin transport to the distal part of the growing axon.

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P1-132 Microtubules provide directional cues for polarized axonal transport through interaction with kinesin motor head

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Post-Golgi carriers of various newly synthesized axonal membrane proteins, which possess kinesin (KIF5)-driven highly processive motility, were transported from the TGN directly to axons. We found that KIF5 has a preference to the microtubules in the initial segment of axon. Low dose paclitaxel treatment caused missorting of KIF5 as well as axonal membrane proteins to the tips of dendrites. Microtubules in the initial segment of axons showed a remarkably high affinity to EB1-YFP, which was known to bind the tips of growing microtubules. These findings revealed unique features of the microtubule-cytoskeletons in the initial segment, and suggested that they provide directional information for polarized axonal transport.

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P1-133 Anterograde and Retrograde Intracellular Trafficking of Fluorescent Cellular Prion Protein

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To investigate the microtubules-associated intracellular trafficking of the cellular prion protein (PrP^C), we performed a real-time imaging of fluorescent PrP^C (GFP-PrP^C) in living cells. Such GFP-PrP^C exhibited an anterograde movement towards the direction of plasma membranes at a speed of 140-180 nm/sec, and a retrograde movement inwardly at a speed of 1.0-1.2 mm/sec. Mapping of the interacting domains of PrP^C identified amino acid residues indispensable for interactions with kinesin family (KIF4): NH2-terminal mouse (Mo) residues 53-91 and dynein: NH2-terminal Mo residues 23-33, respectively. Our findings argue that the discrete N-terminal amino acid residues are indispensable for the anterograde and retrograde intracellular movements of PrP^C.

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P1-134 The Conserved Ser/Thr Kinase UNC51 Functions with Trio and Rac to Regulate Neural Development and Axonal Transport in *Drosophila*.

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Construction of neural networks needs regulated formation of axons and their correct guidance. Despite intensive researches in the past years, mechanisms of axonal development still remain unclear. We have identified the *Drosophila* homolog of *unc51*, which encodes a Ser/Thr kinase and is required for axonal formation in *C. elegans* and mouse. We show that *unc51* is crucial for axonal elongation, guidance, and fasciculation in the *Drosophila* CNS, in which UNC51 interacts with Trio and Rac to regulate axonal development. Moreover, we show that UNC51 plays an essential role in axonal transport and coordination of animal locomotion. Electron microscopic studies suggest that UNC51 may be localized with cytoplasmic vesicles possibly via interaction with other proteins that are crucial for vesicle transport and cytoskeletal organization.

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P1-135 Axon-elongation specific role of microtubule-severing protein, katanin, in cultured neurons

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Process elongation of neurons requires supply of microtubules. One mechanism is that detachment of microtubules from centrosome provides free microtubules that can be transported into processes. Indeed, inhibition of microtubule-severing protein, katanin, has been shown to inhibit process elongation. Here we examined the difference in the roles of katanin on axons and dendrites using RNA interference. Fetal mice cortical neurons were transfected with siRNA for catalytic subunit of katanin and GFP-cDNA by electropolation. After 1 to 2 days' culture, axons were immuno-stained. GFP-positive neurons formed axons, indicating that katanin activity is not critical for axon formation. Length of axons, however, were reduced. Reduction was axon-specific: it was not observed in the length of dendrites. These data indicate that rapid elongation of axons requires free microtubules, but that elongation of dendrites does not require them.

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P1-136 The planar cell polarity (PCP) -related genes are expressed in the CNS.

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Recently, genes controlling the PCP have been studied in *Drosophila*. Although a growing number of PCP-related genes have been identified in both invertebrates and vertebrates, it is not known whether the same genetic programs operate in the developing CNS. To gain insights into the roles played by vertebrate PCP-related genes, we have cloned mouse homologues of the Xenopus Daam genes, which are an essential component of the PCP pathway and the convergent extension movement of cells regulated by Rho signals. Here we report precise expression patterns of Daam1, Daam2 in the central nervous tissues. Expression patterns of these genes suggest that Daam1, Daam2 and PCP-related genes may control the pivotal steps of the polarized division, differentiation and movement. To explore the functional roles of PCP-related genes, we use in utero electroporation assay. Our date suggest that the PCP-related genes regulate migration of neural progenitor cells and axonal projection during CNS development.

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P1-137 Regulation of neurofilament motility along microtubules by phosphorylated and dephosphorylated MAPs

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The translocation of phosphorylated (p-) and dephosphorylated (dp-) neurofilaments (NFs) along microtubules (MTs) was analyzed *in vitro* in the presence of p-/dp-MAP2 or p-/dp-Tau bound either to MTs or to NFs. In the absence of MAPs, dephosphorylation of NFs resulted in an increase in the amount of MT-bound NFs and the percentage of moving NFs. The presence of p-MAPs affected the binding and motility of NFs along MTs, with a stronger inhibition effect of p-MAP2 as compared to p-Tau. In particular, the binding and motion of dp-NFs were totally inhibited by p-MAP2. The dp-MAPs showed similar inhibitory effects, but much milder than those of p-MAPs. These results provide evidence that both dynamic and static MT-NF interactions are regulated through the phosphorylation of NF and MAPs.

P1-138 DNA microarray analysis of gene expression during demyelination

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Overexpression of the proteolipid protein gene causes unique demyelinating disorder in mice, in which normal-appearing myelin forms early, followed by chronic demyelination. We demonstrated that remyelination is severely affected but is not caused by the deprivation of oligodendrocyte progenitors. They were present even at a higher number in demyelinated mice than in wild-type controls, suggesting that their differentiation or maturation is affected due to a change in microenvironment. To identify the factors effecting differentiation/maturation, we utilized microarry to search for genes that are differently expressed between the wild-type and Plp/- mice at 2, 4, 6 and 8 months of age. The results showed that expression of many genes, including cystatin F and cathepsin B, H, L, was higher in Plp/- mice at 4 and 6 months age than those in wild-type mice. These results suggest that an active process including synthesis of new proteins causes the changes in a microenvironment.

P1-139 FcRγ/Fyn participate in the mouse brain during cuprizone-induced demyelination and remyelination

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Inclusion of cuprizon <bis-cyclohexanone oxaldihydrazone> in the diet of mice produces a massive demyelination, and removal of cuprizone permits the study of remyelination. Previously we have shown that the FcRγ expressed in OPCs is the trigger of the Fyn signaling during myelination. To investigate the involvement of FcRγ and Fyn in the process of demyelination and or remyelination induced by cuprizone, we used FcRγ-Fyn doubly deficient mice. As usual, MBP deficit was 20-30% by five weeks of exposure in the cuprizone-treated normal animals. however, level of MBP did not changes in the mutant mice. Furhermore, removal of cuprizone from the diet, there were no changes in their MBP lebels of the mutant mice. These results suggest that FcRγ and Fyn have pivotal roles for cuprizone-induced demyelination and remyelination. Acknowledgement: We thank Dr.T. Takai and Dr.T. Yagi for providing materials.

P1-140 Radial glial cells, astrocyte and ependymal cells express alpha-actinin-1

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Central nervous system (CNS) consists of many types of cells; neurons and glial cells. Most (if not all) of these cells are derived from radial glial cells, which are transiently present in embryonic CNS. To study the lineage of radial glial cells in the CNS, we screened for several monoclonal antibodies that stain radial glial cells immunohistochemically. We found one antibody that labels radial glial cells at embryonic day 15.5 (E15.5) in the spinal cord, and astrocyte and ependymal cells at adult stage. This antibody did not label oligodendrocyte or neurons, and recognized 100 kDa protein by western blotting. We identified the antigen by expression cloning and found that it was alpha-actinin-1. These results suggest that ependymal cells are drived from radial glial cells.

P1-141 Extracellular matricies at the node of Ranvier in the central nervous system.

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Myelinated axons in the central and peripheral nervous systems are organized into distinct domains with glial cell. These domains, which consist of the node of Ranvier and the paranodal and juxtaparanodal regions and the internode, ensure rapid propagation of the action potential along myelinated fibers. The hyaluronan binding chondroitin sulfate proteoglycans, called lecticans are the abundant extracellular matrix (ECM) molecules in the brain. The link proteins are also known to be coordinately present in the brain. Brain link protein-1 (Brall/Hapln2) is colocalized with versican V2, a lectican member, at the node of Ranvier in the CNS. In this study, we have generated mice deficient in the expression of Brall/Hapln2 via homologous recombination. In Brall-deficient mice, versican V2 is barely detectable at the node of Ranvier. Our results suggest that Brall plays an essential role of stabilizing extracellular matricies at the node of Ranvier and is partially concerned with conduction velocity in the CNS.

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P1-142 Effect of lipoproteins on rat cultured microglia: Inhibition of phagocytosis by high-density lipoprotein fraction

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Hyperactivation of microglia probably causes the neuronal degeneration in various CNS diseases. Epidemiological analysis revealed that the incidence of Alzheimer disease is positively correlated to the serum level of LDL and negatively to that of HDL. We examined the effects of lipoproteins on cultured microglia from newborn rat brains. The fractions of lipoprotein, VLDL, LDL, and HDL, were freshly prepared from adult rat serum by Superose 6 filtration chromatography. Addition of HDL in the medium reduced the cell number, assessed by CyQUANT, whereas the others showed no effect. The total mitochondrial activity, assayed by WST-8, decreased by the addition of HDL, but slightly increased by the addition of LDL. Activity of phagocytosis, evaluated by microbeads (1 μm ϕ CM-FluoSpheres) uptake, decreased in the presence of HDL. These results suggest that lipoproteins may play roles for the regulation of microglial activation in the pathological CNS.

P1-143 Neuroprotective effect of TNF released from $P2X_7$ receptor-activated microglia

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Although TNF is a major mediator of inflammation and apoptosis as well as the pathogenesis of neurodegenerative diseases, recent evidence shows that TNF is also implicated in neuroprotection. We previously reported that ATP induces TNF release via $P2X_7$ receptor in rat microglia. However the roles of $P2X_7$-stimulated microglia and TNF have been remained unclear. In this study, we examined the effects of $P2X_7$-activated microglia for neurons using neuron-microglia co-culture system. Glutamate (100 μM) caused neuronal cell death. However, pretreatment of microglia with BzATP, a $P2X_7$ receptor agonist, for 24 h significantly prevented the neurotoxicity. Moreover anti-TNF antibody, TAPI, a TACE inhibitor, and Brilliant Blue G, a $P2X_7$ receptor antagonist, all remarkably suppressed such neuroprotective effects. These results indicate that $P2X_7$-activated microglia might protect neurons through releasing TNF.

P1-144 Generation of Iba1-EGFP transgenic mouse; Visualization of microglia

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Microglia is considered to play important roles in not only injured tissue repairing but also regulation of neuronal activity. To further study the function of microglia, visualization of the cells is essential. Therefore, we generated new transgenic mice with GFP expression under the control of a promoter of ionized calcium binding adaptor molecule 1 (Iba1), which is selectively expressed in microglia/microphage. In the adult brain of the Iba1-EGFP transgenic mice, all of the GFP-positive cells were microglia judging from the immunoreactivity for anti-Iba1 antibody. In the early developing stage, the GFP-positive cells were found in yolk sac, brain rudiment, spinal cord, optic placode, liver, webfoot, and vessel walls. The Iba1-EGFP transgenic mice permit us live observation under a fluorescence microscope, and provide a useful tool for studying *in vivo* function of microglia.

P1-145 Development of oligodendroglial progenitor cells in the central nervous system of rats

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The development of oligodendrocytes was investigated by the expression of 5alpha-reductase (5aR) or 4F2 in the brain and spinal cord of rats. Using the antibody against 5aR type 1, the immunoreactive cells were widely distributed in the central nervous system through neonatal and adult age, and most of the immunoreactive cells were shown to be oligodendrocytes and some were astrocytes. In embryonal period, many immunoreactive cells were widely distributed, and also in the ventricular and subventricular zone. In embryonal day 8 to 9, the immunoreactive cells were located in the neural tube that consisted of a single to a couple of neuroepithelial layers. 4F2, a monoclonal antibody specific to oligodendrocytes, also localized the immunoreactive cells, the distribution of which was similar to that of 5aR from early in the embryonal period through adult age.

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P1-146 Roles for p21 $^{Cip1/WAF1}$ during oligodendrocyte differentiation

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p21 and p27 are Cip/Kip family members of cell cycle-dependent kinase inhibitors. It has been reported that they have non-overlapping functions during the differentiation of oligodendrocytes. While $p27^{-/-}$ cells cannot withdraw from the cell cycle in an appropriate manner, $p21^{-/-}$ cells can efficiently exit the cell cycle but cannot differentiate properly. We will discuss the mechanism that p21 affects the differentiation process of oligodendrocytes.

P1-147 Galectin-1 induces astrocyte differentiation, which leads to production of brain-derived neurotrophic factor

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Brain-derived neurotrophic factor (BDNF) is a neuroprotective polypeptide that is thought to be responsible for neuron proliferation, differentiation, and survival. An agent that enhances production of BDNF is expected to be useful for the treatment of neurodegenerative diseases. Here we report that galectin-1, a member of the family of β-galactoside binding proteins, induces astrocyte differentiation and strongly inhibits astrocyte proliferation, and then the differentiated astrocytes greatly enhance their production of BDNF. Induction of astrocyte differentiation and BDNF production by an endogenous mammalian lectin may be a new mechanism for preventing neuronal loss after injury.

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P1-148 Predominant expression of cystathionine β-synthase (CBS) in the radial glia/astrocyte cell lineage and its relationship to neurological disorders

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The deficiency of CBS gene leads to homocystinuria, an inherited human disease characterized by mental retardation, seizures, skeletal abnormalities and vascular disorders. We examined the regional and cellular localization of CBS in adult and developing mouse CNS. Immunocytochemical studies showed that CBS is predominantly expressed in Bergman glia and in astrocyte throughout the CNS. At early developmental stage, CBS was expressed in neuroepithelial cells in ventricular zone, whereas its high expression was transmitted to radial glial cells and then to astrocytes during the late embryonic and neonatal periods. Moreover, CBS was significantly increased in reactive astrocytes in hippocampus after kainate-induced seizure. These results suggest that CBS play a critical role for the development and repair of the CNS, and also radial glia and astrocyte might be a new therapeutic target for neurological disorders associated with abnormal homocysteine metabolism.

P1-149 Serofendic acid promotes stellation induced by cAMP and cGMP analogs of cultured cortical astrocyte

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We have previously reported that serofendic acid prevented glutamate neurotoxicity in cultured neurons. In the present study we investigated the effect of serofendic acid on primary cultures of cortical astrocytes. Cultured astrocytes ordinarily exhibited flat, polygonal shape, and changed into stellate morphology with several processes in a concentration-dependent manner following exposed to dibutyryl cyclic AMP (dbcAMP), a membrane-permeable cyclic AMP analog and 8-Br-cGMP, a membrane-permeable cGMP analog. Both dbcAMP- and 8-Br-cGMP-induced stellation was significantly accelerated by serofendic acid. DbcAMP-induced stellation and promotion of stellation evoked by serofendic acid were partially inhibited by KT5720, a specific protein kinase A (PKA) inhibitor. These results indicate that serofendic acid promotes dbcAMP- and 8-Br-cGMP-induced stellation and promotional effect of dbcAMP-induced stellation is mediated at least partly by the regulation of PKA activity.

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P1-150 Effects of hydroxy-polychlorinated biphenyls (OH-PCBs) on thyroid-hormone-dependent expression of GLAST, Bergmann glial specific glutamate transporter, in cerebellar astrocytes

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Thyroid hormone (TH) is important for normal brain development. We reported an essential role of TH in the development of mouse Purkinje cells (Develop. Brain Res. 137:55, 2002). TH regulates gene expressions via TH receptors (TR) in neurons, but functional TR has not been identified in astrocytes. We examined the TH-effects on cerebellar astrocytes in serum-free culture. Western blot analysis showed a promoted expression of GLAST by TH. In the presence of TH, glutamate-induced Ca^{2+} uptakes both in astrocytes and neurons were more rapid and greater than those in the absence of TH observed by calcium imaging. Furthermore, low dose of OH-PCBs affected these TH activities. These results indicate that TH regulates functional expressions of several glutamate-related molecules including GLAST in cerebellum via direct or indirect manner.

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P1-151 The mechanisms underlying the inhibitory effect of estrogens on the L-glu uptake by astrocytes

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Recently we discovered that estrogens inhibited L-glu uptake by cultured astrocytes via membrane ERα (mERα). Here we show further evidences indicating that the effect is mediated by mERα and the signal transduction pathway mediating the effect. Western blotting of biotinylated membrane proteins showed the distribution of ERα on the plasma membrane. ERα-specific antibody inhibited the effects of β-estradiol (E2) and E2 conjugated with BSA (E2BSA). E2BSA increased Km of GLAST without affecting Vmax and did not cause the internalization, suggesting the modification of GLAST activity. The effect of E2BSA was significantly inhibited by LY294002, wortmannin, Akt inhibitor, LNA and LNAME. Western blotting of pAkt and quantification of NO indicated the existence of the following signal transduction pathway: mERα>PI3K activation>Akt activation>NO generation>down-regulation of GLAST.

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P1-152 Expression of glutamine transporter in cultured astrocytes

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The system A transporter GlnT (SAT1) has been proposed to mediate the transport of extracellular glutamine (Gln) into neurons to fuel the excitatory neurotransmitter glutamate (Glu) in the mammalian central nervous system. In this study, we have attempted to elucidate possible functional expression of GlnT in cultured astrocytes. Expression of mRNA was seen for GlnT in cultured astrocytes prepared from neocortex, hippocampus and cerebellum of rat brain, in addition to microglia and cerebellar neurons. [3H]Gln was accumulated in a temperature-dependent manner in astrocytes and neurons, with a plateau within 30 min at 37°C. The accumulation of [3H]Gln was significantly inhibited by the inhibitor of system A neutral amino acid transporter (MeAIB) to a lesser extent in astrocytes than in neurons. These results suggest that GlnT may be expressed in cultured astrocytes, in addition to neurons, with unidentified functions in mechanisms associated with the Gln/Glu cycle for recycling of Glu in the brain.

P1-153 Polyamine toxicity in cultured rat brain microglia and astrocytes

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We have previously reported that in the presence of fetal calf serum both spermidine and spermine (SPM) induce dose-dependent cell death in cultured rat brain microglia via an apoptotic process, with higher vulnerability than in cultured astrocytes. In microglia, the toxicity of SPM was not only prevented by aminoguanidine, which is an inhibitor of amine oxidase responsible for conversion of polyamines to aminoaldehyde and hydrogen peroxide to spontaneously yield the aldehyde acrolein, but also attenuated by different antioxidants; including glutathione, cysteine and N-acetylcysteine. In fact, acrolein was much more cytotoxic to microglia than to astrocytes. The endogenous level of glutathione was significantly higher in astrocytes than in microglia. These data suggest that particular polyamines would balance the activities between microglia and astrocytes through production of the cytotoxic aldehyde metabolite acrolein.

P1-154 Sema 4D deficiency up-regulates myelination in mouse brain

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Oligodentrocyte (OL) has an essential role in axon guidance and myelination. We already have reported that Sema4D (S4D) is transiently expressed in some OLs after cerebral ischemia. In this study we examined a possible role of S4D in maturation of OLs using S4D knock out (KO) and wild-type (WT) mice either aged 14 days or 60 days. At 14 days, WT mice showed a poor myelinated structure in the cerebral cortex, but KO mice already developed rich myelination with increased number of MAG+ premature OLs. AT 60 days, KO mice still showed increased myelination and MBP content. O4+ pro-OLs express S4D, but the number of PDGFRa+ OPC is not different between the two strains. These findings suggest that S4D is involved in myelination via the maturation of OLs from pro-OLs to myelinated OLs.

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P1-155 The expression pattern of netrin-1, Sema3A and their receptors in the chick optic nerve

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The migration of oligodendrocyte precursor cells (OPCs) is a critical early step for appropriate myelination to occur throughout the CNS. This migration requires guidance molecules, such as netrin-1 and semaphorin 3A (Sema3A). However, it is not clear that all OPCs are guided by the same cues. We investigated the expression pattern of netrin-1, Sema3A and their receptors in the chick ON by in situ hybridization. At E6, both netrin-1 and Sema3A were expressed in the ventral midline of the 3rd ventricle, where OPCs originate. Netrin-1 was also expressed in the temporal side of ON. Moreover, neogenin, a netrin-1 receptor, and neuropilin1, a Sema3A receptor, were expressed similarly, and some of them were co-expressed with Olig2, marker of OPCs. At E7, Olig2+ OPCs localized in the nasal side of ON, which is complementary to netrin-1 expressing areas. These results suggest that netrin-1 and sema3A force OPCs to enter the ON and that netrin-1 repel OPC to nasal side of ON.

P1-156 In vivo analysis of the conduction velocity of the central neural pathways in the PLP transgenic mice

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Previously we reported the system to measure the conduction velocities (CVs) and relative refractory periods (RRPs) of identified central pathways in mice. We studied the effects of demyelination on the CVs of dorsal column (DC), vestibulospinal / reticulospinal tracts (VRST) and pyramidal tract (PT) in the proteolipid protein(PLP) transgenic mice (PLP Tg) of 2,5 and 8 months old. CVs of DC, VRST and PT were reduced compared to those of wild type (Wt) mice with aging. RRPs was delayed in PLP Tg, compared with Wt. Surprisingly, CVs of all three pathways were significantly reduced in 2-month-old PLP Tg, whose myelin and motor functions were normal. Immunohistochemical study revealed that Nav1.6 was expressed at the node of Ranvier in Wt, while Nav1.2 remained at the node in the age-matched PLP Tg. This finding may correlate with the CV decrement in 2 month old PLP Tg mice.

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P1-157 The expression of SNARE proteins in undifferentiated or differentiated astrocytes

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Several evidences have suggested that astrocytes can directly modulate synaptic transmission. We investigated expression of some SNARE proteins, synaptobrevin and syntaxin, 3 or 7 days after drug application in undifferentiated or differentiated astrocytes by Western blotting and immunocytochemistry. Astrocytes were differentiated by application of a cyclic AMP (cAMP) analog, dibutyryl cAMP (dBcAMP). Although the expression of synaptobrevin increased with prolonged culture time in both undifferentiated and differentiated astrocytes, that was suppressed by application of only a protein kinase A (PKA) inhibitor, H-89. These results suggest that the expression of synaptobrevin is dependent on PKA activity. The syntaxin was detected in both undifferentiated and differentiated astrocytes by immunocytochemistry. These results show that both undifferentiated and differentiated astrocytes express SNARE proteins.

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P1-158 Binding of soluble myelin-associated glycoprotein to specific gangliosides induces the association of p75 NTR to lipid rafts and signal transduction

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Myelin-associated glycoprotein (MAG) is a potent inhibitor of neurite outgrowth from a variety of neurons. Here we show that gangliosides, GT1b and GD1a, as well as Nogo receptor, are functional binding partners for soluble MAG-Fc. Postnatal cerebellar neurons from mice deficient in the GalNacT gene are insensitive to MAG with regard to neurite outgrowth and lack in the activation of RhoA. MAG-Fc or the antibody to GT1b and GD1a elicites recruitment of p75NTR to lipid rafts, specialized microdomain for signal transduction. Disruption of lipid rafts results in abolishment of inhibitory effects of MAG-Fc and the Nogo peptide. These findings establish gangliosides as functional binding partner for soluble MAG. Gangliosides may play a role in translocation of p75NTR to lipid rafts for initiation of the signal transduction.

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P1-159 Distribution and molecular interaction of contactin associated protein (Caspr) 4 in human nervous system.

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Contactin associated protein (Caspr) 4 is a neural cell recognition molecule identified as a member of Caspr family in human. Caspr1 and Caspr2 are known as essential molecules for formation and function of septate-like paranodal axoglial junctions at both central and peripheral myelins, but other members of Caspr3, 4 and 5 have not been characterized yet. Most of Caspr family molecules except Caspr1 contain putative PDZ-binding domains at their c-terminals. To understand the functions of Caspr family molecules, we at first approached to identify proteins associated with Caspr4 using a yeast two-hybrid system in this study. Using the intracellular region of Caspr4 as bait, 46 candidates were identified to interact with Caspr4. Further studies are underway to confirm whether the identified clones interact with Caspr4 in vivo and whether they play a role in Caspr4 intracellular signaling.

P1-160 *Phgdh* gene promoter directs transgene expression to neuroepithelial stem cells and radial glia *in vivo*

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3-Phosphoglycerate dehydrogenase (Phgdh) is a necessary enzyme for L-serine biosynthesis. In developing and mature brain, Phgdh expression is highly regulated in a cell lineage-specific manner, mainly in neuroepithelial stem cells, radial glia, and astrocytes. To gain an insight into the regulatory mechanism, we generated transgenic mice bearing an 8 kb *Phgdh*-DsRed reporter construct. The transgene expression was detected in neuroepithelial stem cell/radial glia in embryonic brain, and was transmitted to Phgdh-positive cells in the subventricular zone, but not to astrocytes in the adult brain. These observations indicate that additional regulatory elements are required for expression of *Phgdh* in astrocytes.

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P1-161 ATPγS induces MCP-1 expression in organotypic cortico-striatal slice cultures

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Monocyte chemoattractant protein-1 (MCP-1, CCL2) is a well characterized CC chemokine involved in the pathophysiology of various brain injury. In this study, we investigated the effects of ATPγS on MCP-1 production in organotypic cortico-striatal slice cultures. ATPγS induced transient MCP-1 expression at the levels of both mRNA and protein in a concentration dependent manner. MCP-1 immunoreactivity was mainly observed in astrocytes. ATPγS-induced MCP-1 production and release were completely inhibited by PPADS, but not suramin. This release was partially inhibited by PD98059 or SP600125, and significantly enhanced by SB203580. These results suggest that ATPγS induces MCP-1 production and release via PPADS-sensitive, suramin-insensitive P2 purinoceptor(s), and that ERK and JNK MAP kinases positively regulate this induction, while p38 MAP kinase suppresses it.

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P1-162 Annexin III expression is induced in microglia in response to motor nerve injury

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To elucidate the molecular mechanism underlying peripheral nerve regeneration, we attempted to identify proteins that are differentially expressed after peripheral nerve injury by using a proteomic strategy. The protein expression profiles in normal and axotomized hypoglossal nuclei were compared by two dimensional polyacrylamide gel electrophoresis methods coupled with mass spectrometry. This approach revealed several candidate proteins, which were up-regurated in injured motor nucleus after axotomy. Amongst those molecules we focused on one candidate, annexin III, because the up-regulation was confirmed by Western blot analysis with its specific antibody. All Annexin III positive cells simultaneously exhibited OX-42 immunoreactivity, suggesting the annexin III positive cells are microglial cells. These results suggest that annexin III may be a novel signal mediator within the activated microglial cells or between the micloglia and nerve injured motor neurons.

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P1-163 Glial cell proliferation induced by the programmed cell death and neural injury in the *Drosophila* adult brain

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For further understanding of the role of cell proliferation in the mature nervous system, we are examining the adult brain of *Drosophila melanogaster*. We reported last year that we found glial cell proliferation around the antennal nerve between 0 and 6 days after eclosion, which was triggered not only by programmed cell death of neurons but also by artificial lesion of the antennal nerve.
To investigate whether glial cells in other brain regions have similar potential to proliferate, we fed flies with BrdU (a marker for DNA synthesis) and stuck a needle to the dorsal side of the brain. After one day of injury, we found the cells that were both BrdU- and REPO- (a glial cell marker) positive near the injured region. These results indicated that a part of glial cells in various regions of the adult brain have the ability to proliferate in response to neural lesion. Such response of glial cells against programmed cell death and injury of neurons may have an important role for brain homeostasis.

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P1-164 Functional analysis of astocytes in lesion site using knockout mouse

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The expression of tenascin, one of the astroglia-derived extracellular matrix proteins, is up-regulated in sites of injured brain of the mouse. In vitro study showed neurite outgrowth was inhibited when neurons were co-cultured with tenascin-expressing astrocytes. In light of these observations it is propagated that tenascin-expressing astrocytes could be involved in the inhibition of CNS regeneration after brain injury. To investigate the role of tenascin played in the regeneration process of the CNS, astrocytes of the tenascin-deficient mouse were studied after a stab wound was made to the brain. The blood-brain barrier permeability in these mice was also analyzed during the wound-healing process.

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P1-165 Effects of GLP-1 on cation channels in cultured microglias from the rat's cortex

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We reported that endogenous glucagon-like peptide-1 (GLP-1) mediates or exacerbates β-amyloid protein-induced amnesia, apoptosis and impairment of LTP in the rat's brain, and that, in the other hand, exogenous GLP-1 improved β-amyloid toxicities. Since microglias play a role in progression of Alzheimer's disease, we examined electrophysiological profiles of GLP-1 on cultured microglias derived from the E18 rat's cortex. Whole-cell patch clamp recording showed that 50 nM GLP-1 suppressed a particular type of K^+ currents. Cell-attached patch clamp recording with the holding potential of +30 mV showed that GLP-1 induced cation channel openings which were sensitive to depletion of Na^+ and Ca^{2+} as well as an inhibitor of cAMP synthesis. Furthermore, GLP-1 facilitated morphological changes from a ramified form into an amoeboid form. Consequently, GLP-1 may modify a functional role of microglias in brain disorders.

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P1-166 PKC modulation of glutamate-induced astrocyte calcium oscillation

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In our previous study, growth factors (EGF and bFGF) treatment altered astrocyte calcium response to neurotransmitters, including glutamate and ATP, from transient to oscillatory, and the glutamate response was pharmacologically attributed to mGluR5. Since mGluR5 is known to be phosphorylated by PKC and to change its calcium increase pattern, we have examined the effects of a PKC activator (PMA), as well as an inhibitor (GF129203X; GFX) on astrocyte calcium responses. As results, glutamate and ATP responses were found to become smaller and transient by PMA and larger and sustained by GFX, however histamine response, which is transient even in the presence of growth factors, was less affected. Although the properties of calcium store have been shown to be affected by the growth factors, they were not affected by PKC modulation. These results suggest that PKC plays an important role in astrocyte calcium oscillation in different manner from growth factors.

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P1-167 Studies on drug dependence (Rept. 396): Involvement of NMDA receptor associated with PKC activation in methamphetamine-activated astrocyte

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The present study was designed to clarify the role of astrocyte underlying neuronal plasticity induced by psychostimulants. Three days in vitro treatment with methamphetamine (METH) into mouse cortical neuron/glia cocultures caused the activation of astrocytes as detected by a stellate morphology and an increase in level of glial fibrillary acid protein (GFAP). Furthermore, this event was blocked by cotreatment with a NR2B-containing NMDA receptor antagonist ifenprodil or a PKC inhibitor chelerythrine. It is of interest to note that glutamate-induced astrocytic activation in neuron/glia cocultures was also reversed by co-treatment with either ifenprodil or chelerythrine. These findings suggest that exposure of METH to neuron/glia cocultures leads to the release of glutamate and the stimulation of NR2B-containing NMDA receptor associated with activated PKC, and in turn activated astrocyte.

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P1-168 Tissue distribution and age-related changes of human and rat GMFB and GMFG

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We developed sensitive and specific two-site enzyme immunoassays (EIA) for glia maturation factor beta (GMFB) and gamma (GMFG) using specific antibodies. These assay systems enabled us to identify GMFB and GMFG (GMFs) in human and rat samples and they were used to investigate the tissue distribution and serum concentrations of GMFs. In the rat, relatively high levels of GMFB were found in the central nervous system, except for the spinal cord, and in thymus and colon. Higher levels of GMFG were found in thymus, spleen and colon. In the rat, the maximum serum concentration of GMFG was at 4 weeks of age. The concentration of GMFB in serum did not change significantly with age. The present EIA systems are sufficiently sensitive for studying GMFs in human and rat organs.

P1-169 Expression of GMFB after cortical cryogenic injury

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Glia maturation factor beta (GMFB) was identified as a growth and differentiation factor acting on neurons as well as glia. We studied the time course of expression and the cellular localization of inducible GMFB during the first 56 days after induction of a cortical cryogenic lesion in rats. Immunohistochemically, astrocytes, hypertrophied with pronounced cell somas and wave processes around the lesion, expressed GMFB immunoreactivity peaking at 14 days after. Cryogenic lesion induced GMFB mRNA in the lesioned brain after 7 to 14 days with a maximum. In the enzyme immunoassay, GMFB protein concentration was peaked at 14 days after in the lesioned brain and peaked at 1 day after in serum. The data indicate that astocytes increased the expression of GMFB around the lesioned area after cortical cryogenic brain injury.

P1-170 Specificities of various glucuronidases to the oligosaccharides containing sulfated glucuronic acids

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Sulfated glucuronic acids (S-GlcA) is now known as the carbohydrate epitope to the HNK-1 Ab. In the nervous system, the HNK-1 epitope is expressed on glycolilpids, proteoglycans, and glycoproteins, such as N-CAM, P0, PASII/PMP22, and MAG. This HNK-1 reactive structure has attracted considerable interest both with regard to cell-cell interactions and demyelinating diseases. When determining oligosaccharide structures, we often need to remove sugars at non-reducing ends. So the specificities of various glucuronidases (GlcAase) were examined. GlcAase from E. coli reacted neither on S-GlcA nor GlcA, whereas did on both from limpet. GlcAase/arylsulfatase from Helix pomatia could remove only GlcA. Then we utilized these specificities for structural determinations of three di-sulfated oligosaccharides. We already knew those entire structures, except for the two sulfated positions. The derivatives were digested with the GlcAases and as a result the positions were determined successfully.

P1-171 The monoclonal antibody, 12F7, recognizes antigens in both oligodendrocytes and astrocytes *in vitro*

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To explore the specific antigen expressing oligodendrocytes, we generated monoclonal antibodies against stage-specific immature oligodendrocytes from E18 rat brain. Of antibodies obtained, a monoclonal antibody, named 12F7, was focused and characterized. Immunocytochemistry showed that 12F7 recognized not only antigens expressing in oligodendrocytes but also in astrocytes *in vitro*. The profile of expression was, at least, different from those of other known myelin-related proteins. In adult rat brain section, an antigen recognized by 12F7 primarily existed in white matter. Western blot showed that the molecular weight of 12F7 antigen was approximately 42 kDa and the expression might change with developmental period of central nervous system.

P1-172 The role of Schwann cell microvilli on the axonal organization

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In the PNS, Schwann cell microvilli surround the node of Ranvier. Microvilli are highly enriched in actin and its binding proteins, including ezrin, radixin, and moesin (ERM). Although this structure is believed to perform special interaction between glia and axon, the role of microvilli is still uncertain. To know the function, we examined the changes of microvilli in the cerebroside sulfotransferase knockout (CST KO) mice. The PNS in CST KO mice shows the disappearance of Caspr clusters and redistribution of K+ channels in the paranode as like in the CNS. However, no remarkable changes of Na+ channel clusters were observed. Localization of ERM was not changed in this mutant, suggesting that microvilli is not influenced by adjacent paranodal disruption. In development, ERM clusters was closely associated with that of Na+ channels. These results indicate that microvilli may play an important role for nodal organization, which is independent from paranodal junction in the PNS.

P1-173 The distribution of QH1-positive cells in the developing quail retina and optic tectum

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The monoclonal antibody QH1, which recognizes quail hematopoietic cells and vascular cells, was used to study the distribution of QH1-positive (Qp) cells in the optic tectum and the retina which remains avascular throughout the life.In the retina, many Qp cells were observed in NFL on the eighth day of incubation (E8). The Qp cell density was highest around the center of retina, gradually decreasing from the center to the periphery where no Qp cells were observed. On E15, Qp cells were also observed in the GCL, IPL and OPL. In the optic tectum, the Qp blood vessels invaded from E5. No Qp single cells were observed until E8. On E9, when the production of neurons was completed, amoeboid and round Qp cells were detectable in SAC. On E15, the amoeboid and round Qp cells occurred in SAC and SO. In other layers, thin ramified microglia-like Qp cells were observed.Our results suggest that Qp cells appear in the nerve fiber tract after the production of neurons ceases.

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P1-174 Changes of protein composition in spinal cord by the defect of paranodal junction

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Myelinated axons are divided at the axonal surface into four discrete functional regions, the node of Ranvier, paranode, juxtaparanode, and internode. At the paranodal regions, paranodal junctions (PJ) are formed between axon and myelinating glia. The cerebroside sulfotransferase knockout (CST KO) mice display disrupted PJ and dislocations of axonal ion channels in addition to channel subtype change. To know the molecular mechanisms of such abnormalities, we examined protein composition of spinal cord in CST KO mice. Immunoblot analysis with various antibodies including anti-phosphoprotein antibodies and 2-dimensional gel electrophoresis were performed and compared with wild type spinal cord. As a result, several proteins including heat shock protein 27 were changed their levels in CST KO mice. These molecules may explain the molecular mechanisms of interaction between myelin and axon through PJ.

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P1-175 Connexin hemichannels regulate microglial activation

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Microglial cell is a brain immune cell, which is activated on neuronal damages, and their activation is controlled by intracellular Ca^{2+} levels. It has been shown that stimulation of microglial cells triggers a rapid Ca^{2+} transient and subsequent sustained increase in intracellular Ca^{2+} levels, which is thought to be responsible for cytokine releases. Connexins, gap junctional proteins, are suggested to form functional ion channels, but the involvement of connexins in the Ca^{2+} entry was not analyzed. Therefore, we used microglial model (MG5) cells expressing Pericam Ca^{2+} sensors and demonstrated that gap junction inhibitors blocked the sustained Ca^{2+} increase but not the initial Ca^{2+} transient. Furthermore, high K^+ stimulation in low Ca^{2+} medium mimicked the receptor-activated sustained Ca^{2+} increase. These results suggest that connexin hemichannels, which are opened by depolarization, may mediate the sustained Ca^{2+} entry, and thus may be functioning for the cytokine releases from microglia.

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P1-176 Changes of neuronal gene expression by disruption of axo-glial communication

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In the CNS, sulfatide is a glycolipid in the myelin sheath. It is synthesized by cerebroside sulfotransferase (CST; EC 2.8.2.11), which is located in the Golgi membrane of oligodendrocyte. CST-null mice lack sulfatide and display disruption of paranodal junctions. Axonal ion channels change their subtypes and locations, suggesting that local signals through axo-glial junctions may influence neuronal gene expression as well as protein trafficking to their axons. To identify neuronal genes those change their expression levels by disruption of axo-glial communication, PCR-based cDNA subtraction analysis was performed. We compared transcripts of the Wd-type mice retina and the CST-null mice one and identified several genes by virtual northern dot blot analysis. Candidate genes were sequenced and further studied. These genes may provide important insight into molecular mechanisms of neuron-glia communication.

P1-177 Upregulation of lipocalin-type prostaglandin D synthase in oligodendrocytes during sleep deptivation

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Prolonged wakefulness or long sleep deprivation (SD) induces an excessive amount of rapid eye movement sleep (REMS) and non-REMS (NREMS) in mammals. However, the mechanism of this sleep rebound is still unclear. We demonstrated that SD for 6 h increased prostaglandin (PG) D_2 content in the brain and induced the strong rebound of NREMS and REMS in wild-type mice, but not in lipocalin-type PGD synthase (LPGDS) gene-knockout mice. *In situ* hydridization and immunohistochemistry revealed that the mRNA and immunoreactivity for LPGDS were upregulated in both perineuronal and interfascicular oligodendrocytes (OLs) of the brain of wild-type mice in a SD time-dependent manner (0-6 h). The LPGDS mRNA level in OLs of the thalamus and hypoyhalamus was increased 1.6-fold after 3 h SD. These results suggest that PGD_2 is produced by LPGDS in OLs during SD and induces NREMS to protect neurons against SD-related stress.

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P1-178 Membrane currents induced by Schaffer collateral inputs in rat hippocampal CA1 astrocytes

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In order to study active roles of glial cells in synaptic transmission, we made whole cell recordings from rat hippocampal astrocytes and analyzed membrane currents induced by stimulating Schaffer collaterals. Recordings were made from cells that showed linear I-V relationship, low input resistance and deep resting potential. The total charge induced by single stimulation was suppressed by CNQX and APV, antagonists for ionotropic glutamate receptors, and was enhanced by picrotoxin, an antagonists for GABA receptor channels. The peak current was not significantly altered by CNQX, APV nor picrotoxin, and was substantially suppressed by TBOA, an antagonist for glial glutamate transporters. When high frequency repetitive input was delivered, synaptically induced current accumulated during the first several inputs and then reached a steady level. The time to reach the steady level was related to the level of extracellular Ca concentration.

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P1-179 Myelinogenesis of the optic nerve in Neonatal diabetic rat model

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To investigate the effect of diabetes mellitus (DM), induced during the early postnatal period, on myelination of the optic nerve, DM was induced in P2 rats by i.p. injection of streptozotocin (STZ) (100 microgram /gm body weight). Triphasic change in the blood glucose level was encountered: initial sever hyperglycemia lased for 3 weeks, normoglycemia for 2 weeks (P20~P34), and mild progressive hyperglycemia after P34. Immunohistochemically, the number of MBP+ cells was reduced in diabetic rats at P5, P8 & P11. Electron microscopic assessment revealed a reduction in the number of ensheathed axons at P7 and a total reduction in the number of myelinated axons at P34. The myelin lamellae were loosely impacted (P34), with abnormally wide and swollen nodes with partial axo-glial dys-junction (P19), in diabetic rats. Data suggest that DM induced during the early postnatal period produces retardation in the process of myelinogenesis with consequent abnormal configuration of nodal and paranodal regions.

P1-180 Microglia transform into cells with neuroectodermal phenotypes

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Microglia massively proliferate shortly after the brain ischemic event, followed by the disappearance of the proliferated microglia and an increase in astrocytes, or astrogliosis. Massive cell death of proliferated microglia during the neuropathologic process does not occur. Similarly, there are numerous proliferating microglia in the developing brain, while few microglia undergo apoptosis. Such observations prompted us to examine the possibility that proliferating microglia are the source of newly generated neuroectodermal cells. In the present study, rat primary microglial cells were cultured in the presence of high concentrations of FCS, and then the cells were turned into promicroglioblasts (ProMGBs). ProMGBs transformed into cells with neuronal, astrocytic, or oligodendroglial phenotypes shortly after transfer into serum-free medium. These results suggest a novel role of microglia as multipotential stem cells to give rise to neurons, astrocytes, or oligodendrocytes.

P1-181 Fluorescence activated purification of brain capillary endothelial cells from Tie2-EGFP transgenic mouse

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It is important in blood-brain barrier (BBB) research to establish highly purified isolated brain capillary endothelial cells (BCECs) due to contamination. The purpose of this study was to establish a new method to isolate highly purified freshly isolated BCECs using Tie2-EGFP transgenic mouse expressing EGFP specifically in vascular endothelial cells. The fluorescent cells from the brain of Tie2-EGFP mice were isolated by FACS Vantage (3.0 x 10^6 cells from 10 mice). No mRNA expression of MAP2 was detected in the isolated cells. The mRNA level of von Willebrand factor (vWF) and the mRNA ratio of vWF to GFAP were 2.8- and 35-fold greater than those in BCECs obtained by the standard method with dextran. These results indicate that contamination by neurons and astrocytes in the newly developed method was greatly reduced compared with the standard method. This method is likely to be useful for the analysis of gene expression at the BBB.

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P1-182 Efflux Transport of 24S-hydroxycholesterol across the blood brain barrier

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24S-Hydroxycholesterol (24-OH-chol) is a major metabolite of cholesterol in the brain. The excretion pathway of 24-OH-chol from the brain is thought to be important to maintain brain cholesterol homeostasis, whereas its mechanism is still unknown. The purpose of this study was to investigate characteristics of the 24-OH-chol efflux transport across rat blood-brain barrier (BBB) directly by the intracerebral injection of [^{3}H]24-OH-chol. Remaining [^{3}H]24-OH-chol in rat brain after the injection was decreased in time-dependent, and its apparent BBB efflux rate constant was 6.27 x 10^{-3} min^{-1}. This efflux transport was inhibited by unlabeled 24-OH-chol at 10 μM in injectate by 76.5%. This is the first direct demonstration that 24-OH-chol undergoes the efflux from rat brain to blood across the BBB, and the results suggest that this efflux involves carrier-mediated processes.

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P1-183 Transport of hibernation-specific protein from blood into CSF in rats

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From previous studies on mammalian hibernation, we have discovered hibernation-specific protein (HP) derived from the liver in chipmunks, rodent hibernators, as a novel molecule regulated by a circannual hibernation rhythm, of which transport into cerebrospinal fluid (CSF) was facilitated by thyroxine through the choroid plexus (CP). In this study, we examined the dose and time dependencies of thyroxine-facilitated HP transport through CP in rats. After intravenous administration of HP-containing plasma of chipmunks, the blood and CSF were collected every 1 hour, and HP was analyzed by western blotting. HP was not detected in CSF in rats without treatment with thyroxine, while thyroxine at lower concentrations effectively increased HP transport into CSF. The highest effect was obtained a few hours after administration. Localization of HP in CP was observed by immunohistochemical analysis using anti-HP antibody. These results indicate that HP is acutely transported from the blood into CSF in rats in the same way as in chipmunks.

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P1-184 Exploration of brain activity during bipedal walking on perturbed and inclined treadmill belt: PET study

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To explore brain activity - bipedal (BP) locomotor behavior relationships in various walking conditions in BP walking monkey model, we employed a non-invasive neuroimaging PET protocol and detected the brain areas. The tasks included 1) BP walking on a level (BP-level), 2) BP walking on an obstacle-attached level (BP-obst), and 3) BP walking on an uphill and downhill (BP-inclined) treadmill belt. The monkey showed a similar activation pattern at M1, SMA, PMd, visual cortex and cerebellum in all these tasks, further the task-specific activity in BP-obst and BP-inclined was also observed. During BP-obst walking, thalamic nucleus was co-acitivated. These results suggest that the monkey utilized basic brain function to elaborate BP walking and add further function to solve the postural-control required walking tasks.

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P1-185 Brain activity during motor execution and imagery in the human precenral gyrus

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To test if parts of the human precentral gyrus (PcG) encode bilateral hand movement, we examined brain activity in 8 right-handed adults using fMRI. The tasks involved sensory-cued execution or imagery of unilateral finger or lower face movement. Auditory cues specified the effector to be involved, and visual cues did the performance mode. Echo-planar images were obtained and analyzed on an individual basis. A small portion of PcG was active during hand movement on either side, but this zone corresponded to the premotor area rather than primary motor cortex. The contra-hand PcG showed almost no imagery-related activity whereas the ipsi-hand PcG revealed substantial imagery-related activity without somatotopy. Unilateral lower face movement induced bilaterally symmetric activity in the PcG, and the same areas showed imagery-related activity with somatotopy. The present results suggest multiple representations of hand movement, each of which has a specialized role in supporting human hand dexterity

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P1-186 Intracortical microstimulation mapping and input organization of the primary motor and premotor cortical areas of the marmoset

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We examined somatotopical representations of the primary motor and premotor cortical areas using intracortical microstimulation (ICMS) in alert marmosets. Analysis of spatial distribution and threshold current of loci where forelimb movements were elicited by ICMS revealed that there exist at least 4 distinct motor areas related to forelimb movements. It was tentatively supposed that these areas corresponded primary motor, supplementary motor, dorsal premotor and ventral premotor cortex of macaque monkeys. The following retrograde tracing experiments and observation of the distribution of thalamocortical neurons showed that such correspondence is certain. The present experiments indicated evolutionary change of cortical motor areas and motor-related thalamocortical system in monkeys.

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P1-187 Direct estimation of arm movements from neuronal activities in the primary motor area of monkeys

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The purpose of this study is to precisely estimate of arm movements from real brain activity, while monkeys perform an arm-reaching task. We have previously reported that the arm movements can be computationally estimated from the neuronal activities in the primary motor area (MI). Then, we developed two methods: 1) reconstructing electromyography (EMG) signals from the MI neuronal activities with linear summation, 2) reconstructing joint angles from the EMG signals by an artificial neuronal network model. These methods allowed us to predict the arm movements from the MI neuronal activities. However, the prediction was not sophisticated and remained difficulty with real-time processing of the cortical signals. Herein we present our updated results, including new methods to reconstruct the arm movements much accurately.

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P1-188 Characteristics of limb coordination in degenerative neurological diseases

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We analyzed the rotational velocity of the pedals with a ergometer for 13 patients with cerebeller degeneration(CD), 27 patients with Parkinson's disease(PD), 26 others and ten normals. For characterization of coordination, five indices were defined. A set was considered as a point located in the five-dimensional space. Sets from PD were divided into four clusters. We classified all into four clusters and located other than PD on the cluster map. 141 data without freezing were classified into cluster 1, 2. 21 data with freezing were classified into cluster 3, 4. Impairment of interlimb coordination in patients with parkinsonism may be represented by patterns of cluster 3, 4.

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P1-189 Impedance control for task optimization

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Although signal dependent noise (SDN) predicts larger motor-command variability during higher muscle co-activation, the relation between impedance and task performance is not obvious. Here, we examined movement variability and muscle activation in reaching with different task requirements. When subjects made reaching movements to different sizes of targets, they increased co-activation for higher accuracy requirements, leading to a reduction in the end-point deviation. When subjects were required to reach a target through a small gate (8 mm wide 3 cm long) at the midpoint of the movement, they increased co-activation and reduced movement variability around the gate compared to when they reached the same target with no gate. The observed reduction of variance by co-activation contradicts the prediction of SDN. Subjects were able to regulate their impedance and control movement variability as the task requirements changed. (supported by NICT)

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P1-190 Effect of spatial attention on human sensorimotor integration studied by trancranial magnetic stimulation

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Recent transcranial magnetic stimulation (TMS) studies have shown that the sensory input can decrease the motor cortex excitability (afferent inhibition). To clarify the effect of attention on sensorimotor integration, we investigated the effect of spatial attention on afferent inhibition. Median nerve electrical stimulation followed, at variable delays (10 - 300 ms), by TMS over the contralateral motor cortex was applied to 8 subjects, during 3 conditions; spatial attention to the right and left hand, and control (no attention) sessions. Inhibition of the motor evoked potential occurred at inter-stimulus interval of 20 ms and 100 ms, which was marked during spatial attention to the right than to the left hand. Enhancement of the afferent inhibition induced by spatial attention to the stimulated side is likely to reflect the interaction between the spatial attention and sensrimotor integration.

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P1-191 Eye-head-hand coordination in food taking movements using a forceps in monkeys

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Monkeys learned a sequential task of grasping forceps placed in front of them with their right hands, using the forceps to pick up a piece of food, and then bringing it to their mouths. The forceps were placed at various locations and directions relative to the monkey and the food. At a beep, monkeys oriented and visually located the forceps but averted their gaze before reaching for them with a grasping hand. They lifted the forceps gazing at the tips and brought them toward the food. Shifting their gaze to the food, they picked it up by closing and rotating the forceps simultaneously with an upward head movement, then brought it to their mouths. Superimposed traces of hand, head and eye movements varied little among trials of the same task, suggesting that the sequencing and timing of the movements were stored in memory and the best matched program was retrieved, as is presumably the case with human touch typing.

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P1-192 The role of ankle joint in the control of postural stability during upright standing on one foot

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The aim of this study is to establish a model to elucidate the inner relationships between lower leg muscular activations and COP deviations during standing. Muscular activations were accessed by sEMG, and COP deviations were measured by a force-sheet. Subjects were asked to stand on force-sheet with left bared food for 30 seconds. sEMG on m. tibialis anterior, m. peroneus longus and Caput mediale m. gastrocnemii were also recorded simultaneously. As a result: 1) ground reaction forces under the foot can be ascribed to three points, make up a triangle; 2) The deviation of COP under the foot is controlled by lower leg muscular contractions; 3) The reciprocal contractions of TA and PL is the cause of the COP deviation in lateral direction.

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P1-193 Somatotopic arrangement of multisynaptic inputs from putamen to primary motor cortex as visualized by retrograde transneuronal transport of rabies virus

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To elucidate the somatotopic organization of multisynaptic inputs from the striatum to the primary motor cortex (MI), rabies virus was injected into hindlimb, forelimb, distal forelimb, or proximal forelimb representation of the MI in monkeys. The injection into the hindlimb region resulted in transsynaptic labeling of striatal neurons in the dorsalmost part of the putamen, while labeling from the forelimb region was located more ventrally. Compared with the distribution of labeled neurons from the proximal forelimb region, that from the distal forelimb region was centered ventrolaterally in the putamen. These patterns of retrograde labeling were in good accordance with the arrangement of corticostriatal terminals from the MI. These data suggest the segregation of the motor cortico-basal ganglia circuit into parallel closed subloops.

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P1-194 Somatotopical organization of the cortico-striato-entopeduncular pathway of rats

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We analyzed somatotopy in the rat cortico-striato (St)-entopeduncular (Ep) pathway using tract-tracing and electrophysiological techniques. Biotinylated dextran amine (BDA) was injected into the vibrissae (V), jaw (J), forelimb (F) or hindlimb (H) area of the motor cortex after intracortical microstimulation. The corticostriatal projections showed distinct somatotopical arrangement. We then identified the St area by examinig orthodromic responses to cortical stimulation in V, J, F or H area, and injected BDA into each area. The St-Ep projections showed less distinct somatotopical arrangement, and the terminal areas were largely overlapped one another. Extracellular recordings of St or Ep neurons revealed that most St neurons showed responses to stimulation in a single motor cortical area, whereas large numbers of Ep neurons showed responses to two or more cortical areas. Thus, the somatotopical information is conveyed in parallel manner from the motor cortex to the St, and then converges on Ep neurons.

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P1-195 Locomotor patterns evoked by stimulation of supraspinal locomotion-inducing sites in decerebrate rabbits

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This study aimed to investigate characteristics of locomotor patterns evoked by stimulation of locomotion-inducing sites identified as the mesencephalic (MLR) or cerebellar locomotor region (CLR) in decerebrate rabbits. After decerebration, the head of the animal (2-3 kg, male) was fixed in a stereotaxic apparatus, and the body was supported by a rubber hummock. Using Wood's-metal-filled glass microelectrodes, 50-Hz stimuli (10-100 μA, 0.2 ms duration) were applied to the MLR and CLR for 5-10 sec. Evoked movements were videoed, and EMGs were recorded from the extensors and flexors of bilateral hindlimbs. When the MLR and CLR were stimulated, the left and right hindlimbs exhibited rhythmic locomotor movements in phase, not alternately. Stronger MLR stimuli increased the locomotor cycle frequency and hindlimb EMG amplitude, whereas stronger CLR stimuli evoked spastic hindlimb extensions. This suggests that the MLR and CLR play a crucial role in the control of locomotion but in a different manner.

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P1-196 Dorsal Premotor Cortex Activation during Observation of Human Gait: A fNIRS Study

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There is growing evidence that the mirror neuron found accidentally in monkey also serves as observation/execution matching system in human. We tested if action observation affects motor performance and cortical activation patterns during human gait using a functional 42-channel near-infrared spectroscopy (fNIRS) in 8 healthy volunteers. Subjects performed locomotor tasks of (1) treadmill-walking at 3km/h, and (2) walking at 3km/h observing human walking or hand movements on VTR. Blood pressure, heart rate, and SpO2 showed no changes during the tasks. During gait under walking observation, oxygenated hemoglobin in the left dorsal premotor cortex (PMd) significantly increased and cadence approximated to that of a walking subject on VTR. Such changes were not found during the other tasks. These results suggest that the left PMd might play a role in matching observation with execution during human gait.

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P1-197 Influences of cerebellar hemispherectomy on generation and control of compound movements

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Cortical field potentials were recorded by electrodes implanted on the surface and at a 2.0-3.0 mm depth in various cerebral cortices, together with three movement traces of eye, neck and trunk, while monkeys learned compound movements of neck and trunk. At an early learning stage when mainly movements of neck or trunk were performed, surface-negative, depth-positive potential (readiness potential) preceding the movement was small in amplitude in the premotor cortex. As the amplitude of the potential became bigger, the compound movements gradually increased in number and skill. After the right hemicerebellectomy, the potential in the ventral part of the left premotor cortex (PMv) was decreased in amplitude, and the compound movement changed in smoothness and in movement pattern composed of the three movement traces. This suggests that the PMv takes part in generation and control of the compound movement together with the cerebellum, possibly through cerebro-cerebellar interactions.

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P1-198 Coordinate frames of Purkinje cell activities for motor control

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To examine coordinate frames for movement-related activities of the cerebellar Purkinje cells (P-cells). We recorded P-cell activities while a monkey performed a behavioral task that dissociates three major coordinate frames for wrist movement (Kakei et al., 1999). The task involves the performance of wrist movements in different directions with different forearm postures. In a monkey trained to perform the task, we recorded task-related activities of P-cells. These P-cells showed systematic changes of activities for different movement directions. In each neuron, we determined a coordinate frame for the neuron activities. Some P-cells were related to a "muscle-like" coordinate frame, while others were related to an "extrinsic-like" coordinate frame. These results suggest diverse roles of the cerebellum in motor control. Supported by PRESTO JST, MESSC (S.K.).

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P1-199 Multisynaptic inputs from prefrontal cortex to primary motor cortex as visualized by retrograde transneuronal transport of rabies virus

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Using retrograde transneuronal transport of rabies virus, we examined multisynaptic projections from the prefrontal cortex to forelimb vs. hindlimb representations of the primary motor cortex (MI) in macaque monkeys. Four days after the viral injections, neuronal labeling occurred explicitly in the prefrontal cortex. In the case of the forelimb injection, the densest labeling was found in the ventral portion of area 46 (area 46v). Areas 9m and 32/24 of the medial wall also contained many labeled neurons. In the hindlimb-injection case, on the other hand, neuronal labeling in the prefrontal areas was generally sparse. In area 46v, only a small number of neurons were labeled. The present results suggest the importance of area 46v in the control of forelimb movements.

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P1-200 Differences of EMG activities of trunk and hindlimb muscles of bipedal-walking rat model (BRM) and normal rat during treadmill locomotion.

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BRM were created by forelimb amputation and training of bipedal walking for 5-10 months. BRM can performed stable bipedal walking on the treadmill. We recorded EMG activities from m. longissimus lumborum (Long) and m. quadriceps (Q) and also movements of rat by video system. During a single step cycle, normal rat showed biphasic EMG bursts of Q and show synchronized bilateral EMG bursts of Long with small amplitude, while rat bipedal-walking model showed the shorter duration and monophasic EMG bursts of Q and the larger amplitude EMG bursts of Long. These results of the present experiments indicate the significant difference of function and neuronal control of Q and Long between quadrupedal and bipedal walking.

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P1-201 Morphological changes in spinal motorneurons after the acquisition of bipedal walking in adult rats

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We examined morphological changes in spinal motoneurons (MNs) in relation to the acquisition of bipedal (BP) locomotion in operantly-trained rats (bipedal rat model: BRM) and compared with MNs in non-training control rats (cont). After 12 months of training, *M. longissimus lumborum* (Long) and *M. quadriceps femoris* (Q) were exposed and WGA-HRP was injected. After 2 days survival periods, the animals were perfused and the spinal cord was removed. The horizontal section were made with 100 μm thickness and reacted by DAB-Nickel method. The results showed 1) larger MNs in Q than Long in both BRM and cont group and 2) the tendency of enlarged Long and Q MNs in BRM. These suggest that, with long-term locomotor learning, rats can plastically modify its MNs for the execution of BP locomotion.

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P1-202 EMG activity of the longissimus muscle and kinematics of the spinal column during level, upslope and downslope treadmill walking in cats

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The purpose of our experiments is to gain insight into the neural mechanisms controlling spinal column movements. Long had two predominant bilateral EMG bursts during level, upslope and downslope walking at the steeper grade. These discharges inhibited the lateral movements of the vertebral column by increasing the stiffness of the vertebral column and producing the inward movements. The timing, amplitude, and duration of the EMG bursts were strongly affected by an increase in the upslope grade as was the relation between the measurements at the different levels of Long. At 5-20degrees of downslope walking, EMG bursts were strongly inhibited. The depression of EMG bursts during downslope walking was related to the well balanced movements of the spinal column in all three kinds of direction. The results suggest the importance of information concerning spinal column movements.

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P1-203 Morphological changes in skeleto-muscular system after acquisition of bipedal walking in adult rats

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Our previous study showed an enhanced lumbar lordosis, and marked lumbar muscle hypertrophy after long-term bipedal (BP) training in monkey. This suggests that not only nervous system also musculo-skeletal system was plastically altered for the execution of BP walking. In this study, we examined the morphological changes in spinal vertebrae and an anti-gravity muscle (*M. longissimus lumborum*: Long). BP walking training (30min/day, 6times/week) was given for 12 months. Age-matched control rats were not trained. After the training, whole body Xp and CT scan was examined, and then skeleton model was made. The results showed 1) spinal lordosis at the lumbar segment, 2) an enlarged facet of spinous and transverse process at whole vertebrae and 3) muscle hypertrophy of Long in the trained rats. These results suggest that the normally quadrupedal rat revised its musculo-skeletal system necessary for the elaboration of BP walking.

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P1-204 Integration of group I and II muscle afferents from various parts of the body on motoneurons innervating m.longissimus lumborum (Long).

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We studied the reflex actions of group I and II afferents from neck, forelimb, trunk and hindlimb muscles to motoneurons inverting Long in the T10, L1 and L5 spinal segments of the decerebrated cats. PSPs recorded from 58 Long motoneurons in 12 cats. The L5 motoneurons showed high responsibility to stimulation of group I and group II afferents innervating trunk and hindlimb muscles , while did not show any PSPs after single electrical stimulation forelimb and neck muscles. The L1 motoneurons showed responsibility to stimulation of group I and II muscles afferents from trunk and group II muscle afferents from hindlimb and neck muscles. The T10 motoneurons showed the responsibility to group II muscle afferents from neck, forelimb and did not show any effects of hindlimb muscles. The results suggest a position-dependent in the control of Long motoneuron activity by group I and II afferents.

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P1-205 Functionally essential neuronal population of spinal motor neurons

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The present study was aimed to determine functionally essential neuronal population for normal motor function in young adult rats (8 weeks) who had received various degrees of nerve crushing to the sciatic nerve at the neonatal stage (P1). The motor function was estimated by the static sciatic index to assess functional loss following injury to the sciatic nerve. After the functional assessment, a retrograde neuronal tracer (BDA-3000) was applied to the common peroneal nerve, the main nerve branch of the sciatic nerve. The total number of intact motor neurons capable of transporting tracer molecules was estimated in the control and experimental animals. The results show a close relationship between the degree of motor function and the number of healthy motor neurons, and implicate functional redundancy of the nervous system. The results also show that normal motor function can be preserved by a relatively small population of motor neurons in sciatic nerve-injured rats at the neonatal stage.

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P1-206 Functional organization of spinal interneuronal systems involved in the control of postural muscle tone

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Microinjection of carbachol into the pontine reticular formation induced muscular atonia in cats. In this study, firing rates of 103 and 95 interneurons in the lower lumbar segments (L6-7) were examined in decerebrate cats (n=24) before and after carbachol injection, respectively. Before carbachol injection, a mean firing rate of cells in the intermediate region and dorsal horn (dorsal cells; 17.7 ± 8.7 Hz, n=56) was higher than that of cells in the ventral part of lamina VII and VIII (ventral cells; 8.3 ± 7.6 Hz, n=47). Dorsal cells mostly excited from volleys in flexor reflex afferents (FRA), while ventral cells largely inhibited by FRA. After carbachol injection, a mean firing rate of ventral cells (24.3 ± 13.7 Hz, n=46) was, however, higher than that of dorsal cells (11.4 ± 7.6 Hz, n=49). These results suggest the presence of topographical organization of spinal interneuronal systems in FRA pathways in relation to the control of postural muscle tone.

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P1-207 Environmental enrichment attenuates motor dysfunction following intrastriatal 6-hydroxydopamine (6-OHDA) lesion

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We studied the effects of environmental enrichment on functional outcome following 6-OHDA injection in the striatum. Male Wistar rats (P25) were housed in normal or environmental enrichment cages (large cage with several equipments) for 5 weeks. 6-OHDA (20μg) was injected into the left striatum and rats were kept under each circumstance for another 5 weeks. Rota-rod and forelimb-use asymmetry tests didn't show any significant difference between the groups. On the other hand, the performance in beam-walk that reflects a fine coordination of motor function was impaired in normal caged but not enriched caged group. The methamphetamine induced rotational asymmetry was lower in enriched caged group than in normal. Thus, the environmental enrichment attenuated the disability in fine coordinated motor function and also the nigrostriatal dopaminergic damage, suggesting the advantage of environmental enrichment in recovery of motor dysfunction.

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P1-208 Reconstruction of axon collaterals arising from single corticospinal neurons in the striatum and thalamus

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The complete corticostriatal and corticothalamic axonal arborizations arising from single corticospinal neurons were analyzed. After injections into the pyramidal tract with sindbis virus that was designed to express green fluorescent protein (GFP), one or several cortical neurons were found to be retrogradely infected and labeled with GFP. By immunohistochemistry for GFP, axons and terminals of the labeled neurons were visualized. About 20% of labeled corticospinal neurons had axonal arborizations in both the striatum and thalamus, and about 40% of the labeled neurons extended their axon collaterals into one of those regions. The whole figures of axonal arborizations arising from the corticospinal neurons were reconstructed. Quantitative analysis for the pattern of axonal arborizations and distributions of synaptic button will be presented.

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P1-209 Downregulation of Metabotrophic Glutamate Receptor 1α in Globus Pallidus of Parkinsonian Monkeys

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The present study aimed at investigating the role of excitatory input through metabotropic glutamate receptors (mGluRs) on pallidal neuron activity in macaque monkeys. Immunohistochemical localization of several subtypes of mGluRs in pallidal neurons was examined in normal and MPTP-treated parkinsonian monkeys. In MPTP-treated monkeys, the expression of mGluR1α was specifically reduced in both the internal and the external segments of the globus pallidus. Extracellular unit recordings revealed that the spontaneous firing rate of pallidal neurons in normal monkeys was increased by a group I mGluRs agonist and decreased by an antagonist. Such effects became obscure in MPTP-treated monkeys.

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P1-210 Pallidal activity during high frequency stimulation of the subthalamic nucleus in parkinsonian monkeys

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High frequency stimulation (HFS) of the subthalamic nucleus (STN) has increasingly been used to treat parkinsonian patients. We observed activity of the internal segment of the globus pallidus (GPi) during STN-HFS in parkinsonian monkeys to clarify its mechanism. Stimulating electrodes were chronically implanted into the forelimb regions of the motor cortical areas of Japanese monkeys. MPTP was administered systemically via the internal carotid artery. A stimulating electrode was inserted into the STN. Single stimulation of the STN evoked a short-latency excitation followed by an inhibition in GPi neurons. Repetitive stimulation decreased firing frequency of GPi neurons and amplitude of cortically-evoked responses. These data suggest that STN-HFS suppresses abnormal activity in the GPi of Parkinson's disease.

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P1-211 Subthalamic neurons coordinate dopamine-mediated behavior through differential neural pathways

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The subthalamic nucleus (STN) is a key relay in basal ganglia circuitry. In the present study, we analyzed the neural pathways through which the STN regulates motor behavior mediated by dopamine (DA) transmission. Conditional ablation of the STN neurons caused hyperactivity of spontaneous movement and reduced motor activation in response to DA stimulation. The ablation modulated spontaneous firing activity of the output neurons, preserving the activity of the globus pallidus (GP) neurons. In addition, the ablation attenuated DA-induced regulation of the firing activity of the output and GP neurons. These results suggest that the STN neurons function to suppress spontaneous behavior through the direct pathway to the output neurons, and that in response to DA they contribute to expression of behavior by acting the output neurons mainly through the GP-mediated pathways.

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P1-212 Axonal arborization of ventral tegmental area dopaminergic neurons: single-cell study

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Dopaminergic neurons in the substantia nigra and ventral tegmental area (VTA) mediate a variety of brain functions, such as motor control, emotion, and reward. We studied the axonal arborization of VTA neurons with Sindbis virus vectors that coded membrane-targeted green fluorescent protein (GFP). After injections of the viral vectors in the rat VTA, the infected neurons were examined whether or not they were immunoreactive for tyrosine hydroxylases, and labeled in a Golgi-like manner by the inimmunoperoxidase method with an anti-GFP antibody. After the reconstruction of projection axons, some neurons were observed to project their axons to the limbic cortices through the caudate-putamen. Another neurons sent axons to the accumbens nucleus. Thus, the present study revealed a difference in the axonal ramifications of single dopaminergic VTA neurons.

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P1-213 Morphological and electrophysiological properties of GABAergic neurons in the superior colliculus(SC) of GAD67-GFP knock-in mice

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It has been shown that burst firing of the neurons in intermediate gray layer (SGI) of the superior colliculus (SC) is generated via disinhibition from local GABAergic neurons as well as the substantia nigra. We studied the characteristics of local GABAergic neurons in SGI by whole-cell patch-clamp recordings in GAD67-GFP mice. The majority of these GABAergic neurons were regular spiking in young rats age (< PND 14) but changed to fast spiking neurons at PND 17-20. We examined the correlation between GABAergic neurons and the expression of Ca-binding protein, and found that 31% of GABAergic neurons were parvalbumin positive. The varied axonal trajectories of intracellularly-labeled GABAergic neurons suggest several patterns of inhibition in the SC.

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P1-214 Cholinergic Responses of Neurons in the Intermediate Layer of Rat Superior Colliculus

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To clarify the role of cholinergic inputs to neurons in the intermediate layer (SGI) of mammalian superior colliculus (SC), we investigated the cholinergic responses of rat SGI neurons. Whole-cell patch clamp recordings were obtained from SGI neurons in Wistar rat SC slices (17-24 days old). Current responses of neurons to 30 μM carbachol alone or with dihydro-β-erythroidine and atropine were investigated in the presence of TTX. Carbachol induced current responses in most of SGI neurons which could be classified into 5 patterns; inward (fast), inward (fast) + inward (slow), inward (fast) + inward-outward (slow), inward (fast) + outward (slow) and outward (slow). Responses in most of the identified projection neurons were classified as the inward (fast) + inward (slow) and inward (fast) + inward-outward (slow) patterns. These data suggest that the cholinergic inputs may differently modulate SGI neuron activities depend on cell types and functions.

P1-215 Stimulation of the rat substantia nigra pars reticulata induces jaw, tongue and facial muscle activity

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To localize the sites of rat substantia nigra pars reticulata (SNr) which induce orofacial motor activity, mapping by electrical microstimulation was performed. Selected sites were electrolytically marked. We used chronically decerebrated rats in which the corticobulbar fibers descending to the brainstem orofacial motoneurons were removed, so that the effect by possible current spread to the cerebral peduncle was neglected. In a total of 40 penetrations at different sites of SNr in five decerebrated rats, microstimulation (333 Hz, 40-60 μA for 61 ms) of 11 sites evoked bilaterally EMG activity in the jaw, tongue and facial muscles, with an ipsilateral predominance. They were characterized by enormous amplitude and low frequency probably due to synchronized motor unit discharge. Ejection in situ of 1.0 μl GABA evoked tonic activity in all muscles. Histological examinations revealed that the effective sites are located in the most ventral layer of the caudal SNr confined to a small middle and lateral region.

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P1-216 Reticular neurons located caudal to the trigeminal motor nucleus form excitatory synaptic connections with jaw-closing motoneurons.

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To understand the neural network responsible for controlling masticatory force according to the physical property of foods, we examined the location of excitatory premotoneurons for jaw-closing motoneurons by using anesthetized rats. Single-pulse stimulation (< 10 μA, 100 μs) applied to the reticular formation (cRF) caudal to the trigeminal motor nucleus (MoV) could elicit a short-latency response of masseter EMG. Application of kainic acid (10 μM, 0.5 μl) to the cRF also evoked masseter EMG activities. Furthermore, neurons located in the cRF were antidromically activated by electrical stimulation of the MoV. Averaging of the masseter nerve after spike potential of the neurons revealed the excitatory response in the masseter nerve. These results suggest that some cRF premotoneurons form excitatory synaptic connections with jaw-closing motoneurons.

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P1-217 Organization of multisynaptic pathways innervating the jaw-closing and jaw-opening muscles in the rat: a transneuronal tracing study using rabies virus.

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Many electrophysiological and neuroanatomical studies have investigated the neural networks controlling masticatory behaviors. However, little is known about the distribution patterns of cortical and subcortical neurons related to jaw-closing vs jaw-opening movements. To address this issue, we injected rabies virus (CVS-11) unilaterally into the masseter muscle or the anterior belly of the digastric muscle, and analyzed the organization of multisynaptic pathways that innervate these muscles specifically. The present results indicated the differential patterns of time-dependent distribution of retrogradely labeled neurons.

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P1-218 Cerebral control of the medullary ventral respiratory neuron group and upper cervical inspiratory neurons in the rat

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Respiratory motor outputs are autonomic rhythmic activities and also are voluntarily controlled. We examined the effects of stimulation of cerebrofugal fibers on respiratory neuron activities of ventral respiratory neuron group (VRGs) in the caudal medulla, upper cervical inspiratory neurons (UCINs) and phrenic motoneurons (Phr. MNs). Animals were anesthetized, immobilized and artificially ventilated. Bipolar stimulation electrodes were placed at the cerebral peduncle (CP) and sensorimotor cortex on the right side. Single or double pulse stimulation of the CP induced facilitation and succeeding suppression on respiratory activities of the phrenic nerve and UCINs on the contralateral side, while stimulation of CP predominantly suppressed inspiratory and expiratory VRG activities on the ipsilateral side. The present results suggest that cerebral excitatory inputs to Phr. MNs and UCINs are not transmitted through VRGs, but rather directly to Phr. MNs and UCINs.

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P1-219 Contribution of histamine H1 receptors to the breathing pattern formation in mice

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We examined the breathing pattern during hypercapnia in wild type (WT) and histamine H1 receptor knock-out (H1RKO) mice in order to elucidate a role of central histamine in the breathing pattern formation. Respiratory variables were measured with whole body plethysmograph during stepwise CO_2 gas exposures before and after vagotomy. Minute ventilation increased with $PaCO_2$ increase in the both mice, however, respiratory rate was lower and tidal volume (VT) was higher in H1RKO mice than in WT mice. VT-inspiratory time relationships showed hyperbolic curve in both mice, but the relationship in H1RKO mice was shifted upward to the right from that in WT mice. The shift in H1RKO mice remained even after the vagotomy. NMDA receptor antagonist, MK801 affected these responses. These results suggest that H1RKO mice raise the threshold in termination of inspiration, indicating that the inspiratory off-switch mechanism is altered.

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P1-220 Measurement of sucking amounts in developing rats

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It is important to provide basic data on sucking amounts in developing animals. Although the lactating mother is always nursing her pups, they are not sucking milk all the time. Thus the amounts of stomach milk usually differ considerably in each pup at a certain observation time. In this study, we tried to show sucking capabilities of rat pups at different developmental stages (P1, 2, 4, 7 and 14). Prior to lactation, we set non-lactation periods (2, 4 and 6 hours) by separating the offspring from the mother and let her lactate for an hour. The pups' sucking amounts were obtained by measuring differences in their body weight just before and after suckling. The results show that the suckled amounts increase greatly as pups grow up, and that longer non-lactation periods are needed with advancing age. These data would be useful for the estimation of sucking capabilities of developing rats both under normal and pathological conditions.

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P1-221 Hypoglossal nerve resection and sucking capability

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The present study was undertaken to know effects of nerve resections of the hypoglossal nerve (XII) components on milk-sucking capabilities. Neonatal pups (P1) received surgical resections of the XII at its main trunk, medial branch and lateral branch unilaterally as well as bilaterally. The bilaterally XII-resected pups did not contain milk in the stomach and never survived. The unilaterally XII-resected pups at the lateral branch began to contain milk in the stomach and showed steady postnatal growth. Parts of the unilaterally XII-resected pups at the main trunk or medial branch failed to suck milk and died, whereas the other pups gradually acquired sucking capabilities and survived, though they showed marked retardation in postnatal growth. The results indicate a crucial role of the tongue, especially of protruding muscular elements innervated by the medial branch of the XII, in breastfeeding. Quantitative data on milk-sucking amounts of control and experimental groups will be presented.

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P1-222 Differential effects of hypoglossal nerve resections on the survival of developing rats

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The hypoglossal nerve (XII) is made up of the medial branch relating to protrusion of the tongue and the lateral branch relating to its retraction. We examined effects of nerve resections of the XII components in breastfeeding and postnatal growth, and obtained the following results. (1) P1. The bilaterally nerve-resected pups never survived. The unilaterally nerve-resected pups were divided into two groups with survival (main trunk & medial branch: 20-40%; lateral branch: 92%) and non-survival. (2) P11. The nerve-resected pups were divided into two groups with survival (unilateral and bilateral resections of the lateral branch) and non-survival (unilateral and bilateral resections of the main trunk or medial branch). (3) P21. Only the rats with bilateral nerve resections of the main trunk did not survive. The results indicate differential effects of XII resections on the survival according to the stage of development.

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P1-223 Cortical control of GABA neurons during cat tongue movements

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Cortical control of CABA neurons during cat tongue movements. Hisao Hiraba. Nihon Univ, Sch of Dent, Tokyo 101-8310. Cat hypoglossal nucleus anatomically receives outputs from bilateral motor cortex (area P in the lateral wall of the presylvian sulcus) (Yasui et al., 1985). ICMS in area P evoked tongue protrusion. In human transcranial magnetic cortex stimulation over the face-associated motor cortex produces bilateral tongue responses (Meyer et al. 1997). We assume from these results that pyramidal cells with bilateral innervation may exist in a unilateral area P, and tongue movement may be regulated on inhibitory circuits in GABA neurons. In this present study, cats were trained to take food from the left and right sides. We measured the regional cerebral blood flow (rCBF) in the area P during the left and right side intake. Further, changes in orofacial behaviors and rCBF were examined after injections in the contralateral area P of GABA agonist (muscimol) and antagonist (bicucurine). Based on the findings, area P GABA neurons may regulate condition of tongue protrusion.

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P1-224 The effect of treadmill running on motor cortex thickness after intracerebral hemorrhage in rats

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Intracerebral hemorrhage (ICH) by striatal collagenase injection induces porencephaly, enlargement of ventricle and shrinkage of cerebral cortex. We have reported that treadmill running after ICH improves forelimb motor function. To investigate the mechanism of the recovery, we measured the thickness of sensorimotor cortex relating forelimb function (1.7 ~ 1.2 mm anterior from Bregma) and hindlimb function (1.8 ~ 2.3 mm posterior). The percentage of thickness of ipsilateral cortex to that of contralateral cortex was higher in forelimb area in treadmill running group than that of control (ICH without training). However, there was no change in the thickness of hindlimb area between treadmill group and control. Data suggest that treadmill running after ICH prevents motor cortex shrinkage, especially in forelimb area, and this may be the basis of motor recovery.

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P1-225 Axonal damage causes motor dysfunction in internal capsule hemorrhage model rats

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We have developed internal capsule (IC) hemorrhage model rats by injection of collagenase solution in the striatum near the IC (1.8 mm caudal to bregma). Injected animals had a small hematoma but relatively bigger motor dysfunction. To detect the damage in the IC, motor neurons were labeled retrogradely by the injection of Fluoro-Gold into the corticospinal tract at C2-3 level, and labeled-cells were counted every 0.5 mm sections. In animals labeled before collagenase injection, the number of labeled-cells decreased (73.9 ± 1.4% of contralateral side) 14 days after the lesion compared to control group (100.8 ± 3.5%) at 0.8-2.8 mm caudal from bregma. Labeled-cells in ipsilateral cortex were shrunken. However, FG labeling after the lesion detected a small number of cells (0.8 ± 0.8% of contralateral side). Data suggest that most axons in the IC were strongly damaged, and this may be the basis of relative big motor dysfunction in this model rats.

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P1-226 Lesion studies on neuronal substrates for rat air righting reflex

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Falling from the supine position,animals may turn their body in the air for landing on the ground (air-righting reflex, ARR). To know the neuronal substrate of the ARR motor program, we studied rat ARR following various lesions given in the lower brain. Electric lesions were performed in Wistar, adult male rats. ARR movements were analyzed on a digital video recorder (30 frames/s). ARR movements of the rats with the fastigial nucleus destructed on the one side varied within the same animals; they showed normal ARR in some case, but in the others turned too much, or too little. Deiters' nucleus lesions forced the animals to turn toward the contralateral side, and they rotated repeatedly during falling. While rats with the unilateral reticular formation lesioned tended to rotate toward the lesion side, the magnitudes were almost normal. ARR motor programs may consist of several components, of which fastigial and reticular components could be not essential but those in the Deiters' nucleus indispensable.

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P1-227 Neural substrates for predicting a dynamic abstract motor sequence: a high-field fMRI study

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We scanned 4 right-handed subjects with a 4T MRI scanner while they were predicting a dynamic motor sequence. Since the sequence of the target position was generated from a logistic map [$x_{(t+1)}=4.0*x_{(t)}*(1-x_{(t)})$, $0.0 < x_{(t)} < 1.0$, t=0-88], this learning task required abstracting the rule from exemplars. After intensive training (> 8 days), two runs of fMRI images were acquired for each subject. After post-processing corrections, we generated t-maps of activated brain regions responsible for predicting the sequence through the learned rule by a conjunction analysis. Overlapped regions between two runs were extracted for each subject. Activated regions common to most of the subjects, including the hippocampus, left inferior frontal gyrus, left parietal cortex, thalamus, basal ganglia, SMA and left premotor cortex, were found and their roles in predicting the motor sequence are discussed.

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P1-228 Minimal voluntary movement of a single finger joint analyzed by a motion analysis system

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In 2001, we reported the minimal travel distance (flexion or extension) of distal phalangeal joint of the index finger was measured by comparing the start and stop position of the small marker painted on its fingertip in video-frame images. The minimal travel was 2.7 mm (1). In the present experiments we traced the marker on fingertips continuously by a motion analysis system to exclude the overlapping travel which was thought to occur by other joint movements. 1. Minimal travel of index finger tip was equal in extension or flexion of distal phalangeal joint and its value was 0.48 mm. which was much smaller than that of our previous report. 2. Maximal travel speed of the finger tip was 14.8 mm/sec.3. The speed was larger at extension than flexion. (1) J.Neurosci. Res. Suppl. 25: S160, 2001.

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P1-229 Effects of Transcranial Magnetic Stimulation to human dorsal premotor cortex during simple visual reaction time task

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We compared the effects of Transcranial magnetic stimulation (TMS) to human motor cortex (M1) during simple visual reaction time (RT) task with the effects to dorsal premotor area (PMd). A single-pulse TMS (higher or lower) was delivered to the left PMd at either timing of 0, 40, 60, 80 or 100ms after the stimulus. We measured motor evoked potential (MEP), RT and integrated muscle activity.
Higher intensity TMS, which evoked MEP in all trials when provided to M1, evoked MEP in 50% of trials when provided to PMd. Only when higher intensity TMS was provided to M1 at the timing of 80 or100 ms, MEP amplitude and integrated muscle activity were enhanced and RT was prolonged, whereas when provided to PMd no such effects were observed. When lower intensity TMS was provided to M1 at these timings, only RT became shorter, but that was not seen on PMd.
The present preliminary data show that role of PMd during simple visual RT task may differ that of M1.

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P1-230 Delay period activity in the central thalamus of monkeys predicts saccade timing

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Evidence suggests that two subcortical structures, the cerebellum and the basal ganglia, are crucial for movement timing. Since the thalamus serves the subcortical gateway to the motor cortices, some thalamic neurons must relay information about movement timing. Monkeys were trained on the memory-guided saccade tasks with a delay of one second for many months. A subset of neurons in the central thalamus exhibited a buildup of activity that peaked around the time of fixation point (FP) offset. Because the activity did not increase any more in trials that were presented at lower probabilities and had a delay of 1.5 s, the activity appeared to predict most probable time of FP offset. Same neurons usually did not respond to visually-guided saccades, but showed activity when the monkeys were required to make memory-guided saccades 800-1600 ms after the offset of a peripheral cue, rather than in response to the offset of FP. The results suggest that the delay period activity in the thalamus might signal timing of internally guided saccades.

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P1-231 Lesion of inferior olivary nuclei reduces abnormal dynamics of reflexive eye movements in a GluRδ2 knockout mouse

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A mutant mouse deficient in the glutamate receptor δ2 subunit (GluRδ2) (δ2-/-) shows impairment in synaptic regulation and motor discoordination. In this study, we analyzed their reflexive eye movements; vestibulo-ocular reflex (VOR) and optokinetic response (OKR). A δ2-/- showed a large phase delay in OKR, a high gain in VOR in the dark and a low gain in VOR in light. Ablation of floccular complex little affected the dynamics of eye movements in both wild-type mice (WT) and δ2-/-s. In contrast, lesion of inferior olivary nuclei (IO) by 3-acetylpyridine treatment substantially reduced abnormalities in δ2-/-s, and also affected the eye movements in WTs. These results suggest that IO play critical roles in the reflexive eye movements. We also compared the effects of lesions in WTs and δ2-/-s with the phenotypes of lurcher mice that have no Purkinje neuron.

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P1-232 The cerebellar floccular region is involved in vergence eye movements

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To pursue objects of interest moving slowly in three dimensional (3D) space, frontal eyed-primates use both smooth pursuit and vergence eye movements. Signals for both systems must be synthesized for pursuit in 3D. The cerebellar floccular region plays an important role in generating smooth pursuit. To investigate whether the floccular region is involved in vergence eye movements as well, we examined simple spike activity of 108 Purkinje (P) cells during both smooth pursuit and vergence eye movements in three Japanese monkeys. The majority of floccular P-cells (65/108=60%) responded to both. Twenty-eight (26%) responded only during vergence, and 15 (14%) responded only during smooth pursuit. These results indicate that the great majority of floccular P cells (86%) are involved in the control of vergence eye movements.

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P1-233 Effects of microstimulation of monkey cerebellar hemispheric lobule VI/VII on eye movements.

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Cerebellar vermis and flocculus/paraflocculus control reflex or voluntary eye movements, but little is known about the role of cerebellar hemisphere. We evaluated the effects of microstimulation of unilateral cerebellar hemisphere. After identifying eye movement responses by unit recording, we inject negative current pulse trains (≤50μA, 40 pulses). Either horizontal or vertical quick eye movements were evoked within hemispheric lobule VI/VII. These results, consistent with old reports using much larger currents in monkeys or cats, suggest that cerebellar hemispheric lobule VI/VII is involved in eye movement control.

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P1-234 GABAergic contribution to feedback-controlled and programmed convergence eye movements

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Human psychophysical studies proposed that the control system of convergence eye movement included both feedback and open-loop controllers. The neuronal basis of this hypothesis was sought in the lateral suprasylvian cortex (LS), one of the extrastriate visual cortex of cat where microstimulation evoked convergence eye movement. Cats (n=5) were trained by a set of stimuli: An approaching LED, and buzz sound preceding the target movement by 4sec. After training, open-loop convergence (convergence eye movement elicited after buzz sound but before the target onset) was found in 60% of trials. Then GABAA agonist, muscimol (4μg/μl,0.5μl x 3) injected locally into their LS. Amplitudes of ordinary convergence were significantly reduced by muscimol injection, while neither those nor probabilities occurence of open-loop convergence were changed significantly. The result suggests that GABAA inhibition plays a different influence on the neuronal circuits in the LS, controlling the ordinary and open-loop convergence.

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P1-235 Classification of the bursting neurons and their functional roles in the frontal eye fields (FEF).

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Cerebral cortex consists of many different types of neurons. In order to examine how these types of neurons operate in the cortex, we recorded neuronal activity from the frontal eye field (FEF) of monkeys performing a saccade task and classified neuron types by their firing patterns. We first classified the neurons whether they had burst episodes or not, and then classified bursting neurons by cluster analysis using interspike intervals and number of spikes within a burst. This analysis allowed us to classify the neurons into four types, regular spiking (RS), fast spiking (FS), fast rhythmic bursting (FRB) and intrinsic bursting (IB) neurons. We found that about 80 % of RS and FRB showed increased activities during visual cue and/or saccade periods, while about a half of FS showed decreased activities during saccade periods.

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P1-236 Visual responses of pursuit neurons in the caudal frontal eye fields (FEF) to motion-in-depth stimuli

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The majority of caudal FEF pursuit neurons discharge during vergence tracking. To understand whether they carry target-motion-in-depth signals, in this study we examined visual responses of pursuit neurons induced by spot- or pattern-motion-in-depth while the monkeys fixated a stationary spot. About half of them discharged for motion of another spot in-depth and also for pattern-motion-in-depth. Their preferred directions for visual responses were similar to those during vergence tracking. Neurons that exhibited visual response in-depth were mostly separate from neurons that showed visual response in frontal plane. Visual responses of the majority of these neurons exhibited sensitivity to the velocity of spot- or pattern-motion-in-depth. They discharged typically 20-40 ms before the onset of vergence tracking. These results suggest that the caudal FEF carry visual signals appropriate to be converted into motor commands for pursuit in three dimensions.

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P1-237 Online gain control for smooth pursuit based on the past experiences

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Previous studies demonstrated that a brief perturbation of a stationary target during fixation induces larger eye movement when primates pursue a moving target repeatedly than when they fixate a stationary target throughout the trials. We studied trial-by-trial changes of ocular responses to the perturbation of the target when human subjects were required to keep their eyes on the target whose movement was determined by a random sequence to elicit either fixation or pursuit. We found that the amplitudes of the ocular responses changed rapidly depending on the time series of the past events, i.e., preceding trials of fixation or pursuit decreased or increased the responses, respectively. The ocular responses were significantly correlated with the past 2 or 3 events. It suggests that the gain of visuo-motor transmission for pursuit initiation is modulated based on a few past events. This "online" gain modulation may contribute to flexible generation of movements suitable to the environment on which primates are facing.

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P1-238 Behavioral analysis of *fosB*$^{d/d}$ mice which express only ΔFosB and *fosB*-null mice lacking both FosB and ΔFosB.

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fosB gene, a member of *fos* family, produces FosB protein and its C-terminally-truncated form, ΔFosB protein by alternative splicing of its transcripts. The expression of FosB is rapidly and transiently induced in brains in response to various stimuli, while ΔFosB chronically accumulates in a specific region of brain after repeated or prolonged stimuli. To understand the biological significance of FosB and ΔFosB, we established *fosB*$^{d/d}$ mice which express only ΔFosB and *fosB*-null mice lacking both FosB and ΔFosB, by means of homologous recombination in embryonic stem cells. We comparatively examined the behavior of mutant and wild-type mice, and found that the exploratory behavior of mutant mice in a novel environment was significantly increased in comparison with wild-type mice, indicating that *fosB* gene products may regulate such activity.

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P1-239 Generation and analysis of mGluR1b-rescue mice

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mGluRs are G protein-coupled receptors activated by glutamate. mGluR1 presents alternative splice variants with differences in the length and sequence of their intracellular C-terminal domains. mGluR1b lack the long C-terminal domain specific for mGluR1a, which interacts with Homer and other scaffold proteins. We showed that the impaired synapse elimination, deficient LTD, and motor discoordination observed in mGluR1 KO mice were all restored in mGluR1a-rescue mice expressing the mGluR1a only in Purkinje cells. Now, we introduced the mGluR1b transgene into mGluR1 KO mice with a Purkinje cell specific promoter. The mGluR1b rescue mice showed apparently normal motor coordination, but impaired synapse elimination, suggesting that proteins interacting the C-terminal region of mGluR1a are involved in synapse elimination.

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P1-240 Development of spontaneous movement of the head and tongue in the mouse embryos lacking glutamate decarboxylase 67

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Development of the motor system is accompanied by spontaneous body movement at late embryonic stages. These activities were analyzed in the GAD67-knockout mouse (KO). *In utero* movement of the embryonic head and tongue recorded by an ultrasonic microscope was not detected at E13.5 and observed at 4-33 times/h in E14.5 wild type (wt). It was suppressed by picrotoxin injection into the lateral ventricle. In E14.5 KO mice the movement was absent or rare. When recorded from C4 ventral root and XII nerve of the isolated brainstem-spinal cord, two types of the discharges were obtained respiratory and body movement-related activities. These activities were infrequent and small in size at E13.5 but well developed at E14.5. The body movement-related activities occurred at 0.81 times/min in wt and at 0.12 times/min in KO at E14.5. Picrotoxin reduced their frequency. Thus GABA controls the activities of a rhythm generator in the embryos.

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P1-241 Neurons Regulating the Duration of Forward Locomotion in *Caenorhabditis elegans*

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The locomotory behavior of *Caenorhabditis elegans* consists of four simple events, forward and backward movements, omega-shaped turns and rests. The wide variety of behaviors of this worm is achieved through a combination of these simple locomotions. To gain insight into the neuronal mechanisms regulating this locomotion, we analyzed the locomotory behavior of *C. elegans* over a long time period. In this study, we revealed the existence of at least two distinct behavioral states - pivoting and traveling - in the forward locomotion of *C. elegans* in the absence of food. Pivoting is characterized by pronounced directional switching and resulting in short-duration forward movement, whereas in the traveling state, forward movement is of longer duration. We showed that, by laser-ablation, neuronal pathways consisting of nine classes of sensory neurons and four classes of interneurons were involved in this regulation. Loss of any one of these neurons altered the locomotory behavior.

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P1-242 Functional relationships of segmentally homologous hindbrain neurons in controlling escape behaviors of zebrafish

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In the zebrafish hindbrain, morphologically homologous reticulospinal (RS) neurons are repeated in adjacent segments. To elucidate functional significance of the serial arrangement of the RS neurons, we imaged their activities during escape behaviors in larval zebrafish and examined the effects of their ablation on the behavior. Confocal calcium imaging of the hindbrain RS neurons revealed that in most cases one of the paired Mauthner (M) cell in segment 4 fired at initiation of fast escapes in response to water pulse stimulus applied to the head. In some cases, however, fast escapes occurred without M-cell firing, in which onset of the behavior was slightly delayed and activities of the MiD3cm in segment 6, a homolog of the M-cell, were observed. Further, even after laser ablation of the M-cell, the fast escapes could be elicited but with stronger stimulus intensities and delayed onset. Thus, there may be duplicated circuits for initiating the escape behaviors, which consist of M-cells and their segmental homologs.

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P1-243 Effect of simultaneous presentation of two attractants on chemotactic response of *C. elegans*

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The chemotactic responses of *C. elegans* to simultaneous presentation of sodium acetate (Na^+) and diacetyl (DA) were examined. The fraction of animals that gathered at the 0.7 M Na^+ location was greater than that at the 0.1 % DA location in the presence of both attractants, although the chemotaxis indexes for 0.7 M Na^+ and 0.1 % DA were similar in the presence of a single attractant. In contrast, the fraction of animals that gathered at the 0.02 % DA location was greater than that at the 0.1 M Na^+ location in the presence of both attractants, although the chemotaxis indexes for 0.02 % DA and 0.1 M Na^+ were similar in the presence of a single attractant. These results suggest that the signals from different attractants are integrated in the neural circuit, and that the manner of the integration changes according to the concentrations of attractants.

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P1-244 Changes in phenotypes of locomotion with aging in wild-type and GABAergic mutants of *C. elegans*

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In the nematode *C. elegans*, the GABAergic neurons participate in regulating locomotion. We studied the differences in the phenotypes of locomotion of the wild-type N2 and two GABAergic mutants, the *unc-25* mutant that lacks glutamic acid decarboxylase, and the *unc-49* mutant that is deficient in a GABA receptor subunit, at each stage from young adult to senescence. The bend angles along the centerline of the body, which are approximately proportional to the curvature at each point, of the *unc-49* mutant, during forward movement on bacteria-free agar plates, were larger than those of the *unc-25* mutant and wild type animals. The bending frequency was lowest in the *unc-49* mutant, among the three strains. The total percentage of backward movement state in both mutants increased with aging.

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P1-245 Function of the LE type neuron in Aplysia food preference behavior

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Aplysia kurodai shows food preferences for various species of seaweeds. They feed on *Ulva* but reject *Pachydictyon* with distinctive patterned jaw movements. When the extracts were applied to the lips, the rhythmic patterned firing was induced in several neurons involved with feeding (jaw-closing JC motor neurons, MA neurons). We previously found that the MA firing activity decreased at each phase during the *Pachydictyon* response compared with the *Ulva* response. Therefore some pre-neurons may regulate the MA firing activity and contribute to a change of these responses. We found that firing of the previously identified LE type neuron shows plateau potentials and produces monosynaptic EPSPs in the MA and JC. During the feeding-like response inhibition of the LE spike activity with large hyperpolarizing currents stopped the MA firing. The LE may contribute to generation of the MA firing pattern during the ingestive response and to a change of the firing pattern in the food preference.

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P1-246 Sensory inputs modulate the frequency of electric organ discharges (EOD) in pulse-type weakly electric fish, Mormyrid.

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The weakly electric fish, *Gnathonemus petersii*, generates EOD according to the output of command neurons situated in the medulla. The electrosensory systems detect local distortions of the electric field as electrolocation, and broad distortions as electrocommunication. We examined how sensory stimuli changed the frequency of EOD in electrolocation and communication. Each fish had own specific frequency during free swimming. A metal rod increased frequency of EOD transiently, while with broad stimuli applied between the head and tail, the fish responded with a EOD tuned to the stimulus frequency, with a minimum latency of c.11 ms. An activator of GABA-A receptor decreased EOD frequency, but with a sharp tuning. Local injection of d-TC near the EO, neither responded to EOD frequency tuning nor fixed latency.

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P1-247 Attentional effect to the biological motion perception: An event-related potentials study

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In this study, we clarified how attention affects neural activities related to visual perception of biological motion (BM) with event-related potentials. Previously, we have found two negative components were elicited by perception of BM. We focused on the amplitude of the second negative component, which was sensitive to BM perception. 13 subjects observed BM or scrambled motion (SM; each point had the same velocity vector as BM, but the initial starting positions were randomized) stimulus with ten noise dots and four rectangles (one of the rectangles rotates 90° or not). Subjects were also required to perform two tasks; *attention-to-motion task*: to judge the motion types (BM/SM), *attention-to-rectangle task*: to detect the rotation of rectangles. We found two negative components (N200, N330) in both tasks. Particularly, N330 amplitude was modulated by attentional condition during BM stimulus was presented. The result implies that attentional process modulates BM perception and might be processed in top-down manner.

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P1-248 Accuracy of object motion perception during smooth pursuit

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Generally, retinal image motion involves a component caused by self-motion, which should be compensated to know real motion of the surroundings. A psychophysical experiment was done to study the capability of this compensation during smooth pursuit in human subjects. The subjects (N=4) were forced to choose rightward or leftward as the perceived direction of moving random dot pattern of large field ($\leq$10 deg/s rightward or leftward), which was presented for 200 ms when they were pursuing a target (5, 10 or 20 deg/s rightward or leftward). Psychometric functions of the probability of subject's choice were calculated as a function of background speeds, and then the compensation gain, i.e., background velocity giving the choice probability of 50% for both directions divided by target/eye velocity then subtracted from 1, was estimated. The averages of compensation gain were 127, 114 and 93% for pursuits of 5, 10 and 20 deg/s, respectively, suggesting that the accuracy of object motion perception depends on pursuit speed.

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P1-249 fMRI Analysis of Brain Activity on Three-Dimensional Structure from Rotational and Translational Motion

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We used functional magnetic resonance imaging (fMRI) to investigate human visual pathways underlying perception of three-dimensional (3-D) structure from motion evoked by two-dimensional (2-D) rotational or translational motion. For 3-D structure-from-motion (SFM) by a rotational motion stimulus, the parieto-occipital area, especially V3A, the intraparietal sulcus (IPS) and the inferior parietal lobule (IPL), and a part of MT+ activated. On the other hand, for 3-D SFM by a translational motion stimulus, the temporal area, especially the superior temporal sulcus (STS), activated. It is suggested that the separated pathways contribute to 3-D SFM by rotational and translational motion.

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P1-250 Active integration of local motion signals revealed by coherency among multiple multistable motion stimuli

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To observe the interaction of local motion signals over time and space, the direction of perceived motion was tested for a multistable motion stimulus that was surrounded by similar stimuli having various temporal phases and spatial positions. For the multistable stimulus, two spots on diagonally opposite corners of a square and those on the remaining corners were presented alternatively, and four types of motion could be perceived. Both the relative temporal phase and spatial position of surrounding stimuli influenced the strength of motion coherency among the stimuli, and strong motion capture was observed in some conditions. It is well known that when spatially separated spots move coherently, we perceive a structure or shape for the group of the spots. In this study, we revealed that this process is not passive. To find some structure or shape from motion, our visual system actively searches for local motion signals that can be grouped, and attracts unstable ones to the group to produce a coherent motion field.

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P1-251 Are orientation maps formed even under the dark rearing condition ?

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To obtain a better understanding of the effect of visual experience on orientation map formation, we conducted optical recording of intrinsic signals from areas 17 and 18 of anesthetized cats reared in complete darkness. Orientation maps appeared in the region which would be anatomically identified as area 18 in normally-reared cats. Orientation selectivity in this region was lower than that of area 18 in normally-reared cats. On the other hand, orientation maps were not observed in the region which would be anatomically identified as area 17 in normally-reared cats. These findings indicate that visual experience assists the formation of orientation maps in area 17, whereas some intrinsic mechanisms contribute to the formation of orientation maps in area 18 and visual experience promotes further maturation of the maps that have appeared before the onset of visual experience.

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P1-252 3-D surface orientation sensitivity in area 18 of the cat.

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How are 3-D surface orientations represented in the visual cortex ? We have examined an idea that tilt of binocular receptive field (RF) structure of A18 neurons encode 3-D surface orientations. Binocular RFs of complex cell were measured by a reverse correlation method. We have found binocular RF profiles with tilts off the front-parallel plane that are not due to recording noise or experimental artifacts. This suggests that neurons in A18 are sensitive to 3-D surface orientations. We examined two possible mechanisms for coding surface orientation. First, output of neurons tuned to different disparities may be hierarchically combined across spatial locations. Second, preferred spatial frequency(SF) of subunits may differ across the eyes. Although there are many instances of a substantial difference in the optimal SFs for the two eyes, the degree of the difference was not correlated with the tilt observed in the binocular RFs. These results suggest that tilt of binocular RFs may be due to a hierarchical organization.

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P1-253 Spatial pooling of subunits in complex receptive fields in the cat area 18

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Complex cell receptive fields are known to be constructed from spatially pooled simple-cell-like linear subunits. However, the extent of this pooling and number of subunits involved are not known for area 18. To address this issue, we have estimated the size of subunit receptive fields and compared with that for complex cell receptive fields. The width and height of complex cell receptive fields were obtained using reverse correlation methods. Next, the width and height of subunit receptive fields were estimated from the inverse Fourier transforms of spatial frequency tuning profiles along the respective dimensions. We find that the receptive fields of complex cells are larger by a factor of 1 to 16 in areal terms than subunit receptive fields. These results suggest a high degree of spatial pooling in the hierarchical organization of complex cell receptive fields in area 18.

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P1-254 Effect of discrimination training on orientation representation in V1 of a monkey.

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In the primary visual cortex (V1), receptive field plasticity can be induced by intracortical microstimulation and passive exposure in anesthetized preparations and environmental manipulation in developing animals. However, whether V1 neurons show such changes following normal learning as reported in the primary somatosensory and auditory cortices is still controversial. To investigate this issue, I recorded an intrinsic signal from V1 of a monkey while the animal performed orientation discrimination. Before introducing discrimination training, the resultant image showed a typical orientation map. After the discrimination training began, however, the cortex showed appreciable modification of orientation preference. Such a change occurred globally across cortical surface and typical orientation map disappeared. In addition, cortical response to the negative stimulus was consistently higher than that to positive stimulus even after discrimination condition was reversed. The results suggest that V1 is involved in behavioral task.

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P1-255 Contrast-dependency of contextual modulation in the cat visual cortex

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In neurons of the primary visual cortex (V1), responses to stimulation of their classical receptive field (CRF) are suppressively modulated by stimuli at the receptive field surround (SRF). As an attempt to understand the behavior of the contextual response modulation, we assessed the contrast-dependency of the modulation in cat V1. First, we investigated the relationship of contrast sensitivity of CRF response and SRF effect, and found that there was no correlation between them although there was a significant correlation for parameters of orientation or spatial frequency. Second, by controlling the contrast of CRF and SRF stimulus independently, we checked if the modulatory effects by SRF stimulus change according to CRF response level. Then we found that there exists the mechanism of shifting the effective contrast range of SRF stimulus depending on the CRF response level. Also we will report the results of geniculate neurons, and discuss a possible origin of modulatory effects seen in V1.

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P1-256 Comparison of contextual modulation by the receptive field surround between V1 and LGN in the cat

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It is well known that neural responses to the receptive field(CRF) stimulation are suppressively modulated by a stimulation of receptive field surround(SRF). To examine underlying mechanisms, we investigated this contextual modulation and compared the properties and strength between V1 and LGN. Eighty-eight LGN neurons and 73 V1 neurons were recorded, and circular variance of orientation tuning of CRF responses and SRF effects was calculated for each neuron. In V1, most neurons exhibited the sharp orientation tuning in CRF responses, but the tuning of SRF effects was broader and weaker than that of CRF response. In LGN, even though neurons did not show orientation selectivity, a population of neurons exhibited orientation-tuned surround suppression which is comparable to that in V1. Our results suggest that the orientation-tuned contextual modulation already exists in LGN which will be enhanced in the network in V1.

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P1-257 Shape selective activities recorded in the area MT of macaque monkeys

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We investigated the involvement of the area MT for the shape discrimination in the shape from motion (SFM) condition, in which shapes were made visible by dots within invisible contours of shapes moving coherently in one direction. Monkeys performed a delayed match-to-sample task in which one of 4 shapes was presented centrally as a sample stimulus, and after a delay period the same shape was presented peripherally as a target with one distracter presented in the opposite. These shapes were defined by moving dots against static dots. Thirty two percent of MT neurons showed shape selectivity and 37 % of them showed higher activity to targets than to distracters. The results suggested the possibility of contribution of the area MT in the shape perception in the SFM condition.

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P1-258 FMRI analysis of color contrast processing in the human visual cortex

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Color appearance depends on chromatic context. Red looks much redder when surrounded by green than by red. We used fMRI to explore human visual areas underlying the color contrast effect. Two subjects were scanned while viewing equiluminant red-green flicker stimuli consisted of two pie-shaped test disks (3° - 11°) and a surround (0° - 15°). Chromatic context was either opposite-phase (OP) or in-phase (IP). The apparent chromatic change of the test was larger for OP than for IP. We observed fMRI responses correlated with the perceptual color change in most of lower and higher visual areas. When the gaps (0.5° or 1°) were larger between the test and surround, the responses of lower visual areas were weaker while those of some higher areas were not. The spatial extent of color contrast effect increases with the hierarchy level of the visual cortical pathway.

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P1-259 Specialization for color processing in the inferior temporal cortex of the monkey

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Lesion studies have suggested that inferior temporal (IT) cortex of the monkey plays a key role in color discrimination. To study the functional specialization within IT cortex with respect to color processing, we recorded neuron activities from IT cortex of awake monkeys. Recording site of each neuron was identified based on the depth profile and MRI image of the cortex as well as X-ray photos taken at each penetration. We tested color selectivity of each neuron using a set of colors systematically distributed in the chromaticity diagram. We also tested shape selectivity using a set of geometrical patterns. We found that neurons having strong color selectivity but with little shape selectivity were concentrated in a restricted region in the anterior part of IT. We identified such a region lateral to the posterior part of the anterior middle temporal sulcus (AMTS). We suggest that color processing in IT cortex is achieved at specialized subregions and the region we observed is one of these.

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P1-260 Color selectivity and functional connectivity of macaque V1 neurons: simultaneous recording from adjacent cells

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Preferred colors of V1 cells as a population cover a wide range across different chromaticities, while those of LGN cells are confined to the red-green and blue-yellow axes in the color space. It is unclear how this change in color representation occurs. In order to address this question, we studied the local clustering and neuronal connections of color-selective V1 cells by recording visual responses simultaneously from multiple adjacent cells in the macaque monkey. Adjacent color-selective cells tended to prefer a similar color. No evidence, however, was obtained that cells with similar color selectivity form columnar clustering. A pair of adjacent cells with a common input had a tendency to have similar preferred colors, while a pair having an inhibitory connection had different preferred colors. The results suggest that neuronal interactions among adjacent cells contribute to color selectivity of V1 cells.

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P1-261 Hemispheric dominance in color discrimination using reaction time task

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We have previously shown that the right hemisphere was dominant in color detection, while the left hemisphere in color discrimination using verbal cues. In the present study we examined the cerebral dominance in non-verbal color discrimination. Eight right-handed subjects were required to press a key when two stimuli were different. The stimuli were red, green or blue circles presented either in the right or left visual field or achromatic ones (12, 14, 18 cd/m^2). Subjects pressed the key by their ipsilateral hands to the stimuli presented on a display. The discriminative reaction time (DRT) to chromatic stimuli was faster in the left hemisphere, however the difference was not significant. The net discrimination time which was calculated by subtracting simple RT from DRT was significantly faster in the left hemisphere. On the other hand, there was no significant difference in achromatic discrimination time. These results suggest that the left hemisphere is dominant in color discrimination.

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P1-262 Color induction contributes to color constancy in the perception of color transparency

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Color transparency is a phenomenon under which color constancy works. In order to clarify how this color constancy works, the contribution of color induction was tested. A circular induction stimulus was presented in various sizes at the surround of a circular target stimulus. The color of the target and induction stimulus was determined by simulating a situation under which a small circular spot was seen through a large circular transparent filter having 50% transmittance. Subjects adjusted the color of a test stimulus until it appeared to be the same as that of the target stimulus. The difference between the color of the target and test stimulus increased with an increase in the radius of the induction stimulus. This difference became larger when the feeling of transparency was enhanced by an auxiliary stimulus. It became clear that the strength of color constancy in transparency perception changes with spatial composition that induces color induction, and color induction additively works with color transparency for color constancy.

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P1-263 The representation of occluded object: an fMRI Study

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We can readily perceive the unity of objects that are partially obscured by occluding surfaces. We tested the possibility that the occluded structure was represented retinotopically somewhere in the visual cortex, using fMRI. Target stimuli were rotating wedges. In a test condition, they passed behind occluders. In a control condition, the occluders & the occluded segments of the wedges were removed. These stimuli caused a transitive neural activity. In V1 & V2, the phase topography consistent with the rotation of the occluded wedges was not observed. But, in V3 & V3A, the phase tended to transit in the regions which retinotopically represented the occluder as if the occluded wedges were interpolated. It is suggested that the structural completion may originate from the middle stage of the visual pathway.

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P1-264 Dose competitive mechanism contribute to the ocular dominance plasticity in the pharmachologically inhibited visual cortex ?

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Monocular deprivation (MD) during the critical period causes a significant retraction of geniculocortical arbors serving the deprived eye. Competitive mechanism between inputs from two eyes underlies this ocular dominance plasticity because the effect of binocular deprivation (BD) is less significant than that of MD. When MD is combined with chronic inactivation of the visual cortex by muscimol, GABAA receptor agonist, active arbors serving the open eye selectively retract while the branches of arbors from the deprived eye remain mostly intact. It is unclear whether these morphological plasticity in opposite directions share similar mechanisms. To address this question, we tested whether the plasticity in the inhibited cortex had competitive mechanisms by analyzing the effect of normal vision and BD on the morphology of arbors in the inhibited visual cortex.

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P1-265 Effect of binocular rivalry on the perception of depth from binocular disparity

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When a visual image presented to one eye is completely different form that to the other, we experience perceptual alternation between the two images. This phenomenon is called binocular rivalry, and is supposed to play some role for the fusion of two correlated images from two eyes leading to the perception of depth. In this study, we examined the interaction of binocular rivalry and stereo-depth perception. As a rivalry stimulus, we presented a vertical grating for one eye and a horizontal grating for the other. A random-dot stereogram overlapping the gratings was presented simultaneously. Subjects reported perceptual alternations among three perceptual states, that is, the perception of vertical grating, horizontal grating, and both. The stability of perception was affected by varying the contrast of the gratings, and the correlation of two random-dot images constituting the stereogram. This result suggests suppressive interaction between the state of binocular rivalry and fusion.

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P1-266 Looming selectivity of cells in the early visual cortex

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Motion-in-depth is an important cue for animals living in a 3D world. Although binocular aspects of motion-in-depth sensitivities have been studied in early visual cortices, relatively little is known about the neural basis of monocular cue representations. Objects that are moving in depth relative to an observer cause changes in apparent size and associated changes in the spatial frequency of object's visual features. To study possible mechanisms by which motion-in-depth information is represented, we have examined the selectivity of cells in cat area 17/18 to sequences of multiple spatial frequencies. There were cells that prefer successive presentations of different spatial frequencies to sequences of identical frequency. Of these, a majority preferred high-then-low frequency sequence rather than the opposite, i.e., they seemed to prefer the looming of objects. Our results suggest that the cells in the early visual cortex can be neural substrates for extracting the monocular motion-in-depth cue.

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P1-267 Synchronous transition between two activity states of neurons in area TE

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In the previous study, we showed that a broad peak (half peak width 125ms) appears in cross-correlogram of spike trains of TE neurons at 0 time delay. In this study, to reveal origin of the broad peak, we (1) calculated auto-correlograms of neurons, (2) compared them with cross-correlogram of the neurons, and (3) applied a regression analysis to cross-correlogram. An auto-correlogram consisted of a sharp peak at the center (0 time delay) and a gradual decay component from the center. This decay component well overlapped with the broad peak in the cross-correlogram that was well fitted with an exponential function (time constant 120ms). These results are explained by assuming each neuron takes two activity states: states with high and low firing activities. State transition is random. Transitions of two neurons, however, are well synchronous, leading to the broad peak in the cross-correlogram. The absence of the sharp peak in the cross-correlogram could be explained by no precise spike timing synchronization.

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P1-268 Spiking neural network model of the primary visual cortex: contrast and stimulus length dependence of neuronal responses

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We conducted computer simulation of cortical dynamics based on a large-scale neural network model composed of spiking units receiving self-organized geniculo-cortical inputs. Firing rate of model cortical cells decreased when the stimulus bar was lengthened beyond the size of the classical receptive field. This end inhibition disappeared when inhibitory neurons were inactivated. Firing rate of the cells in response to different contrasts of grating stimuli exhibited sigmoidal behavior. The saturation of the responses to higher stimulus contrast was attributed to the suppression of activities by inhibitory neurons. Cortical domains activated by a given orientation did not change when the stimulus contrast changed, indicating that orientation tuning of cortical neurons are contrast-invariant. Taken together, it is suggested that the balance between excitation and inhibition serves to realize end inhibition and contrast invariance of neurons in the primary visual cortex.

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P1-269 Topological analysis of GABAergic neurons in optically-recorded primary visual cortex

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Parvalbumin (PV)-containing GABAergic neurons targeting perisomatic domain of pyramidal cells form dual networks through both mutual inhibitory synapses and gap junctions (Fukuda and Kosaka, 2003) and are assumed to be involved in oscillation/synchronization. Here we examined spatial relationship between the PV neuron networks and orientation columns in area 18 of the cat. Serial horizontal sections were cut through the region where optical recording had been performed. Single, reconstructed PV neurons had dendritic fields of several hundred microns in diameter along cortical surface. Their horizontal dendrites freely traversed different orientation domains and made gap junctional coupling with others in EM. All PV neurons located under the recorded region were mapped and found to be distributed densely in a random fashion to make interconnection of horizontal dendrites across different orientation columns, possibly relating to broad synchrony.

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P1-270 Comparison of Functional Maps Revealed by *in vivo* Voltage-sensitive Dye Imaging and Optical Intrinsic Signal Imaging in Monkey IT Cortex

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Optical intrinsic signal imaging (OISI) has revealed that an object image activates multiple spots of columnar size (~0.5 mm)("activity spots") in macaque area TE (Tsunoda et al., 2001). On the other hand, voltage-sensitive dye imaging (VSDI) has demonstrated that the object images activate an area larger than the size of these activity spots. These activity spots and the larger active areas mainly reflect the spiking activity and synaptic potential, respectively. To investigate the spatial relationship between these two distinct activity patterns, in this study we recorded both patterns from identical regions of cortex and compared them.
Activity spots coincided with local peaks of VSD signal except for a few mismatches caused by systematic artifacts. The onset latency of VSD signal at those peaks was shorter than in the surrounding region. This result suggests that the activity spots may correspond to the sites that receive convergent inputs from preceding areas in the ventral visual pathway.

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P1-271 Intra-pulvinar long distance intrinsic connections

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The pulvinar, like other thalamic nuclei, is thought to have only local intrinsic connections. Injections of tracers within medial pulvinar (PM) of macaque monkey, however, provide evidence for more extensive intra-pulvinar connectivity. First, injections of fast blue (n=1) or biotinylated dextran amine (BDA; n=3) result in retrogradely labeled neurons. These are in the vicinity of the injections, but also up to 1.3mm distant. Neurons had multipolar or small spherical somata. Second, axon segments anterogradely labeled by BDA could be followed to the injection site (n=3) or to retrogradely labeled cells (n=6). Neurons had multipolar or small spherical somata. Axons extended for 2.0-3.5mm from the soma, with fine beaded specializations after the proximal 1.0mm. These were confined within PM (n=3), or continued into the adjoining lateral pulvinar (n=4). At least two segments additionally had a collateral going toward the cortex. We suggest that intra-pulvinar connections constitute an important additional associative layer in the pulvino-cortical network.

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P1-272 Retinal target cells of the isthmo-optic projection

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The avian retina receives projection from the isthmo-optic nucleus (ION). We labeled the isthmo-optic (IO) terminals in the Japanese quail retina using anterograde transport of fluorescent tracer injected into the ION, and then labeled target cells for these terminals by means of intracellular tracer injection. Somata of the IO target cells (IOTCs) lie in the innermost zone of the inner nuclear layer of the ventral half retina, and have no dendrites but an axon. The axons run in the inner plexiform layer (IPL) for up to 6 mm and terminate densely in a round or elliptical terminal field, about 90-290 μm in diameter, of the outermost zone of the IPL. Longer axons (> 2 mm) extend dorsally, but shorter ones (< 1 mm) project ventrally or horizontally, and therefore the terminals are distributed widely in both dorsal and ventral halves of the retina. The IOTCs are thought to be "slave" neurons whose output is controlled by the neurons in the brain.

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P1-273 Glutamate responses of the retinal neurons recording with voltage sensitive dye

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The photo information processing in vertebrates retina starts from the release of the glutamate from the photoreceptor cells. Therefore, it can know the mechanism of information processing in the retina by recording the response to extringic application of the glutamate. After making transverse sections of the dace retina, they were stained with voltage sensitive dye (Nihon Kanko Shikiso, NK-2761) and they were put on the microscope stage. A small amount of monosodium glutamate was applied through the glass micro pipette to the each layer of the retina. The responses were recorded as the optical density changes depending on membrane potential. The depolarization was recorded in the horizontal cells when the glutamate was applied to the outer plexiform layer. The depolarization was observed in the inner nuclear and plexiform layer. In some cells in the inner nuclear layer, hyoperpolarizing responses were recorded. It is thought that the mechanism of the photo information processing can be clarified by detailed identification of the cells.

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P1-274 Distribution of leukotriene A_4 hydrolase in the visual and other sensory systems

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To determine a novel neural function of leukotriene (LT) B_4, we studied the distribution of LTA_4 hydrolase (LTA_4H) in the mouse nervous system by immunohistochemistry. LTA_4H showed a characteristic distribution in the visual system. In the retina, amacrine, bipolar, and ganglion cells were immunopositive; furthermore, inner plexiform layer showed a distinctive three-layer pattern. Fibers in the optic nerve and tract were also immunopositive, ending in the superior colliculus. Other sensory structures, such as neurons in the vomeronasal organ, olfactory glomerulus, inner hair cells, spiral ganglion cells, nerve fibers from a vestibular organ, and neurons in the spinal posterior horn, were also positive for LTA_4H. This expression pattern of LTA_4H may suggest a modulatory function of LTB_4 in the visual and other sensory systems.

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P1-275 The Effect of Triamcinolone Acetonide to Retinal Pigment Epithelial Cells by Oxidative Stress

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Age-related macular degeneration (AMD) is a major cause of legal blindness in industrialized nations. The most severe form of AMD is characterized by choroidal neovascularization (CNV). Recently, the therapy of CNV has been tried by triamcinolone acetonide (TA). By this therapy, CNV is diminished, and surrounded by fibrotic cells. However, this mechanism is unknown. In this presentation, we try to propose one of the possible mechanisms of this therapy. Oxidative stress to Retinal Pigment Epithelium (RPE) is one of the causes of AMD, so we used the system of ARPE19 cells stimulated by oxidative stress, hypoxia/re-oxygenation and paraquat (Neuro2001). The changes of the expressions of bioactive substances in oxidative stress stimulated ARPE19 cells by TA were examined using real time RT-PCR analysis and Western blot analysis.

P1-276 Expression of reelin in the dorsal cochlear nucleus of the mouse

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The dorsal cochlear nucleus (DCN) has a distinct laminar structure similar to the cerebellar cortex. Such a laminated structure of this nucleus is cytoarchitectuallly disrupted in the Reelin-deficient mouse, *reeler*. We have studied expression of Reelin protein in this nucleus during postnatal periods to elucidate the functional role of Reelin protein. Anti-Reelin immunopositive products are recognized in the external granular layer and granular layer of DCN during the first postnatal week. Later, neuropil in the molecular layer and fusiform cell layer is immunostained with Reelin-antibody. Next, we examined cytoarchitecture of the reeler DCN with CaMKII alpha immunohistochemistry. CaMKII alpha-immunoreactive cartwheel cells are laminarily distributed in the fusiform cell layer in the normal mouse, but scattered throughout the DCN in the reeler. In conclusion, Reelin may have some roles in laminar arrangement of cartwheel cells.

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P1-277 Activation of muscarinic receptor activation induces hyperpolarization in granule cells and Golgi cells of the mammalian cochlear nucleus

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In mammalian dorsal cochlear nucleus, principal cells receive synapses not olny from auditory nerve fibers but also from parallel fibers which consist of axons of granule cells and convey non-auditory information. As in the cerebellum, granule cells have mutual synapses between inhibitory interneurons, Golgi cells. From morphological studies, it is suggested that both cells might be innervated by cholinergic efferents from the ventral nucleus of the trapezoid body. As expcted, cholinergic agonist, cabachol, induced membrane hyperpolarization with a decrease of input impedance in both cells. Muscarine evoked similar responses. These results indicate that this membrane hyperpolarization is mediated through muscarinic acetylcholine receptors.

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P1-278 c-Fos expression in rat brainstem evoked by aoustic stimuli

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It has been shown that loud acoustic stimuli simulate vestibular nuclei in rat brain stem. This time, we would like to describe the motor nuclei in which c-Fos protein induced after acoustic stimuli.Adult Wister rats received click stimulation of 90 dB above ABR threshold for 40 minutes. After stimulation, rats were perfused and fixed using Karnovsky' solution. Frozen brainstems of the rats were cut into sections and immunohistochemically stained for c-Fos protein.Both he auditory pathway and vestibular nuclei showed strong induction of c-Fos protein. In addition, c-Fos protein induction was observed in the motor and spinal tract nuclei of trigemimal nerve, the facial nucleus, the glossopharyngeal nucleus, the ambigus nucleus and the hypoglossal nucleus and few in III, IV, and VIth nerve. These finding may indicate the etiology for the phenomenon that loud acoustic stimuli evoke a blink reflex or myogenic potentials in neck, and the difference between two column of motor nuclei in brainstem.

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P1-279 Firing properties of glutamatergic and GABAergic neurons of the rat medial vestibular nucleus

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Our previous study using whole-cell patch clamp technique combined with RT-PCR analysis in slices of the medial vestibular nucleus demonstrated that neurons exhibiting delay in spike generation were dominant in GABAergic neurons, whereas neurons exhibiting repetitive firings with constant interspike interval were dominant in glutamatergic neurons. In the present study, we investigated firing properties of the identified glutamatergic and GABAergic neurons in response to ramp current stimulation. Increase in firing rate in response to ramp stimulation in glutamatergic neurons was larger than that in GABAergic neurons. The time differences in the period of repetitive firings between ramp-up and ramp-down phases were small in glutamatergic neurons, whereas the various time differences were observed in GABAergic neurons. These results suggest that firing responses of glutamatergic neurons are relatively linear, whereas those of GABAergic neurons are modified in a state-dependent manner.

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P1-280 Context Dependency on the Interaural-Phase-Difference Representation by Single Neurons and by Neural Populations in the Inferior Colliculus

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We examined the stimulus-history sensitivities of the representation of the interaural phase difference (IPD) in the inferior colliculus (IC). We recorded single-unit responses from the IC of anesthetized gerbils. The stimulus was a dichotic best-frequency tone sequence, consisting of the conditioner (200-ms) followed by the probe (50-ms). The IPDs of the two tones were varied independently. The overall strengths of the probe-driven responses tended to decrease as the conditioner IPD approached the units' best IPDs. Next, we evaluated the conditioner effects on the neural-population representation of IPD (ϕ), defined as the response-weighted average of best IPDs across units. For a given conditioner IPD, the ϕ tended to shift in such a way that would emphasize the contrast of IPDs between the conditioner and the probe.

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P1-281 OLIVOCOCHLEAR EFFERENT FIBERS IN CULTURE OF SLICES OF SUPERIOR OLIVE COMPLEX

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Cochlear hair cells are innervated by efferent nerve fibers from neurons in the superior olive complex (SOC) in the brain stem. To examine whether the cochlear efferent neurons are capable to regenerate nerve fibers in tissue culture, we dissected slices from the brain stem of embryonic or newborn rodents and cultured in a serum-free medium. To verify that the SOC was contained, the slices were prepared so that the roots of facial and abducent nerves were attached (SOC was located between the two roots). When cultured in BDNF-enriched medium, numerous nerve fibers (as identified by immunostain against neurofilaments) were emerged from the slices. Fibers from the SOC were identified by the topographical relationship. Nerve fibers were reduced in BDNF-free cultures, while some long fibers were grown in NGF- or NT3-enriched ones. GABAergic or cholinergic fibers were differentiated by immunostain against glutamic acid decarboxylase or choline acetyl trasferase.

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P1-282 Innervation patterns of afferents in the utricular macula projecting directly to the abducens nucleus in the cat

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Morphological and physiological studies have demonstrated that primary utlicular (UT) afferents project to the ipsilateral abducens (AB) nucleus (Korte 1979, Imagawa 1995) and activate motoneurons and interneurons monosynaptically (Uchino, 1994, 1996). To understand the functional role of the monosynaptic connections between UT afferents and AB neurons, biotinylated dextran amine (BDA) was injected to the AB nucleus. Calyx afferents innervated type I hair cells with calyceal terminals that contained 1 to 4 receptor cells. Five calyx afferents were located in the caudal one third of the UT macula. The other 2 calyx afferents were located in the rostral part of the macula. Bouton afferents innervating the type II hair cells in the bouton terminals were located in the caudal half of the macula. The AB motoneurons and interneurons may receive a wide directional linear acceleration from the ipsilateral UT macula.

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P1-283 Descending auditory pathways through the nucleus anterior tuberis in cyprinids

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The nucleus anterior tuberis (NAT) in teleosts is known as a diencephalic auditory nucleus, which receives auditory projections from the nucleus centralis of torus semicircularis (TSc). In the present study connections of the NAT were studied in detail by tract-tracing methods in cyprinids. The NAT receives abundant descending projections from a telencephalic auditory region (the area dorsalis telencephali pars medialis), but does not send ascending fibers to the telencephalon. The NAT is connected reciprocally with other diencephalic auditory nuclei (nucleus preglomerulosus pars anterior and nucleus dorsomedialis thalami) and mesencephalic auditory nuclei (TSc and nucleus perilemniscularis). The present results suggest that the NAT is an important auditory center interconnected with mesencephalic and diencephalic auditory nuclei and mediates telencephalic influence to these nuclei, presumably to control ascending auditory pathways.

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P1-284 Long-Term Enhancement of Excitability in a Type of Neuron in the Cerebral Cortex

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We investigated whether activity-dependent modifications of intrinsic excitability can be induced in neurons in the cerebral cortex. Using whole-cell recordings in slice preparations, we found that fast-spiking neurons in the rat auditory cortex showed long-lasting increases in their excitability by high-frequency stimulation of excitatory inputs. After high-frequency stimulation, the number of action potentials elicited by a constant current pulse was significantly increased. Potentiation of synaptic potentials often occurred with the change in excitability but lasted much shorter than the increase in the number of spikes. These results suggest that intrinsic excitability can be changed rapidly by input activity in fast-spiking neurons in the cerebral cortex.

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P1-285 Trans-cranial imaging of tonotopic map plasticity in the mouse auditory cortex

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Flavoprotein autofluorescence imaging is useful for visualizing mouse cortical activities, because the transparent skull allows trans-cranial imaging. In this study, neural responses in the auditory cortex elicited by sound stimuli (5-20 kHz for 500 ms) were visualized in mice anesthetized with urethane (1.6 g/kg, i.p.). The responses in the auditory cortex exhibited tonotopic map, as reported previously. Electrophysiological studies have demonstrated that the tonotopic maps can be modified by environmental sounds. We exposed young mice to environmental stimuli (10 kHz, 70 dB) and visualized the plasticity of the tonotopic map. The responses to the sound used as the environmental stimuli were augmented and the response area was expanded. The present study confirmed that acoustic environments are critical for the development of the auditory cortex and the trans-cranial imaging is useful for investigating mouse cortical plasticity without surgical damages on the brain.

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P1-286 Roles of the auditory cortex in sound pitch discrimination in rats

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Sound pitch discrimination could be performed based on absolute pitch or relative pitch. To clarify this point, rats were trained to discriminate between the rewarded sound (S+) of 10 kHz and the unrewarded sound (S-) of 5 kHz on consecutive two days. On the 3rd day, the discrimination between 10 kHz (S+) and 20 kHz (S-), or 20 kHz (S+) and 10 kHz (S-) was tested. The former performance was better than the latter, suggesting the importance of absolute pitch. In the experiments using sounds of 7, 10 and 14 kHz, the importance of relative pitch was suggested. These experiments were performed in the rats with bilateral lesion in the auditory cortex. Although the performance on the 1st and 2nd days was not clearly affected, the performance on the third day was suppressed. These results suggest that absolute pitch and relative pitch were used depending the bandwidth. The auditory cortex might have important roles in re-learning process after changing discrimination paradigm.

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P1-287 Optical imaging of responses to gap- and noise-inserted tones in primary and belt fields of guinea pig auditory cortex

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To study neural mechanisms of temporal induction of sounds, neural activity to 4-kHz pure tones separated by a gap (PGP), white noise (PWP) and band-stop noise (noise without 4 kHz) (PBP) was measured in the primary (AI) and belt fields of the auditory cortex of guinea pigs anesthetized with ketamine using an optical recording technique with a voltage-sensitive dye (RH795). In the AI and belt fields, a phasic excitatory response (R1) was observed at the onset of PGP, PWP and PBP. For PGP, a second phasic response (R2) was observed after the gap when the gap was longer than 60 ms. R2 was inhibited for PWP and the inhibition of R2 was stronger in the belt fields than in the AI. For PBP, the inhibition of R2 was much less than that for PWP. These results suggest that the inhibition of R2 needs a 4-kHz frequency in the noise and this inhibition contributes to temporal induction of sounds.

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P1-288 Neuronal Response to auditory-induction tasks in the primary auditory cortex of guinea pigs

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Auditory induction is the perceptual restoration of missing sounds, which occurs when an appropriate sound is inserted into the gap of silence. This study examined the single-unit neuronal responses to auditory-induction tasks in the primary auditory cortex (AI) of guinea pigs. The tasks consisted of three tones. (1) A long pure tone. (2) A long pure tone with a gap, called inducee. (3) A relatively louder pure tone, called inducer. When the inducer was inserted into the gap of the inducee, it was observed that the ON-response due to the onset of inducee after the gap was attenuated. This response is reduced when the frequency difference between the inducee and the inducer decreases. This result suggests the ON-response amplitude variation in AI to be a possible neuronal basis behind auditory induction.

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P1-289 Neural activities in gerbil auditory cortex responding to sounds related to communication

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In order to examine how animals process data from their sensory organs in real time, this study observed responses to frequency modulated (FM) tones in the auditory cortex of Mongolian gerbils (*Meriones unguiculatus*). Gerbils have their own communication sounds which are mainly FM tones. Sound stimuli used in this study were systematically synthesized by imitating their FM tones. Responses to these stimuli were recorded from the anaesthetized gerbils. From these responses, PSTHs (peristimulus time histograms) were made and compared each other. Some recording sites showed variety of time-response patterns. For example, some neurons responded to a certain sound stimulus with relatively short latencies (about 30ms). However the same neurons responded to different sound stimuli with long latencies (more than 120ms). These results suggest that responses of auditory cortical neurons have different time structures for different types of sounds.

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P1-290 Reversible inactivation of neural activities in gerbil auditory cortex by near-infrared laser.

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A low-level laser irradiation system with a near infrared wavelength has been recently developed to reversibly inactivate neural activities. The system can irradiate focused area and make quick inactivation/recovery process. By using the system we investigated if temporal recovery pattern depended on stimulus frequency. At the same time activities of closely located different neurons were examined. Data showed that recovery pattern depended on frequencies and closely located different neurons presented totally different recovery patterns.

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P1-291 Input output connections and neural activities in the HVC of Bengalese finches

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Male Bengalese finches (BF) sing a complex song which follows the finite state syntax. The song control system in BF brain consists of a set of discrete nuclei including HVC, RA and Area X. The HVC, a higher order song control nucleus, receives auditory inputs and sends axons to both the song motor pathway (RA) and the song learning pathway (Area X). A tracer study showed that about 3-7 neurons formed clusters in HVC, and that these clusters were distributed throughout the nucleus. We also observed that a number of the clusters innervated both RA and Area X. In addition, large bodied neurons in HVC and the majority of RA neurons were AchE positive. We recorded multi unit activities in HVC using 1-3 tetrodes while playing back the bird's own song. Several types of song responding neuron activities were identified. Further, the neural activities were correlated to various features of the song sequences.

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P1-292 Comprehensive classification of Drosophila auditory sensory neurons

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To clarify the mechanisms how animals process acoustic information, we started a comprehensive mapping of auditory neural network in the Drosophila brain. In its first step, we analyzed the innervation patterns of the Johnston's organ neurons (JONs), the insect auditory receptors located in the antennae. By screening the GAL4 enhancer-trap strains that exclusively label JONs in the antennae, we found that the terminal arborizations of JONs spread over broad area of the so called "antennal mechanosensory region" in the brain. Visualization of single JONs using the heat shock-flip out system revealed, however, that each JON innervates only a small subarea. Comparing the projection patterns, we divided the region into eleven subareas and classified JONs into three types according to the combination of subareas they innervate. Interestingly, cell bodies of each type were located in characteristic zones within the JON array. These results suggest that JONs comprise three subgroups each of which might participate in different aspects of auditory reception.

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P1-293 Responses of glomeruli to the mixture of two odorants in the rat dorsal OB

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Individual glomeruli in the rat olfactory bulb (OB) represent a single type of odorant receptor, and typically respond to a range of odorants that share similar molecular structure. Using a large panel of odorants, we previously reported the molecular-feature clusters of glomeruli in the dorsal OB. In this study we used optical imaging of intrinsic signals to examine the responses of glomeruli to the mixture of two different odorants. Glomeruli activated by the mixtures were nearly identical to the sum of glomeruli activated by individual components. When the mixture of two odorants which activated some overlapping glomeruli was used, some glomeruli showed smaller response to the mixture in comparison with the response to an individual component. If we assume that the main intrinsic signal sources reflect the activities of axon terminals of olfactory sensory neurons, these results suggest either an antagonistic interaction at the odorant receptor level or the presynaptic inhibitory process at the level of axon terminal.

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P1-294 Discrimination between chiral pairs in mouse olfactory receptor neurons

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We previously reported enantiomer discrimination capability between R(-)- and S(+)-carvone in murine odorant receptor family, by using Ca-imaging assay. Chiral-discriminating receptors were larger in population of responsive receptors at the lowest effective stimulus concentration than chiral-non-discriminating receptors. However, the non-discriminating receptors became the majority at approximately 10-fold of the lowest concentration. Consequently the previous results leaded to the hypothesis of the sensitivity-dependent hierarchical receptor codes. To further test our hypothesis, we are examining receptor codes for another pair of enantiomers. We will discuss about the odor coding hypothesis on our new data.

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P1-295 Connection pattern of vomeronasal receptor afferents to an accessory olfactory bulb projection neuron

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The mitral-tufted (MT) cell in the accessory olfactory bulb (AOB) receives information from multiple glomeruli. To determine whether a given MT cell gets only one vomeronasal receptor input or multiple receptor inputs, we examined the connectivity between a biocytin filled MT cell and axons expressing VN12 in VN12-tau-LacZ mice. In the most MT cells, one of the dendrites got inputs from a VN12 positive glomerulus, but in some MT cells, all the dendrites got inputs from VN12 positive glomeruli. Furthermore, we found the presence of a glomerulus converging both VN12 and V3R afferents using the RT-PCR method. These findings suggest various connectivity of sensory axonal projections to the MT cell in the AOB, which contrasts with homotypic conectivity in the main olfactory bulb.

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P1-296 Two distinct neural pathways in the zebrafish olfactory system visualized by transgenesis with cell type-specific promoters

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The fish olfactory epithelium (OE) contains two major types of olfactory sensory neurons (OSNs): ciliated and microvillous cells. Previous studies have shown that ciliated and microvillous OSNs are segregated along the apical-basal axis within the OE, however it remains largely unknown how these two types of cells project their axons to the olfactory bulb (OB). Here, we found that ciliated OSNs specifically expressed olfactory marker protein (OMP), while microvillous OSNs expressed transient receptor potential channel TRPC2. To visualize each type of OSNs, we generated transgenic zebrafish lines in which fluorescent proteins were expressed under the control of OMP and TRPC2 promoters, respectively. Histological analyses revealed that ciliated and microvillous OSNs projected axons to mutually different sets of glomeruli in the OB. These transgenic zebrafish lines will be useful for studying molecular and cellular mechanisms underlying the development of the olfactory system.

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P1-297 Electrophysiological bases of odor-induced neural responses in the piriform cortex

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To investigate the neural mechanism underlying olfactory information processing in the anterior piriform cortex(APC), we analyzed the odor-induced response in the isolated whole brain preparation with the olfactory epithelium. A single current pulse stimulus to the lateral olfactory tract(LOT) evoked surface-negative-deep-positive potential in the APC. In contrast, natural odor stimulation induced surface-positive-deep-negative potential. Deep-negative potentials were also recorded when high-frequency electrical stimuli were applied to the LOT or the olfactory bulb. Therefore, repetitive or continuous inputs to the APC might be required to induce the deep-negative potentials. In layer II, the deep-negative potential was often accompanied with generation of spikes in pyramidal neurons. This observation suggests that the natural odor stimulation induced strong excitatory inputs to the layer II pyramidal neurons, which generated deep-negative potentials.

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P1-298 Quantitative analysis of axon collaterals of single neurons in layers IIa and IIb of the piriform cortex of the guinea pig

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To study the axonal distribution of neurons in layer II in the piriform cortex (PC) , axon collaterals of single neurons in layer IIa and IIb were labeled and quantitatively analyzed by intracellular injection of biocytin in the guinea pig. Individual neurons have highly distributed axon collaterals, which display little tendency for patchy concentrations inside as well as outside the PC. The total lengths of axon collaterals of three neurons in layer IIa, including one semilunar cell, one semilunar-pyramidal transition cell, and one multipolar cell, ranged from 54 mm to 256 mm. Their total numbers of boutons ranged 4200 to 23000, out of which 49% to 78% were distributed in the PC. The total axonal lengths of three pyramidal cells in layer IIb ranged 68 mm to 156 mm. Their total numbers of boutons ranged 6000 to 14000, out of which 48% to 85% were distributed in the PC.

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P1-299 The G_o and G_q signaling pathways modulate olfactory responses in *C. elegans*

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The heterotrimeric G protein G_o is abundantly expressed in mammalian brain. In the nematode *Caenorhabditis elegans*, GOA-1 $G_o\alpha$ and EGL-30 $G_q\alpha$ act antagonistically in modulating the neurotransmitter release at neuromuscular junctions.
To elucidate the role of GOA-1 and EGL-30 in more complex behaviors, we focused on chemotaxis behavior, which is mediated by many types of neurons. Both a loss-of-function mutant of *goa-1* and animals overexpressing EGL-30 protein showed an increased chemotaxis to volatile odorants, and had a defect in olfactory adaptation, suggesting that G_o and G_q signaling pathways modulate olfactory responses in opposite directions.
The adaptation defect observed in *goa-1* mutants was rescued by targeted expression of GOA-1 protein either in sensory neurons (olfactory neurons) or in several interneurons. These results indicated that GOA-1 functions in multiple neurons to negatively regulate chemosensory responses.

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P1-300 Distribution of protein kinase C (PKC) in vomeronasal system

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Protein kinase C (PKC) is suggested to involve an olfactory signal transduction. However, the distribution of PKC isoforms in vomeronasal system has not been reported. In the present study, we investigated the distribution of conventional PKC (cPKC) isoforms in vomeronasal sensory cell (VSC) and accessory olfactory bulb (AOB). The frozen sections of rat vomeronasal organ and AOB were applied to fluorescence double staining using anti-cPKC (α, $\beta1$, $\beta2$, γ) and PGP9.5 antibodies. The γ immunoreactivity (IR) was observed in knobs and dendrites, α-IR was observed in dendrites of VSC. In AOB α-IR and $\beta1$-IR were observed in mitral/tufted cell layer (MTL), granule cell layer (GRL) and glomerulus layer (GL). Expression of $\beta2$ in GL of rostral part was stronger than in caudal part. γ-IR was observed in MTL, GRL and ventral GL, $\beta2$ and γ coexpressed partially in GL. We concluded that cPKC isoforms have a specific role because of each distributions are different in VSC and AOB.

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P1-301 Expression and functions of the cephalic homeobox gene *empty spiracles* in the olfactory and gustatory centers of the *Drosophila* Brain

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The *empty spiracles (ems)/Emx* genes have pivotal functions in brain development in both invertebrate and vertebrate. In *Drosophila*, *ems* is essential for the embryonic development of the b2 and b3 neuromeres, which give rise to the olfactory and gustatory centers. We have analyzed expression patterns of the *ems* gene in the larval and adult brains and elucidated the projection patterns of the EMS neurons by axonal tracing with elav-GAL4/UAS-mCD8-GFP and with MARCM mosaic technique. At the larval stage, five prominent EMS clusters per hemisphere are located around the antennal lobe with diverse projection patterns. Four of these clusters are lost after metamorphosis with only one cluster projecting to the subesophageal ganglia. Clonal analyses indicated that cells of the persistent EMS cluster are generated in the early first instar stage. Loss-of- and gain-of function studies are ongoing to elucidate *ems* functions in the complex brains.

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P1-302 *Drosophila* sweet taste perception is mediated by Gs

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We have previously reported that the sugar-taste receptor gene of *Drosophila melanogaster*, *Tre*, encodes a seven-transmembrane receptor. Since the seven-transmembrane receptor family is coupled to a distinct G protein α-subunit (Gα), we examined which Gα is coupled to *Tre*. A mutation in one of Gα genes, *dgs*, which encodes Gs, depressed the amount of sugar-intake and reduced electrical neuronal activities in sugar-taste receptor neurons induced by application of sugar solutions. Furthermore the impairments of behavioral and electrophysiological responses to sugars were rescued by expression of wild-type dgs in *Tre*-expressing receptor neurons. We also demonstrated by immunohistochemistry that dgs is expressed in taste receptor neurons. Taken together, we conclude that sugar-taste signals are processed by Gs in sugar-taste receptor neurons in *Drosophila*.

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P1-303 Gurmarin inhibition of sweet taste responses of the chorda tympani nerve in T1r3 KO, gustducin KO and Trpm5 KO mice

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Gurmarin is known as a specific but partial inhibitor for sweet taste responses in rodents. To study transduction pathways for gurmarin-sensitive (GS) and -insensitive (GI) response components, its inhibition on sweet taste responses of the chorda tympani nerve was examined by using KO mice for the gene controlling sweet taste-related molecule: a receptor component, T1r3, a G protein α-subunit, gustducin, or a cation channel, trpm5. These KO mice showed diminished but not abolished responses to sweet compounds. The residual sweet responses were further inhibited by gurmarin to about 50% in T1r3 or trpm5 KO mice, whereas no such inhibition was observed in gustducin-KO mice. This indicates that T1r3 or trpm5 KO mice possess both GS and GI components whereas gustducin KO mice lack GS. This strongly suggests that gustducin play a key role in GS sweet responsive pathway.

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P1-304 The dpa locus may influence gurmarin sensitive receptor component in mice

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It is known that there are two loci influencing responses to sweet substances on mouse chromosome 4, *dpa* (D-phenylalanine sensitivity) and *Sac* (Saccharin preference). Recently, the *Sac* is found to encode G-protein coupled sweet receptor candidate gene, T1R3. In concerning about *dpa* locus, it has been shown to influence gurmarin-sensitivity at the peripheral taste system by using the dpa congenic strain (CG) having BALB background except a segment including the *dpa* locus derived from D-phe responsible C57 strain, but details remain unknown. In this study, we examined the coexpression pattern of gustducin, T1R1, T1R2 and T1R3 mRNAs in taste papillae and the gurmarin effect on sweet sensitivity in B6, BALB and CG strains. Results suggest that the gene segment derived from C57 may influence either the expression level and pattern of T1R2, T1R3 and gustducin, or gurmarin inhibition of sweet responses.

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P1-305 Expression of α-gustducin and T1r3 in taste receptor cells responding to sweet substances in mice

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Recent studies showed that a taste receptor component, T1r3, and a G protein α-subunit, gustducin, play a key role in receptor and transduction system for sweet taste. More recently, T1r3 KO mice study demonstrated that mice lacking T1r3 had diminished but not abolished behavioral and taste nerve responses to sugars, suggesting the existence of T1r3-independent sweet responsive systems. Here, we showed subsets of sweet-responsive taste cells of mouse fungiform papillae with different expression patterns of α-gustducin and T1r3. After recording of responses of taste cells to sweet substances, molecular expression of these cells was examined by single cell multiplex RT-PCR. Our preliminary results indicated that sweet responsive cells expressed either or both gustducin and T1r3. This suggests a possibility of the existence of multiple receptor and transduction systems for sweet taste.

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P1-306 Relationship between taste perception and the brain monoamine nervous system in rats.

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We investigated changes of the monoamine transport systems in the brain (the hypothalamus, amygdale, limbic forebrain, hippocampus, striatum, cerebral cortex, septum, midbrain and pons) by taste palatability, using high-performance liquid chromatography. We used preferred 5 mM saccharin and aversive 0.3 mM quinine hydrochloride as the taste stimuli. The levels of serotonin in the hypothalamus, amygdala and septum, in rats that received saccharin was higher than those that received quinine hydrochloride. In addition, the levels of dopamine and 4-hydroxy-3-methoxyphenylacetic acid in the hypothalamus, amygdala, septum and limbic forebrain showed similar patterns to that of serotonin. These results suggest that changes in the level of several monoamines including serotonin and dopamine in the brain are involved in taste palatability.

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P1-307 Gustatory Neural Responses in Fronto-Opercular and Adjacent Cortices in Monkeys

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We recorded neuronal responses of 807 units with a pair of microelectrodes from the primary and higher gustatory cortices (PGC: areas G, 3 and 1-2; HGC: PrCO, area 12, OFO and insula) of alert monkeys. Tastants used were four basic tastants (0.1 M NaCl, 0.3 M sucrose, 0.01 N HCl and 0.003 M quinine-HCl), 0.1 M MSG, 100 % orange juice and distilled water. We found 102 gustatory units (PGC: 69, HGC: 33). We applied hierarchical cluster analysis to the collective data matrix of 102 units x 6 tastants (4 basic tastants, MSG and water) because of the small number of samples. Six clusters, each consisting of units best excited by a certain tastant, were segregated. No cluster was related to cortical areas. We recorded 29 pairs of neurons (25 in PGC and 4 in HGC) simultaneously. They significantly responded to the same tastants in 39 response pairs, none of which showed significant cross-correlation.

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P1-308 Analysis of mutants abnormal in the plasticity of salt chemotaxis in *C. elegans*

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We have previously reported a plasticity of salt chemotaxis induced by a combination of two stimuli, starvation and NaCl, in *C. elegans*. Naive wild type animals are attracted to NaCl, but show avoidance behaviors after being conditioned with NaCl and starvation (food-/NaCl+). This behavioral change is not induced under the food-/NaCl- or food+/NaCl+ conditions.
To gain insights into the molecular mechanisms underlying this form of learning, we isolated mutants defective in learning in this assay system. They showed higher chemotaxis than wild type strains after a food-/NaCl+ conditioning. Of these, we focused on 2 strains (*pe401* and *pe402*) that had strongest phenotypes. Neither mutant showed large defects in chemotaxis to NaCl or odorants, and both of them showed normal responses to food and starvation as assessed by the speed of locomotion.
So far, we mapped *pe401* to Chromosome II, and *pe402* to Chromosome V. We are now trying to narrow down the region containing the locus for *pe402*

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P1-309 A Novel Type of Granule Cells in the Olfactory Bulb of Adult Mouse

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While the olfactory bulb (OB) of adult mice contains numerous interneurons, their heterogeneity is not well characterized. In the OB of transgenic mice expressing GFP under the control of nestin promoter, we identified GFP expressing granule cells with characteristic dendritic morphology (referred to as type S cells). Their dendrites formed branches and spines in the granule cell layer, internal plexiform layer and mitral cell layer, but did not reach the external plexiform layer (EPL). This contrasts with typical GCs that extend their dendrites into the EPL and bear spines there. In addition, huge spherical protrusions were observed at dendritic ends of type S cells, some of which were located at the perisomatic region of mitral cells. Type S cells expressed NeuN and GABA, and BrdU-labeling study indicated that they are not at the developmental stage of typical GCs. These results suggest that type S cells have roles distinct from those of typical GCs in the neuronal circuits of the OB.

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P1-310 Histological and pharmacological analysis of the central serotonergic system in the slug *Limax valentianus*

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Limax is a useful model system to study olfactory learning. We investigated the localization of serotonergic neurons in the central nervous system (CNS) and their function in olfactory learning. Many serotonergic neurons, including uniquely identifiable neurons in the cerebral ganglia, were discovered by immunohistochemical staining. In addition, serotonin induced a decrease of the amplitude and an increase of the frequency of the local field potential in the procerebral lobe. Two different antagonists exerted different blocking effects on the amplitude and frequency of the oscillation: one blocked the former and the other blocked the latter. Based on the findings above, there is a possibility that serotonin plays an important role in the CNS of *Limax*.

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P1-311 Activation of p38 MAPK in primary afferent neurons by noxious stimulation and its involvement in peripheral sensitization

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Alterations in the intracellular signal transduction pathway in primary afferents may contribute to pain hypersensitivity. Here, we report intensity-dependent p38 phosphorylation in primary afferents by painful stimulation. c-fiber activation by capsaicin injection induced phosphorylated-p38 (p-p38) in small DRG neurons with a peak at 2 min. We also found a stimulus intensity-dependent increase in labeled cell size and the number of activated neurons after noxious thermal stimuli. Most of these p-p38-immunoreactive neurons were small- and medium-sized neurons, which coexpressed VR-1 and p-ERK. Intrathecal administration of the p38 inhibitor or capsaicin cream, rubbed over the rat footpad skin, reversed the capsaicin-induced thermal hyperalgesia. Therefore, the activation of p38 pathways in primary afferents by noxious stimulation may be correlated with functional activity, and further, may be involved in the sensitization of primary afferents.

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P1-312 Direct action of immunosuppressant FK506 on nociceptive sensory neurons

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To elucidate the mechanisms of transient burning sensation elicited by FK506 (FK), which is often applied topically to the skin of atopic patients for clinical purpose, we examined the action of FK on sensory neurons. Ca^{2+}-imaging study using cultured DRG neurons revealed that FK raised intracellular Ca^{2+} levels in a subpopulation of small neurons (3.1% of neurons). In the neurons from inflamed rats, this incidence increased to 7.4%. FK-sensitive neurons also responded to a subsequent application of capsaicin (Cap) (89.5% in normal, and 100% in inflamed rats) and bradykinin (BK) (31.6% in normal, and 80.9% in inflamed rats). Single fiber recordings in the nerve-skin preparation confirmed the results of cell culture, showing that FK enhanced neuronal discharges of single C-fibers that responded to heat and BK. FK application on inflamed skin may activate nociceptive C-fibers that bear BK receptors and Cap-sensitive heat transducer, TRPV1.

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P1-313 Neurogenic inflammation in the plantar and hip skin areas

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Melittin is the main toxin of bee venom. We have reported that subcutaneous injection of melittin into the rat hindpaw increased the local skin temperature. In order to further investigate whether melittin injected into either the plantar skin or the hip skin causes neurogenic inflammation, skin temperature and blood flow were measured. Any local temperature increase of the hip skin could not be detected by thermography after melittin injection into the hip. The resting hip skin temperature was usually higher than the resting plantar skin temperature in pentobarbital-anesthetized rats. In contrast, the resting blood flow level of the hip skin was lower than that of the plantar skin. Although melittin injection into the plantar skin increased local blood flow, hip blood flow did not show any change after melittin injection into the hip. These data suggest that the plantar skin is easier to detect neurogenic inflammation than the hip skin because the resting blood flow of the hindpaw is sufficiently high.

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P1-314 Effects of ketamine on peripheral glutamate receptors

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We have reported that subcutaneous injection of glutamate into the human forearm evoked pain and produced axon reflex-mediated temperature increase around the injection site. The results suggest glutamate receptors on peripheral primary afferent terminals contribute to peripheral nociceptive signaling.
In order to further investigate which subtype of glutamate receptors is involved in nociception, effect of glutamate receptor antagonist was evaluated in pentobarbital-anesthetized rats. Simultaneous subcutaneous injection of ketamine (NMDA receptor antagonist) and glutamate into the hindpaw produced local skin temperature increase comparable to that produced by isolated injection of glutamate. Unexpectedly, subcutaneous injection of ketamine alone produced both skin temperature increase and paw edema.
These data cannot demonstrate that any contribution of NMDA receptor to vasodilatation and edema induced by subcutaneous injection of glutamate. Besides, it is demonstrated that ketamine alone is capable of producing vasodilatation and edema.

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P1-315 Involvement of spinal glial glutamate transporter GLT-1 in inflammatory hyperalgesia in rats

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The spinal glutamatergic system has been shown to play an important role in the pain transmission. In this study, we examined the role of spinal glial glutamate transporters in inflammatory hyperalgesia induced by intraplantar (i.pl.) injection of 2% λ-carageenan/kaolin in rats. We found that i.pl. carageenan/kaolin induced significant mechanical hyperalgesia for 0.5-8 hr in the paw pressure test, and significantly decreased the expression of GLT-1 mRNA in the spinal cord at 3 and 8 hr after the injection, while the effect on GLAST mRNA expression was less. Furthermore, gene transfer of GLT-1 into the spinal cord by recombinant adenoviruses significantly reduced the carageenan/kaolin-induced mechanical hyperalgesia, although it had no effect on the acute nociceptive responses to the mechanical and thermal stimuli in normal rats. These results suggest that spinal GLT-1 plays an inhibitory role in the inflammatory hyperalgesia.

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P1-316 Expression of L1-CAM in rat dorsal root ganglia and spinal cord following nerve injury

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Cell adhesion molecule L1-CAM plays a key role in morphological changes such as neurite extension, formation of axon tracts and synaptic plasticity in nervous system. We investigated whether 1) peripheral injury affected expression of L1-CAM in pain pathway and 2) the L1-CAM was involved in generation of neuropathic pain. The rats (Sprague-Dawley, weighing 200-250 g) received partial or complete sciatic nerve transection (PSNT or SNT) were used. Following SNT, L1-CAM immunoreactivity (ir) was increased in cell membrane of injured dorsal root ganglia (DRG) neurons somata and in presynaptic button of ipsilateral dorsal horn . However, L1-CAM mRNA showed less change in DRG neurons. Administration of neutralization antibody for L1-CAM significantly improved the pain behavior in PSNT rats. These results indicate that the post-transcriptional regulation of L1-CAM may be involved in the onset and maintenance of neuropathic pain.

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P1-317 Differential temporal expression of temperature-activated transient receptor potential channels in developing dorsal root ganglia

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Several transient receptor potential (TRP) channels are activated at various temperatures and expressed in a distinct subset of nociceptive sensory neurons in adult mice. We examined the developmental expression of three TRP channels, TRPV1, TRPV2, and TRPM8 in the mouse dorsal root ganglion (DRG) neurons. TRPV2 was first observed in DRG neurons at embryonic day (E) 11.5. TRPV1 was detected in DRG neurons at E13.5. The percentage of TRPV2(+) neurons in total DRG neurons was maintained throughout the development (about 15 %), while that of TRPV1(+) neurons continued to increase until postnatal day (P) 14 (44.3% at P14). In addition, TRPM8(+) DRG neurons were first observed at E17.5, and the percentage of these neurons increased mainly in the postnatal period, reaching the adult level at P14 (15.7% at P14). The differential temporal expression patterns in the developing DRG suggest that the expressions of these TRP channels are regulated by distinct factors.

P1-318 Differential activation of MAPK in injured and uninjured DRG neurons after CCI

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To investigate the intracellular signal transduction pathways involved in the pathophysiological mechanisms of neuropathic pain, we examined the activation of ERK and p38 MAPK in the DRG in the chronic constriction injury (CCI) model. The CCI induced an increase in the phosphorylation of ERK in injured medium- and large-sized DRG neurons and in satellite glial cells. Treatment with the MEK 1/2 inhibitor, U0126, suppressed CCI-induced mechanical allodynia and partially reversed the increase in NPY expression in damaged DRG neurons. In contrast, the CCI induced the activation of p38, mainly in uninjured small-to-medium-diameter DRG neurons and in satellite glial cells. The p38 inhibitor, SB203580, reversed the CCI-induced heat hyperalgesia and also the increase in BDNF expression in intact DRG neurons. These changes in injured and intact primary afferents are likely to have a substantial role in pathological states, and MAPK pathways in nociceptors may be potential targets for the development of novel analgesics.

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P1-319 Correlation between morphology and sensory input of excitatory and inhibitory transmission in substantia gelatinosa neurons of adult rat spinal cord *in vitro*

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The major termination of Aδ- and C-afferent nociceptive inputs is the superficial dorsal horn, especially substantia gelatinosa (SG). SG neurons that modulate the transmission of afferent information to higher-order neurons exhibit electrophysiological and morphological heterogeneity. To compare excitatory and inhibitory inputs of individual SG neurons with their morphology, we performed whole cell patch clamp recordings with patch pipettes filled with neurobiotin in a horizontal slice preparation of adult rat spinal cord with an attached dorsal root. Based on the dendritic arborization, there were at least four cell types, and their morphological features were associated with characteristic inputs. This heterogeneity may play an important contribution in processing of nociceptive information.

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P1-320 Cyclooxygenase-2- and GABA-immunoreactivities are often colocalized in cells in the lumbar dorsal horn of the chicken

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Previous studies have shown that cyclooxygenase-2 (COX-2) cells exist in the superficial laminae of the spinal dorsal horn. In this study, we tried to show the relation between COX-2 immunoreactive (IR) cells and GABA immunoreactivity in the superficial laminae of the chicken dorsal horn, using an immunohistochemical double labeling technique and confocal lazer scanning microscopy. We observed that small COX-2-IR cells in the superficial laminae of the dorsal horn often contained GABA. These double labeled cells and the COX-2-IR cells are both contacted by the GABA fibers. As COX-2 is an enzyme of prostaglandins (PGs) synthesis, it is suggested that the double labeled cells might synthsize both PGs and GABA, and that the double labeled cells and the COX-2-IR cells might be affected by GABA fibers.

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P1-321 Effect of chronic inflammation on dorsal horn nociceptive neurons in aged rats

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The present study was designed to elucidate the effect of chronic inflammation on nociception in the aged rats. Foot temperature, thickness and dorsal horn neural activities were studied in the aged and adult rats with CFA. The changes in foot temperature and thickness were much higher and longer lasting in aged rats than those of adult rats after CFA injection. Nociceptive neurons were classified as WDR and NS neurons according to their responsibility to mechanical stimulation of the receptive fields. NS neurons from the inflamed adult rats showed significantly higher responses to noxious stimulation. On the other hand, WDR neurons from both inflamed adult and aged rats and NS neurons from the inflamed aged rats did not show higher responses to mechanical stimulation as compare with those of naive rats. These suggest that chronic peripheral inflammation induces differential effects on pain perception in aged and adult rats.

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P1-322 The effects of substance P on phasic and tonic inhibitory action in deep dorsal horn neurons of mice spinal cord.

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The effect of SP on spinal inhibitory transmission still remains unknown. We studied the effect of SP on inhibitory action not only phasic but also tonic periods in lamina V of mice spinal dorsal horn by using whole-cell patch clamp method. Bath applied of SP increased the frequency of spontaneous IPSC (sIPSC) mediated by GABA and glycine. On the other hand, SP has no effect on the frequency and amplitude of mIPSC recorded under TTX. The tonic inhibitory action can be observed by the application of bicuculline, $GABA_AA$-receptor antagonist, and strychnine, glycine receptor antagonist in deep DH. The application of SP prior to bicuculline made enhancement the inward current induced in lamina V neurons. These results indicate the potentiality that SP induced the enhancement of inhibitory action in the deep DH.

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P1-323 Analysis of gene expression in rat Trigeminal ganglion neurons following maxillofacial inflammation using cDNA microarray

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The cDNA microarray 20500 genes was introduced to identify an inflammation specific gene expression in Trigeminal Ganglion (TG) neurons following peripheral inflammation. TG was extracted 3 days after 50 μl of CFA injection into the right superior lip, and total RNA was purified. The total RNA was labeled with aminoallyl-d UTP and conjugated with Cy-dye fluoroscence. The labeled RNA hybridized 20500 probes on Agilent Rat Oligo Microarray at 60°C for 17h. We used scanning and analyzing specific gene expression by Agilent Scanner. Almost 420 genes with increment of expression were detected following inflammation. In addition, a few genes, CaMKII and KCNK3, were inspected by *in situ* hybridization.

P1-324 Ultrastructural analysis on the peripheral and the central nervous system of an experimental mononeuropathy rat (Bennett's model)

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Our present study was designed to make clear the morphological changes of the peripheral and the central nervous system in the experimental mononeuropathy rats. A peripheral mononeuropathy was produced in adult Wistar rats by placing loosely constructive ligatures with chromic gut around the left common sciatic nerve. After ligature of the sciatic nerve, foot withdrawal response to mechanical stimuli was measured by a dynamic planter aesthesiometer every day. At 14th experimental day, all of rats used in this study showed mechanical hyperalgesia until perfusion. On the periphery of the ligated site of sciatic nerve, a few axons completely degenerated. About 58.7% of fibers in this region showed slightly damaged appearance including irregularity of shape, demyelination, axonal degeneration and remyelination. Small number of macrophages intermingled into these damaged fibers.In DRG, many mitochondria in some small sized neurons swellwd.

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P1-325 C-Fos cells in the cingulate gyrus after cutting the sciatic nerve of rats

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At 1 and 6 hours (h), 1, 3 and 5 days (d), and 1, 3 and 5 weeks (w) after cutting the rat sciatic nerve, the deeply anesthetized rats were perfused. The sections of the rat brains were stained immunohistochemically. Using the sections cells expressing C-Fos-like protein (C-Fos cells) in the laminae II - IV of the cingulate gyrus (CG) relating closely to learning and memory were counted. The number of C-Fos cells increased in the 1 h case, decreased gradually in the 6 h, 1d and 3d cases to the level of the control cases with only perfusion, increased in the 5d and 1 w (reaching to the peak) cases, decreased in the 3 w case to the control level, and increased in the 5 w case. The present study shows that increase-decrease of the number of CG might control periodically the strength of the chronic pain including phantom pain.

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P1-326 Ascending pain pathways to the anterior cingulate cortex in rats: retrograde transneuronal tracing with rabies virus

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The organization of ascending projections to the anterior cingulate cortex was examined in the rat by means of retrograde trans-neuronal labeling with rabies virus. Three to 4 days after the virus injection into the anterior cingulate cortex, a large number of labeled neurons were observed in the parafascicular, submedius, centrolateral nuclei and the limbic-related structures. In addition, retrograde labeling simultaneously occurred in the brainstem regions and the trigeminal ganglion. Many trigeminal ganglion neurons were also labeled after SI injection. Population of the large TRG neurons labeled following SI injection was slightly larger as compared with those after ACCx injection. The present results suggest that the nociceptive information was processed with different population of TRG neurons in the anterior cingulate and SI cortices.

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P1-327 Duration of nociceptive responses encodes stimulus intensity in the medial prefrontal cortex

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Two types of nociceptive neurons, wide dynamic range-like (WDR-L) neurons and nociceptive specific (NS) neurons, evoked by mechanical stimulation encoded absolutely or relatively stimulus intensity in the medial prefrontal cortex (mPFC). Extracellural unit discharges evoked by mechanical stimulation delivered to the rat tail were recorded in the mPFC. WDR-L neurons encoded stimulus intensity with duration of the responses, as well as alternation of discharge frequencies. In the NS neurons, morphine dose-dependently reduced the duration of the nociceptive response, suggesting that it may reflect conscious stage dependent stimulus intensity. These results suggest that the mPFC neurons may transform stimulus intensity into temporal information, allowing it to be appropriately processed for next order activities.

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P1-328 Sex difference in the response of the CNS to formalin-induced nociceptive stimuli as revealed by the expression of phosphorylated cAMP response element binding protein (pCREB)

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We examined whether there was a sex difference in the response of the CNS to formalin-induced nociceptive stimuli by checking the expression of pCREB as a marker of neural activity. The accumbens nucleus (ACB) and the bed nucleus of the stria terminalis (BST) were chosen as the areas to examine this time. Adult male rats and female rats at proestrus and diestrus were injected with saline or 2% formalin into the planter surface of the right hindpaw. Ten min after formalin injection in female rats the number of pCREB cells significantly increased in the ACB and the BST, but not in male rats, indicating sex difference in the response of these areas to nociceptive stimuli. Furthermore, the increase in the number of pCREB cells in the ACB and the BST in female rats at proestrus was larger than that at diestrus rats, suggesting an involvement of estrogen in this sex difference.

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P1-329 A possible spinal pathway of contralaterally projecting coeruleospinal neurons in the rat

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A possible spinal pathway of contralaterally projecting axons from the locus coeruleus/subcoeruleus (LC/SC) to the spinal cord was investigated. Extracellular recordings were made from the left dorsal horn. A hemisection of the spinal cord ipsilateral to the recording sites was performed at the C2 level. Responses of dorsal horn neurons to noxious heat were inhibited during electrical stimulation of the LC/SC. The transection of the dorsolateral funiculus (DLF) contralateral to the recording sites did not affect LC/SC stimulation-produced inhibition. Following transection of the ventrolateral funiculus (VLF) contralateral to the recording sites, LC/SC stimulation failed to inhibit heat-evoked responses. These results suggest that interruption of coeruleospinal inhibition produced by the VLF transections is due to the blockage of axons descending in the ventrolateral quadrant of the spinal cord.

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P1-330 Intrapancreatic neuronal branching of afferent fibers in chronic pancreatitis

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Chronic pancreatitis (CP) is a disease with persistent pain. The histopathologically enlargement of intrapancreatic nerve fibers was observed. Origin of these nerve fibers and mechanisms of the changes, however, have remained unclear. We applied neuronal tracing methods to WBN/Kob rats which genetically cause CP. Injection of HRP and WGA-HRP, retrograde tracers, into the pancreas revealed significant decrease in number of the neurons in DRG innervating pancreas in CP model. Injection of biotinylated dextran amine (BDA), an anterograde tracer, into DRG of T13 revealed significant increase in number of nerve fibers in the pancreas in CP model. These findings suggest that DRG neurons innervating the pancreas make increased axonal branches in CP model. Furthermore, number of c-fos expressing neurons in the dorsal horn increased in CP model, suggesting that such an axonal branching decrease the firing threshold in DRG neurons innervating the pancreas.

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P1-331 Coexpression of vanilloid receptor subtype-1 and acid-sensing ion channel genes in the human trigeminal ganglion neurons

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Previous psychophysical experiments have shown that repeated applications of high concentrations of acids on one side of the dorsal surface of the tongue evoke irritation or pain in human subjects. In our human pain model, direct infusion of acidic solutions (pH > or = 6.0) into human skin caused localized pain, which was blocked by amiloride, an inhibitor of ASICs, but not by capsazepine, an inhibitor of TRPV1. Under more severe acidification (pH 5.0), capsazepine also produced some blocking effect on the acid-evoked pain. *In situ* hybridization histochemistry demonstrated that most of TRPV1-expressing human trigeminal neurons were ASIC1a- or ASIC3-positive. These results suggest that both TRPV1 and ASIC channels are involved in acid-evoked oral irritation or pain.

P1-332 Prostaglandin EP3 receptor-mediated augmentation of bradykinin-induced increase in intracellular calcium

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We have reported that a prostaglandin EP3 receptor (EP3R) agonist augments the nociceptor responses to bradykinin (BK) in vitro canine testis-spermatic nerve preparation. To study mechanism of this augmentation, we established CHO cells stably transfected with canine EP3R cDNAs and examined the effect of an EP3R agonist, ONO-AE-248, on BK-induced increase in intracellular calcium ($[Ca^{2+}]_i$) after transient transfection of BK B2 receptor. BK-induced $[Ca^{2+}]_i$ increase was desensitized when BK was applied at an interval of 6 min. This desensitization was attenuation by pretreatment with ONO-AE-248 at a low concentration (10^{-7}M), which does not elevate $[Ca^{2+}]_i$. The agonist by itself elevated $[Ca^{2+}]_i$ at higher concentrations (10^{-5} or 10^{-6} M) either in the presence or absence of extracellular Ca^{2+}. These observations are compatible with EP3 dependent sensitization of the BK response.

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P1-333 Modulation of TRPV1 activity by prostaglandin E_2

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Prostaglandin E_2 (PGE_2) is one of the inflammatory mediators that play important roles in pain sensation and hyperalgesia in inflamed tissues. In this study, we examined the interaction between TRPV1 and PGE_2. In behavioral analysis, intraplantar injection of PGE_2 produced thermal hyperalgesia in wild type mice. However, the PGE_2 -induced thermal hyperalgesia was attenuated in TRPV1-deficient mice and EP_1 -deficient mice. PGE_2 -induced potentiation of the capsaicin-activated current responses mainly through a PKC-dependent pathway was observed mouse DRG neurons. These data suggest that the potentiation of TRPV1 activity by PGE_2 might be one important mechanism of inflammatory pain.

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P1-334 Modulation of TRPV1 activity by prostaglandin I_2

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Prostaglandin I_2(PGI_2) is one of the inflammatory mediators that play important roles in pain sensation and hyperalgesia. The capsaicin receptor TRPV1 is a nonselective cation channel expressed in sensory neuron and activated by various nociceptive stimuli. Here we show that the functional interaction of TRPV1 with PGI2. PGI_2 potentiated TRPV1 responses through IP receptors in a PKC- or a PKA-dependent pathway in both HEK293 cells expressing TRPV1 and mouse DRG neurons. Furthermore, in behavioral analysis, PGI_2-induced thermal hyperalgesia was diminished in TRPV1-deficient mice and IP-deficient mice. Thus, these results suggest that TRPV1 is important for the development of the PGI_2-indused thermal hyperalgesia.

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P1-335 Functional analysis of anti-phosphorylated TRPV1 antibody

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The capsaicin receptor, TRPV1, is a sensory neuron-specific ion channel that serves as a polymodal detector of pain-producing chemical and physical stimuli. It has been reported that inflammatory mediators such as ATP and bradykinin phosphorylate TRPV1 in a PKC-dependent pathway. Two serine residues in the cytoplasmic domains of TRPV1, Ser^{502} and Ser^{800} were found to be involved in phosphorylation of TRPV1 by PKC. We made an antibody gainst phosphorylated Ser^{800} of TRPV1 because Ser^{800} is known to function as a substrate only for PKC-dependent phosphorylation. We examined the amount of phosphorylated TRPV1 upon treatment with PMA using the antibody in Western blot analysis and immunostaining. We detected the increase in the amount of phosphorylated TRPV1 upon PMA stimulation in both HEK293 cells expressing TRPV1 and cultured rat DRG neurons.

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P1-336 Change in the excitability of large-diameter TRG neurons innervating intact cutaneous area after TMJ inflammation

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The aim of the present study was to test the hypothesis that temporomandibular joint (TMJ) inflammation can alter the excitability of large-diameter (LD) trigeminal root ganglion (TRG) neurons innervating intact skin area. LD-TRG neurons innervating the facial skin were labeled by FG-injection into the facial skin. The magnitude of membrane depolarization provoked by substance P in the FG-labeled LD-neurons of CFA-inflamed rats was larger than that of naive rats. Quantitative RT-PCR showed that expression of mRNA for NK1 receptors in the LD-TRG neurons in inflamed rats became more prominent as compared to that of naive rats. These results suggest that trigeminal inflammatory allodynia in the TMJ disorder may involve up-regulation of NK1 receptors in the LD-TRG neurons.

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P1-337 Frequency-dependent activation of small, medium and large sizes of dorsal root ganglion neurons in vitro

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For the quantification of nerve dysfunction in patients under hyperalgesia or allodynia, it is essential to evaluate objectively the change of threshold of afferent fibers. Recently, an equipment, which produces three sine-wave pulses (2000, 250 and 5 Hz), has been developed (Neurometer) to activate selectively each fibers. However, the selectivity of it has not been rigorously tested. Intracellular recordings were made from identified DRG neurons. Action potentials (APs) were elicited with a suction electrode connected to Neurometer. Aβ-neurons were activated by all three frequencies, while Aδ-neurons were activated by 250 and 5 Hz stimuli. On the other hand, C-neurons were activated only by 5 Hz stimuli. These results indicate that Neurometer could be useful for the selective activation of the primary afferents, and might provide basic evidence for evaluating analgesic drugs and changes in pain sensation in human.

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P1-338 Responses of muscle C-fiber sensory receptors recorded from rat muscle-nerve preparations in vitro after eccentric contraction

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To examine whether sensitivities of muscle C-fiber sensory receptors were changed in hyperalgesic muscle after eccentric contraction (ECC), single C-fiber activities were recorded from extensor digitorum longus muscle-peroneal nerve preparations *in vitro* 2 days after ECC. The response threshold to mechanical stimulation was significantly decreased and the response magnitude was significantly increased in C-fibers recorded from the ECC preparation when compared with those from the untreated control. Discharges induced by chemical (pH 5.5, lactic acid) and thermal (cold, heat) stimulations were not different between two preparations. Augmentation of the mechanical response in muscle C-fiber sensory receptors might be related to the tenderness in delayed onset muscle soreness after eccentric exercise.

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P1-339 Temperature-dependent inhibition of C fiber-evoked excitation via vaniloid and purinergic receptors in the substantia gelatinosa of rat spinal cord slices revealed by optical imaging

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Temperature-dependent changes in C fiber-induced neuronal excitation were studied in the substantia gelatinosa of rat spinal cord slices stained with a voltage-sensitive dye. By a rise of the perfusate temperature from 27 to 33 °C, excitation was depressed by 30%. This inhibition was antagonized by vaniloid receptor antagonist capsazepine and P_2X and P_2Y receptor antagonist suramine, but not by P_2X receptor antagonist TNP-ATP. The excitation in the presence of ionotropic glutamate receptor antagonists AP5 and CNQX was also inhibited by the temperature increase, the result suggesting that C-fibers and their terminals are the primary site of action. Supported by the Grant-in-Aids from JSPS to HI.

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P1-340 In vivo patch-clamp analysis of interaction of excitatory and inhibitory synaptic inputs in the substantia gelatinosa of the rat spinal dorsal horn

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Whole-cell recordings were made from substantia gelatinosa (SG, lamina II of the spinal cord) neurons of urethane anesthetized rat to analyze excitatory and inhibitory synaptic responses evoked by cutaneous natural stimulation. The lumbar spinal cord was exposed and then L1-4 and S1-2 dorsal roots were cut. In most L3 and L4 SG neurons examined, pinch and touch stimuli produced a barrage of EPSCs. Touch stimuli evoked a barrage of IPSCs. The size of receptive field of IPSCs was larger than that of EPSCs. When L5 dorsal root was stimulated by a bipolar electrode, EPSCs and IPSCs were observed in L3 and L4 SG neurons. While L2 SG neurons exhibited IPSCs but not EPSCs in response to the L5 root stimulation and touch stimuli. These results suggest that the extension of inhibitory circuitry in the SG is more widespread than that of excitatory spreading.

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P1-341 Stimulation of the rostral ventromedial medulla facilitates inhibitory neurotransmission in the substantia gelatinosa of the rat spinal cord -In vivo patch-clamp analysis-

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5-HT is one of the major neurotransmitters that modulate noxious transmission at the spinal dorsal horn and is involved in the bulbospinal neurons located in the nucleus raphe magnus and adjacent reticular formation. In the present study, we observed the effect of 5-HT on the inhibitory inputs to the substantia gelatinosa (SG) by electrical stimulation (ES) of the rostral ventromedial medulla (RVM). Under anesthesia, bipolar stimulating electrodes were inserted into the RVM stereotaxically. Then synaptic responses of SG neurons were recorded using an in vivo patch-clamp technique. In the voltage-clamp mode (VH, 0 mV), SG neurons exhibited spontaneous IPSCs (sIPSCs). ES applied to the RVM increases the frequency of sIPSCs in some of the neurons tested. The facilitation of inhibitory inputs to SG neurons may play an important role in the descending inhibitory system of 5-HT.

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P1-342 Acute and late effects on induction of allodynia by acromelic acid, a mushroom poison related structurally to kainic acid

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Intrathecal administration of acromelic acid (ACRO)-A and ACRO-B provoked marked allodynia. Dose-dependency of the acute effect of ACRO-A showed a bell-shaped pattern. NMDA antagonists and JSTX inhibited the allodynia induced by ACRO-A, but not by ACRO-B. Induction of allodynia by low doses of ACRO was lost 1 week after i.t. injection of a sublethal dose of ACRO. In the ACRO-A-treated mice, higher doses of ACRO-A could evoke allodynia dose-dependently, which was not inhibited by JSTX or NMDA antagonists. Properties of the late allodynia induced by ACRO-A were quite similar to those of the acute allodynia by ACRO-B. In the deeper laminae of the spinal cord, ACRO-A could increase $[Ca^{2+}]i$ which was not blocked by JSTX. Taken together, these results demonstrate the stereospecificity of ACRO for the induction of allodynia and suggest the presence of a receptor specific to ACRO.

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P1-343 Involvement of NO in increase of exitatory amino acids level in the trigeminal nucleus caudalis of the rats with nerve injury-induced allodynia

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We have observed that NO-induced increase of exitatory amino acids (EAAs) level in the site of the first synaptic relay of the dental pain pathway (SpVc) plays an important role in development and/or maintenance of allodynia. Therefore, to investigate mechanisms of NO to changes in the EAAs level, the present study examined effect of sulfhydryl reagents on changes of the EAAs level in SpVc. The superficial layer in SpVc was perfused with ringer's solution through microdialysis probe using SD rats. EAAs level was analyzed by HPLC. The application of N-ethylmaleimide (1mM) into the perfusate enhanced EAAs level, but not 2-mercaptoethanol (2-ME, 1mM). 2-ME inhibited capsaicin-evoked increase in EAAs level. These results indicate that NO stimulates EAAs release from synaptosomes by affecting some components of the synaptic vesicle docking and fusion machinery.

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P1-344 Long-lasting tactile allodynia mediated by spinal P2X-purinoceptors in rats

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We examined the effects of i.t. injection of ATP and related compounds in the von Frey filament test in rats. An i.t. injection of ATP (30-300 nmol) or α, β-methylene-ATP, a P2X-purinoceptor agonist (30-100 nmol), induced allodynia within 15 min, and lasted even 7 days after the injection. Such an allodynia was induced also in neonatal capsaicin-treated adult rats. The ATP-induced allodynia was inhibited by co-injection of P2X-purinoceptor antagonists, including a selective $P2X_3/P2X_{2/3}$ antagonist A-317491. It was suppressed by s.c. injection of morphine for first 45 min, but then gradually developed. The allodynia on 1 day after the i.t. injection of ATP was inhibited by i.t. injection of P2X antagonists. These data suggest that development and maintenance of ATP-induced long-lasting allodynia is mediated by spinal P2X (probably $P2X_{2/3}$)-purinoceptors on the capsaicin-insensitive primary afferent neurons.

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P1-345 Effects of intrathecal injection of antidepressants on allodynia in the rat neuropathic pain model

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Antinociceptive effects of antidepressants are derived from reuptake inhibition of serotonin and/or noradrenaline at neuronal terminals. Thus, we compared the anti-allodynic effects of milnacipran, serotonin and noradrenaline reuptake inhibitor (SNRI), and paroxetine or fluvoxamine, selective serotonin reuptake inhibitors (SSRI). Three antidepressants were injected intrathecally into rats that showed allodynia after unilateral sciatic nerve constriction prepared by using polyethylene cuffs. The rat hind paw withdrawal thresholds were measured by von Frey filaments. Intrathecal administration of 10^{-5} M milnacipran significantly attenuated allodynia, whereas two SSRIs were not affected at 10^{-3} M. These results suggest that noradrenergic mechanisms seem to be a significant factor but not serotonergic on the anti-allodynic effect of antidepressants.

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P1-346 Optical Coherence Tomography revealed in vivo the Rise of Cortical Plasticity of Adult Mice in response to Peripheral Neuropathic Pain

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We studied neural plasticity of mice by the use of optical coherence tomography (OCT). To examine the capabilities of this technique in assessing neural plasticity, we chose primary somatosensory (SI) and motor (MI) cortices of mice under the influence of sciatic nerve chronic constriction injury (CCI), which is a model of neuropathy widely utilized in rats. In vivo OCT images revealed that the scattering intensity within the hindlimb area of SI and MI regions was higher in the hemisphere contralateral to the ligated sciatic nerve than in the ipsilateral hemisphere. Synapse and mitochondrion are expected to have high scattering coefficients, since they contain remarkably high concentration of proteins. Thus, our data suggest that the neuropathy gives rise to the neural plasticity within the hindlimb area of SI and MI contralateral to the ligated sciatic nerve.

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P1-347 Effect of systemic milnacipran administration in rats with neuropathic pain.

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Numerous case reports and trials have shown evidence that tricyclic antidepressants (TCAs) have analgesic effect in patients with chronic pain. Milnacipran, a serotonin and noradrenalin reuptake inhibitor (SNRI), seems to be better in tolerance clinically than TCAs. The aim of this study is to show the analgesic effect of milnacipran in neuropathic pain model animals. We used 18 rats with chronic injury in sciatic nerve (Kruger and Mosconi model, 1996). Two weeks after the administration of either milnacipran (1, 5, 10 mg/kg/day, n=12) or saline (control, n=6) by osmotic pomp, the thresholds of withdrawal response were measured by using von Frey test. We found that the thresholds were significantly reduced at 10 mg/kg/day of milnacipran compared with control rats. The results suggest that milnacipran has the analgesic effect in the neuropathic pain model.

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P1-348 Analgesic effect of ramatroban on neuropathic pain

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Prostaglandins are well known to play an essential role in acute and chronic pain not only in the periphery but also in the spinal cord. In the present study, we studied the effect of thromboxane (TP)/CRTH2 receptor antagonist ramatroban on neuropathic pain induced by L5 spinal nerve transection in mice. Mechanical allodynia was evident on day 3 post-surgery and maintained during the 10-day experimental period. Oral administration of ramatroban relieved the mechanical allodynia on day 7 in neuropathic mice in a dose-dependent manner. Following L5 nerve transection, the increase in nitric oxide synthase (NOS) activity stained by NADPH diaphorase histochemistry was observed in the superficial layer of dorsal horn in the spinal cord. TP agonist I-BOP, but not prostaglandin D_2, stimulated NOS activity in spinal slices. These increases in NOS activity were blocked by ramatroban. These results suggest that the analgesic effect of ramatroban on neuropathic pain is mediated by inhibition of NOS activity via TP receptor antagonism.

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P1-349 The molecular mechanisms of neuropathic pain through LPA receptor

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Peripheral nerve injury in human can lead to a pain state referred to as neuropathic pain, with symptoms that include continuous burning pain, and abnormal sensory sensations such as allodynia (pain as a result of non-noxious stimuli) and hyperalgesia (an increased response to a normally painful stimuli). Use of experimental animal models has identified many associated cellular events ranging from peripheral nerves system to central system. Animals with nerve injury develop behavioral allodynia and hyperalgesia paralleled by increased expression of both ion channel and neuropeptide in dorsal root ganglion, ephaptic cross-talk between primary afferent, demyelination in dorsal root, retraction of primary afferent, and increased expression of the protein kinase within the spinal cord dorsal horn. We will discuss the role of LPA and its receptor in these molecular events causing neuropathic pain.

P1-350 Effects of static magnetic field for muscular contraction by pain

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The biological effects of static magnetic field were studied using isotonic contraction of excised vas deferens of guinea-pigs under pain-related drugs (capsaicin and formalin). The preparation was stimulated every minute with a rectangular pulse applied through stimulator in order to elicit the twitch. The distance of 20 mm was used, which produced static magnetic field of 280 mT. The increase in the mechanical response to transmural stimulation observed with capsaicin and formalin. Capsicin administration caused to increase the response after magnetic stimulation. After magnetic field exposure, the enhancing effects of capsaicin and formalin on the twitch was decreased. During magnetic field exposure, Indomethacin and Dynorphin A inhibited the responses of the vas deferens induced by the transmural stimulation. These studies suggest that under pain-antagonist administration, static magnetic field induced to modulate the muscle contraction under pain-antagonist administration.

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P2-001 GABA$_B$ receptor-mediated inhibition of bursting ventral mossy cells in the developing mouse dentate gyrus

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We have previously reported that ventral mossy cells exhibit intrinsic rhythmic bursting activity, and suggested the possible existence of GABA$_B$ receptor-mediated tonic inhibition in these neurons. Here we estimated GABA$_B$ receptor-activated currents in ventral bursting cells using whole-cell patch clamp recordings. First, application of GABA$_B$ receptor antagonist (CGP55845A) to non-bursting cells often induced rhythmic bursting activity. Next, application of GABA$_B$ receptor agonist (baclofen) to intrinsic rhythmic bursting cells abolished their burst firings. The I-V relationship of a baclofen current in ventral bursting cells confirmed the existence of GABA$_B$-mediated potassium currents. These findings demonstrate that GABA$_B$ receptor-mediated currents play an important role in modulation of bursting ventral mossy cells.

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P2-002 Effect of PKC activity on the TTX-R Na$^+$ current in the nodose ganglion neurons

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To determine how protein kinase C (PKC) activity influences properties of the tetrodotoxin-resistant sodium current (TTX-R I_{Na}) in neonatal rat nodose ganglion (NG) neurons, we assessed the effects of phorbol,-12-myristate,13-acetate (PMA), one of the PKC activators, and staurosporine, one of the PKC inhibitors, on the current. PMA (30 and 100 nM) induced an increase in the peak current amplitude of normalized current-voltage curves, a leftward shift in the potential for half activation ($V_{1/2}$) of normalized conductance-voltage curves and a leftward shift of $V_{1/2}$ potential for steady-state inactivation curves. The effects of staurosporine (0.1 and 1 μM) on the peak current amplitude and the $V_{1/2}$ potential for activation were opposite compared with those seen after PMA application. These results suggest that the basal TTX-R I_{Na} obtained from neonatal NG neurons is controlled by the level of PKC activity, which can modulate excitability via alternation of TTX-R I_{Na} in the NG neuron.

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P2-003 Cholinergic oscillation in hippocampus of *tottering* Ca^{2+} channel mutant mice.

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Neurological mutant mice serve as useful in vivo models to investigate channel functions in neural activity. *tottering* (*tg*) mouse is a well known epilepsy model that has mutations in the P/Q-type Ca^{2+} channels α1A subunit gene. While functional abnormalities of *tg* mutant Ca^{2+} channels have been revealed at the molecular level, important impacts of these mutant channels in controlling network activities and behaviors have been poorly understood. The present studies investigate influences of altered properties of mutant Ca^{2+} channels on cholinergic oscillation in hippocampus, to understand mechanism underlying epileptic phenotype in *tg* mutant mice at the network level. High frequency patterns of oscillation are more frequently observed in tg hippocampus. Pharmacological studies revealed that the contribution of inhibitory circuits to cholinergic oscillation in *tg* mice differs from that in wild type.

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P2-004 Predicting spike timings of current-injected neurons

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Typical models of a cortical neuron were examined on the ability to reproduce and predict the spike sequences for a variety of fluctuating currents, using a fixed set of parameter values. We found that with the parameter values determined using the randomly fluctuating current, the models yield spike timings with better accuracy than the models with the parameter values determined using conventional methods. In the successful parameter determination method, the effective membrane time constant is estimated very short, typically about 5 ms. The predicting accuracy was found to be particularly good for the Hodgkin-Huxley models augmented with the Ca^{2+}-dependent potassium current generating slow after hyperpolarization and/or the muscarine-sensitive potassium current.The biological neurons we examined were distinctly classified into two types according to the estimated percent contents of those ionic channels.

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P2-005 Electroconvulsion (ECT) hyperpolarizes neocortex pyramidal cells by inducingHomer1a/Vesl1S expression

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Homer 1a upregulation by seizure activity is known. What is its significance ? We revealed (1) that injection of Homer 1a protein elicited hyperpolarization by 5-10 mV below rest in visual cortex pyramidal neurons in slices, and (2) that activation of mGluR by Homer 1a produces IP_3, thereby causing inositol-induced calcium release (IICR) and a consequent BK channel opening, which finally induces hyperpolarization. We have now made recordings in slices from rats subjected to ECT. We observed hyperpolarization similar to that in Homer-1a-injected cells, and also this hyperpolarization was dependent on mGluRs, IICR and BK channels. Injection of anti-Homer 1a, but not anti-Homer 1c, antibody prevented this hyperpolarization. These findings suggest that an innate feedback regulation of excitability, leading to pyramidal cell hyperpolarization, is switched on by seizure activity, and Homer 1a plays a major role in this feedback.

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P2-006 Localization and function of Na$_x$ sodium channel in glial cells

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Recently, we showed that Na$_x$ sodium channel is expressed the circumventricular organs (CVOs) where is thought to be the sensory loci detecting the extracellular sodium level. Based on several lines of evidence, we hypothesized that Na$_x$ is a sodium-level sensor working in the brain. We here studied immuno-electron microscopic localization of Na$_x$ in the CVOs, and found that Na$_x$-immunopositive signals were specifically associated with fine and thin processes surrounding neurons. These processes were extended from cell bodies of astrocytes and tanycytes, indicating that they are glial end-feet. In addition, we generated glial cell lines stably expressed Na$_x$ using Na$_x$ cDNA. The Na$_x$-positive glial cells were sensitive to an increase of extracellular sodium level. These results suggest that Na$_x$ expressed in the glial end-feet is involved in sodium-level sensing in the CVOs.

P2-007 TEA sensitive and less-sensitive I_{AS} in rat subfornical organ neurons

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Although there are many papers on A-type potassium outword current (I_A) in the subfornical organ (SFO), it is unclear which Kv α-subunits is generating I_A. We investigated I_A in SFO neurons by using RT-PCR and patch-clamp. RT-PCR revealed that the SFO had mRNA of Kv1.4, Kv3.4, Kv4.1, Kv4.2, Kv4.3L, but not Kv3.3 and Kv4.3M. Next, we examined I_{AS} in acutely dissociated 60 SFO neurons of rats. In 5 neurons, only I_K was observed and I_A was not. TEA sensitive I_A existed in 16 neurons, suggesting Kv3.4. Under the presence of 1 mM TEA, TTX and $CdCl_2$, other neurons showed TEA less-sensitive I_A. Further the neurons showing TEA less-sensitive I_A were divided into two groups by recovery rate from inactivation. Recovery time constant in one group was 60.9 ms, suggesting Kv4. Recovery time constants in the other group were 115.8 ms and 11.8 s, suggesting Kv4 and Kv1.

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P2-008 VDCC and neuronal cell death induced by GABAA receptor inhibition

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Although numbers of studies have been made on the neuronal cell death induced by $GABA_A$ inhibition, the mechanism has not yet been determined. In this study, we examined the relation between $GABA_A$ receptor inhibition and voltage dependent calcium channel (VDCC) using a rat hippocampal slice. A hippocampus of Wistar rats (7-8-day post-natal) was sliced into 350-μm - thickness and cultivated on porous membranes for 7 days under 10% CO2 at 35 °C in the modified DMEM (Gibco). Bicuculline (6 μM) was added to the solution to inhibit $GABA_A$ receptors. The cell death was examined using PI-uptake analysis. mRNA was quantified by the real time PCR measurement. PI-uptake result indicated the severe cell death by bicuculline, which was suppressed by a P/Q-type VDCC blocker. The real time PCR result showed that bicuculline stimulation changed the expression level of the P/Q-type VDCC mRNA. These results demonstrated that neuronal cell death induced by $GABA_A$ receptor inhibition was through P/Q-type VDCC pathway.

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P2-009 Effects of channel blockers on the polar excitations in frog sciatic nerves

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Ar+ laser irradiation decreased the amplitude of compound action potential (CAP) generated from the anode more rapidly than that from the cathode in the frog sciatic nerve, and the effect was similar to the case of applying an Ih channel blocker, ZD7288. We examined effects of channel blockers on the polar excitations to reveal whether such effect of ZD7288 is specific or not. Cathode circuit-closed excitation(CE) and anode break excitation(AE) were recorded and their initial amplitudes were standardized with 100%, while giving supramaximal stimuli(10 ms duration) and applying small cotton infiltrated by a channel blocker to the stimulus portion for 30 min. In control(n=3; no applications), amplitudes of CE and AE remained at almost the same level after 30 min; 106±9%(mean ± S.E.) and 102±5%. ZD7288(n=3) decreased the amplitude of AE(18±11%) more rapidly($p<0.01$) than that of CE(50±2%) after 30 min. TTX(n=3) decreased them to 69±2% and 3±2% after 3 min, and to 20±9% and 0±0% after 10 min. Effects of K+ and Ca2+channel blockers will be reported

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P2-010 Inhibitory effects of barbiturates on ATP-sensitive K channels in rat substantia nigra

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Surface ATP-sensitive K (KATP) channels in neurons are shown to control seizure activities during hypoxia. Although barbiturates are often used in patients with brain ischemia, effects of these agents on neuronal KATP channels have not been clarified. We studied effects of barbiturates on these channels in principle neurons of rat substantia nigra pars compacta. Whole cell recordings were made using midbrain slices. When neurons were dialyzed with an ATP-free pipette solution, a hyperpolarization and an outward current developed slowly in a tolbutamide-inhibitable manner. Thiopental inhibited the hyperpolarization and outward current induced by intracellular ATP depletion at 100 and 300 μM. A high dose of pentobarbital also exhibited similar effects on KATP channels. These results indicate that high doses of barbiturates inhibit KATP channels when the channels are activated by low intracellular ATP.

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P2-011 The effects of the synaptic location on the synchronized activity in neural networks of fast rhythmic bursting neurons

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In neocortex, oscillated neural activities in gamma range frequency (20-70 Hz) occur as a result of the fast rhythmic bursting (FRB) firing which work as a pace-maker. In the previous papers, the phase response of two type FRB neuron model are presented. One model is one-compartment model which includes Ca^{2+}-depend cation current, and shows significant transition from anti-phase to in-phase firing state accompanied by the bursting mode changes. Another model is two-compartment model which includes parsistent Na^+ current, and the synchronization properties depend strongly on the synaptic location. Here, we investigated the effects of synaptic location by using the multi-compartment model which based on the cation current model. It is found that the model neurons are more rapidly synchronized or desynchronized than the network of persistent current model, and show phase transition regardless of where the synapse locate.

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P2-012 Homer1a/Vesl1S enhances spike-induced Ca^{2+} influx through L-type channels

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We reported a spike-induced supra-linear increase in $[Ca^{2+}]_i$ in visual cortex pyramidal cells, in which spike-induced Ca^{2+} influx triggers Ca^{2+} release from IP_3 receptors (IP_3Rs) sensitized by a prior increase in IP_3(IP_3-assisted CICR). Activation of group I metabotropic glutamate receptors (mGluRs) suffices to provide the requisite IP_3. Homers, attachable to both IP_3Rs and mGluRs, may be involved in this supra-linear Ca^{2+} increase. We now study whether Homer 1a, presumed to act as a dominant negative regulator, affects spike-induced Ca^{2+} increases. Injection of Homer 1a protein amplified Ca^{2+} increases induced by single spikes. This amplification was attributable to enhancement of Ca^{2+} influx through L-type Ca^{2+} channels, but neither through the other types of voltage-gated Ca^{2+} channels nor release channels. Thus, although a supra-linear increase in spike-induced Ca^{2+} increase was observed after Homer 1a injection, this surprisingly did not depend on IP_3-assisted CICR.

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P2-013 Volume-sensitive Cl^- channels are involved in recovery from excitotoxic neuronal swelling

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During excitotoxic insults, such as ischemia, neurons undergo rapid swelling both in the soma and dendrites. Focal swelling along the dendrites, called varicosity, is known to be induced by excess influx of Na^+ via glutamate receptors and Cl^- via unidentified pathways. If the insult is sublethal, neurons can recover from varicosities after removal of excitotoxic stimuli. Although this recovery process should be accomplished by the efflux of water driven by effluxes of cations and anions, its mechanism is unknown so far. Here we demonstrate that the volume-sensitive outwardly rectifying Cl^- channel is expressed and involved in somatic volume regulation after hypotonic swelling, and that this channel exists on varicosities formed under glutamate excitotoxity and plays an important role in recovery from varicosities by serving as the volume-regulatory Cl^- efflux pathway in cultured mouse cortical neurons.

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P2-014 Depolarizing effects of orexin-A and -B on isolated locus coeruleus neurons are mediated through nonselective cationic and K^+ conductances

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The locus coeruleus (LC) is known to be noradrenergic neurons, which are innervated by orexin (ORX)-like immunoreactive axons, and express orexin receptors (OXRs), both OX_1R and OX_2R. We analyzed effects of ORX-A and -B (ORX-A/B) on dissociated LC neurons by using whole-cell patch clamp techniques. In current-clamp mode LC neurons were depolarized by application of 10^{-7} M ORX-A and 10^{-7} M ORX-B. The firing frequency of action potentials increased by application of ORX-A/B and returned to control levels after removal of these peptides. The depolarizing effects were maintained in the presence of TTX, CNQX and AP5. In voltage-clamp analysis, we demonstrated that ORX-A/B enhanced a non-selective cationic conductance and inhibited the sustained K^+ current. The modulation of these current conductances mediates the membrane depolarization and an increase in firing frequency indicating direct effects of ORX-A/B on LC neurons.

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P2-015 Involvement of Rho and Rho-kinase in the augmenting effect of 5-HT on the depolarization-induced outward current response

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A step depolarization of the membrane from -60 to +10 mV elicits an outward current in the *Aplysia* neuron under voltage-clamp. This current is mainly composed of Ca^{2+}-activated K^+ current triggered by Ca^{2+} entry through voltage-dependent Ca^{2+} channel. Application of 5-HT to the cell markedly augmented the outward current. Intracellular injection of either toxin B or C3 exoenzyme, blocker of small G-protein RhoA-C, depressed the augmenting effect of 5-HT on the outward current. Furthermore, intracellular injection of Y-27632, an inhibitor of Rho-kinase, markedly depressed the augmenting effect of 5-HT on the outward current. It is suggested that the augmenting effect of 5-HT may be mediated by the activation of Rho and subsequent Rho-kinase.

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P2-016 Is Stargazin Involved in the AMPA Receptor Channel Complex ?

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Stargazin (γ-2) is a protein coded by a causative gene for stargazer (stg) mutant mouse. It has been reported to be critical for surface expression and synaptic targeting of AMPA receptors. Here, we found that glutamate-evoked currents through AMPA receptor channels were significantly changed when co-expressed with AMPA receptors and γ-2 subunit proteins except γ-1 and γ-6 in either Xenopus oocytes or HEK293 cells. These effects on current amplitude were independent of the change of AMPA receptor surface expression. Furthermore, coexpression of GluRα1 subunit and γ-2 in Xenopus oocytes produced higher channel activity and cooperativity compared with the GluRα1 subunit expressed alone. These results suggest that γ subunits are functional constituents of the AMPA receptor.

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P2-017 Characteristics of AMPA- and Quisqualic acid-induced current responses in the CA1 pyramidal neuron of rat hippocampal slice

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Application of either AMPA or Quisqualic acid (QA), agonists for AMPA-receptor, induces slow inward current response in the CA1 pyramidal neuron in rat hippocampal slice under whole-cell patch clamp. When one of these agonists was applied repeatedly, the second response to AMPA or QA was occluded at the shorter interval of the agonist application. However, there was no sign of the desensitization in the response, although GABA-induced Cl^--current response examined in the same cell exhibited strong desensitization. Furthermore, perfusion of cyclothiazide (CTZ), a reagent that relief the desensitization of AMPA-receptor, markedly augmented the AMPA- and QA-induced response without affecting the time course of the respopnse. These results suggested that the AMPA-receptor of the pyramidal neuron in the rat hippocampal slice may be lacking a nature of the desensitization.

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P2-018 Purification of recombinant protein of ATP receptor $P2X_2$ using baculovirus expression system.

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We previously observed that the pore properties of ATP receptor $P2X_2$ change depending on the expression level, suggesting dynamic structural rearrangements. However, the structure, even the stoichiometry, remains unknown. Towards the structure analysis, we aimed to purify recombinant $P2X_2$ protein. We first made constructs of $P2X_2$ tagged with FLAG at either N- or C-terminus, and confirmed that they have intact function. We then made them express in Sf9 cells using baculovirus expression system. We solubilized the FLAG-tagged $P2X_2$ proteins in a solution containing 50 mM dodecyl-β-maltoside, purified them using FLAG affinity gel. A single major band was successfully observed by silver staining of SDS-PAGE. We purified the products further using gel filtration chromatography.

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P2-019 Activation of P2Y1 receptors directly excites inhibitory interneurons in the hippocampus

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Despite their abundant expression, the role played by P2Y receptors in the regulation of synaptic transmission in the CNS is still undetermined. We have recently demonstrated that extracellular ATP increased IPSC frequency through activation of P2Y1 receptors in the CA3 pyramidal neurons. To identify the mechanism underlying this effect, responses of interneurons to P2 receptor agonists were analyzed in the hippocampal slices of the young rats. In a subset of interneurons in stratum oriens, 2meSATP generated transient inward currents (~15 pA) and cell firing, an effect was reversibly abolished by MRS2179. Fluorometric measurement revealed several numbers of interneurons responding to 2meSATP with a marked rise in $[Ca^{2+}]_i$. These results point to a mechanism transducing a rise in $[ATP]_o$ into increased inhibitory activity in the hippocampal network through a novel direct coupling between metabotropic ATP receptors and specific cationic conductance in the interneurons.

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P2-020 Sevoflurane affects multiple membrane conductance in the locus coeruleus of the rat

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It is proposed that two-pore-domain K^+ channels (TASK channels), underlying the resting membrane conductance, are one of the targets of the volatile anesthetics. The neurons in the locus coeruleus (LC), which play important roles in the regulation of consciousness and pain sensation, express TASK channels. We recorded membrane currents of LC neurons in the pontine slice of the rat to analyze how TASK channels mediate the effect of sevoflurane. In 6 of 7 neurons, sevoflurane 5% activated an early inward current (-10.7±3.4 pA) and subsequent late outward current (latency, ~220 s; 14.2±9.2 pA) at pH=7.4. At pH=6.5, a condition that suppresses TASK channels, the late outward component was not observed in 6 of 7 neurons, whereas the early inward current was evident. We conclude that sevoflurane at a clinically relevant concentration, activates not only the TASK current but also more rapidly activating unidentified excitatory conductance.

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P2-021 Altered mechanism of transmitter release regulation by G-protein-coupled receptors in the autonomic sensory synapse in mice lacking N-type calcium channels.

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The EPSCs evoked by stimulation of the autonomic primary afferent fibers (eEPSC) were analyzed in the nucleus tractus solitarii (NTS) in the brainstem slice from the mice lacking α_{1B} subunit of N-type voltage dependent Ca channels (Cav2.2-/-), which are reported to show prominent autonomic dysfunctions. We found that, in Cav2.2-/- mice, the eEPSC amplitude was i) not significantly different from that of the wild type, ii) not decreased by ω-conotoxin GVIA (CgTx), unlike in the wild type, and iii) decreased significantly by adenosine, as in the wild type, which was however not affected by CgTx or ω-agatoxin IVA. A crucial modification of the link between presynaptic G-protein-coupled receptors and VDCCs might occur in the NTS of Cav2.2-/- mice.

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P2-022 Mechanisms underlying developmental shortening of presynaptic action potential at the calyx of Held

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Rodents acquire hearing ability at postnatal day (P) 9-13. Across this period, various changes occur at the calyx of Held in the medial nucleus of trapezoid body (MNTB) in the auditory brainstem. For example, presynaptic action potentials (APs) become significantly shorter, thereby possibly contributing to the high-fidelity transmission for high frequency inputs.
To clarify mechanisms underlying the developmental shortening of presynaptic APs, we made patch-clamp recordings of voltage-gated K^+ and Na^+ currents directly from calyceal presynaptic terminals in brainstem slices of rats. Pharmacological properties of K^+ currents were similar between P7 and P14, but those at P14 had a faster rise time and a larger current density than P7. The density of presynaptic Na^+ currents remained similar, but their inactivation time became shorter by 50% from P7 to P14.
These results suggest that developmental changes of both K^+ and Na^+ channels underlie that of the AP waveform.

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P2-023 Expression of CMR1 in the rat sensory neurons innervating the urinary bladder

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Ice-water test (cold stimuli) is used in diagnosis of detrusor overactivities. CMR1, a non-selective cation channel belonging to the transient receptor potential family, responds to cold stimuli. In the present study, we examined the expression of CMR1 gene and protein in the sensory afferents that innervate to the urinary bladder. We also checked whether the expression is changed in the bladder outlet obstruction (BOO) model. In situ hybridization revealed that CMR1 mRNA was expressed in small-diameter neurons of the rat L6-S2 DRG. The DRG neurons, especially of the S1 level, showed co-localization of CMR1 mRNA and a retrograde tracer that was injected into bladder wall. There was no significant difference of CMR1 mRNA expression between normal and BOO rat DRG. We also raised an antibody to rat CMR1 protein. Double labeling of in situ hybridization and immunohistochemistry showed that CMR1 mRNA and CMR1 protein were co-localized in small-diameter neurons of the DRG.

P2-024 pH homeostasis of microglia: The voltage-gated proton channel serves as a pH regulator upon cell acidosis

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The voltage-gated proton (H^+) channel, highly expressed in microglia, is required for phagocytosis since they functionally couple with NADPH oxidases responsible for respiratory bursts. A massive amount of H^+ generated via the oxidases is quickly extruded through the H^+ channel. In this study we examined the role of the channel during transient and reversible cell acidosis in rat microglia. Perforated whole cell recordings revealed that cell acidosis, induced either by removal of pre-loaded NH_4Cl or addition of lactic acid, activated the H^+ channel under actions of intrinsic pH buffers. Enhanced H^+ efflux leads to a rise in intracellular pH, and hence terminates the channel activation. The H^+ channel, thus, serves to maintain pH homeostasis of microglia upon various types of cell acidosis.

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P2-025 Hyperpolarization-activated currents play a minor role in unique distribution of DC field-induced responses in CA1 region of rat hippocampal slice

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We previously reported unique spatio-temporal profiles of DC filed-induced responses in CA1 region of rat hippocampal slice and indicated that the distal dendrites of CA1 pyramidal neurons have a lower membrane resistance using a compartmental model simulation. Hyperpolarization-activated currents (I_h) were reported to be densely in the apical dendrite of pyramidal neurons and they would make distal dendrites "leaky". Here, we examined the contribution of those currents to the responses. CsCl and ZD7288, I_h channel blockers, did not alter the spatio-temporal profiles of DC field-induced responses recorded with optical method. Additionally, we performed patch clamp recordings from soma and apical dendrites of CA1 pyramidal neurons. And the result indicates that the responses recorded optically in CA1 region reflected membrane potential changes of CA1 pyramidal neurons.

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P2-026 IRBIT regulates inositol 1,4,5-trisphosphate receptor in a phosphorylation-dependent manner

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The inositol 1,4,5-trisphosphate (IP3) receptors (IP3Rs) are IP3-gated Ca^{2+} channels on intracellular Ca^{2+} stores. We report a novel IP3R-binding protein, termed IRBIT (IP3R binding protein released with inositol 1,4,5-trisphosphate). IRBIT had a domain homologous to S-adenosylhomocysteine hydrolase in the C-terminal and, in the N-terminal, a 104 amino acid appendage essential for the interaction with IP3R1. Phosphorylation of IRBIT was necessary for the interaction. IP3 dissociated IRBIT from IP3R1 with an EC50 of ~0.5 μM, i.e. it was 50 times more potent than other inositol polyphosphates. IRBIT inhibited IP3 binding of IP3R1 in a phosphorylation-dependent manner. Knockdown of IRBIT by RNA interference resulted in a significant increase in the number of responsive cells to IP3-generating agonist stimulation in HeLa cells. These results indicate that IRBIT is a novel regulator of IP3-induced Ca^{2+} release.

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P2-027 Effect of intracellular ATP and PKA on the hyperpolarization-activated cation current in rat dorsal root ganglion neurons

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Hyperpolarization-activated current (I_h) is reported to be regulated by not only cAMP but also some other intaracellular factors, e.g., $[Ca]_i$, pH and PKA.In rat dorsal root ganglion (DRG) neurons, we found that the intracellular ATP produced a positive shift in the voltage dependence of I_h. We therefore examined the modulatory mechanism by ATP and tested whether phosphorylation-dependent mechanism via PKA is involved in the modulatory process of I_h. An adenylate cyclase inhibitor did not influence the effect of ATP and nonhydrolysable ATP analogs also positively shifted the acitivation curve of I_h. In the neurons dialyzed with the catalytic subunit of PKA, the potentiation of voltage-gated Ca^{2+} current was observed, while the properties of I_h was not changed. These results suggested that cytosolic ATP directly regulates I_h activation without phosphorylation by PKA.

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P2-028 Stability of Synchronous Solutions in Weakly Coupled Neuron Networks

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Previously, we showed that two fast-spiking interneurons synchronize when they are connected by an gap junction and they desynchronize when they are connected by a reciprocal $GABA_A$ connection by using the phase-reduction method. In the framework of the method, the stability of synchronous solution depends on both intrinsic cell properties and connection type, where the intrinsic cell properties are characterized by the phase-response-function which describes the response of a neuron to small perturbations of the membrane potential. In this study, the way in which the form of the phase-response-function and neuronal coupling affect the stability of the synchronous phenomena is reexamined in detail, and the need to stabilize synchrony is demonstrated for several neuron models such as fast-spiking interneuron and low-threshold-spiking interneuron models.

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P2-029 The activation of voltage-gated K^+ channels induced by rosin acid

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Voltage-gated K^+ channels (Kv) are essential for regulation of neuronal activity. Although many blockers selective to Kv channels have been reported, only small number of Kv channel openers has been known. Here, we report a compound, which activates Kv1.x. Cloned rat Kv1.1, rat Kv1.2, mouse Kv1.3 and rat Kv1.6 (delayed rectifier type) as well as rat Kv1.4 and rat Kv4.3 (A type K^+ channel) were heterologously expressed in HEK293 cells. We measured membrane currents under whole-cell voltage-clamp. Pimaric acid (PiMA) is one of major rosin acids and activates large conductance Ca^{2+} activated K^+ channels (Mol Pharmacol, 2002). Application of 10 μM PiMA increased current amplitude of all Kv1.x tested but did not rKv4.3 current. PiMA induced a shift of half activation voltage of rKv1.1 to negative potential by about 10 mV and decreased deactivation rate. These results suggest that PiMA increases the voltage sensitivity of Kv1.x and acts as an opener for them.

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P2-030 Cellular mechanism regulating the upregulation of Nav1.9, a TTX-resistant Na channel, in mouse dorsal root ganglion neurons

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Sensory neurons in dorsal root ganglia express two kinds of tetrodotoxin-resistant isoforms of the voltage-gated sodium channel, Nav1.8 (SNS) and Nav1.9 (NaN). Of special interest is Nav1.9, which is implicated in the pathophysiology of neuropathic pain. Nav1.9 is preferentially expressed in small DRG neurons and mediate a tetrodotoxin-resistant persistent sodium current. We examined properties of the sodium current mediated by Nav1.9 using Nav1.8-null mutant mice. Here, we report on the unusual properties of this current, which has not been reported for any other known voltage-gated ion channels, i.e., the current mediated by Nav1.9 recorded under whole cell patch clamp showed an explosive up regulation of the peak amplitude (kindling of Nav1.9) during recording.

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P2-031 Functional roles of TTX-sensitive and TTX-resistant sodium channels in action potential generation of the mouse dorsal root ganglion neurons.

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Dorsal root ganglion (DRG) neurons express 7 subtypes of voltage-gated sodium channels, 5 of which being sensitive to tetrodotoxin (TTX) and the remaining 2 being resistant. It is not known how these distinct types of sodium channels are involved in the spike electrogenesis of neurons in DRG. We compared the action potential generation under current-clamp recording with the sodium current under voltage-clamp recording in the same neuron. We found that the action potentials in small DRG neurons are largely dependent on Nav1.8, a TTX-resistant isoform. TTX-sensitive sodium channels were switched off in small DRG neurons through a hyperpolarizing shift of the steady-state inactivation, although they mediated action potentials in large DRG neurons.

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P2-032 Subtype-specific modulation of the channel activity of the NMDA receptor by PSD-95 and GKAP

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The NMDA receptor binds PSD-95 which interacts with GKAP (Guanylate Kinase-domain Associated Protein) though the GK domain of PSD-95. We examined the effects of PSD-95 and GKAP on four subtypes of the NMDA receptor. PSD-95 alone potentiated the channel activity of NR1/NR2A, NR1/NR2B and NR1/NR2D, but not of NR1/NR2C. Addition of GKAP further potentiated that of NR1/NR2B but not of NR1/NR2A and NR1/NR2D. The channel activity of NR1/NR2C was potentiated only in the co-presence of PSD-95 and GKAP. The experiments using mutant PSD-95 revealed that the potentiation of NR1/NR2B and NR1/NR2C by PSD-95 and GKAP required the GK domain. These results indicate that PSD-95 and GKAP modulate the channel activity of the NMDA receptor in the subtype-specific manner.

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P2-033 Characteristics of N-type calcium channels stably expressed in BHK N101cells

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Currents through N-type Ca^{2+} channels (consisting of α_{1B}, α_{2a}, and β_{1a} subunits and stably expressed in BHKN101 cells) were measured using the whole-cell recording method. BHKN101cells produced high-voltage activated Ca^{2+} current upon depolarization more positive than -40mV, while the control BHK6 cells lacking α_{1B} subunits did not show any appreciable current. The Ca^{2+} current seen in BHKN101 cells was reversibly suppressed by 10^{-6}M ω-CTX SVIB and irreversibly inhibited by 10^{-6}M ω-CTX GVIA. In contrast, neither ω-agatoxin IVA nor calciseptin affected the Ca^{2+} current. The results indicate that BHK N101 cells mainly express N-type Ca^{2+} channels and the cells offer a useful model for studying the regulatory mechanism of signal-transduction molecules upon Ca^{2+} channels.

P2-034 Reduction of EPSP Shunting by A-channels and its Effect on Neural Interaction

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We elucidate an effect of backpropagating action potentials (BAP)s on neural interaction by the NEURON simulator. First, we focus on the shunting of EPSPs by BAPs in the dendrite. While A-channels and Na-channels in the dendrite are blocked in various combinations, BAPs are paired with EPSPs and somatic EPSPs are observed. These results show that A-channels reduce the shunting of EPSPs. This rises the possibility that in the dendrite BAPs and EPSPs has weak interaction, and these are almost independent functionally due to A-channels. In the simulations, optimal densities of Na-channel and A-channel give the smallest shunting of EPSPs and its optimal value consists of one obtained by previous works. Next, we observe phase response curves of a neuron in oscillatory states. This experiment includes previous experiments for shunting EPSPs, and thus the effect of the shunting of EPSPs on neural interaction can be elucidated.

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P2-035 Characteristics of calcium influx elicited by high-KCl in undifferentiated and differentiated NG108-15 cells

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We have reported that the extent and rate of KCl-stimulated ^{45}Ca uptake by differentiated NG108-15 cells was significantly higher than those of undifferentiated cells. The present work further studies the characteristics of calcium influx underlying the previous results. NG108-15 cells were loaded with the Ca^{2+}-sensitive fluorescent dye fura-2, and dynamic changes in the $[Ca^{2+}]i$ in response to high-K^+ (30 or 60 mM) were monitored using an image analyzer ARGUS-50. The data show that 1) the amplitude of $[Ca^{2+}]i$ increase was greater in differentiated cells, 2) $[Ca^{2+}]i$ responses were suppressed when Ca^{2+} was removed from the bathing solution or by CaS, and 3) ω-Aga IVA, ω-CTX GVIA and SNX-482 exerted no suppressive effect on the $[Ca^{2+}]i$ increase.It is suggested that NG108-15 cells acquire larger numbers of Ca^{2+} channels (mainly L-type) during differentiation.

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P2-036 Helical Structure of S1 in the Hyperpolarization-Activated Cation Channel

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The four subtypes of the hyperpolarization-activated cyclic nucleotide-gated (HCN) channels in mammals show distinct activation kinetics, which may lead to their distinct physiological roles. We previously elucidated that the latter half of S1 is important for the activation kinetics. We focused on the structural basis of S1 in this study and replaced each residue in the latter half of S1 singly with tryptophan in HCN1. Seven out of fifteen Trp mutants expressed hyperpolarization-activated currents in COS-7 mammalian expression system. The periodicity in the effects of the mutations on the expression may demonstrate an alpha-helical structure for S1. The introduction of Trp into the amino acid residues responsible for the different kinetics between HCN1 and 4 also slowed the activation compared with HCN1 wild type. The introduction of Trp into the amino acid residues which are conserved among all HCN subtypes abolished the currents, which may support the hypothesis that these residues face against other transmembrane regions.

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P2-037 Morphological Changes of Tightly Associated Presynaptic Varicosities and Postsynaptic Spines

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Dendritic spines are highly motile structures, but how spines move while keeping their contacts with presynaptic varicosities is not clear. To analyze movements of dendritic spines and axonal varicosities simultaneously, we labeled CA1 pyramidal cells with GFP and CA3 pyramidal cells with rhodamine-dextran in hippocampal slice cultures. Varicosities and spines were visualized using two-photon microscope to detect close association of two components. Time-lapse imaging revealed that they performed rapid morphological changes without losing their contacts. The extent of overall structural changes between varicosities and spines was correlated, while the direction of short-term volume changes was regulated independently. These results suggest that synaptic junctions provide stable attachment sites functioning to correlate presynaptic and postsynaptic motility.

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P2-038 LRP13 is a PSD target protein for PSD-95 and SAP97, regulated by the Ca2+/calmodulin-dependent protein kinase II.

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We have reported a synaptic short form LDLR-related protein (ssLRP). In this paper we re-named it as LRP13 according to systematic nomenclature. The protein was enriched in the PSD fraction. The protein interacted with PSD-95 and SAP97 via its C-terminal tail, and was associated, possibly through these interactions, with both NMDA-type and AMPA-type GluR subunits. CaMKII phosphorylated the protein at Ser1887 and Ser1900 in the C-terminal cytoplasmic region, and the phosphorylation at the latter site negatively regulated the interaction of the protein with PSD-95 and SAP97. These findings suggest important roles of LRP13 for the regulation of postsynaptic signaling and synaptic plasticity.

P2-039 The Number and Density of AMPA Receptors in Single Synapses in Immature Rat Cerebellum

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The number of ionotrophic receptors in synapses is an essential factor to determine the efficacy of fast transmission. Using two-photon uncaging of glutamate and the non-stationary fluctuation analysis in P3-4 rat Purkinje cells (PCs) in slice, we estimated the number of functional AMPA receptors (AMPARs) at single postsynaptic sites. The slice preparations were then fixed for electron microscopy, and areas of postsynaptic membrane specialization at the same synapses were measured by 3D reconstruction. The numbers of functional AMPARs were proportional to the synaptic areas with densities of 1060 - 1300 receptors/μm^2. Moreover, SDS-freeze fracture replica labeling also revealed densities of immunogold particles for AMPARs as 910 ± 36 gold particles/μm^2 in synapse. Our results indicate that the efficacy of synaptic transmission is determined by the synaptic area in P3-4 rat PCs.

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P2-040 Characterization of rat p55, a MAGUK family member, in the synapse

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We have cloned p55, a MAGUK family member, from rat brain cDNA library. Affinity-purified antibody specifically recognized rat p55 but not p55 in human erythrocyte. The rat p55 protein was widely expressed in various tissues including erythrocyte and also distributed in PSD fraction of rat brain. This protein interacted with various PSD proteins, such as PSD-95, SAP97, GKAP, CASK, GRIP, cadherin, tubulin, actin, α-internexin, neurofilament-L and CaMKII, through its PDZ, SH3, HOOK and/or GuK domains. In situ hybridization and immunostaining of cultured cortical neurons supported that rat p55 was localized at synapse. These findings suggest that rat p55 works as a scaffold protein in both synaptic and extra-synaptic regions in rat brain.

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P2-041 NIDD, a novel DHHC-containing protein, targets neuronal nitric oxide synthase (nNOS) to the synaptic membrane through a PDZ-dependent interaction and regulates nNOS activity

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We reports identification and characterization of a novel nNOS-interacting DHHC domain-containing protein with dendritic mRNA (NIDD). Pull-down assays suggested that the C-terminal tail region of NIDD specifically interacted with the PDZ domain of nNOS. The interaction was further confirmed by co-sedimentation assays using COS-7 cells transfected with NIDD and nNOS. Both NIDD and nNOS were enriched in synaptosome and synaptic plasma membrane fractions and present in lipid raft and postsynaptic density fractions in the rat brain. Co-transfection of NIDD and nNOS increased NO-producing nNOS activity. These findings suggest that NIDD plays an important role in the regulation of the NO signaling pathway at postsynaptic sites through targeting of nNOS to the postsynaptic membrane.

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P2-042 BAALC 1-6-8 protein targets to postsynaptic lipid raft by its N-terminal myristoylation and palmitoylation, and interacts specifically with α subunit of Ca2+/calmodulin-dependent protein kinase II.

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We cloned a rat BAALC 1-6-8 isoform, and identified endogenous BAALC 1-6-8 proteins in the brain. The protein was highly selectively expressed in the brain, detected in the synaptic fractions, and localized to both neuronal soma and dendrite. The protein was increased in parallel with synaptogenesis. The protein interacted with the α, but not β, subunit of CaMKII. The interaction was between the N-terminal 35 a.a. region of 1-6-8 protein and the regulatory domain of CaMKIIα. N-terminal portion of the 1-6-8 protein was both myristoylated and palmitoylated and the dual modification was required for targeting of the protein into lipid raft. These findings suggest that BAALC 1-6-8 protein play a synaptic role at the postsynaptic lipid raft through interaction with CaMKII

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P2-043 Roles of the C2B domain of Synaptotagmin I for fast synaptic transmission in *Drosophila* embryos

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Synaptotagmin I (*Syt I*), a major Ca^{2+} sensor for fast synaptic transmission has two putative Ca^{2+} binding domains, C2A and C2B. The C2B domain seems crucially involved in Ca^{2+}-dependent transmitter release and has two Ca^{2+} binding sites, Ca1 and Ca2. To elucidate their roles in synaptic transmission *in situ*, we studied synaptic currents at the neuromuscular junction of *Drosophila* embryos that have mutations in these two binding sites in the *syt I* null background. In a transformant carrying mutations in the Ca1 site synchronized synaptic currents were completely abolished. In another transformant carrying mutations in the Ca2 site, synchronized synaptic currents was rarely evoked, but not abolished. Its quantal content increased with $[Ca^{2+}]_e$ with a slope of 2.4 in the double logarithmic plot, close to 3.1 in the control. We discuss about roles of the Ca1 and Ca2 sites for fast synaptic transmission.

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P2-044 Roles of Tubulin in synaptic vesicle recycling

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We have previously described Tubulin (TUB) dynamics in presynaptic boutons of *Drosophila* larvae during synaptic vesicle (SV) recycling. Here we investigated physiological roles of TUB in boutons using *Drosophila* TUB mutants. When FM1-43 dye was loaded by 90 mM K^+ stimulation to stain recycling SVs, the periphery of boutons (exo/endo cycling pool, ECP) was stained in wild-type (WT). In contrast, in TUB mutants FM1-43 was loaded into whole boutons, including the center (reserve pool, RP), by 90K^+ stimulation. The loaded dye was predominantly released by subsequent 90K^+ stimulation, but some remained in the center. While in WT Cyotochalasin D (Cyt D) inhibited FM1-43 loading into RP, in TUB mutants it blocked dye loading into whole boutons. Accordingly, in Cyt.D-treated TUB mutants synaptic potentials continuously declined during low-frequency electric stimulation. These data suggests that the TUB mutation results in a SV deficiency in ECP and that exocytosed SVs are replenished from RP by the actin-dependent mechanism in TUB mutants.

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P2-045 GABAergic input association with the VGLUT2-expressed terminal at the spine head of cortical neurons

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Our recent finding showed about 1/3 of axon terminals of a few nonpyramidal cell subtypes made synaptic contacts on the spine head with another asymmetrical contact [double innervated (DI) spine] in rat cortex. Two types of vesicular glutamate transporters, VGLUT1 and VGLUT2, show the complementary expression pattern in brain. In neocortex VGLUT1 is expressed in axon terminals of pyramidal cell, whereas VGLUT2 in those of thalamic and cortical layer 4 cells. Postsynaptic target of both terminals are mostly spine heads. We investigated whether VGLUT1 or VGLUT2-positive terminals make synaptic contacts on the DI spines. We found the DI spine heads in 10% of postsynaptic structures innervated by VGLUT2 terminals, but no DI spines postsynaptic to VGLUT1 asymmetrical contacts.

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P2-046 Benzodiazepines facilitate spontaneous GABA release at mouse hippocampal CA1 pyramidal neurons

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In order to explore how benzodiazepines (BZs) can modulate the GABAergic synaptic transmission of hippocampal CA1 region, we investigated the presence of BZ receptors on GABAergic nerve terminals and the functional consequences of their activation. We used mechanically dissociated mouse hippocampal CA1 pyramidal neurons with adherent nerve terminals and conventional whole-cell patch recording of inhibitory postsynaptic current (IPSC). Activation of presynaptic BZ receptors by diazepam and zolpidem facilitated spontaneous GABA release by activating high-threshold Ca^{2+} channels. Bumetanide, a potent blocker of Na-K-Cl cotransporter, diminished the diazepam- and zolpidem-induced facilitatory action of GABA release. Our results indicate that the Na-K-Cl cotransporter accumulates Cl^- inside the nerve terminals so that potentiation of presynaptic $GABA_A$ autoreceptor by BZ receptor agonists causes presynaptic depolarization.

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P2-047 Calcium imaging of pre- and postsynaptic neurons reveals cellular basis for directional tuning properties in cricket cercal sensory system

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Using calcium imaging techniques, we examined activity patterns of ensembles of sensory afferents in response to air-current stimuli from a variety of directions. Different directional stimuli induced different spatial patterns of Ca^{2+} elevation within the terminal abdominal ganglion. We simultaneously recorded the changes in cytosolic Ca^{2+} concentration and membrane potential in the postsynaptic interneuron, and measured directional response characteristics in the local dendritic activity and the overall voltage responses. Based on these results, we proposed that spatial position of the dendritic arbors of the postsynaptic interneuron within ensemble activity pattern maps of presynaptic terminals could determine different directional tuning properties of the interneurons.

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P2-048 Morphometrical analysis of synaptic boutons in the rat hippocampal CA3.

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The hippocampal pyramidal neurons integrate many inputs to process memory signals. We analyzed the laminar distribution of 3,353 synaptic boutons in field CA3 of the rat hippocampus using a large montage electron micrograph (15 X 1,000-mm). Of these boutons, 86.3% contained spherical vesicles (S-boutons), 12% contained flat vesicles (F-boutons), and 1.7% were mossy terminals. In this study, we estimate the intensity of excitatory or inhibitory input to the pyramidal neurons from the sum of the cross-sectional areas of boutons in each stratum. We also report (1) an intimate relationship between synapse distribution and the dendritic structure of pyramidal neurons, (2) the distribution of different types of boutons containing vesicles of various size, and (3) two different plausible foci of inhibition (the upper half of the st. Mol and in the area around the st. Pyr), where F-boutons were distributed densely.

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P2-049 β-Neurexin suppressed VAMP2-EGFP accumulation in axon terminals of zebrafish olfactory sensory neurons

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β-Neurexins are neuronal cell adhesion molecules that are thought to be involved in synapse formation through the interaction with neuroligins. We examined the effect of wild-type and mutant zebrafish β-neurexins (zβ-neurexins) on the axon terminal maturation of zebrafish olfactory sensory neurons visualized with VAMP2-EGFP accumulation. The expression of wild-type zβ-neurexins and mutant zβ-neurexins lacking the cytoplasmic tail suppressed the accumulation of VAMP2-EGFP signals in the axon terminals during synapse formation. On the other hand, mutant zβ-neurexin lacking the extracellular LNS domain exerted little effect on the VAMP2-EGFP accumulation. These results suggested that zβ-neurexin suppressed the VAMP2-EGFP accumulation in the axon terminal of zebrafish olfactory sensory neuron through their extracellular LNS domain *in vivo*.

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P2-050 Involvement of PKC and p38 MAPK in constitutive trafficking of AMPA receptor in cerebellar purkinje cell.

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AMPA type glutamate receptor (AMPA-R) trafficking is one of underlying bases of synaptic plasticity in cerebellar Purkinje cell, however, detailed characteristics of this trafficking has not yet been revealed. We examined whether reduction mechanisms for AMPA-R activity through constitutive endocytosis, isolated by Tetanus toxin (TeTX), share common features with those for long-term depression in parallel fiber (PF) synapse in Purkinje cell. Chelerythrine (10 μM), a protein kinase C (PKC) inhibitor, and SB203580 (1 μM), a p38 MAPK inhibitor, suppressed TeTX-induced reduction in PF-EPSC amplitude. These results suggest that background activity of PKC and p38 MAPK are required for constitutive trafficking of AMPA-R in PF synapse on Purkinje cell.

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P2-051 Characteristics of the Ca^{2+}-dependent binding between myosin-V and syntaxin-1A

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Myosin-V is a processive motor conveying the vesicles and the organelles along F-actin. We found that myosin-V is bound to syntaxin-1A, a membrane protein involved in exocytosis, in a Ca^{2+}-dependent manner. We already estimated the syntaxin-binding site as the neck domain of myosin-V. Here, we obtained another piece of evidence for this confirmation using atomic force microscopy (AFM). We also confirmed that the syntaxin-binding did not change the sliding velocity of myosin-V along F-actin. We made the antibody against the neck domain of myosin-V. Microinjection of this antibody into chromaffin induced the inhibition of catecholamine release using the amperometric assay. Our results suggest that this binding contributes to regulation of exocytosis by the vesicle tethering.

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P2-052 Recurrent excitatory connections between corticostriatal pyramidal cells

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The slow oscillatory depolarization is generated in the frontal cortex and conveyed to the basal ganglia. In the frontal cortex multiple types of pyramidal cells project to the striatum. It remains to be investigated how the excitatory interaction among pyramidal cells induces the slow depolarization and how pyramidal cell subtypes are involved in its transmission to the striatum. We investigated the synaptic property and connection selectivity of one type of corticostriatal pyramidal cells projecting to the bilateral striatum, but not to the brainstem [crossed corticostriatal (CCS) cells], identified by fluorescent retrograde tracers. The CCS cells innervated basal dendrites of other CCS cells with multiple contact sites. The amplitude of unitary excitatory currents depended on the contact site number, and the frequency characteristics of transmission showed paired pulse depression. The same type of corticostriatal cells is excites by the basal dendrite innervation in the frontal cortex.

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P2-053 Thalamic synchronizer: revealed by direct measurement of divergent synaptic connections

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In the brain, a single presynaptic neuron frequently makes divergent contacts with multiple postsynaptic neurons. Such divergent connections are fundamental elements for the network circuitry, but there are few techniques to study divergent connections. Here we designed a new protocol to demonstrate the divergence by combining two techniques: dual patch-clamp recording and "minimal stimulation" of a single presynaptic axon. We applied the protocol to thalamic relay cells in the ventrobasal thalamus (VB cells). We found that inhibitory inputs from nucleus reticularis (nRT cells) had divergent connections on multiple, but only adjacent VB cells. In contrast, excitatory inputs from ascending afferents had no divergent connections on VB cells. The multiple VB cells regulated by an nRT cell exhibited highly synchronous rhythmic activities. Thus, our new protocol provides a precise synaptic arrangement governing synchronous activities among a group of thalamic relay cells.

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P2-054 Cellular mechanisms for the modulation by endomorphins of nociceptive transmission in the rat spinal dorsal horn

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Endomorphin-1 and -2 (EMs) are endogenous ligands for the μ-opioid receptors. We examined the effect of EMs on nociceptive transmission in adult rat substantia gelatinosa neurons with the patch-clamp technique. EMs dose-dependently induced at -70 mV an outward current, the amplitude of which was reduced by a μ-opioid receptor antagonist, CTAP. EMs also decreased the frequency of spontaneous glutamatergic excitatory postsynaptic currents (EPSCs) without changing the amplitude. Like EPSCs, spontaneous glycinergic and GABAergic inhibitory postsynaptic currents (IPSCs) were attenuated in frequency by EMs without any alteration in the amplitude, and the amplitude of electrically-evoked IPSCs was also reduced. We conclude that EMs hyperpolarize membranes and presynaptically depress excitatory and inhibitory transmission by activating the μ-opioid receptors, all of which may contribute to the modulation by EMs of nociceptive transmission.

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P2-055 Network mechanism of GABA-dependent post-tetanic depolarization and seizure-like afterdischarge in the rat hippocampus

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A strong synaptic stimulation induces post-tetanic depolarization (PTD) and subsequent seizure-like afterdischarge (AD) in mature hippocampal slices. We showed that excitatory GABAergic transmission is involved in both PTD and AD, and that interneurons in the strata oriens and pyramidale (s.o./s.p.) may participate in the AD generation. Here we identified interneuron subtypes participating in the PTD generation, and compared with those in the AD generation. The firing rates during PTD were much higher in the interneuron groups in the s.o./s.p., including basket cells, than those in the other layers and pyramidal cells. Depolarizing responses to local GABA application were more prominent in the pyramidal cells compared with the s.o./s.p. interneurons. GAD-staining revealed that minimum number of interneurons to generate PTD and AD was less than 100. Thus, the s.o./s.p. interneurons may play a key role in the generation of both PTD and AD.

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P2-056 Neuropsin/KLK8-induced spine dynamics in hippocampal neurons

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The structures of dendritic protrusions such as spines and filopodia, key determinants of neuronal function, are regulated by the signaling molecules and the extracellural molecules. Changes in the protrusion shape have been documented during development and after stimuli known to elicit long-term potentiation (LTP). More recently, it has been evidenced that serine proteases are involved in activity-dependent synaptic plasticity. Here, we report dynamics of dendritic protrusions by Neuropsin/KLK8, neuronal plasticity related serine protease. We have investigated whether spine motility is regulated by neuropsin and is involved in the degradation of the extracellular matrix. To address the link between neuropsin and spine motillty, we have used the cultured hippocampal neurons. We found that the additional of neuropsin in cultured hippocampal neurons display more spine stabilization than control experiments.

P2-057 Cell type specific measurement of exocytosis by conditionally targeted expression of synapto-pHluorin

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The Cre/loxP recombination system allows the generation of cell-specific somatic mutations in mice. We created transgenic mouse in which the gene of synaptopHluorin (SpH), a GFP-based sensor for exocytosis, is loxed. The SpH fluorescence was found to be restricted to axons and presynaptic terminals of specific type of neurons expressing Cre recombinase under a site-specific promotor. TheSpH fluorescence was transiently increased by neuronal activation which evokes presynaptic exocytosis.

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P2-058 Excitatory and inhibitory innervation on to Purkinje cell soma in calcium channel α1A subunit deficient mice

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In the developing cerebellum, climbing fibers (CFs) first innervate the soma of Purkinje cells (PCs) and then translocate to proximal dendrites. Simultaneous with the translocation, parallel fibers (PFs) and basket cell (BC) axons innervate distal dendrites or somata, respectively. Recently, we have disclosed that calcium channel α1A subunit, which triggers Ca2+ influx upon CF activation, plays an essential role in mono-innervation by CFs and suppression of PF synaptogenesis at proximal dendrites. In the present study, we examined developmental switching of somatic innervation from CFs to BC axons in α1A-deficient PCs. Somatic innervation by CFs persisted in α1A-deficient PCs, whereas that by BC axons was normally established. Thus, α1A appears important to eliminate somatic CF synapses, but neutral to inhibitory synaptogenesis per se.

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P2-059 Distinct regulation of kainate receptors in the corticothalamic and primary sensory (leminiscal) synapses in the ventrobasal thalamus

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We studied properties of EPSCs evoked in relay neurons of the mouse thalamic ventrobasal nucleus (VB) by stimulation of corticothalamic and leminiscal fibers; major excitatory inputs to VB. Corticothalamic EPSCs showed slow decay and paired-pulse facilitation, whereas leminiscal EPSCs showed all-or-none responses, fast decay and paired-pulse depression. The corticothalamic EPSCs, but not leminiscal EPSCs, exhibited the kainate-receptor mediated currents. Furthermore, the activation of presynaptic kainate receptors facilitated and depressed the transmission of corticothalamic synapse at 50 nM and 200 nM of kainate, respectively. Taken together, the corticothalamic synapse, distinct from the leminiscal synapse, is strongly regulated by the activation of presynaptic and postsynaptic kainate receptors, which may be involved in the gain control in the processing of somatosensory information via corticothalamic feedback.

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P2-060 Molecular characterization of VAMP-2, 3 and 4-containing intracellular vesicles

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VAMPs are family proteins involving intracellular vesicular transport. At least seven isoforms are expressed in mammalian cells, however, their respective roles in neurons are not yet clarified. In order to assess their functional roles, we isolated VAMP-2, -3 and -4-containing vesicles from rat brain by using antibody-conjugated magnetic beads. Immunoblotting and liquid chromatographic-tandem mass spectrometry (LC-MS/MS) analysis revealed that VAMP-2 vesicles contained vesicular glutamate transporter, a characteristic protein of excitatory synaptic vesicles. On the other hands, VAMP-3 and VAMP-4 vesicles contained postsynaptic glutamate receptor, GluR2. Interestingly, VAMP-4 vesicles contain several proteins of translational machinery. These results suggest that each VAMP is associated with distinct cellular functions in neurons.

P2-061 SAD-1 is Associated with Synaptic Vesicles and the Active Zone and Implicated in Ca^{2+}-dependent Exocytosis

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A serine/threonine kinase SAD-1 in C. elegans has been shown to regulate synaptic development. We report here the isolation and characterization of mammalian orthologues of SAD-1, tentatively named SAD-1A and SAD-1B. Subcellular distribution analysis of SAD-1A in rat brain indicates that SAD-1A is concentrated in both the synaptic vesicle and postsynaptic density fractions. Immunofluorescence and immunoelectron microscopy has further revealed that SAD-1A protein is abundantly detected in nerve terminals where it is associated with synaptic vesicles and the active zone. Overexpression of SAD-1A results in a reduction in Ca^{2+}-dependent exocytosis from PC12 cells. A point mutation at the ATP-binding site in the kinase domain abolishes the inhibitory effect by SAD-1A. These results indicate that SAD-1 is associated with synaptic vesicles and the active zone and implicated in Ca^{2+}-dependent exocytosis.

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P2-062 Tumor suppressor protein APC affects synaptic signal transduction via clustering of PSD-95

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The adenomatous polyposis coli (APC) tumor suppressor gene is linked to the development of familial adenomatous polyposis and sporadic colorectal tumors. Though APC is highly expressed in the central nervous system (CNS) and directly binds to PSD-95 through the extreme C terminus of APC, the significance of the interaction between APC and PSD-95 in CNS remains unknown. In this study, we investigated whether APC associates neural signal transduction via clustering of PSD-95 in neurons. We performed the calcium imaging in cultured hippocampal neurons. Bath application of 1 μM AMPA increased $[Ca^{2+}]_i$ in control cells. However, this response was inhibited in the dominant negative cells, which were overexpressed a dominant negative molecule (APC-C72) that could disrupt the interaction between APC and PSD-95. This result suggests that APC may affect AMPA signal transduction via clustering of PSD-95.

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P2-063 Postsynaptic AMPA receptor activity maintains presynaptic function in the adult cerebellum

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Maintenance of synaptic strength is important for reliable information transfer and storage. However, its mechanisms are largely unknown. In the present study, we addressed this issue by using climbing fiber (CF)-Purkinje cell (PC) synapse in adult mouse cerebellum. Chronic inhibition of cerebellar neuronal activity by tetrodotoxin as well as postsynaptic AMPA receptors by NBQX caused significant weakening of the strength in CF synapse with reduced glutamate concentration transient at synaptic cleft and decreased frequency of quantal excitatory postsynaptic current. Morphological examination demonstrates diminished CF innervation of PC dendrites after the NBQX-treatment. These results indicate that postsynaptic neuronal activity mediated by AMPA receptors maintains presynaptic function and innervation in the adult cerebellum.

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P2-064 Characterization of the RING-H2 type ubiquitin ligase, hVPS18, in the rat cortical neurons

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The mammalian homologues of Class C *vps*, *vacuolar protein sorting*, are highly expressed in neurons, although their physiological roles still remains unclear. Here we show that the RING-H2 domain of human VPS18 (hVPS18) has a ubiquitin ligase (E3) activity. Using the yeast two-hybrid screening, we identify serum-inducible kinase (SNK), predominantly expressed in the cortical neurons, as a candidate substrate of hVPS18. hVPS18 binds and ubiquitylates SNK *in vivo* and *in vitro*. By using a lentivirus vector which mediates the stable expression of short hairpin RNA (shRNA), we show that the functional involvement of hVPS18 and SNK in the maturation of the synapse in primary cortical neurons. Our data indicate that hVPS18 may play an important role in the degradation of SNK through its ubiquitin ligase activity.

P2-065 Presynaptic inhibition of monosynaptic IPSCs mediated by 5-hydroxytryptamine 1B receptors in rat dorsolateral septal nucleus

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Effects of serotonin (5-HT) on inhibitory synaptic transmission in the rat dorsolateral septal nucleus (DLSN) were examined by intracellular and voltage-clamp recording methods. Bath application of 5-HT (1-30 μM) depressed the monosynaptic fast IPSC in a dose-dependent fashion. The 5-HT-induced depression of the fast IPSC was attenuated by SB 216641, a selective 5-HT_{1B} receptors antagonist and was mimicked by CP93129, a selective 5-HT_{1B} receptor agonist. 5-HT did not change the exogenous GABA-induced current. 5-HT and CP93129 depressed the frequency of mIPSPs without changing their amplitudes. The 5-HT-induced depression of the fast IPSC was blocked by 4-AP (0.5mM) but not by Ba^{2+} (1 mM). These results suggest that activation of presynaptic 5-HT_{1B} receptors depresses the evoked release of GABA via the activation of a potassium conductance in the rat DLSN.

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P2-066 Functional analysis of the *C. elegans* Sec1/Munc-18 family gene *Ce-vps45* involved in membrane traffic

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The docking/fusion of transport vesicles mediated by the SNARE is thought to be regulated by Sec1/Munc-18 (SM) proteins, but their precise roles are still unclear. There are 6 members of SM proteins on the *C. elegans* genome, however, no functional studies have yet been performed. In this work, we focused on the *Ce-vps45* whose orthologous protein in yeast plays an important role in vacuolar protein sorting. The putative null mutation, *tm246*, showed a maternal-effect ts larval lethal phenotype. The ts lethal phenotype of *tm246* was rescued when *Ce-vps45* cDNA was speficically expressed in the intestine. This result suggests that *Ce-vps45* gene products may function in membrane trafficking from Golgi's apparatus to lysosome in mainly intestinal cells where lysosome is highly developed. The mutant *tm246* also shows abnormal endocytic uptake of proteins. We infer that the CE-VPS45 protein is necessary for both biosynthetic and endocytic membrane trafficking pathways.

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P2-067 Tonotopic gradients of membrane and synaptic properties for neurons of the chicken nucleus magnocellularis

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Nucleus magnocellularis (NM) is a division of the avian cochlear nucleus that extracts the timing of auditory signals. We compared the membrane excitability and synaptic transmission along the tonotopic axis of NM. Neurons expressed a Kv1.1 potassium channel mRNA and protein predominantly in the high characteristic-frequency (CF) region. Neurons also showed tonotopic gradients in resting potential, spike threshold, and membrane time constant. Large EPSCs (~10 nA) were generated in an all-or-none manner in the high CF neurons when stimulus intensity of auditory nerve fibers (ANFs) was increased, while small EPSCs (~1 nA) were built up and summation of some synaptic inputs was needed to fire in the low CF neurons. ANF formed enfolding end-bulbs of Held around the cell body in the high and the middle CF region but not in low CF region. Such specializations may be suitable for extracting and preserving the timing information of auditory signals over a wide range of acoustic frequencies.

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P2-068 Expression of precerebellin family in the developing mouse brain and subcellular localization of precerebellin-3 in the adult cerebellum

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Cerebellin was identified as a cerebellum-rich neuropeptide consisting of 16 amino acids. There are 3 members of the precerebellin (Cbln) family, of which Cbln-1 and -3 are transcribed in cerebellar granule cells abundantly or selectively, respectively. Here we examined gene expression of the Cbln family in postnatal mouse brains and protein localization in the adult cerebellum. By *in situ* hybridization, Cbln-1 mRNA was detected highly in the cerebellum, thalamus, olfactory bulb from birth to the adult stage, whereas Cbln-3 mRNA was selective to the cerebellar granular layer from postnatal day 7 on. Using specific antibody, Cbln-3 was localized to the soma, dendrite, axon and terminal of adult granule cells. The granular layer-specific or -abundant expression, early onset during cerebellar development, and wide subcellular distribution suggest that the Cbln family may play a role in differentiation and synapse formation of granule cells.

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P2-069 The Active Zone Protein CAST is a Component of the Conventional and Ribbon Synapses in Mouse Retina

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CAST is a novel cytomatrix at the active zone (CAZ)-associated protein, comprising of two family members CAST1 and CAST2. CASTs form a large molecular complex with other CAZ proteins including RIM1, Munc13-1, Bassoon, and Piccolo in conventional synapses of rat brain. Here, we have investigated the distribution of CASTs in mouse retina. Immunofluorescence analysis reveals that CAST1 and CAST2 show punctate signals in the outer and the inner plexiform layers of the retina, which well colocalize with those of Bassoon and RIM1. Moreover, immunoelectron microscopy reveals that both the proteins , like Bassoon and RIM1, localizes at the synaptic ribbons and localize at the vicinity of presynaptic plasma membrane of conventional synapses. Taken all together, these results indicate that CASTs are components at the presynaptic apparatus of mouse retina and may play an organizational role there by forming the large molecular complex with the other CAZ proteins.

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P2-070 Midazolam increases Miniature IPSCs frequency in layer V but not in layer II neocortical pyramidal neurons via α7 nicotinic ACh receptor

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Midazolam (MDZ) is a benzodiazepine analogue and affects neural excitability in the central nervous system, especially neocortical neurons. Previously, we have reported MDZ increased miniature GABAergic inhibitory postsynaptic currents (mIPSCs) frequency in layer V pyramidal neurons of the somatosensory cortex. The MDZ-induced incremental effect of mIPSCs frequency was blocked by α7 nicotinic ACh receptor (nAChR) blocker methyllycaconitine, but not by α4β2 nAChR blocker dihydro-β-erythtroidine. We also examined MDZ effects on mIPSCs frequency in layer II pyramidal neurons using whole-cell patch-clamp technique in 2-3 week-old rat brain slices. Exposures to 1μM MDZ decreased inter-event interval of mIPSCs in layer V but not in layer II.

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P2-071 ErbB Neuregulin Receptors are Localized to Lipid Rafts

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Neuregulin (NRG), which is concerned with development and repair of nervous system regulates the activity of the ion channels and the neurotransmitter receptors in the cerebellar granule cells. NRG receptor is ErbB, a kind of subfamily of receptor-type tyrosine kinase. It has been discovered that the existence of microdomains (rafts) composed of a special lipid structure plays an important role in the formation of a place for transmitting functional signals. We therefore separated and refined raft fractions, and examined lipid and protein compositions under the condition of low and high KCl concentrations (i.e. variations in the degree of depolarization in membrane potential) in granule cell cultures. In high KCl treatment, the lipid composition shifted to one of typical phospholipids that were observed in raft structure. The composition of synapse control-related proteins such as ErbB in the rafts changed depending on the degree of membrane depolarization.

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P2-072 Effects of dopamine on the inhibitory synaptic transmission in neurons of the rat dorsolateral septal nucleus (DLSN)

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We examined the role of dopamine (DA) on the inhibitory postsynaptic potentials (IPSPs) in neurons of the rat dorsolateral septal nucleus (DLSN) by intracellular recording methods. DA (1-100μM) was administrated by the bath-application. In the presence of bicuculline (15μM), DA (30μM) reversibly inhibited the slow IPSP in all the cells tested (58±3%;n=10). To identify of receptor subtypes involved in the reduction of the amplitude of slow IPSP by DA, we used several dopaminergic agonist. SKF38393 (50μM), a D1 type receptor agonist, slightly inhibited the amplitude of slow IPSP in the DLSN (21±4%; n=11), while PD168077 (30μM), a D4 receptor agonist, remarkably inhibited of the amplitude of slow IPSP in the DLSN (57±2%; n=6). PD168077 (30μM) also depressed the fast IPSP. These results suggest that D4 receptor mainly mediates the action of DA on the inhibitory synaptic transmission in the DLSN.

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P2-073 Inhibition of the dynamic movement of synaphin/complexin by a casein kinase II inhibitor

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Recent studies have indicated that synaptic SNAREs (syntaxin1, SNAP-25 and VAMP/synaptobrevin) are the minimal machinery for the synaptic vesicle fusion with the presynaptic membrane. Cytosolic proteins including NSF, SNAPs, nsec1/Munc18-1 and synaphin/complexin regulate the fusion. We have been using the neuromuscular junction of *Drosophila* larvae to examine the localization of SNAREs and other proteins involved in neurotransmitter release. We previously reported that among these proteins, only synaphin/complexin exhibits a dynamic movement on high K^+-treatment. In this study, we investigated the effects of the specific casein kinase II inhibitor 5,6-dichlorobenzimidazole riboside (DRB) on the localization of these proteins. DRB abolished the high K^+-induced movement of synaphin/complexin. The localization of other proteins examined did not change by DRB treatment.

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P2-074 Properties of inter-areal circuits between primary auditory cortex and the surrounding areas in rat cerebral slices

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Adjacent areas in sensory cortices are connected with inter-areal circuits in gray matter, and such pathways may have important roles in sensory information processing. We investigated the connections between primary auditory cortex (area 41) and the surrounding areas in rat slices. Neural activities, elicited by repetitive stimulation, were visualized using flavoprotein autofluorescence imaging. The activities propagating in a short range (~0.5 mm) from area 41 into the secondary auditory cortices (area 20 and area 36) were more prominent than those in the opposite direction. This asymmetry was also observed in the activities propagating in a long range (~1.5 mm) between area 20 and the middle part of area 41. The propagation of activities was not affected by cutting white matter between the areas. Now we are investigating the possibility these inter-areal circuits might be modified by sensory deprivation in the slices obtained from deafened rats.

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P2-075 Optical and chemical analyses of synaptic transmission in neonatal rat marginal zone (MZ)

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MZ plays important roles in construction of cerebral cortex. Details of synaptic transmission in MZ are yet to be elucidated. Tangential slices were made from MZ of P0-3 rats. Spread of excitation evoked by single electrical stimulation was recorded by optical imaging using voltage-sensitive dye, JPW1114. Spread of excitation in MZ was not affected by glutamate receptor blockers, whereas largely inhibited by $GABA_A$ receptor antagonists, and glycine receptor antagonist. Furthermore, Na^+, K^+-$2Cl^-$ cotransporter inhibitor, bumetanide, reduced it. Microdyalysis revealed taurine was released in MZ by electrical stimulation. The results suggest GABA and taurine facilitate excitatory transmission provided high $[Cl^-]_i$ due to Cl^- uptake.

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P2-076 Regulation of excitatory synaptic transmission by a neuronal ubiquitin ligase

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Both abnormality of synaptic transmission and dysfunction of the ubiquitin-proteasome system have been linked to neurological diseases. Therefore, it is important to identify the ubiquitin regulatory enzymes that reside at synapses, and to determine how such enzymes regulate synaptic function. Here, we identify a neuron-specific ubiquitin ligase (E3) that selectively reduces excitatory synaptic transmission. GFP-tagged E3 was overexpressed in hippocampal neurons and mEPSCs obtained using whole-cell recordings. Overexpression of E3 decreased both the amplitude and the frequency of mEPSCs. However, expression of E3 had no effect on mIPSCs. Moreover, neurons expressing E3 exhibit shrunken dendritic spines and have fewer synapses. We propose that the neuronal ubiquitin ligase regulates synaptic transmission and may promote synapse elimination.

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P2-077 Difference in dynamical property to paired-pulse facilitation of two LTP-inducible hippocampal-prefrontal cortex pathways of rats

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Influence of stimulus intensity and interpulse interval (IPI) were examined on paired-pulse facilitation (PPF) and relationship between PPF and LTP in the posterior dorsal and the ventral hippocampal-prefrontal cortex pathways (pdHP- and vHP-PFC). In the pdHP-PFC, stronger stimuli (0.24-0.32 mA) inhibited the second-pulse-evoked response at 25 ms-IPI but facilitated it at 50~100 ms-IPIs in comparison with the respective initial response, whereas the weaker stimuli did not follow this pattern. After LTP induction, the PPF ratio decreased at longer IPIs and stronger stimuli, but increased at 25 ms-IPI and weaker stimuli. In the vHP-PFC, the PPF ratio tended to be high at 25 ms-IPI and changed little after LTP. These indicate that the stimulus intensity characterizes the property of IPI-controlling PPF in two pathways.

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P2-078 Experience-dependent pruning of dendritic spines in mouse visual cortex by tissue-type plasminogen activator

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Ocular dominance (OD) plasticity is reversibly impaired in tissue-type plasminogen activator (tPA) knockout mice (KO). To determine whether tPA couples physiological perturbation of sensory input to synaptic rearrangement, we examined morphological refinement of layer II/III pyramidal neurons in the binocular zone. Total number of dendritic protrusions were counted from randomly labeled apical dendrites. In both tPA KO and wild type (WT) mice, dendritic protrusions continued to increase in density after eye opening. In contrast, dendritic plasticity was high only in WT mice during the critical period. The number of protrusions decreased after 4-days of monocular deprivation (STMD) with a similar time course to the physiological changes of OD. Interestingly, the pruning of protrusions by STMD was restored only nearest the cell body in tPA KO mice by exogenous tPA injections, which also rescued functional OD plasticity. Dendritic plasticity by tPA may underlie the appropriate refinement of local circuits in visual cortex.

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P2-079 Activity-inducible protein Homer1a promotes redistribution of postsynaptic protein Homer1c in cultured rat hippocampal neurons

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In cultured rat hippocampal neurons, over-expression of Homer1a, an inducible protein upregulated by seizure or long-term potentiation, caused a reduction of punctuate distribution of a postsynaptic protein Homer1c in dendrites, without affecting the protein amount. Clusters of F-actin were also reduced, suggesting that disassembling Homer1c cluster by Homer1a is associated with rearrangement of actin-cytoskeletons. Induction of endogenous Homer1a expression by BDNF or a proteasome inhibitor also led to dispersion of Homer1c puncta. This process was found to be reversible, where the punctuated distribution of Homer1c recovered in several hours after stimulation. These results indicate that the accumulation of Homer1a, either exogenously expressed or endogenously induced, causes redistribution and dispersion of postsynaptic clusters of Homer1c and F-actin, suggesting important roles of Homer1a in synaptic remodeling.

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P2-080 Long-term potentiation in retinotectal synapses in the optic tectum of rainbow trout

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We investigated long-term potentiation (LTP) in the retinotectal synapses in the optic tectum of rainbow trout. Field-excitatory postsynaptic potential (fEPSP) in the synapses was potentiated by repetitive stimuli to the retinotectal afferents. The application of APV during the induction stimulation had no effect on the induction or maintenance of LTP, but the application right after the induction suppressed the LTP. The maintenance of LTP was not prevented by APV at 30 min after the induction. When the test stimulation was absent for 30 min following to the induction, the fEPSP was gradually potentiated after resumption of test stimulation. The magnitude of potentiated responses is the same as that in the case of continuous application of test stimuli. These results indicate that the induction of LTP needs activation of NMDA receptors after the induction.

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P2-081 Interaction between Arc and CaMKII monitored by fluorescence resonance energy transfer

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Activity-regulated synaptic protein Arc accumulates in the postsynaptic density. We previously reported that Arc binds to several synaptic proteins including CaMKII. To further investigate Arc-CaMKII interaction, we measured fluorescence resonance energy transfer (FRET) between CFP-tagged Arc and YFP-tagged CaMKII pairs. A screening system that combined random insertion and yeast 2-hybrid (Y2H) assay was developed; GFP variants were introduced into Arc and CaMKII using transposon-based gene transfer, and then, clones that maintained the integrity of Arc-CaMKII interaction were screened using Y2H. Optimal pairs were selected based on FRET efficiency in COS cells. We successfully isolated several Arc-CaMKII pairs that gave strong FRET signals. The results indicate that the Y2H approach may be widely useful to generate optimized pairs of tagged molecules for FRET studies in intact cells.

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P2-082 Extracellular stimuli regulate polyadenylation of *vesl-1* mRNAs that encode synaptic scaffold proteins

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The *vesl-1* (*homer-1*) gene encodes a scaffold protein, Vesl-1L, that interacts with mGluR1/5 and RyRs to modulate synaptic functions. The gene also encodes two shorter forms, Vesl-1S and Vesl-1M, that counteract the functions of Vesl-1L. Expression of the shorter forms is driven by neural activities that modulate the alternative splicing of *vesl-1*. Each functional poly(A) site forms a different part of the 3'-terminal exon, with promoter-proximal and -distal sites at the end of exons corresponding to the short and long form Vesl proteins, respectively. Analyses of transcripts, by RNase protection assay and Real time PCR, from plasmid in which *vesl-1M*- and *vesl-1L*-poly(A) sites locate in a tandem manner suggest that the function of *vesl-1M*-poly(A) site is directly regulated by extracellular stimuli. These results suggest an importance of polyadenylation event in the neural activity-dependent regulation of *vesl-1* alternative splicing.

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P2-083 Characterization of AMPA receptors that mediate presynaptic inhibition of cerebellar GABAergic transmission

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Repetitive activation of climbing fibers (CFs) inhibits GABA release at cerebellar basket cell (BC)-to-Purkinje cell (PC) inhibitory synapses. We previously reported that this CF-induced heterosynaptic inhibition is mediated by activation of AMPA-type glutamate receptors (AMPARs). However, the properties of AMPARs underlying the presynaptic inhibition of GABAergic transmission are not known. We thus examined receptor mechanisms involved in the AMPAR-mediated inhibition by using patch-clamp recordings in rat cerebellar slices and AMPAR-specific pharmacological tools, such as philanthotoxin-433, bromohomoibotenic acid and γ-D-glutamlyglycine. Results from this pharmacological analysis suggest that CF-induced inhibition of cerebellar GABAergic transmission is mediated by the CF transmitter that spills out of CF-PC excitatory synapses and acts on Ca^{2+}-impermeable AMPARs, possibly GluR2/GluR3 heteromers expressed in BC terminals.

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P2-084 Morphological changes of hippocampal CA1 pyramidal cells caused by 17beta-estradiol

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The density of dendritic spines on hipocampal CA1 pyramidal neurons is dependent upon circulating 17β-estradiol during the 5days estrus cycle in the adult female rat. Recently, we showed that sex hormones (17β-estradiol, testosterone) were synthesized de novo within hippocampal neurons in the adult male rat, and electrophysiological result indicate that 17β-estradiol, which is de novo synthesized within hippocampus may rapidly modulate synaptic plasticity. So, we analyzed dendritic morphological changes by 17β-estradiol. Hippocampal neurons were injected iontophoretically with Lucifer yellow, and high resolution images were collected by confocal laser microscopy. We analyzed spine density and morphology on each cells. The results showed that 17β-estradiol caused the rapid (within 2 hours) morphological changes of dendritic spines in the adult male rat hippocampus.

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P2-085 Activity-dependent synaptogenesis in cultured hippocampal neurons by repetitive PKA activation.

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We previously reported that the repetition of transient activation of PKA with Sp-cAMPS (a membrane-permeable cAMP analog) induced a long-lasting (for 1 week or more) synaptogenesis in dissociated cell cultures prepared from the rat hippocampus as well as in slice cultures. Here we show further details of this long-lasting synaptogenesis, especially in the stimulus conditions and the functionality of the newly formed synapses in the dissociated cell cultures. When a fluorescence dye FM1-43 was applied to the cultures, the numbers of presynaptic sites taking up the dye was larger in the Sp-cAMPS-applied group than in the control group, supporting the functionality of the synapses. When we applied tetrodotoxin to the culture, the increase of synapse number was reversed, suggesting also the increased synapses were functional.

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P2-086 Identification of genes differentially expressed between V1 and area TE of the macaque monkey

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The cerebral cortex of the macaque monkey contains a number of functionally and structurally different visual areas. Little is known, however, about how these areas differ at the molecular level. We applied the method of PCR-based cDNA subtraction and northern blotting to identify genes differentially expressed between two different areas. Here we focused on the primary visual cortex (V1) and area TE of the inferior temporal cortex, the first and final stages in the ventral cortical pathway, which is crucially involved in visual recognition of objects. Although we could not find genes more abundantly expressed in V1, 5 genes were found preferentially expressed in area TE. Interestingly, all the 5 genes encode proteins that are potentially involved in the dynamism of synapses or dendrites. The results provide a new aspect of the molecular characterization of V1 and TE.

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P2-088 Different phosphatases regulate stargazin family in cortical neurons.

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Stargazin (cacng-2) plays important roles in trafficking AMPA receptors to the plasma membrane and targeting them to the synapse. Three isoforms (cacng-3, -4 and -8) as well as stargazin are able to associate interacting with AMPA receptors, however, little is known about the disparities between the isoforms in synaptic functions. We examined the differences of these isoforms using rat cortical neurons and brain homogenate. Cacng-2, -3 and -8 were localized in discrete fractions when they isolated by detergent extraction and separated by sucrose gradient fractionation. As in the case of cacng-2, we found that cacng-3 and -8 are also phosphorylated in cultured cortical neurons and in rat brain homogenate. Cacng8 was predominatly dephosphorylated by occurred by PP1 and or PP2A, whereas cacng-2 and -3 were by PP2B. These results suggested that each stargazin family may be concerned with different phosphatase-depedent synaptic functions.

P2-089 Loss of synapses provoked by repeated inductions of LTD in cultured hippocampal slice

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Activity-dependent changes in synaptic strength are called synaptic plasticity and are assumed as the cellular bases of memory in the mammalian brain. For the study of long-term plasticity in vitro, long-term potentiation (LTP) and long-term depression (LTD) in fresh brain slices are inadequate, since they are maintained only for hours. We previously showed that repeated inductions of LTP in the cultured hippocampal slice led to a persistent synaptic enhancement accompanied by synaptogenesis. Here we show an apparently symmetric phenomenon; repeated inductions of LTD led to a slow and persistent reduction in synaptic strength accompanied by the loss of synapses. Electrical measurement of population EPSP, light-microscopic observation of pre- and postsynaptic structures and electron-microscopic examination of synaptic ultrastructures, all indicated that this phenomenon fills the requirements for the long-term synaptic plasticity.

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P2-090 Activity-dependent maturation of excitatory synaptic connections in solitary neuron cultures of neocortex

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We examined the role of activity in the establishment and maturation of excitatory synapses using solitary neuron cultures of mouse neocortex. With the days in culture, the amplitude of evoked EPSCs increased and short-term depression decreased. These changes were prevented by activity blockade with tetrodotoxin (TTX). The number of synapses estimated by recycled synaptic vesicles with FM4-64 was larger in control than TTX-treated neurons. However, miniature EPSC amplitude remained unchanged during development irrespective of activity. Transmitter release probability, assessed by use-dependent NMDA-EPSC blockade with MK801, was smaller in control than TTX-treated neurons. These results indicate that activity-dependent EPSC enlargement is mainly ascribed to the increase in synapse numbers, while activity-dependent alleviation of short-term depression was mostly ascribed to the decrease in release probability.

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P2-091 Long-lasting synaptogenesis after repetitive exposures to glutamate depends on the induction of LTP

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We previously reported that repetitive exposures to glutamate (100μM, 3min, 3 times in 24h intervals) induced a long-lasting synaptogenesis in rat hippocampal slice cultures. To elucidate whether the synaptogenesis depends on the induction of LTP, we investigated the correlation between the dose of glutamate and the induction of synaptogenesis. Whereas the treatments with 10-30μM glutamate produced a transient enhancement of fEPSP in CA3-CA1 synapses, those with 100-300μM induced LTP lasting for more than 1h. Repetitive exposures (3 times in 24 h intervals) of 100-300μM glutamate, but not 10-30μM, could induce the synaptogenesis, when monitored by electrophysiological and immunohistochemical indices. These results indicate that the repetitive induction of LTP, but not the repetitive excitation (or STP) triggers the cellular processes leading to the formation of new synapses.

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P2-092 Reduction of synapse density after long-term adaptation of horizontal optkinetic response in the mouse flocculus

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Adaptation of horizontal optkinetic response (HOKR) eye movements is a typical form of cerebellar motor learning, which builds up during several days of consecutive daily training. This long-term adaptation involves down regulation of synaptic transmission between parallel fibers (PF) and Purkinje cells (PC) in the middle one third of the flocculus. In the present study, we examined changes in number of PF-PC synapses in the mouse flocculus following adaptation of HOKR. Synapse density in the flocculus after 5-day training of HOKR was significantly lower (by 33%) than that in the control flocculus and that in the paraflocculus of the trained animals. Moreover, the synapse density returned to the control level after a recovery from the HOKR adaptation. These results suggest that reduction of synapses is involved in the mechanism of the long-term adaptation of HOKR.

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P2-093 Chronic exposure of DHEAS primes LTP induction in rat hippocampal CA1

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Acute exposure of memory-enhancing neurosteroids dehydroepiandrosterone sulphate (DHEAS) reversibly facilitates the transmission in CA3-CA1 synapses, but not LTP induction. However, chronic effects of DHEAS on LTP induction remain to be elucidated. We examined the LTP induction at CA3-CA1 synapses in hippocampal slices from DHEAS-treated (20mg/kg, 7days) adult rats using tetanic stimuli of 5-200 pulses at 100Hz. In control rats, only the stimuli over 50 pulses could induce LTP in a pulse-number dependent way. Chronic DHEAS exposure significantly lowered the threshold (20 pulses) for LTP induction. However, this treatment failed to alter the basal synaptic transmission (input-output relationship) and short-term plasticity (paired-pulse facilitation), implying that the facilitated LTP induction by DHEAS may be of postsynaptic origin.

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P2-094 Noradrenergic activation restores ocular dominance plasticity in adult cats irrespective of normal or paradoxical shift

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Brief monocular deprivation induces a shift in ocular dominance (OD) toward the non-deprived eye as long as a sensitive period (OD Plasticity). To verify the noradrenaline (NA) hypothesis in the regulation of OD plasticity, we administered L-thero-DOPS, a synthetic precursor aminoacid for l-NA daily (100 mg, b.i.d., PO) to matured cats (> one year old), concomitantly with monocular lid suture lasting for about 4 weeks. During the monocular deprivation, muscimol (10 mM in an osmotic minipump), a GABA-A receptor agonist, was injected into the left striate cortex. Single unit recordings in the medial bank of striate cortex showed that OD was shifted toward the deprived eye in the region close to the muscimol infusion site, while normal shift in OD was observed in the control hemisphere. This is the first experimental evidence supporting that the central NAergic system controls OD plasticity, irrespective of the direction of OD shift, which may be determined by activity-dependent mechanisms.

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P2-095 Regulation of ERK 1/2 activation by visual input in the developing visual cortex

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It is still not clear why visual cortex expresses ocular dominance plasticity only in the critical period. Recently it was reported that inhibition of extracellular signal-regulated kinase 1/2 (ERK 1/2) disturbs this plasticity. If ERK 1/2 is involved in the mechanism of the plasticity, monocular deprivation (MD) might change the activity of ERK 1/2 only during the critical period. To test this possibility, we examined effects of MD on the phosphorylated ERK 1/2 proteins in the rat visual cortex by immunohistochemistry. Phospho-ERK 1/2 was found mainly in the cytoplasm of layer 2/3 cells. MD significantly decreased the number of immunopositive cells. This effect was observed in both young and adults. On the other hand, MD enhanced nuclear translocation of ERK 1/2 only in young rats. These results suggest that ERK 1/2 activation might be regulated by different mechanism between young and adults.

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P2-096 Effects of a lack of CRE-mediated transcription factor ICER on amygdala kindling

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Kindling is an experimental model for long-lasting neuronal plasticity. Dynamic gene regulation and newly protein synthesis are required for development of kindling. A lot of immediate early genes including ICER, an inducible CRE-mediated transcription factor, are induced transiently in the kindled brain after the electrical stimulation. To understand the functional role of ICER on kindling, we examined kindling development in ICER-knockout mice. While the afterdischarges were evoked normally in the amygdala after the initial stimulation, development of kindling was significantly accelerated in the knockout mice. These results suggest that this transcriptional repressor suppresses an acquisition of irreversible seizure susceptibility through the downregulation of gene expression. Thus, the downstream genes of ICER might have a critical role for regulating long-lasting neuronal plasticity.

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P2-097 Cholinergic regulation of hippocampal synaptic plasticity via M1 muscarinic receptors

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The cholinergic system is involved in higher brain functions, including learning and memory. This cholinergic action has been reported to be mediated by muscarinic acetylcholine receptors (mAChRs). The hippocampus, which is implicated in certain kinds of memory, is innervated by cholinergic fibers originating from the medial septum, and expresses all five mAChR subtypes (M1-M5). In order to reveal the mechanism of cholinergic modulation of hippocampal synaptic plasticity by mAChRs, we performed electrophysiological experiments in the CA1 region of hippocampal slices. A low concentration of carbachol, a nonselective cholinergic agonist, as well as physiologically released endogenous acetylcholine by repetitive stimulation of cholinergic fibres, facilitated long-term potentiation, and this priming effect was absent in M1 knockout mice. These results suggest that the cholinergic activity induced by physiological stimuli can facilitate synaptic plasticity via M1 receptors.

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P2-098 Role of neuropsin in hippocampal long-term potentiation in vivo

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Neuropsin (NP), an extracellular serine protease, was specifically expressed in the hippocampus, being involved in memory formation. We investigated the role of NP for the induction of long-term potentiation (LTP) in the CA1 in vivo. We found that the NP activity was rapidly increased 5 min after theta-burst stimulation (TBS), peaking 6 min, and then sustained at low level for 60 min. Furthermore, to examine the functional role of activated NP, we injected active form of recombinant NP into the hippocampus of adult mice. Application of NP led to the long-lasting synaptic enhancement at Schaffer-collateral pathway. Like LTP, NP-induced potentiation did not affect paired-pulse facilitation. Our results imply that the proteolytic activity of NP augments synaptic efficacy. However, NP-induced potentiation did not occlude the subsequent TBS-induced LTP, suggesting NP-induced potentitation probably occurs by a mechanism independent of conventional LTP.

P2-099 Modulation of excitatory neurotransmission by 17β-estradiol in the hippocampus

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Though wide range of effects on postsynaptic mechanisms have been reported, little information is available on presynaptic modulation by 17β-estradiol(E2). Here we report results indicating that E2 modulates synaptic transmission through presynaptic mechanisms. Population excitatory post-synaptic potentials (EPSPs) were recorded in vitro from subfields CA1 and CA3 of the hippocampus of male rats. At Schaffer collateral-CA1 synapses, application of E2 rapidly potentiated EPSPs. The effect was reversible after the removal of E2, which was in contrast to that observed at mossy fiber-CA3 synapses. E2-induced potentiation was accompanied with a decrease in paired-pulse facilitation. At CA1 synapses, application of E2 in the presence of DPCPX, an adenosine A1 receptor antagonist, induced long-lasting potentiation. The results obtained here are quite similar to the reported effects of forskolin, an adenylyl cyclase activator, indicating that E2 potentiates EPSPs by increasing transmitter release from presynaptic terminals.

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P2-100 Bistable membrane properties as a determinant of heterosynaptic CA3 LTP induction

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Hippocampal CA3 pyramidal cells receive two independent afferents from the entorhinal cortex, i.e., a direct input via the temporoammonic pathway (TA) and an indirect input via the mossy fiber (MF) of a dentate granule cell. Recently, we found that LTP at TA synapses is heterosynaptically induced when MFs alone are activated at a high frequency rate. Here we investigated what mechanisms underlie the interplay between TA and MF synapses. A CA3 pyramidal cell fired repetitive spikes in response to TA activation only after a brief burst stimulus of MFs. This was likely due to a decrease in spike threshold, which was unaffected by CNQX applied to the area of associational/commissural synapses while CNQX prevented heterosynaptic induction of TA LTP. We thus focus on the intrinsic properties of a CA3 pyramid, i.e., bistable membrane potential. Indeed, DOWN states, not UP states, gated the induction of heterosynaptic LTP. The intrinsic properties may determine the throughput of inter-synaptic interactions.

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P2-101 Presynaptic nicotinic acetylcholine receptors regulate transmitter release in adult songbird motor control nucleus

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The zebra finch forebrain nucleus RA is a critical location for integration of sensory and motor signals during song learning. We previously showed independent regulation of postsynaptic NMDA receptors and presynaptic release probability between synapses from HVC and LMAN to RA. We have begun to examine possible mechanisms of presynaptic regulation. In the present study, we tested α7 nicotinic acetylcholine receptors (nAChRs) effects using electrophysiological and morphological methods. We found a high density of punctate staining against α7-nAChRs localized to RA of adults, but not in young birds. In addition, in the presence of the specific α7-nAChRs antagonist methyllycaconitine (MLA), the release probability was significantly decreased only for input from HVC, but not from LMAN. MLA did not affect either input in young birds. These results suggest that presynaptic function at 2 functionally distinct inputs to RA neurons may be selectively regulated in adults.

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P2-102 Identification of a Dendritic Targeting Element in the 3'UTR of Arc mRNAs

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Dendritic sorting of mRNAs and the regulated translation near the synapses have been suggested to play important roles in synaptic plasticity. In the present study, we focused on the dendritic targeting of Arc (Activity-regulated cytoskeleton-associated protein) transcripts. Arc is an immediate early gene that can be induced by neuronal activity. The unique feature of Arc is that the newly synthesized Arc mRNA is rapidly delivered to the activated region within dendrites that have received intense synaptic activation. Therefore, Arc mRNA is expected to harbor the unique dendritic targeting element (DTE) to the activated synapses. To identify a DTE, recombinant mRNAs containing various region of Arc sequences were expressed in primary hippocampal neurons and their subcellular distribution was analyzed by in situ hybridization. We delineated the DTE within 350 nt in the 3'UTR of Arc mRNA.

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P2-103 Involvement of BDNF in the long-lasting synaptogenesis after repetitive activation of PKA

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We previously reported that the repetitive activation of PKA induced a long-lasting synaptogenesis in dissociated cell cultures of the rat hippocampus. In search for the signals in the downstream of PKA, we investigated BDNF (brain-derived neurotrohic factor) as a possible outcome of PKA activation. A transient application of BDNF (100ng/ml, 15min) induced an immediate synaptogenesis within 1hr, as monitored by immunohistochemistry and immunoblotting using antibodies against synaptic proteins (synaptophysin and PSD-95). Repeated applications of BDNF (24 hr intervals) induced not only the immediate synaptogenesis but also a long-lasting one. In addition, the PKA activation with Sp-cAMPS (a membrane-permeable cAMP analog) led to the expression of TrkB (BDNFreceptor), which was enhanced by the repeated Sp-cAMPS treatments. Thus BDNF meets the requirement for the candidate signal molecule.

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P2-104 The protein kinase CK2 is required for the induction of NMDA receptor-dependent long-term potentiation in rat hippocampus

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The protein kinase CK2 (formerly known as casein kinase 2) is abundant in the brain, and its activity increases transiently in the induction of hippocampal long-term potentiation (LTP), which is thought to contribute to learning and memory processes. CK2 is shown to phosphorylate many kinds of proteins associated with synaptic plasticity. But it is not clear whether CK2 is involved in LTP. Here we examined the effect of DRB, a specific inhibitor of CK2, on LTP at Schaffer-CA1 synapses in hippocampal slices. DRB significantly inhibited NMDA receptor-dependent LTP induced by 1 sec stimulation at 100 Hz. In contrast, DRB did not affect NMDA receptor-independent LTP induced by strong stimulation (10 trains of 200-msec stimulation at 200 Hz delivered every 5 sec at strong intensity) in the presence of a NMDA receptor antagonist, AP-5. These results suggest that CK2 participates specifically in a NMDA receptor-dependent process during LTP.

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P2-105 Inhibition of phosphoinositide 3-kinase impairs LTP in the main olfactory bulb

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We previously demonstrated that phosphoinositide 3-kinase (PI-3 kinase) in the main olfactory bulb (MOB) was activated during long-term aversive olfactory learning and the selective PI-3 kinase inhibitor, wortmannin blocked olfactory learning by inhibition of CREB phosphorylation in young rats. In the present studies we examined whether application of wortmannin has effects on LTP in the MOB slices, we found LTP evoked by tetanic stimulation of the lateral olfactory tract was blocked by application of wortmannin during tetanic stimulation, but not at 60 min after LTP induction. These results indicate that PI-3 kinase is essential for induction but not maintenance of LTP in the MOB in young rats.

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P2-106 Noradrenaline enhancement of LTP in the accessory olfactory bulb: a cellular mechanism

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To investigate the cellular mechanisms of alpha2-adrenergic enhancement of LTP, we used whole cell recordings from mitral and granule cells. Noradrenaline (NA) and the alpha2-adrenoceptor agonist clonidine decreased voltage-gated calcium currents in mitral cells, but not in granule cells. Both of drugs reduced the frequencies of sEPSCs in granule cells without affecting their amplitudes. In contrast, clonidine had no effect on mIPSCs in mitral cells. In granule cells, both NA and clonidine decreased plateau depolarization and increased number of action potential firing during theta frequency stimulation. These results suggest that NA inhibits mitral-to-granule cell synaptic transmission via presynaptic alpha2-adrenoceptors, thereby leading to a smaller depolarizing plateau and thus to increased firing probability in granule cells that may contribute to enhancement of LTP induction during theta frequency stimulation.

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P2-107 Mechanism of heterosynaptic LTD in neonatal hippocampus

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Long-term potentiation (LTP) of synaptic transmission is thought to be involved not only in learning but also in modifying neural circuits during development. In neonatal CA1 area of hippocampus, CaMKII-mediated robust LTP is not fully expressed in order to avoid too much excitation in immature neural circuits that have only poor inhibition (Yasuda et al., 2003). Instead of fully enhancing activated synapses by robust LTP, we found that in neonatal CA1 area inactivated synapses were heterosynaptically depressed when modest LTP was induced in tetanically activated synapses and thereby tetanized pathways are highlighted more. And this heterosynaptic long-term depression (heterosynaptic LTD) becomes weak during development. We investigated the mechanism of the induction of heterosynaptic LTD in developing hippocampus. Heterosynaptic LTD was partially blocked by a cannabinoid receptor antagonist and occluded by its agonist.

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P2-108 Long-term analysis of LTP in the mouse hippocampal slice using multielectrode array recording

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Multielectrode array system has provided a powerful means for monitoring neural electrical activity of brain neural circuits non-invasively and simultaneously. In order to study late LTP which is dependent on syntheses of mRNA and proteins, we applied multielectrode array system to mouse hippocampal slices and re-evaluated the effects of a protein synthesis inhibitor, anisomycin, on LTP. LTP in the CA1 region could be monitored for a long-term period (6.5h). In the presence of anisomycin, induction and early LTP in the CA1 region were not affected by anisomycin but the potentiated EPSP returned gradually to the base line.

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P2-109 Re-organization mechanisms of grown-up neuronal circuit in dissociated hippocampal pyramidal neurons

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Neuronal circuit is formed and organized with activity dependent manner during development. But, little is known about the plasticity of grown-up neuronal circuits. We used hippocampal grown-up pyramidal neurons (21 DIV) to investigate the issue. Suppression of neural circuit activity leaded to an increase in neuronal connectivity accompanied with surface delivery of NR2B-subunit containing NMDA receptors. Blockade of NMDAR activation prevented the increase in neuronal connectivity without disturbing the surface-expression of NR2B-NMDARs. These results indicate that suppression of circuit activity induces surface delivery of NR2B-NMDARs and the activation is critical mechanism for circuit re-organization with enhanced neuronal connectivity.

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P2-110 Enhanced mossy fiber plasticity in mice with increased kainate receptor function

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Kainate receptors (KARs) are thought to contribute to short-term facilitation and modulate induction of long-term potentiation (LTP) at hippocampal mossy fiber - CA3 pyramidal cell synapses. We tested this hypothesis by comparing paired-pulse facilitation (PPF), frequency facilitation (FF) and LTP of excitatory postsynaptic currents (EPSCs) in hippocampal slices from wildtype and GluR6ECS mice, in which GluR6-containing kainate receptors have increased conductance and calcium permeability. Mossy fiber EPSCs from GluR6ECS mice had a similar PPF but larger FF compared to wildtype EPSCs. Surprisingly, LTP induced by trains of 100 Hz stimulation was larger in GluR6ECS mice. GluR6ECS mice also demonstrated a lower threshold for induction of LTP using a 25 Hz stimulation paradigm. These data support a critical role for GluR6-containing KARs in modulation of synaptic efficacy, and further suggest that they can affect the magnitude as well as the induction of long-term mossy fiber plasticity.

P2-111 Direct Focal Detection of Axonal Terminal-Branch Variability in *Drosophila* Learning Mutants: The Role of cAMP in Transmission Precision, Output levels, Activity-Dependent Plasticity, and Ca^{2+} Regulation

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Although synaptic transmission of individual axonal branches can be independently regulated, it is not known whether synaptic strength of individual boutons is similar along a branch. Focal recording from *Drosophila* neuromuscular junctions revealed that synaptic strength is comparable along a terminal branch. Interestingly, the branch-output levels were severely depressed in a learning mutant *rutabaga* (Ca^{2+}/Calmodulin-activated adenylyl cyclase), leading to increased variation among branches. In *rutabaga* terminal branches, activity-dependent plasticity of synaptic transmission became unpredictable and stability of transmission was compromised. Altered excitability and Ca^{2+} handling along a terminal branch were detected and attributable to Ca^{2+} channel function. Our results suggest that cAMP-dependent modulation of the whole branch can provide a powerful mechanism for activity-dependent plasticity.

P2-112 Effects of Capsaicin on Neocortical Long-term Depression in Awake and Freely Moving Rat

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In the present study we examined the effect of Capsaicin (Cap)- induced C-fiber depletion on Long-term depression (LTD) induction in the somatosensory cortex. In this work we treated neonatal rats with Cap in the first day of birth. After three months, a bipolar stimulation and a recording electrode were implanted into corpus callosum and somatosensory cortex respectively. Two weeks after the surgery evoked filed potential responses were recorded from alert animal. Then callosal low frequency stimulation was delivered everyday for 12 days. In control animal, LTD appeared after 3 days of stimulation and reached its pick in about 9 days. Cap treatment postponed the development of LTD. This result suggests that the depletion of C-fiber alters the normal function of cortical neurons.

P2-113 Ventral shift of the site of oligodendrocyte origin in the netrin-1 deficient mouse spinal cord

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Oligodendrocyte precursor cells (OPCs) in the netrin-1 deficient spinal cord was examined to study involvement of netrin-1 in the oligodendrocyte development. OPCs were labeled with Olig1, a lineage specific bHLH protein, and with PDGF receptor alpha. OPCs in the spinal cord first appeared in the E12.5. They were localized in the ventral ventricular zone forming bilateral foci. In the netrin-1 deficient mouse, the site of OPC origin in the spinal cord was slightly, but definitely, shifted towards the ventral direction. In accordance with this, ventral part of the dorsoventral (D-V) axis is shifted ventrally. The alteration of D-V axis formation started by E11.5. Lesser cell proliferation or survival was not occurred in the ventral spinal cord of netrin-1 knockout mouse. Although precise signaling cascades were yet unknown, netrin-1 may be directly involved in D-V axis formation in the developing spinal cord.

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P2-114 Large-scale screening of topographic molecules in the embryonic mouse retina using oligo DNA microarrays.

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A fundamental aspect of neural networks in the nervous system is the formation of topographic maps, in which the spatial order of one set of neurons are precisely maintained in their synaptic connections to a second set of neurons. The formation of one such map, defined by the connection of ganglion cells in the retina to their targets in the tectum or superior colliculus, is thought to depend upon interactions between retinal guidance molecules and tectal or collicular guidance molecules.To understanding the molecular mechanisms for the establishment of topographic maps, we performed a large-scale screening of molecules with asymmetrical expression patterns along the DV or NT (AP) axis in the embryonic mouse retina using oligo DNA microarrays. We successfully identified a large number of topographic molecules including many genes previously identified as topographic molecules. On the other hand, several unknown genes with asymmetrical expression patterns were also included.

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P2-115 Neuronal generation and migration in the mouse hippocampal primordium as revealed by in utero electroporation

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The formation of hippocampal layer structure was examined by labeling the ventricular surface cells with EGFP expression vector. The vector was transfected into the spatially and temporally restricted neuroepithelium of the primordium by in utero electroporation, and the morphology of EGFP-labeled migratory neurons and interrelation with the radial glia were observed. Pyramidal cells in hippocampus proper migrated radially along glial processes from the wide area of neuroepithelium between E14-15. By contrast, dentate granule cells originated later than E16 from the restricted neuroepithelium adjacent to the fimbria. Their initial migration was rapid and independent of radial fibers. Subsequent tangential migration in the subpial space and final settlement into the forming dentate gyrus showed close association with radial glia. Above findings indicate the clearly distinct origin and mode of migration between the neurons in hippocampus proper and dentate granule cells.

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P2-116 Selective ablation of radial glia through the expression of suicide gene by in utero electroporation

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The promoter of tenascin-C gene (tn-c) is specifically activated in the radial glia within the neocortical primordium. Taking advantage of this promoter, a method to selectively ablate radial glia by expression of herpes-simplex-virus-thymidine-kinase gene (hsv-tk) was devised. A plasmid harboring tn-c and hsv-tk was transfected into the ventricular surface cells of the mouse embryonic cerebrum by in utero electroporation. Subsequently, ganciclovir was administered and the embryonic neocortex was histologically examined. Most of the nestin-positive radial glia was ablated in the region where the plasmid was transferred. Radial migration of neurons was observed up to the cortical plate, although their numbers were reduced. Thus, radial glia is considered to be involved mainly in the generation of neurons as their precursors, but dispensable for their radial migration.

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P2-117 Birth-date dependent alignment of GABAergic interneurons in the developing mouse visual cortex

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In the developing cerebral cortex, projection neurons and interneurons arise in distinct places and migrate into the pallium in different ways. While the projection neurons arise in the pallial ventricular zone and migrate radially toward the pial surface, most interneurons are known to derive from the ganglionic eminences and migrate tangentially into the pallium in mice. To clarify the mechanisms of the cortical histogenesis, we labeled the interneurons with bromodeoxyuridine at each developmental stage, and analyzed their ultimate locations in the mouse visual cortex. Here, we show that, interneurons as well as projection neurons, are aligned in "inside-out" pattern in the cortical plate, but the relatively early arised interneurons are distributed around two peak locations. Difference between the two peak locations of the early arised interneurons will be discussed.

P2-118 Identification and neural expression of mouse Ccd1 (Coiled-coil-DIX1).

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The Wnt signaling plays an important role in cell growth, differentiation, and polarity formation. Recently we isolated a novel zebrafish gene encoding Ccd1 which has coiled-coil and DIX domains. Ccd1, forming homomeric and heteromeric complexes with Dishevelled (Dvl) and Axin, contributes to the neural patterning through Wnt activation. Here we report the isolation and characterization of mouse *Ccd1*. We found 14 putative isoforms that show different expression patterns in various mouse tissues. In contrast to the zebrafish Ccd1, any isoform of the mouse Ccd1 examined did not activate or only weakly activated TCF-dependent transcription *in vitro*, while it robustly potentiated Dvl-dependent Wnt activation. *Ccd1* mRNA and protein were highly expressed in the embryonic and adult brain. These results indicate that mouse Ccd1 functions as a positive regulator of the Wnt/β-catenin signaling and implicate *Ccd1* in neural development and brain fucntion.

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P2-119 Abnormal Formation of Entorhinohippocampal Projection of Reeler Mutant Mouse

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Reeler mouse is a neurological mutant mouse. We labeled entorhinohippocampal projection by injection of biotin dextran amine (BDA) into the entorhinal cortex to clarify whether the course and terminal endings of this projection of the reeler mouse are normal or not. We found that in the reeler mouse, BDA-labeled, which crossed the hippocampal fissure in the normal mouse as a perforant pathway, turned around the this fissure instead of crossing it. The distribution pattern of terminal endings of this projection of reeler was different from the normal counterparts. Next, we examined the developmental changes of the hippocampal fissure by immunohistochemistry and DiI labeling. At postnatal day 0 (P0), GFAP-immunopositive cells were abundant nearby the hipocampal fissure in both strains. However, in the normal mouse, GFAP-positive cells were decreased during development, whereas in the reeler, GFAP-positive cells were accumulated. Perforant fibers were recognized in reeler mouse at P1 to P3, but those fibers completely disappeared by P4.

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P2-120 The rhythmic bursts of the respiratory activity neurons in medullary half-slice preparations of neonatal rodents

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In the rhythmic brainstem slice preparation, spontaneous respiratory activity of pre-Boetzinger complex (PBC) is generated endogenously and can be recorded as output activity from the hypoglossal XII nerve. We used reduced medullary slice preparations of neonatal rats and/or mice and analyzed changes of spontaneous respiratory activity to elucidate the mechanisms for respiratory rhythm generation. To determine the respiratory bursting frequency generated in the XII nerve, consecutive respiratory cycles were measured and compared before and after mid-sagittal transections. Similar respiratory bursts with slightly decreased frequency were observed after mid-sagittal transections. We also examined the effects of serotonin (5-HT) on the spontaneous respiratory rhythm in half slice preparations. These results demonstrate that PBC neurons in one half slice are capable of generating rhythmic respiratory bursts and 5-HT modulate their rhythm.

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P2-121 Expression analysis of *SulfFP1* mRNA in the adult mouse brain

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Heparan sulfate (HS) plays an important role in neural patterning. Molecular heterogeneity in HS chains specifically regulates the signaling of heparin-binding factors. Recently we isolated a novel sulfatase, SulfFP1, which degrades the 6-O sulfate of intact heparin at neutral pH. SulfFP1 is localized in the Golgi apparatus inside the cell and also secreted into the extracellular space, suggesting that it may affect extracellular signaling by altering the HS microstructure. To gain the insight of the *SulfFP1* functions, we examined the expression of *SulfFP1* in the adult mouse brain. *SulfFP1* is highly expressed in the primary olfactory cortex including the olfactory tubercle, piriform cortex, entorhinal cortex, and olfactory nuclei. It is also expressed in the layer 6 of the cerebral cortex, choroid plexus, and Bargmann glia in the cerebellar cortex. Co-localization of SulfFP1 signal with NeuN and GFAP indicate that *SulfFP1* is expressed in both neuron and glia.

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P2-122 Postnatal development of corticotectal projection of the reeler mouse

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We have examined the cytoarchitecture, myeloarchitecutre, and afferent system of the superior corriculus (SC) in the Reelin-deficient mouse, *reeler*. The laminar structures of the superficial layers of the SC are disorganized and intermingled into a single layer in this mutant. Myelinated fibers are scattered within the SC instead of laminar distribution. Next, we have investigated the postnatal development of corticotectal (CT) projection with DiI and BDA. Although the time schedule of the extension and terminal maturation of CT is normal in the reeler, anterogradely labeled CT fibers take an aberrant course within the SC. reelin mRNA and Reelin protein are both recognized in the superficial layers of the normal SC, but not in the *reeler* counterpart. These results suggest that abnormal positioning of neuronal components in the superficial SC of the *reeler* causes an aberrant course of CT fibers.

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P2-123 Functional analysis of zebrafish *SulfFP* genes during neural development

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Heparan sulfate proteoglycans (HSPG) play a crucial role in mediating important signals such as Wnt, Hedgehog and FGF. Recently we isolated novel sulfatases, SulfFPs, which degrade the 6-O sulfate of intact heparin at neutral pH. SulfFP proteins are localized in the Golgi apparatus inside the cell and also secreted into the extracellular space, suggesting that it may affect morphogenetic signaling by altering the HSPG microstructure. In order to examine the function of *SulfFP* genes, we cloned the zebrafish *SulfFP1*, *SulfFP2*, and sulfatase modifying factor 1 and 2 (*SUMF1* & *SUMF2*), the enzyme that are required for generating active sulfatases. We found that the combinatorial expression of *SulfFP* and *SUMF in vitro* resulted in robust activity of degrading an artificial sulfatase substrate. We thus examined the morphological, biochemical, and physiological changes of zebrafish embryos after inducing the SulfFP activity by injecting *SulfFP* and *SUMF* RNAs.

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P2-124 A role of the zinc finger genes *fez* and *fezl* in the forebrain formation

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fez and *fezl* are the olfactory- and forebrain-specific zinc finger genes that encode transcriptional repressors containing an Engrailed-type repressor motif. We found that *fezl*- and *fez*-deficient mice had defects in the formation of thalamocortical axons and subplate neurons, and the olfactory bulb and sensory neurons, respectively. We generated *fez;fezl* double knockout (dKO) mice. At E15.5, the neocortex was small, and the hippocampal region and the ventral/dorsal thalamus did not form in the dKO embryos. Reduction of the *Gbx2*-expressing dorsal thalamsus and absence of the *Dlx1*-expressing ventral thalamus were detected at E12.5. Reduced expression of *Emx2* and absence of the *shh* expression in zona limitans intrathalmica was observed at E10.5. The data indicate that Fez and Fezl function redundantly in the formation and patterning of telecephalon and diencephalon at the early embryonic stage.

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P2-125 Differential gene expression analysis of anterior and posterior thalamus in rat embryo

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Mammalian cerebral cortex is divided into functionally different regions such as motor, somatosensory, visual, and auditory area. During this regionalization, special sensory axons project to cortical specific areas via anterior thalamus, and nonsensory axons project to frontal lobe including motor cortex via posterior thalamus. While several transcription factors involved in cortical regionalization have been identified, detailed mechanisms are still unknown. This study is focused on the molecules, which are related to the thalamocortical projection during late embryonic stage. To identify differentially expressed molecules, we analyzed subtracted cDNA from the rat anterior and posterior thalamus at embryonic day 18. A number of gene fragments enriched either anterior or posterior thalamus were detected using virtual northern blot analysis. These molecules may be involved in accurate thalamocortical projection and functional regionalization.

P2-126 Proteomic analysis of rat embryonic forebrain

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Mammalian forebrain has crucial roles for cognitive and emotional functions. During the late embryonic stage, forebrain has been differentiated and constructed dynamically. In this stage, many of genetically regulated transcription factors that organize the development of forebrain are identified. However, the downstream proteins expressed by these are still unknown. To identify such downstream proteins, we used proteomic approach. We isolated several fractions from rat cerebral isocortex and thalamus-hypothalamus. These fractions at each embryonic day 16, 18, and 20 were analyzed by two-dimensional electrophoresis. The protein spots were visualized by silver staining. While most of the protein spots were identical among these fractions, several spots were detected differentially. The spots detected differentially are potential candidates that play roles for the development of cerebral isocortex, thalamus, and hypothalamus.

P2-127 Role of bombesin-like peptide receptors on developmental chick brain

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Bombesin-like peptides and their receptors are known to act as a neuromodulator. The role of these receptors, however, on brain development is not elucidated. To examine the effect of overexpression of the receptors on neural development in vivo, we constructed RSV-derived retroviral vectors carrying the mouse bombesin-like peptide receptors and infected to chick embryos at E1.5. Compared to control ones at E20, the cerebellum of embryos expressing GRP-R showed marked dysplasia. In addition to the small size of cerebellum, Purkinje and granule cell layers were disorganized and minimal foliation was observed. The layer of tectum was also misarranged. GFAP-positive astrocytes were increased in number at ventral hyperpallium in the telencephalon. Embryos expressing NMB-R increased in size especially in the telencephalon, tectum and cerebellum. These results suggest that two receptors may be involved differently in early neural development.

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P2-128 A project on superfine digital atlas of the development of the mouse nervous system

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The highly complex adult nervous system is developed from a single egg through multiple cell division, movement and differentiation. Although many molecules expressed at the brain were identified, their functions are still not understood well. It was difficult to map their expression accurately because the high-resolution map was not available. It was difficult to assess abnormal develomment of the knockout mice. To establish the accurate standard, we initiated a project on superfine digital atlas of the development of the mouse nervous system. Mouse embryos at E8.5-E18.5 and postnatal brain at P0-P56 were fixed and embedded in paraffin. The complete sets of serial coronal and sagittal sections at right angles were made and stained with HE. The digital images at a single cell resolution were made and integrated into large images of the whole embryo and brain. The area of interest can be zoomed in and out continuously at any magnification. The 3 D digital images at a single cell resolution will be integrated with the knowledge- based information in the future.

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P2-129 In situ hybridization screen for genes involved in mouse cerebellar compartmentalization

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The mammalian cerebellum is anteroposteriorly and mediolaterally compartmentalized. To elucidate the molecular mechanisms underlying the establishment of functional cerebellar compartment, gene clones from our CDT (Cerebellar Developmental Transcriptome) project were examined for their patterned expression. To search "early onset" genes expressed prenatally and playing a role in the establishment of the mediolateral compartment, the CDT clones were subjected to whole-mount in situ hybridization. Not a few known and unknown genes were found to be expressed in a pattern of parasagittal bands in the mouse embryonic cerebellum. These parasagittally expressed genes were classified in comparison with the expression pattern of En2 and Wnt7b genes, which probably correlate with the birth date of Purkinje cells. Our accumulating data suggest that not only so-called patterning genes, also genes supposed to be related in later events in neural development such as synaptogenesis were expressed as "early onset" genes.

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P2-130 Axonogenesis in the medaka embryonic brain

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The axons were stained immunocytochemically using HNK-1 and acetylated tubulin antibodies. The fiber systems developed correlating to the organization of the longitudinal and transverse divisions of the embryonic brain. The first axons extended from the synencephalon in the ventral longitudinal zone of the neural rod. In the neural tube, throughout the entire brain two pairs of longitudinal fiber systems, one ventral series and one dorsal or intermediate series, and four pairs of transverse fiber tracts in the rostral brain were formed sequentially during the first 16 h of axon production. The overall pattern of the longitudinal fiber systems in medaka brain is similar to that in mouse, but apparently different from that in zebrafish. We propose that a ventral tract reported in zebrafish partially belong to the dorsal fiber system, and that the longitudinal fiber systems in all vertebrate brains pass through a common layout.

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P2-131 Layer disorganization in reeler piriform cortex.

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Previous study showed cerebral cortex develops abnormal layering in reeler mutant mice. With the aids of several layer specific markers, we examined the cytoarchitectonic organization of the piriform cortex in normal and reeler mice. Most of Reelin expressing layer IIa cells are broadly distributed from the outer zone to the inner zone of the piriform cortex of the orlean reeler mutants. The layer IIb cells, expressing a novel gene WSS-1, are displaced in the deep inner zone near or within the endopiriform nucleus. BrdU labeling or several in situ hybridization experiments indicated that the population of the outer zone of reeler piriform cortex corresponds to the subpopulation of layer III in normal cortex. We will discuss how does Reelin regulate the positioning of cortical cells in piriform cortex

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P2-132 Upregulation of necdin expression in bFGF-treated neural stem cells *in vitro*

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Necdin is a growth suppressor expressed in postmitotic neurons. We report here that necdin expression is upregulated by bFGF in the neural stem cell (NSC) culture. Immunostaining of E12 mice revealed that necdin was concentrated in MAP2-positive neurons in the preplate of the forebrain. To study the expression pattern of necdin during neurogenesis, NSCs prepared from the cortices of E12 mice were cultured in the presence of bFGF, which was withdrawn from the cultures 2 days later to induce neuronal differentiation. Unexpectedly, necdin was expressed in NSCs at day 0 as highly as in neurons at day 8 after bFGF deprivation. To test the possibility that bFGF modulates the expression levels of necdin, NSCs were maintained in the presence or absence of bFGF for 4 days. Western blot analysis revealed that bFGF increased the necdin expression in a dose-dependent manner. These findings suggest that necdin expression in NSCs is positively regulated through bFGF signal transduction system.

P2-133 Simple procedure for production of neural stem cells and neurons from embryonic stem cells

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We establish a method to produce a lot of neural stem cells and neurons from mouse embryonic stem (ES) cells. In free-floating culture using astrocyte-conditioned medium with FGF-2 and EGF, colonies of undifferentiated ES cells cause spheres, Neural Stem Spheres (NSSs), composed of abundant neural stem cells. Next, by culturing the NSSs on an adhesive substrate with mitogens, only neural stem cells migrate onto the substrate. The cells can be expanded, preserved by freezing, and differentiated into functional neurons. This NSS method surely provides ES-derived neural stem cells and neurons as donor cells for neuronal transplantation.

P2-134 The distribution of Neurogenesin-1 in the developing and adult rat brain

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Neurogenesin-1 (Ng1) is a 521 amino acid protein containing a N-terminal signal peptide and 3 cysteine-rich domains to which BMP-4 is bound. Ng1 binds to BMP-4 and alters the fate of adult hippocampal neural stem cells from glia to neurons in vitro. In this study, we studied the expression pattern of Ng1 in the developing and adult brain with a polyclonal anti-Ng1 antibody. Ng1 was abundantly expressed in the developing and adult rat brain. To characterize which cell types express Ng1, we performed double immunochemical experiments with several cell marker antibodies. These investigations showed that astrocytes expressed Ng1. The abundant Ng1 expression in the developing and adult brain indicates that Ng1 plays important roles in both brain development and adult neurogenesis.

P2-135 Excitatory GABAergic inputs promoting adult neurogenesis in dentate gyrus

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New neurons continue to be produced in the hippocampal dentate gyrus throughout adult life. A series of reports have proposed the activity-dependent control of the process in which neuronal progenitor cells differentiate into new neurons; however, little is known about how these progenitor cells make connections with the hippocampal circuitry. In this study, we found that neuronal progenitor cells receive GABAergic inputs via $GABA_A$-R activation. The high $[Cl^-]i$ of these cells contributed to the depolarizing effects of GABA as seen in gramicidin perforated-patch recordings. Systemic injection of phenobarbital, a $GABA_A$-R agonist, into mice promotes neuronal differentiations of neuronal progenitor cells in the adult dentate gyrus. Here we propose that GABAergic connections of neuronal progenitor cells with hippocampal GABAergic system mediate the activity-dependent regulation of adult neurogenesis.

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P2-136 Expression of *Drosophila* MAGE in brain neurons

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Necdin is a member of MAGE (melanoma antigen) family and acts as a growth suppressor in postmitotic neurons. Although ~30 members of human MAGE family have been identified, *Drosophila melanogaster* has only a single MAGE (d-MAGE) whose sequence is similar to those of necdin-homologous MAGE family members. In this study, we analyzed the spatiotemporal expression pattern of d-MAGE mRNA and its encoded protein. We cloned d-MAGE cDNA and raised a rabbit polyclonal antibody against the d-MAGE protein. Northern blot analysis revealed that d-MAGE mRNA was expressed at all stages of *Drosophila* development. *In situ* hybridization histochemistry demonstrated that d-MAGE mRNA was enriched in brain neurons of adult fly. By double immunostaining for d-MAGE and neuron-specific elav proteins, the d-MAGE protein was detected in virtually all neurons including photoreceptors and mushroom body neurons in adult fly. These results suggest that d-MAGE has a necdin-like function in *Drosophila* brain neurons.

P2-137 Intracellular domain of delta provides signaling for neurogenesis

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Delta (D) is a ligand which activates Notch pathway. Although the intracellular domain of D (DIC) is believed to be non-functional, we provide evidences of the cleavage of D in intramembrane and of the nuclear localization of DIC. As it is thus possible that DIC controls transcriptions of certain genes, we screened transcription factors which bind to D (mouse Dll1) IC. Nuclear extracts from mouse neural stem cells were incubated with 150 species of DNAs, which represent binding sequences of known transcription factors. After immunoprecipitation with antibody against DIC, DNAs eluted from the precipitated complexes were hybridized to arrays that were spotted with the same set of DNAs. Smad binding sequence showed hybridization and co-immunoprecipitation revealed that DIC strongly binds to Smad2 and 3, that mediate TGF-β/Activin signaling. Furthermore, we demonstrate that DIC enhances Smad depending transcriptions.

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P2-138 Olig2 positive cells differentiate into GABAergeic neurons and/or Oligodendrocyte progenitors in the forebrain

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Olig2 is a bHLH transcription factor regulating fate decision of motor neuron and oligodendrocyte precursors (OLPs) in the spinal cord. However, function of Olig2 in the forebrain remains unclear. Here, lineage tracing of forebrain Olig2 positive cells was performed to examine functional involvement of the gene in cellular differentiation. Fate of Olig2 cells was analyzed with tamoxifen inducible Cre-loxP system, and also with double-labeling of Olig2 and cell type specific markers. Some $Olig2^+$ cells expressed PDGF receptor alpha, a markers for OLPs. Other populations of $Olig2^+$ cells were positive for GABA, GAD65 and calbindin, all of which are marker for GABAergic neurons. This observation was also confirmed by the Cre-loxP system. The present results elucidated that at least certain fraction of Olig2 lineage cells differentiated into GABAergic neurons and/or OLPs.

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P2-139 Enhanced Neurogenesis in the Dentate Gyrus of Non-Human Primates after Focal Ischemia

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Adult neurogenesis in dentate gyrus (DG) is modulated by growth factors, physical conditions, and lesions. We studied effects of focal ischemia on neurogenesis in DG of cynomolgus monkeys. Animals underwent MCA occlusion-induced focal ischemia and were given BrdU injections 1 to 9, 13, and 14 days after ischemia, and then perfused 28 days later. Brains were assessed for BrdU immunohistochemistry and double immunofluorescence with BrdU and neuronal markers. Ischemia caused a 4.7-fold increase of BrdU-labeled cells in the ipsilateral DG to ischemia as compared with control (non-operated). However, there were no significant differences in the proportion of neuronal differentiation. In all groups, ~40% of BrdU-labeled cells were double-labeled with NeuN. Our data suggest that a focal ischemia insult enhances the proliferation of neuronal precursors in DG, but does not promote the neuronal differentiation.

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P2-140 Time-lapse observation of dendrite formation of cerebellar Purkinje cells in vitro

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Cerebellar Purkinje cells (PCs) have the most elaborate dendritic trees among the neurons in the CNS. To investigate dynamical aspects of dendrite formation of PCs, we performed a time-lapse observation of PC differentiation using cerebellar cell cultures derived from glutamate decarboxylase 67-green fluorescent protein (GFP) knockin mice. This method allowed us to visualyze the living GABAergic neurons including PCs as the GFP-expressing cells during 4 weeks in vitro. PCs extended several primary dendrites in the first 2 weeks in vitro. In the next 2 weeks, the shortest primary dendrite was retracted in some PCs, while extensive branching occurred in the other dendrites. Thus, the final morphology of PCs is achieved not only through extension but also through partial retraction of their dendrites. We are currently analyzing morphological characteristics of the retracted dendrites in detail.

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P2-141 In utero fate mapping of Emx- and Dlx-expressing cells in the mouse forebrain

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Glial cells are functionally diverged. However, the mechanism of their subclass identification is yet unknown. We hypothesized that the regionally distinctive neuroepithelial cells along with dorsal/ventral and anterior/posterior axes are settled into various glial subclasses during early development. To resolve this issue we have established a cell fate mapping strategy using in utero electroporation of Cre reporter mice. In this study we first identified enhancer/promoter region of Emx2 and Dlx5/6 genes to precisely control Cre-expression in a region specific manner. A 5.5 kb fragment from 5'-flanking and a 500 bp fragment from 3'-flanking regions of Emx2 gene replicated endogenous Emx2 expression in the dorsal forebrain. A 400 bp fragment from Dlx5/6 intergenic region recapitulated Dlx expression in the basal forebrain.

P2-142 A novel basic helix-loop-helix (bHLH) transcription repressor, NeuroAB, expressed in bipolar and amacrine cells in the developing chick retina

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bHLH transcription factors are implicated in cell-fate determination and differentiation in neurogenesis. We identified a novel bHLH transcription factor, NeuroAB, from chick retina. *In situ* hybridization and immunostaining indicated that NeuroAB is expressed in bipolar cells and GABAergic amacrine cells in the developing retina. Reporter and DNA pull down assays indicated that NeuroAB functions as a transcriptional repressor by binding to the E-box sequence, and its activity is modulated by phosphorylation at a specific serine residue in the consensus phosphorylation site for glycogen synthase kinase 3β (GSK3β). These findings suggest that NeuroAB is presumably involved in the maturation of bipolar cells and GABAergic amacrine cells, and regulation by GSK3β plays an important roll in retinogenesis.

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P2-143 Phenotypes of postnatally generated cells in hippocampal slice culture

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We have previously reported the postnatal neurogenesis in hippocampal slice culture. In the present study we examined the phenotypes of newly generated cells which are differentiated in the slice culture. The new cells were identified by the expression of EGFP inoculated with retrovirus vector. Four weeks after inoculation 25% of newly generated cells expressed NeuN, a marker of mature neuron and 25% expressed Tuj1, a marker of immature neuron. Some of them expressed morphological features of dentate granule cells as well as the molecular marker, calbindin D 28k. The others are immunoreactive with GFAP, a marker of astrocyte and radial glia or with nestin, a marker of neural progenitor. It is suggested that some postnatally generated cells differentiate into neurons while others remain undifferentiated as reserve cells.

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P2-144 A long-term lineage analysis of Olig2-expressing cells by CreER™/loxP system

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Olig2 is a bHLH transcriptional factor that is essential for generation of motoneurons and oligodendrocytes. However, it is not known whether Olig2-expressing progenitors generate only motoneurons and oligodendrocytes.
We performed long-term lineage tracing by using double transgenic mice that have an Olig2 allele replaced with tamoxifen-inducible Cre recombinase, CreER™, and a reporter allele that expresses lacZ gene after CreER™-mediated recombination. We administered tamoxifen to these animals at various embryonic stages and assessed lacZ expression by X-gal staining at later times.
In this study, we show that GFAP-positive astrocytes at the glia limitans and ependymal cells at the ventricular zone are also derived from Olig2-expressing progenitors.

P2-145 Primitive neural stem cells from the mammalian epiblast differentiate to definitive neural stem cells under the control of Notch signaling

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FGF2-responsive definitive neural stem cells first appear in E8.5 mouse embryos, but not in earlier embryos although neural tissue exists at E7.5. Here, we demonstrate that LIF-dependent sphere-forming cells are present in the E5.5-7.5 epiblast/neuroectoderm of the mouse embryo. The resultant clonal sphere cells possess self-renewal capacity and multipotentiality within the neural lineage, cardinal features of the neural stem cell. However, they also retain some non-neural properties suggesting that they are the in vivo cells equivalent to the primitive neural stem cells that form in vitro from embryonic stem cells. The generation of the in vivo primitive neural stem cell was independent of Notch signaling, but the activation of the Notch pathway was necessary for the transition from the primitive to full definitive neural stem cell properties.

P2-146 Nature, migration and fate of hilar proliferating cells in the postnatal rat dentate gyrus

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In the dentate gyrus, most of the granule cells (GCs) are generated postnatally. In this study, we examine the development of newly generated cells into the GCs in early postnatal rats. To detect the newly generated cells, we injected bromodeoxyuridine (BrdU) into 5-day-old rat. Thirty minutes after the BrdU injection, many BrdU-labeled cells were seen in the hilus and more than 60% of the BrdU-labeled cells expressed S100β, an astrocytic marker. Subpopulation of these cells co-expressed GFAP, nestin or GLAST. Thereafter, the percentage of the BrdU-/S100β-positive cells became less than one third of the total BrdU-labeled cells 14 days later (P19). At P19, most of the BrdU-labeled cells were located in the granule cell layer (GCL) and expressed Hu, a neuronal marker. These results suggest that the subpopulation of the proliferative hilar astrocytic cells should generate immature neurons which migrate to the GCL and become GCs.

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P2-147 Gab1 mediates EGF signal inducing selective proliferation of Olig2-expressing neural progenitor cells in the developing spinal cord

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EGF is well-known as a mitotic signal for neural progenitors (NP). However, it has not been shown in what type of NP EGF-mediated signal plays roles for and what signaling mediators function in its downstream. To address these questions, we focused on the development of mouse spinal cord. Olig2, a bHLH transcription factor, induces motoneurons in cooperation with Ngn2 and subsequently specifies oligodendrocytes in cooperation with Nkx2.2 after E12.5. We found that Olig2$^+$ cells expressed EGF receptor at E12.5 in spinal cord and the percentage of Olig2$^+$ cells was increased when cultured in the presence of EGF. Moreover, lack of Gab1, an adaptor molecule in a downstream of EGF, resulted reduction of the number of Olig2$^+$ cells in E12.5 spinal cord and their EGF-dependent proliferation *in vitro*. These results indicate that EGF signal via Gab1 is required for the expansion of Olig2$^+$ NP in the developing spinal cord.

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P2-148 Search for roles of the L3/Lhx8 on retinoic acid-induced neural differentiation in neuro2A by in vitro approaches using the siRNA

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LIM homeobox genes encode a family of transcription regulators, and they all contain two tandemly repeated cysteine-riche double-zinc finger motifs called LIM domains, in addition to a homeodomain. L3, also called Lhx8 (L3/Lhx8), is a member of the LIM homeobox gene family and is specifically expressed in the embryonic basal forebrain and oral mesenchyme. Our previous in vivo study demonstrated that the cells expressing cholinergic neuron markers were significantly reduced in the basal forebrain of the mutant mouse with a targeted deletion of the homeobox region of L3/Lhx8 gene. To further explore roles of the L3/Lhx8 gene, we employed retinoic acid (RA)-induced neural differentiation model in murine neuroblastoma neuro2A cell line, in which L3/Lhx8 mRNA was expressed. We report the effects of siRNA against the L3/Lhx8 mRNA on neural differentiation in this system with special reference to transmitter phenotype.

P2-149 Pax6 haploinsufficiency impairs hippocampal neurogenesis

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Pax6 transcription factor is expressed in brain regions where postnatal neurogenesis occurs such as the SVZ and the SGZ of the hippocampal dentate gyrus. We have shown that Pax6-expressing cells in the hippocampus have a stem cell-like character (nestin+, GFAP+, NeuN-, BrdU incorporation) and that BrdU-incorporation is 30% reduced in the Pax6 deficient (rSey2/+) rat as compared with the wild type. In the present study, we found that Pax6-expressing cells did not express PSA-NCAM, a late progenitor marker. Electron microscopy revealed that Pax6-expressing cells had dark cytoplasm and glial filament. In BrdU pulse/chase experiments, the ratio of Pax6 co-expression was about 100% at 30min after BrdU incorporation and it decreased as time passed. In the rSey2/+ rat, Pax6-expressing cells were small in number, had light cytoplasm, and sometimes co-expressed PSA-NCAM. Our results suggest that Pax6 gene dosage is critical for hippocampal neurogenesis especially at the early stage.

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P2-150 In vitro differentiation of cholinergic neurons from human neural stem cell/progenitor cell (hNSPC) using chondroitinase ABC

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The research to graft hNSPC to the disease part has been attempted. However, it is reported that to control multipotency of grafted hNSPC is very difficult. To overcome these problems, we developed the novel method to effectively generate cholinergic neurons from hNSPCs *in vitro*. Human NSPCs expressed chondroitin sulfate proteoglycans (CSPGs) and secreted these CSPGs to the neurospheres conditioned medium (CM). We found that when CSPGs were activated through chondroitinase ABC the hNSPCs were easily differentiated and some Tuj1 (+) cells differentiated by this method expressed choline acetyltransferase which is the marker of cholinergic neurons. These findings suggested that hNSPCs-derived CSPGs activation by chondroitinase ABC would be very useful to induce cholinergic neurons and could expect to apply for treatment of Alzheimer's disease.

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P2-151 Dorsal-to-ventral migration of Olig3 lineage cells in the fetal mouse hindbrain and spinal cord

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Olig3 is a member of Olig gene family, bHLH transcription factors. To trace the Olig3 lineage cells, we generated knock-in mice that carry lacZ gene in Olig3 locus. X-gal staining was performed in the hindbrain and spinal cord. X-gal-positive(+) cells appeared in the rhombic lip by E11.5. They composed the subpial migratory streams towards the ventral direction, and were subsequently settled in the precerebellar nuclei. In the spinal cord, the X-gal+ cells appeared in the dorsalmost portion by E9.5, except in the roof plate. At E10.5, X-gal+ cells spread ventrally, and some of them were positive for neuronal markers. On E11.5 onwards, these cells were located in the intermediate and ventral parts of the spinal cord, indicating that Olig3 lineage cells that arose from the dorsal part could migrate ventrally. These results revealed the presence of dorsoventral migration of Olig3 lineage cells in the fetal CNS.

P2-152 Cortical and striatal expression of tyrosine hydroxylase mRNA in neonatal and adult mice

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Dopaminergic (DA) neurons were localized primarily in the substantia nigra and olfactory bulb in postnatal mouse brain. Using a sensitive in situ hybridization technique, we demonstrated specific cell populations in the cortex and striatum expressed mRNA for tyrosin hydroxylase (TH), the first enzyme in DA biosynthesis. TH mRNA-expressing cells in the cortex and striatum, first detected at postnatal day 5, showed a gradual developmental increase in number. Transgenic mice harboring a 9kb promoter/EGFP construct were examined to detect TH promoter activity. Many cells especially in the cortical layer V and VI exhibited overlapping expression of EGFP reporter and the endogenous gene. These findings suggest that partial DA differentiation may occur in some cortical and striatal cells, but that full expression of phenotype requires as-yet unidentified factors.

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P2-153 Expression analysis of ATBF1 and ZFH4, the zinc-finger and homeodomain family proteins, in developing rat brains

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ATBF1 and ZFH4 are the members of the ZFH transcription factors, and are suggested to have functions in neural cell differentiation. We generated the specific antibodies, and firstly detected the proteins of 404kDa and 390kDa corresponding to ATBF1 and ZFH4, respectively, in the rat brain by Western blot. The expression levels of both were high in the brainstem at perinatal periods and insignificant in the cerebral cortex in Western blot, Northern blot and immunohistochemistry. Major cellular localization of both proteins was nuclei of postmitotic neurons. Precursor cells in the thalamus and inferior colliculus were intensity labeled for ATBF1 but not for ZFH4, while ZFH4 but not ATBF1 was highly expressed in the subcommissural organ. These results indicate the similar roles of both factors in the region specific CNS differentiation, while they might function distinctively in the specific regions.

P2-154 Non-surface-dividing cortical progenitor cells: Their decision to leave the surface during cell cycle progression

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To understand histogenetic events that trigger the emergence of the cortical plate in the mouse cortex, we used a slice culture technique. Most divisions at the ventricular surface generated paired cycling daughters (P/P divisions) and the majority of the P/P divisions were asymmetric in daughter cell behavior; they frequently sent one daughter to a non-surface position, the subventricular zone (SVZ), within a single cell-cycle length while keeping the other daughter for division at the surface. The NS-dividing cells were mostly Hu+ and their daughters were also Hu+, indicating their commitment to the neuronal lineage. The release of a cycling daughter cell to SVZ was achieved by collapse of the cell's ventricular process during S and G2 phases. Cell cycle-dependent expression of neurogenic transcription facotrs may be involved in the choice of mitosis position, as well as the pattern of interkinetic nuclear movement (Miyata et al., Development, 2004).

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P2-155 Mutation analysis of the *Drosophila* neural C2HC-type Zn finger gene *dnzf*

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NZF/MyT proteins have evolutionarily conserved DNA-binding domains of a C2HC-type zinc finger. Functions of these genes remain elusive, although *Xenopus* MyT1 was shown to play a critical role in neuronal differentiation. We identified a *Drosophila* homologue dNZF that contains only two C2HC zinc fingers. Expression pattern analysis showed that the transcripts and the proteins are expressed in neuronal subsets of the central and peripheral nervous systems (including RPs and vch1). To elucidate *dnzf* function, we generated three *dnzf* mutants by the imprecise excision of a P-element upstream to the *dnzf* gene. Antibody staining revealed that these mutants appear to lack the proteins, suggesting that they are null mutants. Using the GFP balancers, we found that almost mutants die at the late pupal stage (as pharate adults). We are studying on phenotypes of these mutants using various neuronal markers.

P2-156 Systematic analysis of central nervous system-specific promoters in ascidian tadpoles

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The ascidian tadpole larva has a central nervous system (CNS) derived from the dorsal neural tube and consisting of about 330 cells, of which only about 100 cells are neurons. The larval CNS of ascidians can serve as a simple model system for studying the development and function of the chordate CNS. We have identified about 100 genes that are specifically expressed in the larval CNS of the ascidian *Ciona intestinalis*. We have computationally searched the upstream regions of these genes for *cis*-regulatory elements responsible for cell type-specific transcription. Several DNA motifs were found to be over-represented in upstream regions of co-expressed gene sets. Statistics of distribution patterns of the motifs suggest their functional importance, which was confirmed by *in vivo* studies of the promoter activity. We also identified *cis*-regulatory regions required for gene expression in distinct types of neurons by introducing *GFP* fusion constructs into *Ciona* embryos.

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P2-157 Characterization of the proliferating cells in early postnatal mouse cerebellum

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We fractionated the dissociated cells from early postnatal mouse cerebellum by a discontinuous Percoll gradient. The small cell fraction was comprised mainly of granule cell precursors, while the large cell fraction contained the cells expressing GFAP weakly. In the presence of epidermal growth factor (EGF), the number of these cells increased extensively. Most of the cells expressed nestin, brain-lipid binding protein, and RC2. When we removed EGF from the culture, the cells stopped proliferating, lost nestin expression, and began stronger GFAP expression. When we cultured these cells individually in microwells on a non-adherent surface in the presence of EGF, some of them formed floating cell aggregates. A part of the cells expressed neuron specific β-tubulin III, when the aggregates were transferred onto poly-lysine-coated substrates. We are now trying to find the conditions under which most of the cells differentiate into neurons.

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P2-158 Role of aligned neurite bundles of granule cells in determination of Purkinje cell dendrite orientation in 3-dimensional (3D) cerebellar cultures

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The Purkinje cell (PC) has an espalier-like dendrite that is oriented perpendicular to the axonal bundles of the granule cells, the parallel fiber, in the cerebellar cortex. To clarify the factor determining such orientation, we immunocytologically analysed the orientation angles of PC dendrites to neurite bundles of granule cells in a 3D Matrigel culture of dissociated mouse cerebellar cells and in a cerebellar microexplant culture. In the 3D gel, the flattened dendrites of PCs extended at right angles to the aligned neurites of granule cells. In the microexplant proper or nearby, dendrites, but not axons of PCs 3Dly developed preferentially toward the neurites of granule cells and were oriented orthogonal to the aligned neurite bundles. Therefore, the juvenile dendrites of PCs may extend at right angles to the parallel fiber by perpendicular contact guidance in vivo, which was first discovered in the migration behavior of granule cells in vitro.

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P2-159 Chondroitin sulfate proteoglycans in the milieu of neural stem cell

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Behaviors of cells are generally considered to be regulated by their environmental molecules, but molecules in the milieu of neural stem cells have been studied poorly. We found that chondroitin sulfate (CS) existed in surroundings of nestin-positive cells in the rat telencephalon at embryonic day 14. E-unit, a highly sulfated CS disaccharide, was contained in a CS prepared from the telencephalon. A commercial preparation of CS-E promoted proliferation of neural stem cells in culture to form neurospheres, and removal of CS by chondroitinase ABC from neurospheres resulted in adhesion of the cells to a plastic surface. Immunoblot analysis revealed the existence of CS-proteoglycans (CSPGs), neurocan, phosphacan/RPTPβ/ζ, and neuroglycan C in neurospheres as well as the telencephalon. These results suggest that these CSPGs are involved in proliferation and adhesion of neural stem cells as an environmental factor.

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P2-160 The effect of neural activity on cell proliferation and differentiation in the cerebral cortex of adult rats

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Cortical spreading depression (SD) is characterized by propagation of neuronal/glial membrane depolarization throughout the unilateral cerebral cortex and has been linked to several neurological disorders, including migraine aura and epilepsy. We prepared SD model rats using a method of photo-oxidation of a local area in the cortex by external photoirradiation following injection of photosensitizing dye. After SD stimuli, BrdU was injected intraperitoneally into the rats for 3 days. Cortical SD increased BrdU-incorporated cells in the cerebral cortex in proportion to the number of elicited SD events. In the SD-induced cortex, 52.9% of the BrdU-labeled cells were immunopositive for NG2 and 24.6% were immunopositive for OX42. These data indicate that functional excitation of the cerebral cortex induces proliferative response in cortical cells, which may subsequently differentiate into glial progenitor or microglia within 3 days after the stimuli.

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P2-161 Dynamics of nestin-positive cells in the adult dentate gyrus after kainate administration

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It has been reported that kainate-induced seizures promote neurogenesis in the dentate gyrus. In our previous studies we identified two types of nestin-positive neuronal progenitor cells in the adult dentate gyrus using nestin-EGFP transgenic mice : the early progenitors with radial-glia like properties and the late progenitors with neuronal excitatory (S. Fukuda et al., (2003)). So we examined which of the two progenitors would increase in number in response to kainate. Within three days after kainate-injection, the ratio of the early progenitors labeled with BrdU to total BrdU-positive cells increased significantly. Patch-clamp method showed that these early progenitor cells were received perforant path input, and this excitation was blocked by a glutamate-transporter inhibitor L-trans-2,4-PDC. Thus, our study indicates that kainate administration increases the number of the early progenitors in the dentate gyrus.

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P2-162 The lineage tracing of Olig2 expressing cells in the adult mice brain after cryo-injury using tamoxifen inducible Cre/loxP system

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We crossed transgenic mice that express tamoxifen-inducible CreER in oligodendrocyte progenitors under the control of the Olig2 promoter with ROSA-EGFP reporter line. This double transgenic mouse enabled us to perform lineage tracing experiment of Olig2 positive cells after tamoxifen treatment. We have previously reported that proliferating cells after injury have oligodendrocyte progenitors-like characters at early stages after injury in the adult mice brain and thereafter tend to differentiate into astrocytes. We also detected Olig2 mRNA induction after cryo-injury. In this study, we traced cells that express Olig2 shortly after injury in the double transgenic mice and characterized them immunohistochemically.

P2-163 The expression and functional studies of Cystatin C in the developing mouse central nervous system

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Cystatin C is an inhibitor of cysteine proteases, such as cathepsin family, and is recently implicated in the survival of neural stem cells. We have shown that cystatin C enhances astrocytic differentiation in vitro in the primary culture system. Here we show that the expression pattern of cystatin C dramatically changes in the embryonic brain and spinal cord, as well as in the developing eye. At E11.5, cystatin C mRNA was detected only in the optic stalk, and later at E16.5, the gene was expressed in the pigment epithelium and optic nerve head. In the forebrain, cystatin C mRNA was present in the tissue surrounding the ventricles at E12.5 and later. Using neutralizing antibodies in the primary culture of E14.5 mouse brains, inhibition of cystatin C activities facilitated oligodendroglial differentiation and suppressed astrocytic differentiation. Thus, cystatin C may regulate glial fate determination.

P2-164 Neuronal differentiation is promoted by treatment with G1/S cell cycle blockers to neurospheres

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To investigate the effect of cell cycle arrest on neuronal differentiation, neurospheres from E12.5 rat ventral mesencephalon (VM) were prepared and differentiated for 3 days after cell cycle regulation with G1/S phase blockers (deferoxamine etc.). The numbers of β-tubulin III (+) cells became twice after treatment with the cell cycle blocks. Expressions of mRNA and protein of MAP2 were increased while that of GFAP protein was decreased. With the differentiation after BrdU labeling for 5 hr just before treatment of cell cycle blockers, number of β-tubulin III/BrdU double-positive cells was increased while that of GFAP/BrdU double-positive cells was decreased, suggesting the induction of neuronal differentiation but not glial one. No significant change in expression of p21, p27 and p57 was found just after the treatment of cell cycle blockers for 8 hr. Data suggest that neurospheres were promoted to differentiate to neuronal phenotype by treatment with the G1/S blockers.

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P2-165 Functional Characterization of a Novel Neuron-Specific Protein Family, BRINP

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We have recently identified a family of neuron-specific proteins, BRINP whose expression is regulated by BMP and retinoic acid during development. Three BRINP proteins, BRINP1-3 are unique proteins in that they posses no conserved motif except for a signal peptide and a coiled-coil domain. Genomic localization of human *BRINP1* homolog within a candidate bladder tumor suppressor region suggested its function as a tumor suppressor. In the present study, we examined the effects of overexpression of these proteins in mammalian cells. We found that BRINPs require the signal peptide for glycosylation and appropriate subcellular localization in PC12 and NIH3T3 cells. Flow cytometric analysis revealed that overexpressed BRINPs suppressed the cell cycle progression at G1/S transition in these cell types. These findings suggest that BRINPs play important roles in the cell cycle arrest of terminaliy differentiated neuronal cells.

P2-166 Distribution of GAP-43 in the medulla oblongata and sensory ganglion of rat during postnatal development

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To investigate the function of growth associated protein (GAP)-43 in developing nervous system we examined the distribution of GAP-43 in the medulla oblongata and trigeminal and vagus ganglia of postnatally developing rats. Dense GAP-43-immunoreactive (IR) terminals were seen in the medulla oblongata widely including spinal trigeminal, solitary and hypoglossal nucleus and reticular formation by postnatal day 10 (P10). After P14 GAP-43-IR labels localized in solitary and spinal trigeminal nucleus and in spinal trigeminal tract as fiber-bundles showing TRPV1-immunoreactivity also. GAP-43-IR neuronal soma and fibers were observed in trigeminal, nodose and jugular ganglion in postnatal all stages. Moderate immunoreactivity was seen in majority of nodose ganglionic neurons of P5 rats. The ratio of TRPV1-IR neurons in GAP-43-IR ones increased at later stages. Our results suggest that GAP-43 functions for neural development widely in the medulla oblongata at postnatal early stage and then converges in sensory neurons.

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P2-167 Influence of prenatal exposure of Genistein, a phytoestrogen, on the catecholaminergic neurons in the locus coeruleus in the rat

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To investigate the effect of prenatal exposure of Genistein, a phytoestrogen, on the development of the catecholaminergic neurons in the locus coeruleus, a sexually dimorphic nucleus (SDN), Genistein was subcutaneously injected daily from gestational days 15 to 19, into pregnant SD rats at the dose of 0 (control), 1.5 (low dose) or 30 (high dose) mg/kg/day. At 56 days of age, 4 male and 4 female offsprings from each group, were perfused with 4 % formaldehyde. The coronal sections of 18-μ thickness were immunostained with tyrosine-hydroxylase-antibody. Then the numbers of A4 and A6 catecholaminergic neurons in the locus coeruleus and/or subcoeruleus were counted. The total numbers of LC neurons (A4+A6) in the control group are about 3000-3500. The numbers of these neurons decreased dose-dependently both in male and female. Our result suggests that the sex difference of the number of LC neurons is abolished by the effect of prenatal genistein exposure.

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P2-168 FGF2 signaling regulates proliferation and maintenance of multipotentiality of neural stem cells via the glycogen synthase kinase3β pathway.

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FGF2 signaling controls neural stem cell proliferation and differentiation. We found that FGF2 stimulation caused gsk3β phosphorylation in neuroepithelial cells. FGF2 also induced the mRNA and protein levels of cyclin D1, which is known as a downstream target of gsk3β. An Akt inhibitor LY294002 impaired the FGF2-mediated gsk3β phosphorylation and cyclin D1 induction. In addition, FGF2 enhanced the promoter activity of Hes1, which is a Notch target gene and maintains neural stem cells in the undifferentiated state. The activation of the Hes1 promoter by FGF2 was blocked by Akt inhibitor LY294002 and dominant active gsk3β. Taken together, these results suggest that the effects of FGF2 on proliferation and maintenance of multipotentiality of neural stem cells are, in part, mediated by inactivation of gsk3β via PI_3K-Akt pathway.

P2-169 Biochemical study of neuronal RNA binding protein Hu

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Neuronal Hu RNA binding proteins are expressed immediately after the neuronal progenitor withdraw from cell cycle and continues to be expressed in postmitotic neurons. Recent studies indicate that overexpression of Hu is sufficient to induce neuronal differentiation and bind with UTR region of mRNA for genes involved in cell cycle inhibitor, such as p21 and p27 and neuron specific structural genes. To reveal molecular mechanism underlining timing-control of neuronal differentiation through posttranscriptional regulation by Hu, we identified Hu associating proteins. We isolated RNP complexes associated with Hu from exstracts of Hu-adenovirus infected cultured cells and three proteins were identified by MALDI-TOF Mass. We demonstrated that one of Hu associating proteins, hnRNPK bound to p21 mRNA 3'UTR and repressed p21 translation in nonneuronal and neuronal cells. We will discuss the role of two RNA binding proteins, Hu and hnRNPK in neuronal differentiation through post-transcriptional regulation of p21 mRNA.

P2-170 A comparative analysis of layer-specific genes in mammalian neocortex

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Mammalian neocortex consists of six layers containing neurons of similar size, morphology and stereotypical connections. Several studies have identified genes with high layer specificity, suggesting the difference of molecular genetic properties of neurons that constitute each layer. These layer-specific genes can be used as an excellent marker to examine the region- and species-specific differences of cerebral cortex. Here we report the detailed expression analyses of a panel of genes that are preferentially expressed in layers 4, 5, 6 and their sublayers in monkey cortex. We have used the fluorescent double in situ hybiridization technique to accurately determine the layer borders and distinguish distinct subpopulations in the same layer positions. These analyses revealed not only the conserved aspects of cortical laminar structure but also many variations present in different areas and species.

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P2-171 Effects of prostaglandin E2 on ischemia-induced neurogenesis

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Neurogenesis in the dentate gyrus (DG) of adult rodents is elicited by brain ischemia. Cyclooxygenase (COX)-2, a limiting enzyme for prostanoid synthesis, is also induced by ischemia. To clarify whether prostaglandin (PG) E2 synthesis by COX is involved in neurogensis, PGE2 and PGD2, a major product of the arachidonate cascade, were injected into the hippocampus. Incorporation of BrdU in the DG was accelerated by PGE2 though PGD2 has no effect on cell proliferation. Since PGE2 receptor has four subtypes, we studied to identify the receptor that stimulates proliferation of neural progenitor cells using a receptor specific agonist. EP1 and EP4 agonist caused an increase in the number of BrdU-positive cells in the subgranular zone (SGZ) but not EP2 and EP3 agonist. On the other hand, EP1 is detected in the DG neurons adjacent to the SGZ using immunohistochemical method. These results suggest that PGE2 plays an important role in the proliferation of cells in the SGZ via EP1. At present, we are examining in detail localization of EP4 in the DG.

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P2-172 N-myc downregulated gene 1 (NDRG1) is expressed in developing neural cells in the postnatal brain

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N-myc downregulated gene 1 (NDRG1) is induced by DNA damage, hypoxia, intracellular calcium elevation. NDRG1 is also upregulated during cell differentiation. We have previously reported that, in the hippocampus, the cellular localization changed from the pyramidal neurons to the astrocytes between postnatal day 10 and 20. We have immunohistochemically investigated the overall expression pattern of NDRG1 in the postnatal brain. NDRG1 was expressed in radial glia, migrating and/or immature astrocytes and oligodendrocytes in the early postnatal neocortex. In the cerebellum, immature neurons in the external germinal layer, as well as Bergman glia expressed NDRG1. In the adult brain, NDRG1 was expressed in the cortical astrocytes and Bergman glia. It is suggested that NDRG1 is expressed in the astrocytic lineage throughout life, but is upregulated in differentiating neural cells.

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P2-173 Notch-RBP-J signaling regulates cortical laminar fates.

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The developmental stage at which progenitors exit the cell cycle determines the functional type and migration target of neurons. However, the molecular mechanism of the regulation of this cell cycle exit is largely unknown. To examine involvement of Notch-RBP-J signaling in the cortex laminar formation, we deleted RBP-J from neural progenitors in anatomically restricted areas by in vivo electroporation of Cre-expressing plasmids into embryonic ventricles of RBP-J-floxed mice. Such studies revealed that RBP-J deficiency caused transformation of glutamatergic pyramidal neurons in layerII/III to layerIV glutamatergic neurons with concomitant loss of astrocytes. Similarly, layerII/III interneurons changed their fates to layerV interneurons in RBP-J-deficient mice. The results showed that Notch-RBP-J signaling regulates the timing of the cell cycle exit of neuronal progenitors, and determines the laminar and cellular fates of neural progenitors of both glutamatergic and GABAergic types.

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P2-174 Functional subdivisions of the hand representation in the Japanese monkey second somatosensory cortex

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We previously reported the hand representing area in the Japanese monkey second somatosensory cortex (SII) which occupies the upper part of the parietal opelculum (Taoka et al 2000). In the present study, we found another representation of the hand which exists in more rostral and deeper part of SII. Electrophysiological studies using conscious animals revealed that the receptive fields (RFs) of the caudal hand region (c-hand-SII) usually included both side hands and often covered another body parts, the foot and/or face, while RFs of the rostral hand region (r-hand-SII) were almost always restricted to the contralateral hand. After injecting retrograde tracers, we found that c-hand-SII received inputs from areas 1, 2 and 5 of the postcentral hand region, while r-hand-SII received inputs from only area 3b suggesting that SII hand representation is composed of two functionally different subdivisions.

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P2-175 Effects of interleukin-2 on histamine-induced itch-associated responses and intracellular Ca^{2+} concentration in primary sensory neurons in mice

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Although interleukin-2 (IL-2) has been suggested to be involved in itch, pruritogenic mechanisms of IL-2 are unknown. In mice, an intradermal injection of IL-2 (3-100 mU/site) did not induce scratching, an itch-associated response. Although histamine (10 nmol/site) alone did not elicit scratching, the response was dose dependently increased by intradermal pretreatment with IL-2 (3-100 mU/site). Immunostaining showed that the murine dorsal root ganglion neurons expressed IL-2 receptor. IL-2 (0.1-1 mU/ml) enhanced high K^+ (20 mM)-induced increase in intracellular Ca^{2+} ion concentration, with a peak effect at 0.5 mU/ml. IL-2 (0.1-1 mU/ml) itself did not affect intracellular Ca^{2+} ion concentration. The results raise the possibility that IL-2 increases itch through enhancing the responses of primary sensory neurons to itch mediators.

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P2-176 Representation of The Secondary Somatosensory Cortex in local rubbing stimulation: an fMRI study

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The purpose of this study was to investigate the somatotopic organization of different skin areas (right index finger, fifth finger, elbow, knee and foot) in the human secondary somatosensory cortex. Eleven healthy volunteer were examined on a 1.5T MRI scanner to acquire fMRI data. The mentioned skin areas were rubbed according to a block design. In all skin areas, we could detect significantly activation in the contralateral somatosensory area during stimulation. These results are consistent with the somatotopic representation of S1 reported by Penfield et al.. S2 was activated near the parietal operculum. Highest activation in the stimulation of the leg was found caudal and lateral of the parietal operculum, whereas activation of the arm was found within the parietal operculum. The results for the leg differ from other reports.

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P2-177 Differential roles of the thalamic VPm and POm nuclei in a directional discrimination task with a single whisker of the rat.

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For a rat, the whisker is one of the most important sensory organs to acquire spatial information. Sensory information received with a whisker is relayed to the barrel cortex through two thalamic nuclei, VPm and POm. In order to examine functional difference between the two nuclei for discrimination of stimulus direction, we trained rats to discriminate direction of stimuli applied to a single whisker. In this task, bending the whisker forward instructed the rats to lick (i.e. "Go"), while bending the whisker to the opposite direction instructed not to lick (i.e. "No-Go"). All the rats responded correctly for more than 90% of the trials. When we made a selective lesion in VPm, rate of correct response dropped significantly. In contrast, when a lesion was made in POm, we found no significant change in performance. These results suggest that VPm plays more important roles in discrimination of stimulus direction.

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P2-178 Anesthetic response and abnormal behavior to menthol and cold stimulation in rice fish

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Effects of menthol and cold stimulation to rice fish were examined in a tank. A concentration of 0.5 mM menthol addition to the tank induced an anesthetic response but not an abnormal swimming behavior in 56% of the fish tested. All of the fishes showed the anesthetic response to menthol more than 0.5 mM. A latencies of the response in the anesthetic response was to be a shorter in a dose dependently manner. A high concentration of menthol (3.3 mM) also produced the abnormal swimming behavior followed by the anesthetic response. Rapid temperature change from 22°C to lower temperature also produces the anesthetic responses and a threshold temperature was around 7.8°C. Addition of menthol (0.17, 0.33 mM), which never produced the anesthetic response itself, lowered the threshold temperature to produce anesthetic response by the cold stimulation. The present results suggest the possibility that the fish has menthol receptors and cold nociceptors.

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P2-179 Efferent connections of the sensory trigeminal nucleus in the carp

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The trigeminal nerve in teleosts conveys general somatosensory information from the head region to the sensory trigeminal nucleus (STN). To identify targets of the secondary sensory trigeminal projections, efferent connections of the STN were examined by tract-tracing methods in the carp. Injections into the STN mainly labeled terminals in the bilateral nucleus ventromedialis thalami, stratum album centrale of the optic tectum, nucleus lateralis valvulae, torus semicircularis, with the preference for the contralateral side. Labeled terminals were also observed in the bilateral nucleus ruber of Goldstein, nucleus nervi oculomotorii and inferior reticular formation, in the ipsilateral inferior lobe and eminentia granularis, and in the contralateral sensory trigeminal nucleus and descending trigeminal nucleus. The present study revealed that the organizations of ascending trigeminal pathways in teleosts are basically similar to those in mammals.

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P2-180 Fiber connections of the nucleus preglomerulosus pars commissuralis in a cichlid fish, *Tilapia*.

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The nucleus preglomerulosus (PG) in the diencephalon relays various sensory information to the telencephalic dorsal area. In percomorph teleosts, the PG is divided into three parts: pars lateralis (PGl), pars medialis (PGm), and pars commissuralis (PGc). The PGl relays gustatory inputs, and the PGm relays octavolateral inputs to the telencephalon. However, fiber connections of the PGc remained to be clarified. In this study, we examined the connections of the PGc by means of a tract-tracing method using biocytin in *Tilapia*. The results showed that the PGc recieved projections from the corpus mamillare pars parvocellularis (CMp) and sent ascending fibers to the telencephalic dorsal area. The CMp received projections from the nucleus diffusus lobi inferioris (NDLI) and torus lateralis of the inferior lobe. Since the NDLI received the gustatory and general visceral sensory inputs, the PGc probably relays such information to the telencephalon.

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P2-181 GAP-43 mRNA is increased in the motor cortex of the recovered brain-injured monkey

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GAP-43 is involved in synaptic sprouting. To investigate the neuroanatomical basis of motor recovery, we analyzed the expression of GAP-43 mRNA in the motor areas of macaque monkey after recovering from brain injury. Before lesion, the monkey retrieved small food pellet smoothly using index finger-to-thumb-opposition. Ibotenic acid injection into digit representation region of the primary motor area resulted in hand paralysis. After one month of postlesion rehabilitative training, smooth movement with affected hand was recovered. In the recovered brain tissue, GAP-43 mRNA was more intensely expressed in the damaged hemisphere, especially in the ventral premotor area than in the undamaged hemisphere. The result suggests that synapse formation in the damaged hemisphere is involved in motor recovery.

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P2-182 Ectopic corticospinal and corticothalamic neurons in the cerebral necortex of *fukutin*-deficient chimeric mice

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Fukuyama-type congenital muscular dystrophy, which is mutated in *fukutin*, manifests muscular dystrophy and brain malformation. We had previously generated *fukutin*-deficient chimeric mice to study a functional role of fukutin. We injected Fluoro-Gold into the spinal cord to label corticospinal (CS) neurons in chimeric mice and two days later the animals were perfused for laminin imunohistochemistry. Next, we injected WGA-HRP into the thalamus to label corticothalamic (CT) neurons in adult chimeric mice. Some neurons were ectopically distributed just beneath the pia mater in chimeric mice. Ectopic CS neurons were distributed just beneath the disrupted laminin-positive basement membrane, suggesting that fukutin is important for the formation of the laminar organization of the cerebral cortex.

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P2-183 Differential evaluations of rat hind-limb function after spinal cord injury

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In order to establish the ideal evaluation system for motor function after experimental spinal cord injury, we compared three methods. Under general anesthesia, spinal cord was carefully exposed and compressed by 20-g weight for 20-60 minutes. The evaluation of hind-limb function was done at third, seventh and 14th day after the injury. Evaluations were done by BBB scale, forced walking distance measured by treadmill, and counting of standing frequencies. At 3-7th day after the compression, significant decrease of BBB score was observed in 40-60min compression models. Walking distance and standing frequencies showed significant decrease in 20-60min compression models. At 14th day after the injury, significant result in BBB score was observed only in 60min compression models while walking distance and standing frequencies showed significant change in 20-60min compression models.

P2-184 Central Administration of Leukemia Inhibitory Factor Disrupts Dopaminergic Signaling and Latent Inhibition

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Leukemia inhibitory factor (LIF) is a pleiotrophic cytokine induced by nerve injury and implicated in etiology and/or pathology of various psychiatric diseases including schizophrenia. Here we evaluate the effects of continuous infusion of LIF into the right striatum on neurochemical and behavioral parameters. LIF treatment significantly decreased the contents of dopamine, and increased its turnover and the expression of dopamine D1 receptors. In parallel, the rats treated with LIF displayed abnormal behaviors in motor activity and latent inhibition of contextual learning. These results demonstrate that LIF is a potent regulator for dopaminergic function and cognitive ability in the brain and might contribute to pathology of psychiatric impairments.

P2-185 Striatal Epidermal Growth Factor/ErbB1 Signal Alters Dopamine Metabolism and Functions

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Epidermal growth factor (EGF) and its receptor (ErbB1) signal exert neurotrophic activity on various neurons such as midbrain dopaminergic neurons. In the present study, we investigated whether EGF administration into adult rat brain influence dopamine metabolism as well as its related behaviors including methamphetamine (METH) sensitivity. Intrastriatal EGF infusion markedly altered monoamine synthesis and turnover. In the locomotor test, however, we failed to find any effects of EGF but EGF treatment enhanced sensitivity to repeated METH (1mg/kg) treatments. In addition, startle response was increased and PPI was decreased in the EGF-treated group. In context fear conditioned task, however, their memory retentions were normal but their latent learning was disrupted. These results indicate that abnormal EGF/ErbB1 activity in the brain disrupt dopamine transmission and cognitive functions.

P2-186 NMDA receptors are increased in the hippocampus of heterozygous Reeler mice.

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Heterozygous Reeler mice (HRM) display several phenotypes resembling schizophrenia. As disturbances in NMDA-Rs are hypothesized in this disorder, we measured the amount of NMDA-R proteins in the brain of HRM by immunohistochemistry and Western blotting. The immunoreactivities of NMDA-R subunits were increased in the hippocampus of HRM in comparison with wild-type mice. When we measured the amount of NMDA-R mRNAs using Quantitative Realtime PCR, no difference were detected between HRM and wild-type mice. On the other hand, the protein phosphorylation of NMDA-Rs were increased in HRM, which may result in the prevention of proteolysis of NMDA-R subunits. In schizophrenia brains, the decrease in Reelin and the increase in NMDA-Rs were also reported.Therefore, these results are another schizophrenia-like pathology of HRM as well as propose a novel pathway between Reelin and NMDA-Rs.

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P2-187 Generation and characterization of the *Als2* knockout mice

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The loss-of-functional mutations in *ALS2* accounts for a number of juvenile recessive motor neuron diseases (MNDs) including a type of juvenile ALS (ALS2), juvenile-onset PLS, and infantile-onset ascending hereditary spastic paralysis. To delineate the molecular pathogenesis for these MNDs, we here generated mice homozygous for disruption of *Als2* by a standard homologous recombination strategy. Disruption of *Als2* and null expression of the ALS2 protein were confirmed by southern and western blotting, respectively. The *Als2* knockout mice were born and grew with no obvious developmental and reproductive abnormalities by 10 months of age. Behavioral and pathological analyses of these mice are currently underway. Generation of the *Als2* knockout mice will provide additional clues to the understanding of the molecular pathogenesis for MNDs caused by *ALS2* mutation.

P2-188 Mice with down-regulated expression of neuroglycan C, a CNS-specific transmembrane proteoglycan, show abnormal behaviors

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Neuroglycan C (NGC) is a membrane-spanning chondroitin sulfate proteoglycan (CSPG) that is expressed predominantly in the brain. To study the role of NGC in brain development, we planed to generate NGC-deficient mice by conditionally-targeted disruption of the NGC gene. On the way, we found that the expression of NGC was down-regulated in various regions of the brain in incomplete NGC-knockout mice. The NGC-knockdown mice were viable and fertile. The mice did not show major differences in gross brain anatomy. However, the mice showed obvious behavioral alterations in open field tests; the NGC-knockdown mice have an extremely low motility, suggesting NGC takes part in functional development of the brain.

P2-189 Alterations in the expression of genes involved in exo- and endo-cytosis in the pancreatic islets of the diabetic GK rats

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The Goto-Kakizaki (GK) rats which develop diabetes at maturation are considered as a model of human type II diabetes. In pancreatic islets from GK rats, a decrease in insulin secretion has been shown. Using two types of DNA microarrays, a high-density array and our original Synaptoarray, we compared gene expression profiles between the pancreatic islets of GK and normal rats at 4, 6, and 10 weeks of age, to find the regulatory factors in the exocytotic process which share many of its components with the nerve terminal. At 10w, genes for metabolic enzymes, membrane transport factors, and mitochondrial proteins were down-regulated, while stress-response genes were up-regulated in GK. Additional SNARE-related genes were found to be differentially expressed using the Synaptoarray. The results suggest the impairment in the exocytotic process as one of the causes for the diabetic condition in GK rats.

P2-190 Generation and analysis of α-synuclein transgenic mice using a Cre/loxP system

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To assess the degenerative mechanism of dopaminergic (DA) neurons in human Parkinson's disease, we generated α-synuclein (αS) transgenic (Tg) mouse lines using a Cre/loxP system. Human αS cDNAs encoding WT, A30P, A53T, or S129A variants were joined downstream to CAG promoter-loxP-EGFP-loxP cassette, and the resulting constructs were microinjected. The obtained αS Tg mice were then crossed to TH promoter-Cre Tg mice, obtaining double Tg mice that express αS innately in DA neurons in substantia nigra (SN). Otherwise, the rAAV containing Cre-expression cassette was injected stereotaxically to SN of the αS Tg mice, to express αS in DA neurons in adult mice. These mice are now under the behavioral, physiological, and biochemical analyses. The usefulness of the Cre/loxP-Tg model will also be discussed.

P2-191 The development of PGP9.5-immunopositive neural network structure using the confocal laser scanning microscopy in the embryonic rat intestine.

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The enteric nervous system has a 3-Dimensionally complicated structure. To investigate the development of this structure, we applied confocal laser scanning microscopy to whole-mounts of embryonic intestine, after immunostaining with PGP9.5 antibody with spacer-equipped slides. At embryonic day 12.5(E12.5), immunopositive(IP) nerve cells connected by a few nerve fiber bundles were scattered in the intestine. At E15.5, the number of IP bundles increased significantly, and a neural network with a IP meshlike structure appeared at this time. The distribution of IP nerve cells and form of this IP network were similar to those features in neonate. The area of IP neuronal network and the number of IP nerve cells kept decreasing until E16.5. The meconium passed through the intestine from E17.5, the time at which we consider the ENS to be morphologically established.

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P2-192 thalidomide- or valproic acid- induced autism model rats show serotonergic abnormalities

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Previously, we reported autism model rats induced by thalidomide (THAL) or valproic acid (VPA)(Pediatric Research, 52; 576: 2002). In those rats, abnormalities of serotonergic system, which are often observed in human autism, were observed. We next examined whether early serotonergic neuronal development is perturbed by THA/VPA. When THA or VPA was exposed on embryonic day 9, a dramatic shift of the distribution of serotonergic neuron in the dorsal raphe nuclei was observed on postnatal day 50. This alteration is thought to reflect abnormality of serotonergic neuronal differentiation and migration. In vitro studies revealed that VPA retards the maturation of serotonergic neuron from ES cell-derived neuronal progenitors, and exogenously added Sonic hedgehog, a morphogen which has been implicated in serotonergic cell fate, partially compensated the retardation. These results indicate that distraction of early serotonergic neuronal development might be responsible for autism.

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P2-193 Animal model of autism: possible implication of thyroid hormone

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Thyroid hormone is essential for mammalian brain development. It is well known that maternal hypothyroidism can lead to neurological deficits in the offsprings. We have reported that rat pups exposed to antithyroid agent, propylthiouracil (PTU) exhibit hyperactivity and spatial learning impairment. Recently, knockout mice lacking of thyroid hormone receptors (TRKO) have been suggested to show behavioral abnormalities. We investigated the development of cerebellum and several other brain regions in PTU rat pups and TRKO (α, β). Hyperactivity was obvious in PTU rat pups and TRKOα. Time course of apoptosis in the cerebellum was delayed in PTU rats. Furthermore, expressions of several gene families, which are known as organizers in ishmus, were also delayed in the cerebellum of PTU rats. It is thus suggested that developmental hormone deficit may provoke derangement of gene expressions in the cerebellum and related structures.

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P2-194 An experimental targeting protocol for gamma knife irradiation on rat brain.

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We propose a new targeting protocol for gamma knife irradiation on the study of rat brain. Using a Regis-Vallitioni frame and MRI imaging, the target was determined relative to the front and rear end, dorsal surface and midline of the cerebrum, which were easily observed with MRI images. Gamma irradiation of 150 Gy was applied with 4 mm collimator focusing on the left striatum. The positions of the center of the resulting lesions were surveyed histologically. The deviations of the aiming from the target plan were less than 1mm in any direction. The center of the lesion was occupied by the necrotic area, which was surrounded by vascular delatation, gliosis and chromatine aggregations. The irradiated rats showed the apomorphine-induced circling behavior after 4 weeks from the irradiation. The patterns of circling behavior were comparable with that resulted from 6OHDA injection, suggesting the alterations of dopaminergic functions at the irradiated striatum.

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P2-195 Development of neurogranin mRNA in the monkey cerebral neocortex

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We previously reported that mRNA expression of GAP-43, a marker molecule for axonal sprouting, is high in the fetal and early postnatal period, and decreased to the adult level at postnatal day 70 (P70). In the present study, we investigated the expression of neurogranin, the dendritic counterpart of GAP-43, in the developing monkey neocortex. The Northern blot analysis showed that the amount of neurogranin mRNA was low during fetal periods, increased to its peak value at P70 or at postnatal month 6, and decreased gradually until postnatal years 2-3. The in situ hybridization experiments also showed transient increase of neurogranin mRNA around P70, especially in layers II and III. The present results suggest that dendritic development in the monkey cerebral neocortex continue even after axonal development has completed.

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P2-196 Proteomic analysis in rat brain with neonatal serotonin depletion using two-dimensional electrophoresis

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Serotonergic dysfunction was suggested in some developmental disorders such as infantile autism. We prepared neonatal 5-HT depleted rats with i.c.v. injection of 5,7- dihydroxy-tryptamine (5,7- DHT) at 3 and 6 days after birth. We previously reported that in 5,7-DHT treated rats, the serotonergic fibers were significantly reduced in the cerebral cortex but not in the brainstem, but that the serotonergic neurons were significantly reduced in the dorsal raphe nucleus. In this study, we investigated the changes of protein expression level in the model rats using two-dimensional electrophoresis, and found the up- or down-regulation of several proteins in the cerebral cortex and brainstem. These results suggest that neonatal serotonin depletion affects the expression level of some proteins in the rat brain which may influence on brain development.

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P2-197 Relationship between neuronal migration and expression of L1 molecule in the brain following prenatal exposure to irradiation

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The relationship between neuronal migration and neuronal cell adhesion molecule L1 in the brain were investigated through comparison between rats prenatally exposed to X-rays and controls. The pattern of neuronal migration was identified by distribution of BrdU-labeled cells in the cereberal cortex. Expressing L1 in the brain was analyzed by SDS-PAGE after isolation of L1 by immunoaffinity chromatography. The results showed that neuronal migration disorder and reduction of expression L1 appeared in the irradiated brains, suggesting migration of neural cells may be dependent on neural cell molecule L1. A decrease in L1 expression may be one of reasons of abnormal neuronal migration.

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P2-198 Requirement of a POZ/zinc finger transcriptional repressor, RP58, for cortical lamination and pathway formation

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We have generated mice lacking the RP58 gene, a sequence-specific transcriptional repressor. Birth-date analysis using bromodeoxyuridine indicates that neuronal migration in the mutant shows a reeler-like outside-in pattern. Furthermore, in the mutant, cortical efferent axons did not invade the striatum, and thalamocortical axons were reduced in number and ran aberrantly in the cortical plate. Restricted expression of RP58 transcripts in the cerebral cortex suggests that abnormal development of cerebral cortical neurons in this mutant causes both abnormal lamination and pathway formation.

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P2-199 Effects of repeated stress on Fos expression in the maternal and fetal rat hypothalamic paraventricular nucleus in late pregnancy

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This study was designed to elucidate effects of repeated stress (restraint and immobilization, daily for 30 min from gestational day 17 to 21) on Fos expression in the maternal and fetal hypothalamic paraventricular nucleus (PVN). We assessed apoptosis and cell death in the fetal PVN after acute and repeated stress, in addition to the number of Fos-immunoreactive cells in the fetal and maternal PVN. In the parvocellular region of the maternal PVN, repeated mild (restraint) but not severe (immobilization) stress revealed significant decreases in the number of Fos-positive cells compared to acute stress. In the fetal PVN, reduced Fos expression following repeated stress occurred following severe but not mild stress. Since the fetal PVN in the repeated severe stress group revealed marked apoptosis and cell death, the reduced Fos expression in the fetal PVN of this group may be due to apoptosis and cell death.

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P2-200 Effect of perinatal hypothyroidism on gene expression in the developing mouse cerebellum studied with an original oligoDNA microarray

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To study the roles of thyroid hormone in brain development, we analyzed the time course of gene expression changes in the cerebellum of experimentally hypothyroid and control mice during a perinatal period of up to 4 weeks using original oligoDNA microarray "Synaptoarray". In addition to synaptic protein and relevant genes were selected experimentally using a high-density array, resulting in the array with 496 probes for 177 genes and capable of detecting ±20% changes in expression ratio. Hypothyroidism resulted in;[A] up-regulation of growth-associated genes at P15, [B] up-regulation of stress response genes, and [C] down-regulation of TH-related genes and genes involved in later stages of synaptogenesis. Retardation of growth and synaptogenesis as well as induction of stress response by hypothyroidism was clearly detected by the Synaptoarray, which was confirmed morphologically.

P2-201 Attention-deficit and hyperactive neurobehavioural characteristics by perinatal hypothyroidism in rats

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We examined behavioural alterations caused by transient perinatal hypothyroidism using a series of behavioural tests consisting of the open field test, passive and active avoidance test, and measurement of spontaneous activity, focusing particularly on attention-deficit/hyperactivity disorder (ADHD)-like behaviours. Various abnormalities might coincide with those found in case of ADHD. In addition, we will demonstrate the gene-expression profile perturbed by perinatal hypothyroidism in cerebral cortex of male neonatal rats.

P2-202 Perinatal exposure to BPA affects ER expression in rat hippocampus

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We have reported the rats perinatally exposed to low dose bisphenol A (BPA) expressed hyperactivity and impaired memory. Estrogen receptor alpha (ER alpha) expression in the hippocampus was investigated to further clarify the mechanism. Pregnant SD rats were treated with BPA and/or ICI from the pregnant 11th day to the 21st day after childbirth. The male pups were tested in open field and Morris water maze. ER alpha of hippocampus was studied with the methods of immunohistochemistry, Western blot and real-time PCR. The male pups from BPA+ICI showed no difference from control group, while hyperactivity and impaired spatial memory was observed in BPA group. In CA1, CA3 and DG, the control and BPA+ICI groups showed highest immunoactivity for ER alpha on 7day, but in BPA group the peak appeared on 11day. The results of Western blot and real-time PCR for ER alpha indicated same tendency as above. These findings suggest that ER alpha is involved in the effect induced by perinatal exposure to BPA.

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P2-203 Fetal exposure to bisphenol A impairs sexual differentiation of brain functions in rats

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Perinatal exposure to Bisphenol A (BPA), a well-known endocrine disrupters, has been shown to influence the sexual differentiation of brain and behaviors. In this study, BPA was exposed to the pregnant Wistar rats at a dose of 0.1ppm during the last week of prenatal period. The offspring were tested using an open field test, an elevated plus maze test, a passive avoidance test and a forced swimming test. Olfactory responses of medial amygdalar neurons were also examined. In the BPA treated group, sex differences of the number of rearing, the duration of struggling, and the pattern of neuronal responses to odors were abolished. The duration of immobility, an index of depressive behavior, in the forced swimming test was elongated. These findings suggest that fetal exposure to bisphenol A impairs the sexual differentiation of brain functions and enhance depressive response.

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P2-204 Oxidative stress and microglial activation in the substantia nigra following intrastriatal injection of MPP^+

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The present study aims to study substantia nigra (SN) cells immunohistochemically following intrastriatal MPP^+ injection and to characterize temporal and sequential alterations occurring in both dopaminergic neurons and glial cells. Heme oxygenase-1 (HO-1), a marker of oxidative stress, first appeared in dopaminergic neurons in SN at 1 day postlesion. Subsequently, microglia in SN exhibited morphological changes indicative of activation. At 7 days postlesion, those findings increased severity and a significant reduction in the number of dopaminergic neurons was observed. Additionally, the immunoreactivities of GRP78/Bip increased in the dopaminergic neurons in SN, suggesting that unfolding protein stress is induced during the degenerative process. The present finding suggests that extensive oxidative stress and secondary- induced neuroinflammation play a relevant role in MPP^+- induced retrograde dopaminergic neuron degeneration.

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P2-205 MPTP-induced oxidative damage in dopaminergic neurons of mouse substantia nigra and neuro-protection by MTH1 encoding an oxidized purine nucleoside triphosphatase

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We have previously reported that 8-oxoguanine (8-oxoG) accumulate in the cytoplasm in dopaminergic neurons of substantia nigra of Parkinson disease patients and the expression of MTH1 carrying an oxidized purine nucleoside triphosphatase activity is increased in these neurons. These results suggested that oxidative damage of nucleic acids is involved in the dopaminergic neuron death. We administrated MPTP to wild-type mice and found an increased accumulation of 8-oxoG in the dopaminergic neurons of substantia nigra during the dopaminergic neuron death. MTH1-null mice exhibited an increased dysfunction of TH-positive neurons in striatum after MPTP administration in comparison with wild-type mice. Thus we first time present that MTH1 protects the dopaminergic neurons from increased oxidative stress induced by MPTP administration.

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P2-206 ^{1}H-MRS identify changes in energy metabolism in the striatum of MPTP-treated C57BL/6 mice

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We utilized proton magnetic resonance spectroscopy (^{1}H-MRS) at a field strength of 7 T to identify changes in energy metabolism in the striatum of MPTP-treated C57BL/6 mice. One to 4 systemic injections of 20 mg/kg MPTP dose-dependently caused increase in the peak signal for lactate, which is an index of anaerobic oxidation, occurring at 1.3 ppm chemical shift. The rise occurred as early as 2 h after 4 injections of MPTP, and then returned to the basal level at about 7 h. A selective monoamine oxidase B inhibitor deprenyl or dopamine reuptake inhibitor GBR-12909 completely attenuated the MPTP-induced increases in striatal lactate signal and subsequent a loss of dopaminergic nerve terminals. The present study indicates that ^{1}H-MRS is a sensitive and noninvasive measure of biochemical alteration of the brain in models of Parkinson's disease and the increases in striatal lactate may be associated with dopaminergic neurodegeneration induced by MPTP.

P2-207 Induction of Bip/Grp78 by 6-hydroxydopamine (6-OHDA) is regulated by p38 MAPK activation in human neuroblastoma SH-SY5Y cells

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Several lines of evidence indicate that endoplasmic reticulum (ER) dysfunction may induce neuronal cell injury in Parkinsons disease. Neurotoxin 6-OHDA has been extensively used for making an animal model of Parkinsons disease. We have reported that 6-OHDA induces a highly specific unfolded protein response, which involves ATF6 proteolysis, followed by expressions of ER stress-response genes such as Bip/Grp78 and CHOP/Gadd153. In the present study, we tested whether the expression of Bip/Grp78 by 6-OHDA required a signal transduction pathway mediated by p38 MAPK. Western blot analysis revealed that p38 MAPK inhibitor SB203580 prevented the expression of Bip/Grp78 induced by 6-OHDA. These results provide that p38 MAPK is in part involved in mechanisms underlying the expression of Bip/Grp78 by 6-OHDA.

P2-208 Recovery of dopaminergic neurons damaged by 1-methyl-4-phenylpyridinium in vitro

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Regrowth of surviving dopaminergic (DA) neurons after the administration of 1-methyl-4-phenylpyridinium (MPP^{+}), a potent and selective DA neurotoxin, was examined in vitro. Dissociated cell cultures were prepared from embryonic rat mesencephalon and striatum. MPP^{+} (10μM, 24h) was administered on culture day 7 and succeeding changes of DA neurons were investigated for up to 50 days. There was a 75% reduction of DA neurons in number following administration of MPP^{+}, and surviving neurons exhibited severely deformed terminals. Regrowth of DA terminals was seen 2 weeks after MPP^{+} administration and continued during the observation period. Regrowth was slight in mesencephalic culture lacking striatum, indicating that target tissue is important for recovery. The ratio of number of Calbindin D28k-positive DA neurons was 3 times greater in MPP^{+} treated culture than that in control, suggesting that this protein has protective role against this neurotoxin. In conclusion, surviving DA neurons damaged by MPP^{+} has strong regrowth capability in vitro.

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P2-209 Degradation of Pael-R by human HRD1 involved in ER-associated Degradation (ERAD)

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Endoplasmic reticulum-associated degradation (ERAD) is a system in which, once terminally misfolded, proteins synthesized in the ER are retrotranslocated from the ER to cytosol and degraded by 26S proteasome. We have identified a human homolog (HRD1) of *S. cerevisiae* Hrd1 which is an ER membrane protein that acts as an E3 ligase in ERAD. Accumulation of Pael-R, a substrate of Parkin, has been proposed to lead to neuronal death in autosomal recessive juvenile Parkinsonism (AR-JP). We found that HRD1 was co-localized with Pael-R in the ER and interacted with Pael-R through proline rich region of HRD1. HRD1 ubiquitinated Pael-R *in vitro* and *in vivo*, and Western Blot, pulse-chase, and immunocytochemistry analyses demonstrated that HRD1 can degrade Pael-R. Furthermore, we found that ATF6 and XBP1 which induce HRD1 promoted the degradation of Pael-R. Thus, up-regulation of HRD1 could decrease accumulation of Pael-R in AR-JP, instead of dysfunctional Parkin.

P2-210 Dopamine and levodopa promote polyubiquitination in dopaminergic cells: possible mechanisms in Lewy body formation in Parkinson disease

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In order to clarify how Lewy body (LB) is formed in dopaminergic neurons of Parkinson disease (PD), we analyzed what kinds of factors were related to the growth and increase of intracellular aggregates, using experimental inclusion body model in neurally differentiated PC12 cells. Proteasome inhibition induced the appearance of ubiquitin- and alpha-synuclein (SYN)-positive aggregates, similar to LB. Tyrosine hydroxylase (TH) inhibitor, alpha-methyl-tyrosine (aMT) dramatically reduced the formation of aggregates under proteasome-inhibited condition. Furthermore, aMT also decreased the amount of polyubiquitinated proteins. Neither dopamine nor levodopa enhanced the oligomerization of SYN itself. These data suggest that dopamine and levodopa are enhancers for LB formation by producing aggregates of the ubiquitin-ubiquitin complex in dopaminergic neurons.

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P2-211 Effect of mild mitochondrial dysfunction on primary cultured mouse mesencephalic neurons

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Oxidative stress and impairment in protein degradation have been suggested as possible causes leading to the selective loss of dopaminergic neurons (DA neurons) in SNpc in Parkinson's disease. In the present study, we examined the effects of mild mitochondrial dysfunction induced by rotenone at low-doses (1pM-1nM) in primary cultured ventral mesencephalic neurons including DA neurons. After exposure to 100 pM rotenone for 24h, survival rates of DA neurons and total neurons were 49% and 92% of control, respectively. Similar exposure to rotenone at 10pM did not induce cell death, but induced neurite retraction only in TH-positive DA neurons. Other neurons were without apparent morphological change, indicating the selective vulnerability of DA neurons. Alternations in gene expression under these conditions are being investigated using our original oligo DNA microarray to analyze cellular differences in the response to rotenone.

P2-212 Thalamic β-band rhythmic activities contingently induce the parkinsonian rigidity

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During the thalamotomy for Parkinson disease (PD), excess β-band rhythmic activities are observed in the thalamic ventroanterior/ventrolateral nuclei (VA/VL). We analyzed conditions in which these activities were related to the surface electromyograms (EMGs) of rigid limb muscles. Six PD patients gave their informed consent to undergo the surgery. With a bipolar concentric semi-microelectrode we recorded and filtered the VA/VL activities as local field potentials (LFPs) and multiple unit spikes (MUSs). We found that the VA/VL β-band LFPs (β-waves) became transiently synchronized with the complex spikes in individual muscle EMGs in conditions such that the patients were alert with increased or even rigid muscle tone. The negative-going deflection of β-waves with concurrent MUSs preceded the spike EMGs. Thus, the excess VA/VL β-band rhythmic activities contingently induce the parkinsonian rigidity presumably by driving individual or a population of muscles to contraction through the thalamocortical pathways.

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P2-213 Upregulation of cerebellar degeneration-related genes in HEK293 following stable expression of the cellular prion protein PrPC

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Mice having a cytoplasmic accumulation of PrPC showed cerebellar neurodegeneration, although its mechanism remains unknown. To identify the genes whose expression is affected by PrPC, we established a stable PrPC-expressing HEK293 cell line named P1. The gene expression profile on a 12,814 cDNA microarray was studied in P1 and PrPC-non-expressing F1. Among 33 genes expressed differentially between P1 and F1, 14 genes were involved in neuronal and glial function, 6 in ECM production, and 2 in the complement cascade. P1 showed upregulation of brain-specific protein phosphatase 2A beta, a causative gene of SCA12, and cerebellar degeneration-related autoantigen CDR34, and downregulation of secreted frizzled-related protein-1, a dominant negative receptor for Wnt. These results indicate that a persistent accumulation of PrPC induced dysregulation of various genes important for cerebellar neuronal function which is responsible for PrPC-induced neurodegeneration in the cerebellum.

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P2-214 Hsp90β modifies the conformation of recombinant mouse prion protein *in vitro*

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In mammals, prions reproduce by recruiting the normal cellular isoform of the prion protein (PrP^C) and by stimulating its conversion into the pathological conformational isoform (PrP^{Sc}). The conversion process is thought to be required for another factor(s). In order to establish an *in vitro* assay system for the identification of the factor(s), we first constructed and purified *E. coli*-expressed mouse PrP^C as a substrate protein (recPrP). The highly-purified recPrP was "correctly" refolded by 1 M arginine solution. Using this substrate, we found that the extract of neuroblastoma cells contained a conformation modifying activity of recPrP due to the exposure of trypsin sensitive sites. After the several steps of chromatography, we purified the activity to homogeneity and found that it was Hsp90β isoform from mass spectrometric analysis.

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P2-215 Molecular function of nuclear-cytoplasmic shuttle proteins which bind to the novel *cis*-element in the *HD* gene promoter

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Huntington's disease (HD) is a neurodegenerative disease. The study of the conditional HD mouse gave a new aspect of the HD pathogenesis by means of perpetual expression of the mutated *HD* gene. However, molecular base of the transcriptional regulation of the *HD* gene in neurons has not been understood as yet. To delineate the transcriptional regulation for the *HD* gene, we identified two proteins, HDBP1 and HDBP2, which bind to the *HD* promoter region. In this study, we showed that HDBP1 and HDBP2 shuttled between cytoplasm and nucleus, and the C-terminal conserved regions of both proteins were responsible for binding to the unique 7-bp sequence (GCCGGCG) in the *HD* promoter region. Interestingly, reporter gene assays demonstrated that the 7-bp sequence was essential for the *HD* promoter activity in IMR32 cells, but not in HeLa cells. Our study implies that HDBP1 and HDBP2 in conjunction with 7bp *cis*-element trigger off the *HD* gene expression in neuronal cells.

P2-216 Analysis of mutated GFAP dynamics

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Alexander disease is a falal neurological disorder caused by heterozygous mutation in GFAP. The pathological hallmark is the presence of astrocytic inclusion body called Rosenthal fibers, which contain GFAP. To understand the pathophysiology of this disease and utilize those results to the understanding of normal astrocytic function in vivo, we studied the dynamics of mutated GFAP in culture. We expressed wild type or mutated human GFAP gene in mouse primary astrocytes using adenoviral vectors and detected them using anti human GFAP specific antibody. Wild type GFAP formed normal appearing filaments with thick bundles or fascicular aggregations, whereas mutant GFAP did not form the fibrous structure but occasionally formed bulging bodies. When the cells expressing mutant GFAP were treated with proteasome inhibitor, the number of the cells harboring bulging bodies increased. These findings suggest that 1) mutant GFAP is incapable of forming the filament, 2) the formation of inclusion body caused by mutant GFAP is dependent on the proteosomal activity.

P2-217 Analysis of neurofilament-L mutations linked to Charcot-Marie-Tooth disease on filament assembly

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Charcot-Marie-Tooth disease (CMT) is one of the most common hereditary peripheral neuropathies. Mutation of NFL gene is recently identified to be a cause for some forms of CMT. NFL is a major subunit of neurofilaments (NFs), neuron-specific intermediate filaments. In order to elucidate the mechanism by which the NFL mutation induces axonal degeneration, we analysed the effect of CMT mutations (N97S and A148V) of NFL on neurofilament assembly. We expressed each NFL mutant in SW13 cells and observed filament organization by confocal microscopy. NFL-N97S distributed diffusely throughout the cell without filament formation, whereas NFL-A148V did form filaments. We speculate that Asn-97 has an important role in filament formation. To analyse their ultrastructure in detail, we are going to purify the mutant proteins from E. coli and observe assembled filaments in vitro by electron microscopy.

P2-218 Perturbed calcium regulation of endoplasmic reticulum in a peptide-delivery model of polyglutamine-induced neuronal death

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The mechanism by which polyglutamine (polyQ) expansion causes common features of neuronal death remains unclear. By delivering polyQ expansions directly into cultured sympathetic neurons, we have shown that early apoptotic signals are replaced by necrosis via perturbed Ca^{2+} homeostasis. Here we have examined Ca^{2+} signaling of the endoplasmic reticulum using cultured cortical neurons. Neurons left untreated or treated with expanded polyQ tracts were permeabilized and preloaded with $^{45}Ca^{2+}$. The neurons were then stimulated either with caffeine or IP_3, and release of $^{45}Ca^{2+}$ from caffeine- or IP_3-sensitive stores, respectively, was measured. Ca^{2+} release from IP_3-sensitive stores remained unaltered upon treatment with the polyQ tracts, whereas that from caffeine-sensitive stores was impaired. Moreover, treatment with dantrolene rescued these neurons from neurodegeneration. These findings suggest that polyQ tracts altars Ca^{2+}-induced Ca^{2+} release leading to perturbed calcium homeostasis.

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P2-219 Decreased expression of hypothalamic neuropeptides in Huntington disease transgenic mice with expanded polyQ-EGFP fluorescent aggregates

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We generated transgenic mouse lines expressing expanded polyQ-EGFP. The expanded polyQ-EGFP cytoplasmic and nuclear aggregates appeared almost simultaneously and increased progressively with age in various regions of the brain, such as striatum, hippocampus and hypothalamus of transgenic mice. We carried out a high-density oligonucleotide array analysis, followed by TaqMan RT-PCR and in situ hybridization. In addition to the already-reported transcriptionally down-regulated genes in striatum of R6/2 mice, we found expressions of some hypothalamic peptides mRNAs, such as oxytocin and vasopressin were down regulated in HD190QG brains without observing significant loss of hypothalamic neurons. These results indicate that striatum and hypothalamus were more susceptible to aggregate formation in those mice and that may down-regulate expression of specific genes in both areas.

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P2-220 Identification of Ubiquitin-interacting proteins in the purified polyglutamine aggregates

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[Purpose] In polyglutamine (polyQ) disease, one of the main features is the presence of intracellular ubiquitin (Ub)-positive aggregates (Agg). It is known that the components of aggregate-interacting proteins (AIPs) reflect either the cellular defense against the toxicity of polyQ Agg or the cellular machinery affected by polyQ Agg. The purpose of our study is to clarify the whole AIP components. [Method] We used a Neuro2a cell line that is stably transfected with exon 1 of huntingtin with 150 (CAG)-EGFP-NLS and nuclear polyQ Agg was induced by ecdysone. Nuclear polyQ Agg were then purified and analyzed by mass spectrometry.[Result] We have identified ubiquilin 1, ubiquilin 2 and Tollip as new AIPs. We also confirmed that these proteins associated with intranuclear inclusions in a mouse model of Huntington disease.

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P2-221 Analysis of interaction between Dorfin and VCP, forming ubiquitylated inclusions in neurodegenerative disorders

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Dorfin is a ubiquitin ligase(E3), which can ubiquitylate mutant SOD1, a causative gene of familial ALS. Dorfin is located in ubiquitylated inclusions in various neurodegenerative disorders. High through-put analysis using masspectrometry revealed that VCP was a Dorfin-associated protein. VCP interacted with Dorfin in vitro and in vivo. These two proteins were colocalized in aggresomes in HEK293 cells and UBIs in the affected neurons of ALS and PD. A dominant-negative form of VCP deteriorated the E3 activity of Dorfin. Overexpression of Dorfin inhibited the ERAD pathway in HEK293 cells. Our results indicated that Dorfin and VCP functionally regulated each other through their interaction and their functional properties may be related with the process of ubiquitin-positive structures in neurodegenerative disorders.

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P2-222 Mitochondrial dysfunction in PQBP-1 transgenic mice

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PQBP-1 interacts with the polyglutamine disease gene products and plays a crucial role in the pathology of these diseases such as spinocerebellar ataxia type-1. Our previous findings from transgenic mice overexpressing human PQBP-1 suggest that excessive action of PQBP-1 causes a late-onset and gradually progressive motor neuron disease like phenotype, and loss of neuronal cells in the cerebellum as well as that of spinal motor neurons. To clarify the mechanism on the neuronal cell loss, we investigated the change of gene expression at ventral spinal cord in PQBP-1 transgenic mice by microarray analysis. Surprisingly, a half of the affected genes turned out to be those transcribed in mitochondria. Electronmicroscopic analysis showed that mitochondrial ultrastructure is changed in spinal motor neurons. These results suggest that mitochondrial dysfunction underlies the neuronal death by PQBP-1 overexpression.

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P2-223 Per oral plant-lycopene administration can delay the progression of paralysis of the limbs in motor neuron degeneration(*Mnd*)mutant mouse during postnatal growth

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The *Mnd* mouse shows an ascending motor paralysis of limbs and premature death.Per oral daily administration of an antioxidant-lycopene-containing diet to *Mnd* mouse recovered both descending enzymic activities of cytochrome oxidase(CO), a key enzyme in aerobic energy production metabolism,and superoxide dismutase(SOD), a detoxicating enzyme for cytotoxic reactive oxygen radicals,in the CNS tissues during postnatal growth.Also,lycopene uptake is effective to suppress the continuous rise of transglutaminase(TGase), a marker of tissue degeneration process in *Mnd* mouse during growth.Progression of motor paralysis in *Mnd* mouse was attenuated by lycopene uptake,and the mouse survived rather longer period with less disturbances of limbs movements.These data suggest that lycopene uptake is quite effective for prevention of the progression of motor disabilities in *Mnd* mouse.

P2-224 NEDL1, a novel E3 ubiquitin ligase for Dishevelled-1 (Dvl-1), targets mutant SOD1 to form intracellular aggregates in familial amyotrophic lateral sclerosis (FALS)

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About 20% of FALS arise from germline mutations in the *SOD1*. The neuronal ubiquitin ligase, NEDL1, physically binds to TRAP-δ, and also binds and ubiquitinates mutant but not wild-type SOD1 proportionately with the disease severity caused by that particular mutant. Immunohistochemically, NEDL1 is present in the central region of the Lewy body-like hyaline inclusions in the spinal cord ventral horn motor neurons of both the FALS patients and the mutant SOD1 tg mice. Mutant SOD1 also interacts with Dvl-1 in the presence of NEDL1 and inhibits its function. Our results suggest that an adverse interaction between misfolded SOD1, NEDL1, TRAP-δ and Dvl-1 forms ubiquitinated protein complex to be included in potentially cytotoxic protein aggregates, leading to motor neuron death in FALS.

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P2-225 An ALS rat model by AMPA receptor-mediated excitotoxicity

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Based on the AMPA receptor-mediated neuronal death hyphothesis of ALS, we aimed to develop as yet unavailable ALS animal model by continuous infusion of kainic acid (KA) into rat spinal subarachnoid space. Motor function of the KA-infused rats measured with a rotarod was significantly decreased, which was accompanied with morphological changes of spinal motor neurons after 4 or 8 weeks of KA infusion. They displayed selective motor dysfunction with both neuropathological changes and the upregulation of GluR3 subunit mRNA expression selective in motor neurons in KA-treated groups. Addition of cyclothiazide exaggerated the pathological change. The GluR3 upregulation may increase the proportion of GluR2-lacking (ie. Ca^{2+}-permeable) AMPA receptors, thereby inducing the selective vulnerability of motor neurons to KA. This model rat may be useful in the investigation of mechanism underlying motor neuron death including that of ALS.

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P2-226 Expression of serine protease protease M/neurosin in demyelination and remyelination

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Protease M/neurosin is expressed by mature oligodendrocyte in the CNS. Therefore, protease M/neurosin may be important to the processes occurring after the maturation of oligodendrocytes such as myelination or turn over of the protein in the myelin. In this study, we investigated the contribution of protease M/neurosin in demyelination and remyelination using the oligodendrocyte-specific toxin, cuprizone (bis-cyclohexanone oxaldihydrazone). Luxol fast blue staining demonstrated demyelination in the corpus callosum by 7 day treatment with 0.7 % cuprizone. Protease M/neurosin mRNA expression decreased at 7 day treatment of cuprizone compared with control, but increased at 7 day after remyelination. Protease M/neurosin mRNA expression in myelin basic protein-deficient shiverer mice brain was not detected by RT-PCR analysis. These observations indicate that protease M/neurosin have important roles in the turnover of the protein in the myelin during demyelination and remyelination.

P2-227 GeneChip analysis (Part. 2) of mouse brain stimulated by repetitive Transcranial Magnetic Stimulation

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rTMS is a noninvasive technique to induce electric current in the brain and is supposed to be beneficial for depression, schizophrenia and neurodegenerative disorders. But there are few reports about gene expression pattern induced in the brain by rTMS. So we performed GeneChip analysis on mouse brain stimulated by chronic rTMS at 24hrs after 20days stimulation. Increase 70 and decrease 17 genes were identified. c-fos and junB were decreased opposite to the previous report. Then we checked c-fos and junB mRNA after stimulation of rTMS on mouse brain . Consistent with the previous report, transient increase of c-fos and junB were confirmed at 1hr just after 1 times stimulation, but decrease at 24hrs after 20 days chronic treatment. On the other hand, at 24 hrs after 20 days stimulation, mRNA related to monoaminergic system were changed in GeneChip results and the results genes were confirmed by TaqMAN assay. These results indicate that chronic rTMS modulate monoaminergic system.

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P2-228 A role of FK506-related immunophilines for neuroprotective properties FKBP-binding immunophilin ligands

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We focused on immunophilin isoforms in order to clarify the neuroimmunophilin, which were identified as targets for the immunophilin ligands (IPLs) to elicit the neuroprotective effect. Although the expressions of five FKBPs mRNAs were detected in both the SH-SY5Y and U251 cell lysates, the FKBP12 mRNA expression was detected in only the SH-SY5Y cells, but not the U251 cells. However, we found that the SH-SY5Y and U251 cells were equipotent in the intensity of cellular protection of FK506 (immunosuppressive) and GPI1046 (non-immunosuppressive), indicating that the protective effect and GSH activation of FK506 and GPI1046 had little need to bind FKBP12. Therefore, we concluded that the neuroprotective and antioxidant properties of IPLs are independent on the FKBP12 pathway.

P2-229 Reversal of Orofacial Dyskinesia by FK506

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Tardive dyskinesia caused by long-term use of neuroleptics, is associated with oxidative stress. FK506 reported to be an effect antioxidant , was tested in reversal of Tardive dyskinesia.Vacuous chewing movements (VCMs) in rats was, induced by daily administration of haloperidol (1.0 mg/kg) for a period of 21 days. Animals with VCMs were given FK506 for a period of 4 weeks and behavioral scoring was done every week before administration of FK506. Chronic haloperidol (1.0 mg/kg for 21 days) treatment significantly induced VCMs and tongue protrusions in rats and FK506 (0,5-3mg/kg for four weeks) significantly reversed VCMs and tongue protrusions. Biochemical analysis revealed that haloperidol treatment significantly induced lipid peroxidation, decreased glutathione (GSH), levels in the brains of rats. FK506 (0,5-3 mg/kg for 4 weeks) significantly reduced lipid peroxidation and restored the GSH, levels. The results of the present study clearly indicated that FK506 has a protective role in reversing haloperidol-induced orofacial dyskinesia.

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P2-230 Modulation by stress of BrdU incorporation in adult mouse dentate gyrus

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Adult male mice were intraperitoneally injected with 5-bromo-2'-deoxyuridine (BrdU; 50 mg/kg) every 12 h twice for labeling cells newly synthesizing DNA, followed by perfusion with 4% paraformaldehyde (PA) 12 h after the last injection and subsequent dissection of coronal frozen sections for immunohistochemical analysis. Immunoreactive BrdU was exclusively detected as clusters of plural cells in the hippocampal dentate gyrus (DG) and subventricular zone. Animals were stressed with immobilization in a restraint cage immersed in water at 25 °C up to the individual clavicle for 3 h, before and after the first injection of BrdU. Stress for 3 h induced a significant decrease in the number of cells labeled by BrdU in the DG irrespective of the timing of BrdU injection. These results suggest that stressful manipulation could modulate the proliferative activity of neural progenitor cells expressed in the DG of murine hippocampus under adult neurogenesis.

P2-231 Relationship between the dynamics of working memory and the emergence of cognitive deficits in psychiatric patients: A computational study

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A number of studies indicate that the prefrontal cortex is intrinsically linked to working memory and that dopamine critically modulates working memory activity. Goldman-Rakic and her colleagues modeled the biphasic modulation of working memory function by dopamine; there is an optimal level of D1 receptor occupancy for efficient physiological signaling and performance. According to their model, we constructed an electrophysiological circuit model for spatial working memory. The results of our simulation with this model show that the two phases have different electrical properties. In this study, we investigate these differences in the characteristics of working memory and discuss what "hypofrontality" in psychiatric disorders such as schizophrenia, Parkinson's disease, and attention deficit-hyperactivity disorder is.

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P2-232 Repeated treatment with phencyclidine impairs latent learning in mice: association with the cortical Ca2+/calmdulin kinase II

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To investigate the effect of phencyclidine (PCP) on latent learning, we used a one-trial water finding task in mice. The trained mice showed significantly shortened finding latency in the test trial in comparison with the naïve mice that had not been trained. When mice were administered PCP repeatedly, the finding latency and CaMKII activation were significantly prolonged and decreased respectively in comparison with that in the saline-treated mice. Clozapine ameliorated both impairments in the PCP-treated mice. This ameliorated effect of clozapine was antagonized by D4 receptor agonist. These finding suggest that D4 receptor antagonism are involved in the ameliorated effect of clozapine, and are mediated through CaMKII activation.

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P2-233 Effects of systemically administered phencyclidine can be mimicked by the local perfusion in the ventral hippocampus

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We examined effects of local application of phencyclidine (PCP), a psychotomimetic drug that induces schizophrenia-like symptoms in man, into the ventral hippocampus (vHIP) on firing activity of PFC neurons in freely moving rats. PCP locally perfused (2 mM) into the vHIP increased spontaneous discharges of PFC neurons during perfusion with augmentation of locomotor activity. Local application of a more selective NMDA receptor antagonist, MK801, to vHIP neurons under anesthesia increased the spontaneous firing rates of most neurons directly projecting to the mPFC, whereas no mPFC neurons exhibited excitatory responses to locally applied MK801. The present results indicate that tonic excitatory inputs from the vHIP to the PFC may initiate development of behavioral abnormalities.

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P2-234 Antidepressants enhance BDNF-induced release of glutamate in cultured cortical neurons.

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Alteration of BDNF-regulated neuronal function has been implicated in the pathogenesis of depressive disorder. We investigated effects of antidepressants (imipramine and fluvoxamine) on BDNF-induced glutamate release in cultured cortical neurons. Dissociated cortical neurons from postnatal 2-3 days-old rats were maintained for 5 days prior to application of antidepressants. We found that BDNF-induced glutamate release was enhanced after treatment with antidepressants for 48 hours. Previously, we reported that the BDNF-induced glutamate release occurs in a PLC-γ/IP3/Ca pathway-dependent manner. In accordant with this, antidepressants potentiated the BDNF-elicited increase in intracellular Ca. Furthermore, imipramine treatment increased endogenous expression of PLC-γ and enhanced its activation stimulated by BDNF, suggesting that antidepressants reinforce the BDNF-dependent neuronal function via up-regulation of PLC-γ pathway.

P2-235 Effect of co-administration of sulpiride and fluvoxamine on extracellular monoamine levels in rat prefrontal cortex

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The antipsychotic sulpiride (SUL) acts as antidepressant at low doses, and combination of SUL with selective serotonin (5-HT) reuptake inhibitors is clinically used for treatment-resistant depression. In this study, we examined the effect of co-administration of SUL and fluvoxamine (FLV) on *in vivo* release of 5-HT, dopamine (DA) and noradrenaline (NA) in rat prefrontal cortex and striatum. SUL and FLV alone did not affect DA release in the prefrontal cortex, while their combination caused a significant increase in DA release. SUL alone did not affect cortical 5-HT and NA release, while FLV alone caused a marked increase in 5-HT release and a slight increase in NA release. SUL did not affect the FLV-induced 5-HT and NA release. Co-administration of SUL and FLV did not affect DA release in the striatum. A possible neurochemical basis for clinical efficacy of the combination therapy is discussed.

P2-236 The antidepressant-like effect of selegiline hydrochloride in the mouse forced swimming test

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Selegiline hydrochloride (HCl), used in the treatment of Parkinson's disease, is a selective inhibitor of monoamine oxidase type-B. We investigated the effect of selegiline HCl on forced swimming test and locomotor activity in mice. Mice were administered selegiline HCl 1 h before behavioral test. Selegiline HCl significantly reduced the immobility time at dose of 10 mg/kg, which had no effect on locomotor activity in mice. A dopamine D1 receptor antagonist reduced the antidepressant-like effect of selegiline HCl. In contrast, dopamine D2 receptor antagonist, serotonergic receptor antagonist and noradrenergic receptor antagonist had no effect. These results suggest that selegiline HCl exerts the antidepressant-like effects on forced swimming test. These actions of selegiline HCl on forced swimming test may be attributed to a stimulation of dopamine D1 receptor.

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P2-237 Genomic polymorphism of the peripheral-type benzodiazepine receptor (PBR) and its relation to the trait anxiety

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PBR expression in platelets is correlated with trait anxiety in normal human subjects (Nakamura et al., Psychopharmacology 162: 301-303, 2002). We have further studied this correlation at the genomic level in 51 normal human subjects. Genomic DNA was prepared from lymphocytes, and sequence variations on the PBR gene were analyzed within exon 4. Single nucleotide substitution G485A (Arg162His) was identified in the coding region. Seventeen subjects (33%) with G485A hetero-zygosity showed lower densities of platelets PBR ($p<0.01$) and lower scores of the trait of anxiety ($p<0.05$) as compared with 31 subjects (61%) with G485G homo-zygosity. The results suggest that the genomic polymorphism of PBR may be associated with the trait of anxiety.

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P2-238 Effects of anxiogenic drugs on BTG2 mRNA in the mouse brain

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A variety of drugs produce anxiety, which include FG7142, yohimbine and mCPP. Common substrates and pathways may be involved in the induction of the symptom. BTG2 plays a role in th regulation of cell cyle. We investigated effects of FG7142 (20 mg/kg), yohimbine (5 mg/kg) and mCPP (2 mg/kg) on the BTG2 expression in the adult mouse brain. The mice were killed 1hr after the injection, and the brain areas (neocortex, hippocampus and striatum) were rapidyly removed in the cold. Total RNA of the tissue was extracted by Qiage RNeasy Midi system and single-strand cDNA was then synthesized using SuperScript Preamplification system. The resulting cDNA was used for quantitative RT-PCR analysis by means of LightCycler system. FG7142 and yohimbine induced a significant increase in the BTG2 mRNA in the three areas, while mCPP produced a marked increase in the striatum. The present study indicates that the drug-induced anxiety can be coupled to the regulation of cell cycle in the brain.

P2-239 Peripheral Administration of Interleukin-1α To Rat Pups Impairs Cognitive /Behavioral Development

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Abnormal cytokine signals induced by maternal inflammation, viral infection, and obstetric complication are implicated in psychiatric diseases of neurodevelopmental origin including schizophrenia. In the present study, we evaluated the potential contribution of peripheral Interleukin-1α activities to abnormal behavioral/cognitive development or psychiatric diseases using rats. Interleukin-1 was subcutaneously administered to neonatal rats. After adolescence, rats exhibited cognitive/behavioral abnormalities in startle response, prepulse inhibition. Neonatal interleukin-1α treatment resulted in abnormal monoamine metabolism and responses at adult stages.These animal experiments suggest that, during infantile stages, inflammatory interleukin-1 activities in the periphery influence brain development and disrupt future psychobehavioral and/or cognitive function.

P2-240 Enhancement of hippocampal neurogenesis by lithium augmentation

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Decreased volume and functional impairment of the hippocampus due to stress-induced hippocampal cell loss and/or repression of neurogenesis may underlie the pathophysiology of depression. In international treatment guidelines and algorithms, lithium augmentation which lithium is used in addition to an antidepressant is considered a first-line treatment strategy for refractory depression. In this study, we investigated the effect of lithium augmentation on neurogenesis in the adult hippocampus. Neurogenesis was assessed using bromo-deoxyuridine (BrdU) incorporation assay. Male Sprague-Dawley rats that received daily fluvoxamine injections for 2 weeks (30mg/kg) showed increased numbers of BrdU-positive proliferating cells in the dentate gyrus relative to control. Moreover, they received same injections with 0.2% LiCO3 for 3 weeks showed more BrdU-positive cells. These results indicated that lithium augmentation enhanced the antidepressant effects on neuorgenesis.

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P2-241 Subchronic senstization induced by a low dose of methamphetamine develops with the progress of neurotoxic morphological changes in the medial prefrontal cortex of the rat

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The present study aimed to determine whether the development of behavioral sensitization induced by a relative low dose of methamphetamine (MAP) is associated with neurotoxic morphological changes produced in mFC. Rats were daily injected with MAP (5mg/kg, i.p.) for 12 days. Sensitization of stereotypic behavior reached a basic plateau level on day 4, and then a little higher levels on day 6 and day 12. Morphological study showed that tyrosine hydroxylase immunoreactive fibers reduced by about 30% in density in mFC on day 4. Many TUNEL positive cells were observed in mFC on day 12. In summary, the development of behavioral sensitization induced by MAP roughly corresponded in time-course with the progress of the neurotoxic changes produced in mFC.

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P2-242 Increased cell proliferation in the adult mouse hippocampus following chronic administration of mGluR2/3 antagonist (MGS0039) with antidepressant-like effects

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Various pharmacological and histochemical studies have implicated the role of group II glutamate receptors (mGluR2/3) in the control of emotional states, including depression and anxiety. Recent studies suggest that the behavioral effects of chronic antidepressant treatment are mediated by the stimulation of neurogenesis in the hippocampus. In the present studies, we examined the effects of MGS0039, a potent and selective antagonist of mGluR2/3, on cell proliferation in the adult mouse hippocampus. MGS0039 was administered chronically to male ICR mice. Immunohistochemical analyses after BrdU injections for labeling the dividing cells revealed that chronic MGS0039 treatment enhanced BrdU-positive cells in the dentate gyrus. Moreover, MGS0039 had dose-dependent antidepressant-like effects in two despair models of depression.

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P2-243 Behavioral and molecular changes of olfactory bulbectomized rats: effects of a sigma$_1$ receptor agonist, SA-4503

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In the present study, olfactory-bulbectomized (OB) rats exhibited not only abnormal hyperactivities in response to acute stress resembling the psychomotor agitation symptom in depressive patients, but also anxiety behaviors and cognitive deficit resembling the cognitive disfunction observed especially in comorbid anxiety-depressive patients. OB rats also showed the decrease of phosphorylation ratio of CREB and CaMK II, and protein content of GAP-43 and NMDA receptor NR1 subunits in several regions of limbic system, some of which were observed in depressive patients and remitted by the typical antidepressant treatment. Several behavioral deficits and molecular abnormities were attenuated by chronic treatment with SA-4503. These findings suggest that the sigma$_1$ receptor agonist SA-4503 has antidepressive and anxiolytic effects, and may be effective for poor responsers of typical antidepressants.

P2-244 Synaptic plasticity in LA and fear conditioning in PTSD model rats

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Memory disturbances, such as flashback, in PTSD patients resemble to conditioning. Using putative PTSD model rats, we studied the effects of strong stress on synaptic plasticity in amygdala and fear conditioning. SD male rats were exposed to single-prolonged stress and analyzed immediately (acute), 1 day (1D) or 1 week (1W) after the stress exposure. Slice field recordings indicated that LTP in lateral nucleus was persistently attenuated in SPS rats. Acute and 1D groups indicated significant suppression of I/O relationship, while there was no difference in paired-pulse responses. Behaviorally, contextual fear conditioning and startle response were enhanced in 1W rats, but not in 1D rats. There was no difference in pain threshold. The apparent discrepancy between synaptic plasticity and fear conditioning should indicate peculiar effects of SPS, which might reflect pathophysiology of PTSD.

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P2-245 Circuit simulation of the mesocorticolimbic system

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Dopamine (DA) plays critical roles in cognitive functions. Disruptions in DA cells activity impair these functions and induce clinical conditions such as schizophrenia and Parkinson's diesease. To investigate the mechanisms of these processes, we constructed a computational model of the network consisting of the prefrontal cortex (PFC), the nucleus accumbens (NAc) and the ventral tegmental area (the mesocorticolimbic system). By the circuit simulation of this system, we suggest that the increase of inputs from the PFC enhances the cortical DA release but decreases the DA release in the NAc. The reduction of the NAc DA level is due to the GABAergic transmission within the mesolimbic structure. The increase in the DA release results in decreses in the neuronal activities of the PFC and NAc. This simulation, therefore, suggests an unsymmetrical modulation of DA levels between the mesocortical and the mesolimbic systems.

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P2-246 Cerebral Blood Flow Change following body weight recovery in Anorexia Nervosa: A SPECT study analysis using statistical parametric mapping

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Some studies revealed the changes of regional cerebral blood flow (rCBF) in patients with Anorexia Nervosa (AN). However, most of them were assessed rCBF of AN patients only at low body weight using region of interest method. We performed single photon emission computed tomography (SPECT) to 8 female AN patients following body weight recovery. SPECT images analysis were performed with a voxel-based analysis using statistical parametric mapping (SPM) software. Compared with AN at low body weight, AN after body weight recovery showed significant hyperperfusion in precuneus. The rCBF of precuneus was also correlated to Eating Disorder Inventory subscale 7 (Interoceptive awareness). This change of rCBF may reflect the cognitive function and psychological changes.

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P2-247 Activation of medial prefrontal cortex neurons by MK801 is mediated via non-NMDA glutamate receptors

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Recent studies show that long-lasting activation of the medial prefrontal cortex (mPFC) induced by systemic NMDA-antagonists like PCP or MK801 plays a pivotal role in development of behavioral abnormalities. However, a receptor or receptors mediating such activation is not yet specified. The present study examined effects of local application of CNQX, an AMPA/kainate glutamate receptor antagonist, on elevated firing activity of mPFC neurons after systemic MK801 administration in anesthetized rats. Once clear tonic activation of mPFC neurons was established after MK801 injection (0.5 mg/kg, i.p.), CNQX was ejected with nitrogen gas pressure. Locally applied CNQX (25 μM, 30 nl/min) reduced MK801-induced elevation of firing activity to the baseline level. These results suggest that NMDA antagonist-induced activation of mPFC neurons is mediated via AMPA/kainate glutamate receptors.

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P2-248 Genetic Analysis of the human striatum-enriched CalDAG-GEFI gene with Japanese schizophrenia

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We previously reported the isolation of striatum-enriched novel guanine nucleotide exchange factor (GEF) that has both calcium (EF-hand) and diacylglycerol (DAG) binding domains. CalDAG-GEFI mRNA is expressed almost exclusively in the brain and hematopoietic system. It is shown that CalDAG-GEFI proteins are localized at the synaptic terminals of GABAergic output neurons in the striatum. Since the dysregulation of the striatal GABAergic output neurons are implicated in the pathophysiological mechanisms of schizophrenia, CalDAG-GEFI gene can be a good candidate for molecular studies of schizophrenia. We have genotyped 8 SNPs in the *schizophrenia patients (n=214) and normal controls (n=236) to evaluate* association between schizophrenia phenotype and these markers using direct sequencing and single nucleotide extension methods. No significant differences in the allele frequencies of the 4 SNPs were found. Four other SNPs were monomorphic in our sample.

P2-249 DISC1 interacts with a centrosomal protein, kendrin

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Disrupted-In-Schizophrenia 1 (DISC1) was identified as a novel gene disrupted by a translocation that segregated with major mental diseases. We screened a human brain cDNA library using the yeast two-hybrid system and identified a giant centrosomal protein, kendrin/pericentrin-B, as an interactor of the DISC1 protein. Amino acids 446-533 of DISC1 were essential for the interaction with kendrin. Immunocytochemical analysis revealed the colocalization of *DISC1* and *kendrin to the centrosome. Moreover, we* analysed *in situ* localization of kendrin mRNA in mouse brain. The pesent study indicates that DISC1 localizes to the centrosome via interaction with kendrin, suggesting the possible involvement of DISC1 in the pathophysiology of mental diseases due to its putative effect on centrosomal function.

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P2-250 Search for dysbindin binding partners by yeast two-hybrid systemSearch for dysbindin binding partners by yeast two-hybrid system

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Schizophrenia is a debilitating psychiatric disorder with a lifetime morbidity risk of 1%. Genetic factors are known to be important in the etiology of schizophrenia, and recent genome-wide linkage studies have revealed several susceptibility genes for schizophrenia, including dysbindin at 6p22.3, neuregulin1 on chromosome 8p-p21, and G72/G30 at chromosome 13q34. Dysbindin is an evolutionarily conserved protein with one coiled-coil secondary structure, and a novel member of Duchenne Muscular Dystrophy Complex (DPC), which appears to be involved in signal transduction at postsynaptic densities (PSD). However the precise mechanism how dysbindin functions at PSD remains unknown. In the present study, we report the result of yeast two-hybrid screen for dysbindin binding partners using human brain cDNA library.

P2-251 Identification of a novel binding partner for schizophrenia susceptibility gene dysbindin

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Dysbindin, emerging as a major schizophrenia susceptibility gene located in region 6p22.3, encodes an evolutionarily conserved 40kDa coiled-coil-containing protein. Dysbindin was originally identified in yeast two-hybrid screen for interacting proteins with beta-dystrobrevin, a member of Duchenne Muscular Dystrophy Complex (DPC) which is a component of synapses in muscle and brain. In the brain, dysbindin immunoreactivity is detected primarily in synaptic terminals in the cerebellum and hippocampus (Benson et al. 2001). To better understand the role of dysbindin in the brain, we performed a yeast two-hybrid screen for dysbindin binding partners using human brain cDNA library. Here we report a novel binding partner of dysbindin, which is involved in a number of cellular processes.

P2-252 Dysbindin, one of schizophrenia susceptibility genes, is involved in protein trafficking

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Dysbindin, originally identified as interacting protein with beta-dystrobrevin, is an evolutionarily conserved protein with one coiled-coil secondary structure, and a novel member of Duchenne Muscular Dystrophy Complex (DPC), which is involved in signal transduction at postsynaptic densities (PSD). *Recent reports have revealed that dysbindin is one of susceptibility* genes for schizophrenia. Dysbindin is also reported as one of causative genes for Hermansky-Puldak syndrome (HPS-7), which is a genetically heterogeneous diseases characterized by oculocutaneous albinism, prolonged bleeding and pulmonary fibrosis due to abnormal vesicle trafficking to lysosomes and related organelles. In the screening for dysbindin binding partners by yeast two-hybrid system, we found that one of sorting factors interacts with dysbindin, which suggest a cross-talk between schizophrenia and protein trafficking disruption in the central nervous system.

P2-253 Enriched environment influences depression-related behaviors and hippocampal neurogenesis

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We investigated the possible protective effect of enriched environment (EE) against the development of depression-related behaviors. Young adult mice were raised in EE for 5 weeks and then subject to behavioral tests. In the open field test, EE mice exhibited decreased locomotion and longer time spent in the center, compared to control mice. EE mice showed longer duration per contact in the social interaction test, and reduced immobilization in the forced swim test. As hippocampal neurogenesis might contribute to the pathophisiology of depression, we examined neurogenesis quantified by BrdU-labelled immunohistochemistry. We found increased survival of BrdU-positive cells in EE mice. Our results suggest that EE facilitates emotional stability, reduced anxiety and stress resistance, and that these behaviors may be related to enhanced neurogenesis.

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P2-254 Age-Dependent Interactive Changes in Serotonergic and Noradrenergic Cortical Axon Terminals in F344 Rats

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In the aging rat frontal cortex, we found an increase in sprouting of the noradrenergic (NA) axons originated from the locus coeruleus (LC). Although the serotonergic (5-HT) axons originated from the dorsal raphe (DR) share the same cortical area, their age-dependent changes and interactions with NA axons were still unclear. To compare quantitatively the densities of axon terminals of DR and LC neurons in the frontal cortex, we extracellurally recorded both DR and LC neurons for each animals and antidromically stimulated to 32 cortical points (a pair of stimulating electrodes was moved at 100-μm intervals from 500 μm to 2000 μm in depth). In addition, to examine the effects of degeneration of 5-HT axons on NA axons, and vice versa, we used specific neurotoxins for 5-HT (PCA) or NA (DSP-4) axons. Results suggested that 5-HT axons sprouted between 15 and 17 months of age and NA axons accelerated the age-dependent change of 5-HT axons.

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P2-255 Mitochondrial dysfunction in brain of the Down syndrome mouse model; Ts1Cje

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Down's syndrome (DS) caused by trisomy of chromosome 21(HSA21), is the most common genetic cause of mental retardation and affects every major organ in the body. The DS mouse model with partial trisomy 16, Ts1Cje covers HSA21 syntenic region and extends from Sod1 to Znf295, but excludes Sod1 and App that has been suggested to be critically involved in the DS phenotypes. In the present study, we observed impairment in mitochondrial function in primary cultures of Ts1Cje neurons and astrocytes, morphology and distribution of the mitochondrial alterations, in addition to behavioral defects. Collectively, our results suggest that the trisomic segment of Ts1Cje excluding Sod1 and App plays a critical role in the mitochondrial dysfunction and behavioral abnormalities, possibly contributing to the Ts1Cje DS mental retardation like phenotype.

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P2-256 Studying the neurodegenerative phenotype in Ts1Cje, a partial Trisomy 16 mouse model for Down Syndrome

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Down syndrome (DS) or trisomy 21, the most common genetic cause of mental retardation, affects one in 800 live born human beings. Ts1Cje is a segmental trisomic DS mouse model in that the trisomic segment is syntenic to human chromosome 21q22. The mice show learning and behavioral abnormalities mimicking DS mental retardation, and the trisomic segment has therefore been suggested to be involved in the DS phenotype. Our present analysis of both E16 fetus and adult Ts1Cje mouse show abnormal morphometric profile, growth retardation and region specific abnormal brain- neuron morphology. Our results suggest that the trisomic segment of Ts1Cje excluding *App* and *Sod1* genes plays a critical role in the neuronal degeneration possibly contributing to the DS mental retardation-like phenotype of Ts1Cje mouse.

P2-257 Dosage-dependent over-expression of genes in the trisomic region of Ts1Cje mouse model for Down syndrome

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Down syndrome (DS) is the most common chromosomally-caused form of mental retardation and is caused by trisomy for chromosome 21. The over-expression of genes located on the trisomic region has been assumed to be responsible for the phenotypic abnormalities of DS, but this hypothesis has not been fully confirmed. We have therefore investigated global gene expression profiles in Ts1Cje, a mouse model for DS. DNA microarray analyses of six Ts1Cje and six normal littermate (2N) mouse brains at postnatal day 0 revealed that the expression levels of most genes in the trisomic region were increased about 1.5-fold. In contrast, the expression levels of genes on outside of the trisomic region were largely the same in Ts1Cje and 2N mice. These results indicate that the genes in the trisomic region of Ts1Cje are over-expressed in a dosage-dependent manner.

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P2-258 Polyglutamine aggregates induces autophagy via activation of ER stress

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The ER stress leads to the activation of genes possessing unfolded protein response, and excess amounts of unfolded proteins are degraded by a ubiquitine-proteasome system called the ER-associated degradation system (ERAD). However, if these systems do not sufficiently work, prolonged stress activates apoptotic pathways. Recently, the relationship between ER stress and conformational diseases has been focused. Some types of the polyglutamine (polyQ) aggregates also decrease ERAD activity and stimulate ER stress signals. PolyQ aggregates also induces the autophagy. 3-methyl adenine inhibits the autophagy and promotes the polyQ aggregates, suggesting that autophagy is involved in the degradation of malfolded protein. In the present study, we examined the relation between the autophagy and ER stress. PolyQ aggregates induced the autophagy via activation of ER stress.

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P2-259 A new compound that inhibits the ER stress-mediated cell death

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Unfolded or malfolded proteins in the endoplasmic reticulum (ER) activate stress signals via ER stress sensor proteins that induces ER stress. When these stress signals are unable to rescue cells, various apoptotic pathway is activated. Recently,the relationship between ER stress and conformation diseases has been focused. Unsufficient degradation by ER associated degradation system (ERAD) induces accumulation of malfolded proteins resulting in ER stress-mediated cell death. Little is known about the translocation of unfolded and malfolded proteins from ER into the ERAD. In the present study, we show the novel compound that inhibits the ER stress-mediated cell death via promoting the translocation of unfolded and malfolded proteins from ER. This compound suppressed the GPR37(Pael-R)-induced ER stress and ER stress-mediated cell death. This compound may be useful for one of the therapeutic approaches to the conformation diesease including parkinson disease.

P2-260 Prevention of muscular necrosis by inhibition of hematopoietic prostaglandin D synthase

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Duchenne muscular dystrophy is X-linked muscular abnormality caused by the loss of dystrophin and is one of the most gravely genetic disorders. We have recently been found that hematopoietic prostaglandin (PG) D synthase (H-PGDS) is induced in the expanded necrotic muscle of patients of this disease. In this study, we applied an H-PGDS inhibitor HQL-79 to a bupivacaine-induced muscular necrosis model and to genetically dystrophin null mice (*mdx*) to examine the pathological role of PGD_2 produced by H-PGDS on the expansion of muscle necrosis. Oral administration of HQL-79 prevented the wound expansion and accelerated the accumulation and activation of macrophages in the wounded area in these model and *mdx* mice. These results indicate that PGD_2 produced by H-PGDS is involved in the wound expansion in the animal models of the muscular dystrophy.

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P2-261 Visualization of 6-hydroxydopamine-induced nigral lesions in rats by magnetic resonance imaging

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Size and location of lesions after 6-OHDA injection (2-16 μg) in the rat SN were evaluated by T2-weighted magnetic resonance imaging (MRI). Hyperintense area in MRI was found in a dose-dependent manner, which was correlated with the loss of tyrosine hydroxylase-positive cells. The shape of hypertintense area was formed with two components, circular and longitudinal regions. Administration with higher doses (8-16 μg) accompanied damages beyond the SN. However, rotational behavior induced by methamphetamine was maximal over 4 μg injections. These results suggest that MRI provides highly valuable information for verifying the size and location of intended lesions in individual animals as well as for determining the optimal dose of neurotoxins to elicit specific neurodegeneration.

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P2-262 Enhancement of the release of GABA and glycine during ischemia in rat spinal dorsal horn

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A change in spontaneous inhibitory postsynaptic currents (sIPSCs) following ischemia was examined in substantia gelatinosa (SG) neurons of adult rat spinal cord slices by use of the whole-cell patch-clamp technique. About 10 min after superfusion of an oxygen- and glucose-free medium, the amplitude and frequency of sIPSC was remarkably increased compared with control. In a phase of the increase in sIPSC activities, GABAergic and glycinergic sIPSCs, which were observed in the presence of strychnine and bicuculline, respectively, with tetrodotoxin, were largely increased in frequency with a minimal change in the amplitudes. It is concluded that the ischemic simulation increases the spontaneous release of GABA and glycine to SG neurons from nerve terminals; a part of this enhancement is possibly due to an increase in the spontaneous activities of inhibitory interneurons. GABA so released might serve to inhibit the release of L-glutamate from nerve terminals.

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P2-263 Neuroprotective role of endogenous IL-6 signaling in the ischemic mouse brain

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Cerebral ischemia rapidly induces interleukin-6 (IL-6) expression in the brain. To study physiological meaning of the IL-6 expression, we administrated the anti-IL-6 receptor antibody (IL-6RA) that blocks the IL-6 signaling to mice after middle cerebral artery occlusion (MCAO). The IL-6RA administration resulted in a significant reduction in the amount of phospholyrated Stat3 protein in the peripheral part of the ischemic brain tissue at 6h after the MCAO. We found that the blockade of the IL-6 signaling increased the infarct size and aggravated neurological function at 24h after ischemia. These results suggest that the endogenous IL-6 has a critical role to protect neurons in the acute phase of brain ischemia, which might be mediated by the Stat3 activation.

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P2-264 17β-estradiol can attenuate delayed neuronal death in gerbil hippocampus

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Delayed neuronal death in gerbil hippocampus CA1 area following transient ischemia/recirculation treatment can be produced by excessive release of excitatory neurotransmitter, glutamate, from abnormally stimulated neuron terminals into surrounding extracellular (EC) space. High affinity sodium dependent glutamate transporter is effective to reduce the disorders. Daily intraperitoneal (IP) administration of 17β-estradiol (17β-ED) to the animal after ischemic attack recovered amount of glutamate transporter, GLT-1 and GLAST, in hippocampus within 7 days. The decreased fluorescent intensity stained with GLT-1 or GLAST antibody on the tissue slices of CA1 tended to increase by daily IP injection of 17β-ED for a week after the treatment. These results suggest that administered 17β-ED can accelerate glutamate transporter functions to reduce toxic glutamate from EC space into glial components and protect the progression of neuronal death in the brain.

P2-265 Attenuation of mitochondrial injury by poly (ADP-ribose) synthetase siRNA in experimental model of cerebral ischemia

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Poly (ADP-ribose) synthetase (PARS) is a nuclear enzyme that is activated by DNA strand breaks. We reported that PARS activation plays a critical role in the mitochondrial injury in the ischemic neuronal cell death. In the present study using the RNA interference (RNAi) technique, we analysed the mitochondrial injury after oxygen-glucose deprivation (OGD) and reoxygenation in rat cortical neurons. A 2-hr OGD induced neuronal apoptosis and mitochondrial injuries; (1) enhanced ROS generation and reduction of membrane potential in mitochondria, and (2) a release of cytochrome c (cyt c) and AIF from mitochondria. The cytosolic cyt c activated caspase 3, that cleaved PARS. The suppression of PARS expression by siRNA transfection inhibited neuronal apoptosis and the release of both cyt c and AIF. These results indicate that PARS activation induces mitochondrial injury, leading to apoptosis in caspase-dependent and -independent manner.

P2-266 In vivo detection of the cerebral hydroxyl free radicals in a rat model of transient focal cerebral ischemia -a microdialysis study-

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We measured the cerebral hydroxyl free radicals by using microdialysis with the salicylic acid trapping method during ischemia and reperfusion. The method was based on the principle of aromatic hydroxylation and 2,3-dihydroxybenzoic acid (2,3-DHBA) levels were a reliable hydroxyl marker in vivo. The level of cerebral 2,3-DHBA was increased during ischemia and reperfusion, especially during the early reperfusion stage at the periphery of the infarct area. Free radicals such as hydroxyl radicals may play an important role in the ischemia reperfusion injury process at the penumbral area of the brain. Additionally, decrease of monoamine levels especially in the reperfusion stage reflected the levels of the cell damage. Finally, we also applied this method to assess a radical scavenging property of neuro-protective drugs.

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P2-267 Involvement of γPKC in neruroprotection on focal brain ischemia through G protein-coupled estrogen receptor

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There is increasing evidence protein kinase C (PKC) is activated by stimulation of estrogen receptor on the plasma membrane. Rapid signaling of estrogen in hypothalamic neurons are reported to involve a G-protein-coupled estrogen receptor that activates phospholipase C (PLC), leading to PKC activation. We hypothesized the neuroprotective effects of estrogen on ischemic injury is due to the activation of γPKC, a neuron-specific PKC subtype in the brain. We demonstrated estrogen-induced neuroprotection on transient focal cerebral ischemia was attenuated in γPKC-KO mice. We also visualized the rapid and reversible translocation of γPKC-GFP by estrogen stimulation in living cells. These results suggest the activation of γPKC is involved in the estrogen-induced neuroprotection from focal brain ischemia.

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P2-268 Preinjury administration of naloxone promotes early functional recovery after spinal cord injury in adult rats

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To investigate whether naloxone has the effects on the spinal cord injury (SCI), a contusive SCI was made in female Wistar rats with a weight after laminectomy at Th 9/10. Naloxone was treated using osmotic pump (0.1 mg/day for 7 days, s.c.) that was implanted at 4 hours before (Pre-Group) and just after SCI (Post-Group). Improvement in BBB score was found earlier in the Pre-Group compared with both controls and the Post-Group, however no significant difference was observed between the Post-Group and controls. Immunohistochemical analysis showed that the number of microglia and caspase-3-activated cells in the ventral white matter in 10 mm caudal were significantly decreased in the Pre-Group. These data suggest that pre-treatment with naloxone facilitated functional recovery in early phase after SCI partly with modification of microglia and apoptotic cells in the ventral white matter.

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P2-269 Synaptic re-organization after motoneuronal axotomy in the rat

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The axonal injury of the motoneurons (MNs) leads to various morophofunctional consequences in the motor nuclei. In aiming to identify the mechanism linking the axonal damage to the perisomatic alteration, we analysed the changes in the synaptic activity of the MNs after unilateral facial nerve axotomy in the neonate-young rats. On the first post-operational day, the postsynaptic currents recorded from the facial nuclei in the brainstem slices were significantly more frequent and larger in amplitude in axotomized MNs than in the non-axotomized ones, both in the absence and presence of TTX. However, after the third post-operational day, synaptic inputs to the axotomized MNs became less frequent and smaller than the non-axotomized ones. These results are suggestive of a re-organization of the premotor-to-MN synapses occurring at an early stage after axotomy, which might lie upstream to the cell degeneration and apoptosis at later stages.

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P2-270 Induction of hematopoietic prostaglandin D synthase after traumatic brain injury

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Prostaglandin (PG) D_2 is produced by hematopoietic PGD synthase (HPGDS) in microglia in the CNS and is considered to act as an inflammatory mediator by stimulating two distinct types of PGD_2 receptors; DP and CRTH2 receptors. In this study, we found that HPGDS was induced in activated microglia/macrophages around the stab-wounded lesion of the mouse brain. DP receptor was also induced in activated astrocytes localized in the edge of the wounded lesion. The wound injury was severer in human HPGDS-overexpressing transgenic (TG) mice and milder in the HPGDS-KO mice than the WT mice, as judged by histological examination and by Evans-blue dye exclusion assay. Administration of an HPGDS inhibitor HQL-79 suppressed the expansion of wound injury in HPGDS-TG and WT mice. These results indicated that PGD_2 produced by H-PGDS in microglia/macrophages aggravates the brain injury by stimulating DP receptors on the activated astrocytes.

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P2-271 Effective application of Chondroitinase ABC to spinal cord injury

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Recently, several investigators have revealed that the chondroitin sulfate proteoglycans(CSPGs) inhibit axonal regeneration after spinal cord injury and that axonal regeneration is promoted by intrathecal infusion of Chondroitinase ABC(C-ABC) in injured spinal cord reducing the amount of CSPGs. They applied C-ABC to the injured spinal cord by repeated infusion to subarachnoid space via catheter every other day because enzyme activity of C-ABC decreases rapidly in vivo. To determine the best way of C-ABC delivery to reduce the CSPGs, which express robustly after spinal cord injury, we compared two ways of continuous intrathecal infusion and intralesional injection of C-ABC to contusive injured spinal cord of rats regarding digestion of CSPGs.

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P2-272 The influence of the aggregational state of amyloid-β-peptides on microglial activation.

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Microglia activated by amyloidβ-peptides (Aβ) have been reported to damage or kill neurons by the release of neurotoxic molecules such as tumor necrosis factor-α (TNF-α), nitric oxide, or reactive oxygen species. Although the relationship between the aggregational state of Aβ peptides and their neurotoxicity has been well investigated, little is known about the relationship between the aggregational state of Aβ peptides and their ability to activate microglia. In the present study, we thus performed both structural and biochemical studies to clarify the relationship. The present results suggest the putative existence of distinct β-turn content thresholds of Aβ peptides for fibrillar aggregation and microglial activation.

P2-273 Immunohistochemical study of APP in the old dog brain

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Whole brains were removed from 16 dogs (3 to 19 years old), and the brains were immersed in 4% paraformaldehyde for fixation. The cerebral frontal cortex was serially sliced at a thickness of 50 mm with a microslicer. Immunohistochemistry was performed using antibodies against APP for b-amyloid precursor protein 695 and NeuN for neurons. APP immunopositivity was fine granular in the neural cytoplasm. In addition, many APP-immunopositive small-sized globular structures, which also confirmed the immunopositivity of neurofilaments, existed in the gray and white matter. The number of APP-immunopositive globular structures was counted under the microscope. Furthermore, APP- immunopositive patchy structures in the gray matter were revealed, and they morphologically resembled the deposition of Ab. We report the relationships between APP-immunopositive small-sized globular structures, the Ab-positive area, the number of neurons, age and dementia score.

P2-274 Sensitive analysis of Aβ related peptide with immunoprecipitation and liquid chromatography ion trap mass spectrometer

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A significant accumulation of Aβ amyloid is major pathological feature in the brain in patients with Alzheimer's disease (AD). Aβ is derived from its precursor protein, APP, by sequential proteolytic processing involving β- and γ-secretase. Recently, it was shown that γ-secretase processed near the cytoplasmic membrane boundary of APP C-terminal fragment (named ε-cleavage), as well as at γ-secretase cleavage site in the middle of the membrane domain. However, the relation between γ- and ε-cleavage is still unknown. In this presentation, to analyze the relation between γ-cleavage and epsilon cleavage, I carried out sensitive analysis of Aβ related peptide with immunoprecipitation and liquid chromatography ion trap mass spectrometer, and found that longer Aβ peptides with C-termini between residues 43-52 were secreted under normal condition. This method is a sensitive, reliable, and powerful method for Aβ analysis.

P2-275 Changes of neuropeptides expression in MK-801 treated Alzhimer's model rat brain and the effects of therapeutic drugs.

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Alzheimer's disesase (AD) is a progressive neurodegenerative disorder, characterized by hallmarks; extracellular beta-amyloid deposition, phospharylated tau and decrease of neuropeptide levels. MK-801 is a non-competitive NMDA receptor antagonist. MK-801 administration induces neuronal cell death in the cortical limbic system and posterior singulate/retrospinal (PC/RS) cortex involved in early stage of AD. To explain the connection between AD pathogenesis and neuropeptides expression, we measured somatostatin, cholesystokinin, and substance P peptide and mRNA levels in rat brain regions after MK-801 treatment. Furthermore, we investigated the effects of typical/atypical anti-psychotic drug, haloperidol and clozapine, pretreatment against MK-801 toxicity. These results revealed neuropeptides expression involved in AD pathogenesis and effect of therapeutic drug.

P2-276 Syntaxin 5 interacts with Presenilin holoproteins and suppresses the production of Aβ peptides

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We have previously shown that syntaxin 5 (Syx5) interacted only with the full-length presenilin (PS) in the early secretory pathway. We further examined the interaction of PSs with Syx isoforms, the effects of Syx5 overexpression on PS holoproteins and βamyloid precursor protein (βAPP), and the Aβ peptide production. Among Syxs, the ER-type isoform of Syx5 bound to holoproteins of PSs. Interaction domains in Syx5 were mapped to the transmembrane domain and the cytoplasmic region that is distinct from SNARE motif. Among FAD-mutants of PS1, the PS1ΔExon9, which lacks the endoproteolytic cleavage site, showed markedly decreased binding to Syx5. Overexpression of Syx5 resulted in accumulation of PS and βAPP holoproteins, and reduced the secretion of Aβ40 and Aβ42. These results indicate that Syx5 binds to full-length PSs and affects the generation of Aβ peptides.

P2-277 Amyloid β-protein aggregation in the membrane-mimicking environment

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A fundamental question regarding the development of Alzheimer's disease and hereditary amyloid angiopathy is how amyloid β-protein (Aβ) deposits in the brain. Here, we report that membrane-mimicking environment is sufficient for amyloid fibril formation from soluble Aβ. Furthermore, hereditary variants of Aβ, including Dutch (E22Q), Flemish (A21G), and Arctic (E22G) types, show mutually different aggregation behavior in these environments. In the presence of sodium dodecyl sulfate or GM1 ganglioside, Arctic-type Aβ shows markedly rapid and higher level of amyloid fibril formation in contrast to wild type and other variant forms of Aβ. These results suggest that there are favorable local environments for fibrillogenesis of Aβ.

P2-278 ER stress influences the production of amyloid beta peptides

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Endoplasmic reticulum stress (ER stress) has been reported to be involved in the neuronal cell death in several neurodegenerative disorders such as Alzheimer disease (AD). The progressive accumulation of the amyloid beta peptide (Aβ) that is composed of 40 or 42 amino acids is a key feature of AD. Here we studied the association between Aβ production/secretion and ER stress signaling. The secreted Aβ from SH-SY5Y neuroblastoma overexpressed APP was decreased by the treatment with tunicamycin or thapsigargin, the both of which induce the ER stress, and concurrently APP was retained in the ER and the maturation was inhibited. These results indicate that ER stress affects the trafficking and processing of APP, and Aβ production/secretion. Our data may show the new insight of cross talk between the neuronal cell death and the Aβ production in the brain of AD patients.

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P2-279 The PGD_2-mediated inflammation is enhanced around amyloid plaques in the Tg2576 mouse model of Alzheimer's disease

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Alzheimer's disease (AD) is a progressive and age-related dementia characterized by inflammatory responses induced by amyloid-β deposition. PGD_2, a major prostanoid in the CNS, is increased in several neurological disorders, however, the role of PGD_2 in AD has remained elucidated. We investigated expression of PGD_2- synthase (PGDS) and its receptors in Tg2576 mice. Immunocytochemistry revealed that hematopoietic PGDS (H-PGDS) and D-type PG receptor (DPR) are localized in microglia and astrocytes, respectively, around amyloid plaques. Quantitative RT-PCR also demonstrated the progressive increase of H-PGDS and DPR in Tg2576 mice. Our data suggests that HPGDS and DPR may play an important role in the chronic inflammation around amyloid plaques in AD.

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P2-280 Disruption of calcium homeostasis by Alzheimer's β-amyloid protein and prion protein

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Alzheimer's β-amyloid protein (AβP) and prion protein share several similarities such as formation of amyloid fibrils with β-sheet structures, abnormal deposition in the brain, cytotoxicity, and formation of calcium permeable pores on membranes. We have revealed that AβP and prion protein fragment peptide (PrP106-126) cause the elevation in intracellular calcium levels ($[Ca^{2+}]i$) of cultured neurons and hypothesized that the disruption of calcium homeostasis may be based on the molecular mechanism of their neurotoxicity. Here, we investigated the detailed characteristics of $[Ca^{2+}]i$ changes of cultured rat hippocampal neurons induced by AβP and PrP106-126. Although several agonists or antagonists of neurotransmitters or channel blockers did not influence, the elevation in $[Ca^{2+}]i$ was disappeared after the removal of extracellular calcium. These results suggest the similar pathological mechanism of these diseases.

P2-281 On the mechanisms by which β amyloid peptides inhibit long-term potentiation

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The fibrillar structures of β amyloid peptide (Aβ) are known to contribute to pathogenesis of Alzheimer's disease (AD). Recently, not only the fibrillar Aβ but also the soluble oligomeric Aβ has been suggested to relate to neuropathology of AD, in particular the memory loss. Also it has been reported that low concentrations of the soluble oligomeric Aβ inhibit long-term potentiation (LTP), a cellular model for learning and memory, at short time exposure in vivo and in vitro. We now studied how the oligomeric Aβ affects synaptic plasticity by whole-cell recording from rat hippocampal CA1 pyramidal cells. Brief perfusion of slices with low concentration (1μM) of $Aβ_{1-42}$ significantly inhibited LTP induction without affecting the basal synaptic transmission. The same concentration of Aβ affected neither NMDA current nor paired-pulse facilitation.

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P2-282 Presenilins negatively regulate antiapoptotic activity of FKBP38

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Presenilins (PS) are multifunctional membrane proteins localized predominantly at the ER and Golgi apparatus, of which mutations are responsible to the majority of familial Alzheimer disease (FAD). Modulation of apoptotic susceptibility has been described as a notable activity of PS. Here we demonstrate that FKBP38, an immunophilin residing in the mitochondria, is a PS-interacting protein and that PS downregulate FKBP38 and translocate it to ER and Golgi to antagonize an antiapoptotic function of FKBP38. The PS deletion induced resistance to mitochondria-mediated apoptosis, while the overexpressed PS sensitized cells to apoptosis. FAD-linked PS mutations conferred a gain-of-function effect on this activity, whereas the overexpressed C-terminal fragment exerted a dominant-negative effect. Our results provide a molecular mechanism underlying apoptosis modulation by PS.

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P2-283 Prostaglandin D_2-mediated inflammation in glial cells associated with senile plaques in Alzheimer's disease.

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Alzheimer's disease (AD) incidence and progression are reduced in patients with long-term treatment of nonsteroidal anti-inflammatory drugs (NSAIDS), which prevent production of prostaglandins (PGs). We investigated the expression of PG receptors and synthases in AD and non-AD brains. Receptors and synthase for PGD2 were induced in activated microglia and reactive astrocytes in senile plaques (SPs) of AD brain, whereas all other PG receptors and synthases were almost unchanged between two patient groups. Our findings indicate that PGD_2 is actively involved in chronic inflammatory within SPs Therefore, PGD_2 synthase inhibitors and its receptor antagonists developed as anti-allergic drugs could be reconsidered as anti-AD drugs.

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P2-284 Deletion of Presenilin C-terminal tail enhances Aβ generation.

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Previously, a C-terminal tail of Presenilin (PS) was shown to be required for the endoproteolysis and stabilization of PS. Here, we examined the relation between the role of PS C-terminus and PS cofactors in Aβ generation. We found that a small deletion at PS1 C-terminus (PSΔC) caused an increase in Aβ generation in PS-null cells. The expression of PEN-2, which is one of the PS cofactors, enhanced Aβ generation from wt PS1 but no more enhanced that from PSΔC. Moreover, the expression of a short fragment containing the PS1 C-terminal sequence completely inhibited Aβ generation from PSΔC.These results suggest that the PEN-2 has a role in attenuating the inhibitory effect of PS C-terminal tail on γ-secretase activity.

P2-285 Search for the proteins which interact with N-terminal 126 amino acids of Microtubule-associated protein (MAP) 1B

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Previously we reported overexpression of MAP1B fragments containing N-terminal 126 amino acids promoted neuronal death and neurite outgrowth. These results suggest that overexpression of full length MAP1B might act as the effector of cell death in neurodegenerative disorders related to cytoskeletal abnormalities.
Then to reveal the mechanism of MAP1B N-terminal induced neuronal death, we are searching for the proteins that interact with N-terminal of MAP1B by two-hybrid system. Now 2 known prtoeins and 1 unknown peptides are proved to interact with MAP1B N-terminal 126 amino acids. Other 10 candidates are under confirmation of in vitro interaction with MAP1B N-terminal 126 amino acids by pull-down assay.

P2-286 Microtubule Binding Protein Tau and 14-3-3 Form a Multi-protein Complex.

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Neuron-specific microtubule binding protein tau plays a critical role in controlling stability of microtubule networks. Tau has as many as 25 possible phosphorylation sites, and its physiological function is regulated by phosphorylation status. Abnormal phosphorylation impairs physiological function of tau resulting in neurodegenerative disorders including Alzheimer's disease.
We have found that a scaffold protein, 14-3-3ζ, binds to tau as a substrate effector, (M. Hashiguchi et al.(2000) J.B.C. 275: 25247). Binding of 14-3-3ζ to tau is not phosphorylation-dependent, while 14-3-3 binds mainly to phosphoproteins. To clarify the binding mechanism in which 14-3-3ζ associates with tau, we have conducted chemical cross-linking assay by DSS. DSS promoted aggregation of tau in the presence of 14-3-3. Not only a tau dimer but also much heavier protein bands were detected. That tau and 14-3-3 form a multi-protein complex is clearly indicated.

P2-287 Effect of FTDP-17 mutations on tau phosphorylation by cyclin-dependent kinase 5

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Various mutations of tau have been found in frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17). Mutant tau is highly phosphorylated and aggregated in patient's brains. Cdk5 has been suggested to be one of tau protein kinases, but phosphorylation of FTDP-17 tau mutants by Cdk5 is not analyzed yet. We examined phosphorylation of K257T, P301L, P301S and R406W tau mutants by Cdk5. Although all tau mutants were less phosphorylated than WT tau, phosphorylation sites were same among WT, K257T, P301L, and P301S. Ser404 was less phosphorylated in R406W compared to other mutants. However, when a synthetic peptide containing R406W mutation was phosphorylated, Ser404 was phosphorylated as much as a WT tau peptide. R406W mutation may induce the conformational change which reduces phosphorylation at Ser404 by Cdk5.

P2-288 Proteomic analysis of mouse brain lipid rafts rich in amyloid precursor protein

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Lipid rafts are membrane microdomains enriched in cholesterol and sphingolipids, and act as platforms for conducting a variety of cellular functions. Recent studies suggested that lipid rafts would be crucial sites for processing of amyloid precursor protein (APP) to amyloid-beta peptide. We hypothesized that the partitioning of APP into the lipid rafts would be regulated not only by lipid environment but also by raft proteins. To search for candidate proteins, we obtained raft fractions rich in APP from mouse brain tissues by using immunomagnetic isolation. Mass-spectrometric and immunological analyses demonstrated enrichment of a distinct set of proteins, which suggests functional specialization of the APP-containing rafts. Systematic identification and characterization of enriched proteins will provide new insights into the regulatory mechanism of APP processing and the physiological functions of APP in neurons.

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P2-289 A Novel Action of Apolipoprotein E (ApoE): ApoE Inhibits HDL-Mediated Cholesterol Release

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We show that apoE3-HDL induced strong cholesterol release from neurons, while apoE4-HDL induced little. ApoE3- or apoE4-lipid emulsion (EM) complex was used to elucidate this mechanism. With increasing number of apoE associated with EM, neither apoE3- nor apoE4-EM induced cholesterol release. However, when apoE at certain concentrations was bound to EM, apoE3-EM markedly induced lipid release, while apoE4-EM didn't. This is because apoE4 has higher lipid binding affinity than apoE3 and the binding affinity of apoE3 increased by β-mercaptoethanol treatment,suggesting that the dimerization of apoE is responsible for the isoform-specific effect of apoE-HDL or apoE-EM on cholesterol release.

P2-290 Impairment of LTP in the Hippocampus of APP Transgenic Mice in VIVO by Neprilysin-Deficiency

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To explore whether the increased Aβ level by deficit of neprilysin activity may contribute to the dysfunction of hippocampal synapses, we examined changes in synaptic plasticity including hippocampal long-term potentiation (LTP) in vivo in neprilysin knock-out APP transgenic mice. The model was established by crossing the APP 23 mice, and neprilysin knock-out mice. Our study demonstrates that synapse plasticity is inhibited in neprilysin knock-out APP transgenic mice compared with APP transgenic mice, suggests that the decreased activity of neprilysin may contribute to impaired synaptic plasticity in hippocampus by way of Aβ accumulation in brain.

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P2-291 Gene expression in frontal cortices of patients of Alzheimer-type dementia

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We examined gene expression in frontal cortices of Alzheimer-type dementia (ATD) patients and compared with that of non-ATD control using DNA microarray technique and quantitative reverse transcriptase-polymerase chain reaction. 14 Genes were confirmed to be up-regulated and 3 genes down-regulated in ATD. Up-regulated genes included those for insulin-like growth factor II (IGF-II) and cellular retinoic acid-binding protein I (CRABP I), and down-regulated genes included IGF-I gene. Immunoblotting also showed that levels of CRABP I protein in frontal cortices were greater in ATD. These observations suggest that the differential expression of these genes in frontal cortex might be related to ATD pathology.

P2-292 Different age-dependent changes of noradrenergic innervations in the hippocampus and frontal cortex

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The locus coeruleus (LC) is the largest nucleus of noradrenergic (NA) neurons, and these neurons project to the frontal cortex and hippocampus. Our previous electrophysiological studies suggested that the projection from LC to the frontal cortex and hippocampus is gradually decreased, but axonal branching in frontal cortex and hippocampus was increased dramatically between 15- to 17-month-old. Therefore, we investigated age dependent changes of NA innervations in the frontal cortex and hippocampus by immunohistochemistry and western blotting analysis of TH, norepinephrine transporter and Glial cell line derived neurotrophic factor (GDNF) expression.
In this study, we used 6-, 13- and 25-month-old male F344/N rats. The NA fibers density in the hippocampus was reach maximum level at 13-month-old, but not altered by aging in the frontal cortex. Moreover, western blot analysis also showed some difference of aging pattern between the hippocampus and frontal cortex.

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P2-293 Exogenous and endogenous cGMP inhibits upregulation of GLAST induced by β-amyloid in cultured astrocytes

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Although mounting evidence has implicated amyloid β-protein (Aβ) as a key protein in the pathogenesis of Alzheimer's disease, little is known about the mechanism by which Aβ causes dementia. In our previous results, we found that prolonged treatment of Aβ augments glutamate uptake in cultured astrocytes due to the translocation of glutamate transporter (GLAST) to the cell surface. Here we show that exogenous and endogenous cGMP inhibits the effect of Aβ. Coapplicaion of 8-Bromo-cGMP, a cell-permeable cGMP analog, with Aβ25-35 for 48hr attenuated Aβ-induced upregulation of GLAST and enhancement of glutamate uptake. The uptake was also inhibited by cGMP-specific phosphodiesterase (PDE) inhibitor zaprinast and atrial natriuretic peptide (ANP) which activates particulate guanylate cyclase (pGC). But NO donor SNAP or YC-1 which activates soluble guanylate cyclase (sGC) was ineffective. cGMP level was not increased by these treatment probably due to the low expression of sGC.

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P2-294 Lost of acetylcholine dependent autoregulation of cerebral blood flow and memory disturbance in mice

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Disturbances in the acetylcholine muscarinic receptor may play a role in a number of pathophysiological conditions including Alzheimer's and Parkinson's disease, depression, schizophrenia, and epilepsy. *M5R*$^{-/-}$ mice display cerebrovascular insufficiency caused by the lacked of autoregulation of cerebral blood flow (CBF) and significantly reduced CBF in cortex and hippocampus. Pyramidal neurons show an increase in spine-loss and dendritic atrophy in cortex and hippocampus. Moreover, hippocampus dependent memory was impaired in *M5R*$^{-/-}$ mice. Our findings suggest that deficit of M5R may play a role in the pathophysiology of cerebro-circulatory disturbances, and an attractive therapeutic target in cognitive deficits caused by impairments in cerebrovascular function.

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P2-295 Memantine protective effects on the glia activation and the changes of neuropeptides and peptide-degrading enzymes in NBM-lesioned Alzheimer dementia model rat brain regions

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Glia activation play a critical role in the progression of Alzheimer disease (AD). We investigated the changes of glia activation and neuropeptides and their degrading enzymes in rat brain after ibotenic acid (IBO) infusion into rat nucleus basalis magnocellularis, an animal model of AD, and the possible protection by memantine treatment, a clinically tolerated and therapeutic drug for AD. Substance P and somatostatin levels decreased in the frontal cortex and striatum 1 week after IBO infusion, and recovered with memantine treatment, indicating the involvement of neuropeptides in AD pathology. The immunostaining of GFAP and CD11b in the cortex and striatum increased in IBO-treated rat brain. Co-administration of memantine reduced the glial proliferation.

P2-296 Mechanism of Action of Galantamine on N-Methyl-D-Aspartate Receptors in Rat Cortical Neurons

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Galantamine, a new Alzheimer's drug is known to inhibit acetylcholinesterase activity and potentiate acetylcholine-induced currents in neurons. However, since both cholinergic and NMDA systems are down-regulated in the brain of Alzheimer's patients, we studied the effects of galantamine on NMDA receptors. NMDA currents potentiated by bath and U-tube applications of galantamine at 10 - 10000 nM, showing a bell-shaped dose-response relationship. Addition of glycine at 1-3000 nM to 30 μM NMDA enhanced NMDA currents, the galantamine- and glycine induced potentiation of NMDA currents were additive. These results suggest that galantamine did not interact with the glycine binding site in the NMDA receptor. The potentiation of NMDA receptor activities by galantamine is deemed partially responsible for improvement of cognition, learning and memory in Alzheimer's patients.

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P2-297 Interaction of the memory gene *amnesiac* and the aging gene *methuselah* in longevity and memory

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Age-related memory impairment (AMI) results from a specific decrease in *amnesiac* (*amn*)-dependent memory. Young *amn* mutants are already defective for the memory component that is lost upon aging. Thus, *amn* mutants may be prematurely aging. However, we show here that *amn* mutants show a significant extension (approximately 40%) of lifespan compared to wild-type. In addition, we show that a long-lived mutant, *methuselah* (*mth*) displays mild memory defects. We further found that a *mth* mutant can suppress the memory defects of an *amn* mutant, indicating that these two genes interact genetically. This interaction also occurs with respect to lifespan. *amn* and *mth* single mutants show extended lifespan while double mutants show a reduction of lifespan. Intriguingly, the *amn* gene encodes putative neuropeptides while the *mth* gene encodes an orphan G-protein coupled receptor. Our results suggest an interaction between *amn* and *mth* and a genetic linkage between memory and longevity.

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P2-298 Induction of Caspase 3 activity and accumulation and phosphorylation of tau in NG108-15 cells expressing tau by cAMP

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To explore changes to the tau molecule in Alzheimer's disease, we studied the effect of tau expression in stably transfected neuroblastoma NG108-15 cells (tau cells). When treated with cAMP, tau cells began to form neurites within 2hr. Subsequently, these neurites became longer and thicker than those of wild type cells. Farther more, an accumulation and increased phosphorylation of tau were observed and caspase 3 activity was increased upon treatment with cAMP. Caspase 3 activity was activated after the initiation of morphological change, and before the accumulation of tau in tau cells. However, under these conditions, apoptotic cell death was not observed. We also found the accumulated tau did not associate with tubulin and were dislocated around the nuclei of tau cells. These observations have implications for the cellular causes of Alzheimer's disease where the accumulation and mislocation of tau occur concomitant with neuronal degeneration.

P2-299 Brain activation and memory acquisition during picture encoding in the elderly; an approach to screening system of pre-dementia state

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We examined the relationship between brain activities during encoding and actual acquisition of memory, in order to develop the screening system of pre-dementia state. In the encoding experiment, we presented pictures of landscape to subjects, and, in the retrieval, asked them to judge whether the pictures, half of which were modified, had been seen or not before. In these experiments, a correlative relationship was seen between increased hippocampal activities and acquired memory, which suggests that diagnosis of pre-dementia. We also show the results indicating that the degree of brain atrophy is not paralleled with that of neural activities in memory-related brain regions.

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P2-300 Modification of calcium entry channel in the aged rat supplemented with arachidonic acid

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One plausible mechanism to preserve LTP in aged animals supplemental with arachidonic acid is switching the contribution of calcium entry from NMDA to voltage dependent calcium channel (VDCC). We examine whether the primary calcium entry channel altered during aging and AA diet complemented the degraded calcium entry channel from NMDA to VDCC. Aged rats administered AA ester (OA) or control diet (OC) or of 2 months old young rats supplemented with control diet (YC) were used to record LTP from hippocampal slice with 36 electrode array. VDCC LTP (vdccLTP) tended to be dominant than NMDA LTP (nmdaLTP) in OC compared with YC, whereas nmdaLTP seemed to be more prominent than vdccLTP in OA as YC. This raised the possibility that oxigenated unsaturated fatty acid in the membrane of hippocampal neuron was complemented with AA diet in aged animal.

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P2-301 Effect of monoamine oxidase inhibitor pretreatment on methamphetamine-induced changes in brain monoamine metabolism in mice

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Clorgyline is characterized as a monoamine oxidase (MAO)-A inhibitor. Pretreatment of a repeated s.c. administration of clorgyline (1.0 mg/kg, once per day for five consecutive days) blocked hyperlocomotion induced by a single i.p. administeration of methamphetamine (METH, 1.0 mg/kg) in mice, without any effect on spontaneous locomotion. The blockade was also observed when mice were pretreated with a single administration of clorgyline. In contrast, single or repeated pretreatment of *l*-deprenyl (0.3 mg/kg, s.c.), a MAO-B inhibitor, had no effect on METH-induced hyperlocomotion. Clorgyline pretreatment, both single and repeated, showed significantly different changes in apparent serotonin turnover after METH challenge between control (saline) and clorgyline pretreated mice in the region of the thalamus and hypothalamus. These results suggest that clorgyline tends to oppose METH-induced hyperlocomotion via hypothalamic/thalamic serotonergic system.

P2-302 Inhibitory effects of Leu-Ile on morphine-induced dependence

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We investigated the effects of Leu-Ile, which can induce the syntheses of GDNF and TNF-α *in vivo*, on morphine (MOR) -induced dependence. The immunoreactivity of TNF-α was increased after the co-administration of Leu-Ile and MOR compared with Leu-Ile or MOR treatment alone in the nucleus accumbens, striatum, hippocampus of mice. Similar results were obtained even when the treatment of Leu-Ile was started after the cessation of MOR treatment. Leu-Ile blocked the acquisition of MOR-induced place preference (PP) and sensitization of locomotion (SL) in wild-type mice. Moreover, even after the acquisition of PP or SL, Leu-Ile treatment attenuated them. Lle-Ile failed to inhibit the acquisition of PP in TNF-α knockout mice. These results suggest that Leu-Ile could be useful as a novel treatment agent for drug dependence.

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P2-303 The MMP/TIMP system is involved in the sensitization induced by Methamphetamine in the rat

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Matrix metalloproteinase (MMP) constitutes a family of extracellular enzymes, which function to remodel the pericellular environment. MMP activity is regulated by the interaction with tissue inhibitors of MMP (TIMP). MMP/TIMP system has been implicated in neurological diseases. In this study, we investigated whether MMP/TIMP system is involved in methamphetamine (MAP)-induced sensitization which might be resulted from a remodeling of neuronal circuit. MAP treatment induced sensitization and markedly increased TIMP mRNA expression and protein content in the brain. MMP activity was increased by MAP treatment. Infusion of antisense TIMP oligonucleotide aggravated the sensitization induced by MAP. These results suggest that MMP/TIMP system might be associated with arrangement of neural network, such as behavioral response to MAP dependence.

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P2-304 Inhibition of GIRK channels by various antidepressants

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G protein-activated inwardly rectifying K^+ channels (GIRK; Kir3) are gated by various Gi/o protein-coupled receptors. GIRK channels play an important role in the inhibitory regulation of neuronal excitability in most brain regions and the heart rate. Modulation of GIRK channel activity may affect many brain functions. Here, we show using Xenopus oocyte expression assays that GIRK channels are inhibited by micromolar concentrations of various antidepressant drugs: imipramine, desipramine, amitriptyline, nortriptyline, clomipramine, maprotiline, fluoxetine and citalopram except fluvoxamine, zimelidine and bupropion. In contrast, Kir1.1 and Kir2.1 channels in other Kir channel subfamilies were insensitive to all of the drugs. Our results suggest that inhibition of GIRK channels by the antidepressants may contribute to some of the therapeutic effects and side effects, especially seizures and atrial arrhythmias in overdose.

P2-305 5MeO-DIPT-induced dopaminergic cytotoxicity

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Recently, N, N-diisopropyl-5-methoxytryptamine (5MeO-DIPT) has become a popular recreational rave drug which abused widely by young adults in Japan. However, neurotoxicity of the drug is still obscure. In the present study, we examined cytotoxicity of 5MeO-DIPT using dopamine (DA)-containing neuronal cells. Exposure to 5MeO-DIPT showed dose-dependent increases in LDH release from DAergic cells and apparent cell death, accompanying apoptotic morphological changes in nucleus. Fluvoxamine maleate, selective serotonin transporter inhibitor, enhanced 5MeO-DIPT-induced cytotoxicity. Furthermore, non-toxic dose of methamphetamine aggravated 5MeO-DIPT-induced LDH release and apoptotic cell damage. These results suggest that acute exposure of 5MeO-DIPT promotes apoptotic cell death in DAergic cells through changing synaptic serotonin, and that co-abuse of 5-MeO-DIPT with methamphetamine is highly toxic in DAergic neuronal cells.

P2-306 Study of neuronal plasticity in morphine-induced tolerance and dependence - new strategy of locus-specific rescue of gene into knock - out mouse brain by electroporation

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Opioid systems are present at multiple locations on analgesic and addiction pathways. The clinical utility of opioid analgesics is often overshadowed by the development of tolerance and dependence following chronic use. It has been proposed that some non-opioid neurons including anti-opioid neuron may cause a counter-adaptation to show such side effects. A major question is whether opioid neuronal plasticity by anti-opioid system in morphine analgesic tolerance and dependence could be caused in specific loci or in downstream of opioid neuron in the brain. To specify the brain locus, we performed the locus-specific rescue of gene into the brain of knock-out mice. Here we found morphine tolerance and some withdrawal signs result from brain locus-specific plasticity of neuronal circuits.

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P2-307 Nociceptin receptor knockout mice show reduced preferences for ethanol

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Several lines of study suggest that the novel neuropeptide nociceptin (known also as orphanin FQ) suppresses ethanol reward and seeking. In order to understand the role of endogenous nociceptin in the control of these processes, we studied ethanol intake in nociceptin receptor (NOP) knockout mice using a bottle choice paradigm. At low concentrations (3 and 6%), NOP knockout mice showed similar preferences for ethanol solutions as wild-type mice. At higher (10 and 20%) concentrations NOP knockout mice showed reduced preference for ethanol solutions. However, closer analysis revealed that this was due in part to an increased water intake. Further study of taste preference using saccharine and quinine solutions found no differences between the genotypes. These studies suggest that, similar to exogenous nociceptin, endogenous nociceptin regulates ethanol consumption.

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P2-308 cDNA subtractive screening for ethanol-responsive genes in the rat brain

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Ethanol (EtOH) intake at more than moderate levels over years may result in physical and psychiatric disorders, including alcoholism. To elucidate the molecular mechanisms of EtOH action on the central nervous system, we investigated changes in gene expression in the rat brain after chronic EtOH treatment. Male F344 rats were fed EtOH-containing milk, a condition under which physical dependence on EtOH was established. EtOH-treated and control brains were removed and dissected into several regional blocks. Poly(A)$^+$ RNA was extracted and gene expression changes were analyzed by cDNA subtraction. Over 20 candidates gene were up-regulated by EtOH-treatment in the telencephalon and/or cerebellum. Quantitative RT-PCR further identified significantly elevated synaptotagmin mRNA levels in the brains (cerebral cortex, striatum, diencephalons, and midbrain) of EtOH-treated animals, suggesting altered efficiency of synaptic transmission in the brain after chronic exposure to EtOH.

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P2-309 KCC2 expression is reduced in human epileptogenic cortical dysplasia

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Focal cortical dysplasia (FCD) characterized histologicaly by disorganized cortical lamination and cytomegalic cells is one of major causes of intractable epilepsies. Alterations in Cl^- homeostasis may underlie depolarizing GABA actions. Thus, we investigated NKCC1 and KCC2 to regulate of $[Cl^-]_i$, expressions in FCD tissue obtained by surgery from patients of intractable epilepsy. In dysplastic cells, KCC2 mRNA and protein expressions were decreased in small-sized dysplastic neurons as compared to cytomegalic dysplastic neurons in FCD and non-dysplastic neurons in histologicaly normal portion located in periphery of excised cortex. These results suggest that decreases in KCC2 expression in small-sized dysplastic neurons might change Cl^- homeostasis, so that impairment of GABAergic actions could occur in FCD being involved in epileptogenesis.

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P2-310 Effects of laser irradiation on the threshold of paroxysmal discharge in the rabbit hippocampus

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We have demonstrated that Ih blockers can increase the threshold of paroxysmal discharge (PADT) in the rabbit hippocampus, and Ar+ laser irradiation has a selective suppressive effect on the anode break excitation in the frog sciatic nerve. We examined effects of laser irradiation on the PADT in the hippocampus CA1. Semiconductor laser (YVO4; 532 nm) was introduced through an optical fiber (125 μm) into the apical dendritic layer where the electrical stimulation (1 ms pulse, 50/s) was applied for 1 s to induce PAD. The PADT was 2.9±0.2 V (mean±SE; n=10) before laser irradiation, whereas it changed to 2.8±0.2 V (n=3), 3.6±0.1 V (n=4), and 6.0±0.6 V (n=5) after 10 min irradiation of 50 mW, 75 mW and 100 mW laser, respectively. In some cases, the histological lesion (diameter: 1.0-1.5 mm) was observed. It is suggested that laser irradiation has an anti-epileptic effect.

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P2-311 A network mechanism of absence epilepsy in Ca2+ channel mutant mice tottering

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Ca2+ channel mutant mice tottering (tg) show not only cerebellar ataxia but epilepsy. The EEG and pharmacological similarities suggest that it can serve as a model of the human absence epilepsy. To identify the mechanism of epileptogenesis, we studied the effect of the mutation on the thalamocortical network. IPSC amplitudes recorded from layer IV pyramidal neurons in response to thalamic stimulation became disproportionally reduced, compared with EPSC amplitudes, in later developmental stages (P21-30). Similar results were obtained by local stimulation. The reduction was not seen in layer V pyramidal neurons. Recordings of multiple extracellular potentials revealed that cortical excitation (evoked by layer IV stimulation) spread in the horizontal direction more strongly in epileptic tg than in control or younger tg mice. These results suggest that the defect of feed-forward inhibition in the cortical input layer is critical for the generation of absence epilepsy in tg.

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P2-312 Biophysical Analyses of Voltage-gated Na^+-Channel Gene Mutations Identified in Intractable Epilepsies with Severe Mental Decline

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Heterozygous sporadic mutations of the voltage-gated Na^+-channel genes, SCN1A and SCN2A, have been described in intractable epilepsies associated with severe mental decline. Recordings of Na^+ current from human cells expressing mutant channels showed drastically attenuated or no currents, suggesting haploinsufficiency of channels as the molecular pathology of these epilepsies. Moreover, recordings from human cells coexpressing the mutant and wild-type channels showed dominant-negative effects, suggesting a novel pathogenic mechanism of the epilepsy. We further discuss the biophysical implications of the mutations in intractable epilepsies with variable phenotypes.

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P2-313 Antiepileptic action induced by a combination of vigabatrin and tiagabine

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We have investigated the effects of individual and combined application of vigabatrin (VGB) and tiagabine on GAT1 expressed in Xenopus oocytes, on epileptiform discharges in CA3 area of brain slices of genetically epileptic El and control ddY mice, and on occurrence of seizures in El mice. In Mg^{2+}-free solution, co-treatment by the two drugs produced additive inhibition in slices from both mouse strains, but pre-treatment with VGB in produced synergistic inhibition in slices only from ddY mice. A contribution to the synergistic inhibition of epileptiform discharges in brain slices may have originated from the complex interaction with GAT1. In experiments on the GAT1 expressed in oocytes it could be demonstrated that synergistic inhibition occurs only at low (0.1 nM) concentration of VGB. The results present a novel interpretation of synergistic inhibition of certain epileptic discharges using VGB and another drug.

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P2-314 Towards the identification of genes responsible for juvenile myoclonic epilepsy

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Among idiopathic generalized epilepsies, juvenile myoclonic epilepsy (JME) is the most common, accounting for 10-20 % of all epilepsies. Mutations of several genes including GABRA1 and CACNB4 have been reported in a few JME-associated cases, but most JME cases still do not show mutations in those genes. To understand the molecular pathology of JME and to improve the diagnosis and therapy, further JME genes should be identified. Previously, we mapped a JME locus on chromosome 6p12-p11 and recently narrowed it to 3.5cM region that should harbor a major JME gene. We have mapped eighteen genes in this region including c6orf33, that is predominantly express in brain, and an EF-hand domain containing gene, that would be a calcium modulator. Now we are performing mutation analyses of these candidates, and are characterizing their function.

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P2-315 Magnetic Resonance and Biochemical Studies in Pentylenetetrazole-induced Epileptogenesis. - the relationship between NO, nNOS and seizures

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To the best of our knowledge this is the first time that NO detection and monitoring were attempted after multiple injections 40 mg/kg/day of pentylenetetrazole (PTZ) in animals. The major aim of this study was to elucidate the role and the function of NO in epileptogenesis. This study is to determine the role and contribution of NO in PTZ-kindling epileptogenesis. These results indicate that the induction of NO by the daily administration of PTZ is associated with an increase in the amount of nNOS. We demonstrate that status epilepticus is caused by NO induction during the persistence up-regulation of nNOS expression and NO induction is not associated with severe generalized seizures following treatment with challenging dose of PTZ after withdrawal.

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P2-316 New approach examining enhanced cerebral blood flow in rat hippocampus during treadmill running with laser-Doppler flowmetry combined with microdialysis.

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In order to examine regulating mechanism behind enhanced cerebral blood flow (CBF) in rat hippocampus during exercise, we establish the new approach monitoring hippocampal CBF (hCBF) in rat with slow treadmill running (10 m/in), employing laser-Doppler flowmetry combined with microdialysis. We found that hCBF increased by 142% with treadmill running and the effect was mostly abolished by tetrodotoxin(15μM) infused through dialysis probe. The metabolic parameters (pH, PCO_2, PO_2) in venous blood concurrently altered respectively, but the extents of which were small and consistent during running, allowing stable hCBF monitoring. These results suggest that treadmill running would increases hCBF through activation of hippocampal neurons, and our approach would thus be useful to study the coupling of CBF to neural activity during exercise.

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P2-317 Neural mechanism of central fatigue, as revealed by fMRI study

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For understanding of neural mechanism of fatigue, we examined regional activities of brain those were task-dependent and independent during the session of fatigue using fMRI in the control and chronic fatigue syndrome (CFS) patient groups. Percent signal change per hour in the visual cortex after the session of fatigue by prolonged visual search was similar between the two groups. Before the session of fatigue, auditory cortex was activated by turning off the scanner sound independently of visual search trials for 1 s. During the session, activation in this brain region was decreased in the CFS group. These results suggest that decreased activity in the task-dependent brain region might be induced by adaptation, and that in the task-independent brain region might be induced by central fatigue.

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P2-318 Wavelength-dependent effect of near-infrared laser on ATP content in rat hippocampal slices

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Low power laser with near-infrared wavelength (diode laser, 830-nm) or a visible one (He-Ne laser, 632.8 nm) has been used to suppress various kinds of neuropathic pain. However, the action spectrum of the effect of laser has not yet been studied. We previously reported that irradiation with the 830-nm-laser light to the brain tissue increased ATP content both in anesthetized rat and in the hippocampal slices. Here we tested the effect of laser light with three different wavelengths on ATP content in the slices. Pre-incubation of the slices in the glucose-free medium for 60 min at 31 °C decreased in basal ATP content to 50%. Subsequent treatment with 830-nm-laser for 15 min recovered the content. On the other hand, laser with 652 nm or 940 nm had no effect. These suggest that laser irradiation modulates cellular metabolism in the wavelength-specific manner.

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P2-319 Localization of Glucokinase in Rat Hypothalamus

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Glucokinase (GK) has been proposed to play a role in the glucose-sensing mechanism, but the mechanism remains unknown. In this study, we measured glucose phosphorylating activities and GK mRNA expression in the hypothalamus and each nucleus in the hypothalamus from male Wistar rats, and data were compared with those from streptozotocin (STZ)-induced diabetic rats. Glucose phosphorylating activities were measured by fluorophotometric assay, and GK mRNA expression was measured by real time RT-PCR. GK activities were high in the arcuate nucleus, moderate to low in the ventromedial nucleus, lateral hypothalamic area and paraventricular nucleus, and very low in the cortex. The order of GK mRNA expression was the same as that of GK activities. GK activities in STZ diabetic rats were almost the same as those in nondiabetic rats.

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P2-320 Glutamine is a preferential energy substrate over glucose for neurons

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We investigated the metabolic trafficking between neurons and astrocytes in primary culture using ^{13}C-NMR spectroscopy and HPLC. Neurons, astrocytes and co-culture cells with transwells were incubated with DMEM (glucose free) for 12 hours and then incubated for 8 hours after an addition of [1, 6-^{13}C]-glucose into the medium. ^{13}C-NMR spectra of neuronal extracts showed the ^{13}C incorporation into [4-^{13}C]-Glu but not into Gln. Astroglial extracts showed the ^{13}C incorporation into [4-^{13}C]-Gln and only slightly into [4-^{13}C]-Glu. Neuronal extracts obtained from co-cultures, however, showed the ^{13}C incorporation into [4-^{13}C]-Gln. After 8 hour incubation, over 90% of ^{13}C signals were observed from [3-^{13}C]-lactate in the astrocyte and from ^{13}C-glucose in the neuron cultures. HPLC measurement of the medium revealed that Gln consumption of neurons and astrocytes was 0.45 and 0.14μmole/mg-protein/h, respectively. These results indicate that Gln may be a preferential substrate over glucose for neurons in primary culture.

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P2-321 Calcium and PKC dependent metabolic process for synaptic utilization of lactate in hippocampus

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We have reported that replacement of glucose with lactate can trigger calcium influx to sustain synaptic function. The aim of this study is to investigate this metabolic process. Synaptic function was estimated by the amplitude of population spikes, recorded in rat hippocampal granule cells and CA1 pyramidal neurons. Replacement with lactate induced the decline of synaptic function, followed by spontaneous recovery. Applications of inhibitors for NMDA and L-type calcium calcium channels or protein kinase C (PKC) suppressed the recovery of synaptic function. We measured the incorporation of 14C-lactate in the cultured hippocampal cells after replacement of glucose with lactate. Incorporation of lactate was gradually increased, which was suppressed by the applications of inhibitors for NMDA and L-type calcium calcium channels or protein kinase C. Results indicate that calcium influx and activation of PKC could induce some metabolic process toward lactate utilization to sustain synaptic function.

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P2-322 The effect of hemodilution on changes in local cerebral blood flow and field potential in rat somatosensory cortex evoked by hindpaw stimulation.

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The effect of hemodilution on changes in local cerebral blood flow and field potential in rat somatosensory cortex evoked by hindpaw stimulation. [a,b]Masakatsu Ureshi and [a]Iwao Kanno[a] Department of Radiology and Nuclear Medicine, Akita Research Institute for Brain & Blood Vessels, Akita, Japan[b] JSPS. We have investigated the effect of hemodilution on the relationship between local cerebral blood flow (LCBF) and field potential (FP) in rat somatosensory cortex. The LCBF was measured by Laser-Doppler flowmetry and the FP with a tungsten electrode while electrically stimulating the hindpaw during hemodilution induced by switching blood with hetastarch. We found that Hct decreased from 36.3±2.6% to 18.8±2.3% and the baseline LCBF increased by 42.5%. The FP evoked by hindpaw stimulation did not change during hemodilution. Also, the percentage change in evoked LCBF response was not significantly altered during hemodilution despite the increase in baseline LCBF.

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P2-323 Changes in amino acids levels caused by mental fatigue

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We assessed the change of various substances including general metabolites, neurosteroids, endocrine hormones, and amino acids after the session of mental fatigue. Fatigue session consists of Advanced Trail Making Test, Japanese KANA pick-up test, and mirror drawing test. The subjects were nine healthy volunteers (27.6 ± 5.5 years, 5 males and 4 females). We found that plasma branched-chain amino acids and aromatic amino acids levels were altered after the session of fatigue. These amino acids levels might be used as biomarkers for quantification of extent of fatigue.

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P2-324 Recovery and prevention from physical and mental fatigue: effect of fursultiamine on behaviors in an animal model of fatigue

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To evaluate the effect of vitamin B1 derivative (fursultiamine; FT) on fatigue, weight-loaded forced swimming test (WLFST) was performed after administration of FT (10-50 mg/kg, i.p.) for five days.
The swimming time of WLFST was increased in FT-treated rats as compared with that in saline-treated rats ($p<0.05$). Fatigued rats that were kept in a cage filled with water to a height of 1.5 cm for five days showed shorter swimming time than control rats. Swimming time was increased after administration of FT for five days in the fatigued animals ($p<0.05$). However, swimming time was not increased by administration of vitamin B1 (thiamine) in the control and fatigued animals.
These results indicated that FT might be effective for the attenuation of fatigue.

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P2-325 Functional brain mapping of thinking in monkeys and humans

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Logical thinking can be defined as the ability of problem-solving in order and in rational ways. The neural mechanism for logical thinking in macaque monkeys and humans was compared by positron emission tomography with $H_2{}^{15}O$ and functional magnetic resonance imaging, respectively. Subjects of both species (two monkeys and eight men) were measured regional cerebral blood flow when performing a rule-related task where they were required to move a shovel toward a given target presented on the table via joystick based on the rule between moves of joystick and shovel, followed by scanning a randomly varied rule each trial, compared to the joystick operation based on a fixed rule across trial. Monkeys may use the same cognitive strategy for the task as humans, where may involve the equivalent brain regions (prefrontal cortex, parietal cortex and cerebellum) in both species. These findings suggest that even monkeys as well as humans can think in rational manner.

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P2-326 Long time multiple-site optical recording system for neural activity

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We have newly developed an optical recording system for detecting neural activity continuously from 1020-site at the time resolution of 1 msec. The preparation is stained with a voltage-sensitive dye and placed under the system. Green LEDs provide the excitation light and fluorescent light is focused on a magnified real image using a tandem lens optic system. The fluorescent light intensity is detected simultaneously by a 34 x 34-element photodiode array. The outputs from each pixel are amplified, multiplexed, digitized and sent to a computer. The transferred data are written in a pair of memory blocks alternatively. After 4 sec (8 MB) data are written and the memory block switch over to another, the data are fed into a hard disk. It takes about 3.2 sec to read out 4 sec data and store in the hard disk, so this system makes it possible to record continuously for at least 1,000 sec. Since this limitation comes from the maximum file size, 2 GB, it is easy to expand the limitation. We applied this system to the rat somatosensory cortex.

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P2-327 Brain Activation during Algebraic Calculation - an fMRI study

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Brain activity during arithmetic (AR) and algebraic (AL) calculation were examined using fMRI. The experiment was ablock design that consisted with visual presentation of rest, AR, and AL. Subjects solved problems and chose answers by pressing one of the 3 buttons. The activation fields in AR-AL were in the precentral and paracentral lobule, frontal and fronto-parietal operculum, insula, superior temporal gyrus, supramarginal gyrus, hippocampus, and lenticular nucleus. Those in AL-AR were found in the middle, superior and medial frontal gyrus, supeiror and inferior parietal lobule, precuneus, lingual and fusiform gyrus. The results indicate that greater activation in the prefrontal and parietal cortices are necessary for AL compared to AR, and that the activation in AR-AL were attributable to the higher rate of arithmetic operations in AR than in AL.

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P2-328 Effects of serotonin concentration on *in vivo* binding of two PET tracers [^{11}C]McN5652 and [^{11}C]DASB to serotonin transporters

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[^{11}C]McN5652 and [^{11}C]DASB are the radiotracers used for quantification of the serotonin transporter (5-HTT) density in the living brain using PET. Recently, both tracers have been reported to be sensitive to detect changes in 5-HTT density due to MDMA, "Ecstasy." neurotoxicity. However, it is unknown whether these tracers are sensitive to a change in endogenous 5-HT concentration in the living brain. To assess this, we tested the effect of acute injection of 5-hydroxy-L-tryptophan (5-HTP), which is converted to 5-HT in neurons, on the binding potential (BP) of [^{11}C]McN5652 and [^{11}C]DASB in conscious rhesus monkeys. In parallel, we also measured the concentration of 5-HT in the brain by microdialysis technique to evaluate the effects of 5-HT on BP of both radiotracers.

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P2-329 Remains of infant days: Cortical magnetic responses from first generation American English speakers

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We examined an impact of the first language acquisition during infancy on second language representation in the auditory cortex by recording cortical magnetic responses. Subjects were American English (AE) speakers who were born and educated in United States and regarded English as their primary language. In one group, parents of the speakers were monolingual AE speakers (Monolinguals, n=11). In the second group, they learned AE only after they started to attend preschool because their parents were not native AE speakers (Late-Learners, n=11). We used a passive oddball task (deviant, 15%, standard 85%) using synthesized /la/ and /ra/ sounds. A mismatch field (MMNm) and P3a were consistently observed only for Monolinguals and these components were significantly reduced for Late-Learners.

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P2-330 Phase synchronization in theta rhythm indexes global cortical networks: A study of simultaneous fMRI and EEG

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In human scalp and intracranial EEG, the long-range phase synchronization was reported to associate with several cognitive functions, while limits of the spatial resolution in EEG prevent to uncover the spatio-temporal structure of the coherent ensemble in the whole brain. In the present paper, we proposed a method to grasp phase-synchronization associated neuronal activities from simultaneous fMRI and scalp EEG. Using this method in a mental calculation task, we demonstrated that task-dependent synchronization in theta rhythm is associated with negative BOLD in the posterior cingulate, perirhinal, and medial prefrontal cortices and with positive BOLD in the parietal areas. These findings indicate that global cortical networks including a limbic circuit are dynamically organized with phase synchronization in scalp EEG and suggest a possible role of theta rhythm in their control.

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P2-331 Neural activation during memory-dependent reasoning: An fMRI study

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We investigated fMRI activations during the memory-dependent reasoning. 12 normal healthy subjects participated. Before fMRI, subjects learned the complete (4-scene) and incomplete (3-scene) version of the 4-scene comic strips. During fMRI, subjects performed the story guess (SG), story recall (SR) and control (CN) tasks. In SG and SR, subjects were presented with the 1st scene and 2 selections of scenes, and were required to choose one scene appropriate to the last. In the former task, we employed the stimuli learned as the incomplete version whereas in the latter as the complete one. The two contrasts of SG with CN and SR with CN showed common activations in the bilateral superior parietal lobule and left middle frontal gyrus. In the contrast of SG with CN, the right middle frontal gyrus, left middle and inferior temporal gyrus, angular gyrus, and precuneus were specifically activated. The results suggest that these areas specific to SG may contribute to the memory-dependent reasoning.

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P2-332 Group I mGluR-induced calcium oscillation of CA1 pyramidal neuron in rat hippocampal slice culture preparation

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Group I metabotropic glutamate receptors (mGluR I), which couples to Gq, is expressed both in neurons and astrocytes in hippocampus and its selective agonist, DHPG is known to possess excitatory as well as inhibitory effects on neuronal activities and to induce transient and/or oscillatory calcium response in astrocytes. In order to investigate DHPG-induced hippocampal network activities, calcium responses of individual cells in rat hippocampal slice culture preparations were examined using confocal microscopy, and then DHPG was found to induce asynchronous calcium oscillation in CA1 pyramidal neurons. These oscillations were not affected by pre-synaptic inhibition using an adenosine receptor agonist, CCPA, however, abolished by an ionotropic glutamate receptor antagonist kynureic acid. Recent evidences of calcium dependent·glutamate release from astrocytes are supposed to underlie the mechanism for the present phenomena.

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P2-333 Brain activations associated with telling a lie and its importance: An fMRI study

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In the present study, we investigated neural activations associated with lying and its importance by using fMRI. Before fMRI, subjects were required to choose a card, on which a numeral was printed, and to memorize it. During fMRI, subjects were required to answer whether the numeral presented on the screen is larger or smaller compared with the one memorized previously. Subjects were required to answer the question honestly or telling a lie, alternatively. Subjects were instructed to be fined when the experimenter found the number. The penalty was selected either 100 yen or 1000 yen, so as to manipulate the importance of the lie. We found that the bilateral middle frontal gyri were significantly activated in lying. The right amygdala was apparently activated when a high penalty was charged. The former activation may reflect the inhibition of truth response during telling a lie. The amygdala activation suggests that a high cost of the penalty may cause the emotional response.

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P2-334 Visualizing system for ongoing translation from a specific mRNA using translation termination complex.

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We attempt to visualize ongoing translation by detecting an intermolecular reaction in the course of translation termination, using fluoroscense resonanse energy transfer (FRET). We transiently express a pair of FRET probe and an mRNA which was tagged with MS2 binding site just downstream of its terminator codon. The expressed mRNAs were tethered by one of the FRET probe whose entity was a Upf2-Upf3 fusion protein modified with an MS2 protein and YFP. The other FRET probe comprises of eukaryotic release factor (eRF3) and CFP. In this preparation, if translation from the tagged mRNA with YFP probe is promoted, an interaction between Upf2-Upf3 and eRF3 increases and this reaction can be detected as FRET. We show the result by applying this system to COS7 cell and PC12 cells and demonstrate that this can reflect a real-time imaging of translation level from the specific mRNA.

P2-335 A novel approach for visualizing spatiotemporal regulation of localized translation in dendrite of rat hippocampal neuron.

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Recent years a considerable number of studies have been made on the regulated localization of specific mRNAs in dendrite and/or axon. But little is known direct evidence of local protein synthesis in dendrite, not yet. So we take notice of eukaryotic initiation factors (eIFs), that are known to localize in dendrite, and aim to assemble of real time visualization system of local protein synthesis in dendrite by fluoroscense resonanse energy transfer (FRET). We examined detection of local protein synthesis in dendrite of rat hippocampal neuron stimulating by growth factors (BDNF etc) resulted increase emission ratio by FRET. Furthermore, region of upregulating translation was confinement in dendritic spine. So this new detection system is one of the potential manners of elucidation of molecular mechanism of synaptic plasticity in terms of local protein synthesis.

P2-336 Analysis of the origin of optical signal recorded in the primary motor cortex of the monkey brain.

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Real-time optical imaging of the brain activity with a voltage-sensitive dye allows us to detect neuronal activity with a high spatio-temporal resolution. Currently we are studying the functional architecture of the motor region of monkey brain with the method. However, it is not easy to explain the origin of the optical signal, since the signal is the sum of various kinds of membrane potential changes, e.g., EPSPs and action potentials of the neurons of several kinds. The aim of this study is to get a clue with which we analyze the origin of the optical signal obtained in the *in vivo* optical recording. In this study, for example, we stimulated neurons of the primary motor cortex (MI) orthodromically or antidromically, and the resultant optical responses were compared. Antidromic stimulation to MI neurons was performed by the electrical stimulation to the corticospinal axons, whereas orthodrmic one was made by stimulating the neurons of premotor cortex projecting to MI neurons.

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P2-337 Inspiration-related alpha band synchronization in human olfaction: locations of source generators estimated by a dipole tracing method.

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The present study was performed to determine whether the respiratory pattern is affected by pleasant and unpleasant olfactory stimuli at the odor threshold and recognition levels, and whether the phase from inspiration onset each odor stimulus is related to EEG cortical activity. We also sought to investigate the locations of dipoles estimated from averaged EEG potentials triggered by the onset of inspiration during an odor stimulus. During olfactory stimulus, inspiration-related alpha band synchronization was found in the averaged potential triggered by inspiration onset. EEG dipole tracing identified the location of dipoles from the potentials in the limbic area and the cortex; the entorhinal cortex, hippocampus, amygdala, premotor area, and centroposterioe orbitofrontal gyrus subserve odor threshold, and the rostomedial orbitofrontal gyrus subserve odor recognition.

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P2-338 Autofluorescence imaging of functional connections between S1 and S2 in rat somatosensory slices

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Somatosensory information is processed by the primary (S1) and secondary (S2) cortices. We investigated the functional connections between S1 and S2 in rat cortical slices. Neural activities elicited by repetitive electrical stimulation were visualized with flavoprotein autofluorescence imaging. The stimulation applied in S1 produced neural responses spreading into S2. However, the neural activities were not invaded into S1, when S2 was stimulated. These responses were almost completely inhibited by a mixture of CNQX and APV, suggesting the polysynaptic nature. We also investigated the functional connections with field potential recordings. Slow negative potentials were elicited by repetitive stimulation. Distribution of the slow potentials confirmed the results obtained with flavoprotein autofluorescence imaging. The anisotropic propagation of neural activities demonstrated in vitro probably reflects the physiological information flow from S1 to S2 in vivo.

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P2-339 NO and prostanoids mediate activity-dependent cerebral vasodilation in mouse somatosensory cortex

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We investigated the mechanisms underlying activity-dependent cerebral vasodilation with flavoprotein autofluorescence imaging. C57BL/6 mice were anesthetized with urethane and the surface of the skull was covered with clear acrylic resin for keeping the transparency of the skull. Cortical images of green autofluorescence in blue light were recorded in the primary somatosensory cortex through the intact skull. The autofluorescence reflecting neural activities was increased by vibratory stimulation applied on the contralateral hind paw skin. Darkening of the arterial images reflecting vasodilation followed these responses. Local application of N-nitro-L-arginine (100 μM), an inhibitor of NO synthase, on the surface of the cortex selectively suppressed the vasodilation. Indomethacin (10 mg/kg, i.p) weakly attenuated the vasodilation without changing neural responses. From these results, we concluded that NO and prostanoids mediated the activity-dependent vasodilation in the mouse somatosensory cortex.

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P2-340 fMRI and NIRs data fusion: a dynamical model based on a bottom-up approach

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The simultaneous recording of fMRI and NIRs represents nowadays an advanced technique to explore brain activations. However, there is a dilemma related to methodological and technological restrictions that resided in the physical basis of BOLD signal acquisition and in the inverse problem rising from the optical tomography. In this paper, a new methodology to properly use data obtained from separated observation of BOLD and optical signals is proposed on the basis of: a) a ROI selection criterion based on images standardization, b) non-linear regression to remove signal drifts, c) the introduction of a factor that control the effective transparency of capillary bed and d) finally, using of a bottom-up model for a common non-linear dynamics, where physiological parameters are fitted by using LL filter strategy.

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P2-341 Impact of modeling heteroscedastity on fMRI time series analysis

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In serial regression analysis, it is important to grasp the character of error term. For valid statistical inference, we must require the justification of assumptions of residuals, such as independence and homoscedasticity. We reported that when the particular form of the task-correlated motion was contaminated in fMRI data, the time series showed task-related heteroscedasticity. In this study, we will investigate the impact on modeling heteroscedastity in fMRI time series analysis. Both autocorrelation and heteroscedasticity was modeled using appropriate covariance constraints and estimated though Restricted Maximum Likelihood scheme. Finally, we evaluated the effects of modeling the heteroscedastity in terms of an improvement in the specificity and sensitivity of statistical inference.

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P2-342 Trans-cranial imaging of awaking mouse cortical activities using flavoprotein autofluorescence

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The transparent skull of mice allows trans-cranial imaging of cortical activities using flavoprotein autofluorescence. We applied this method in awaking mice. A C57BL/6 mouse was anesthetized, and the skull was thinned and covered with transparent resin for preventing infection and keeping the transparency of the skull. A few days after the surgery, the head of the awaking mouse was fixed by screwing a metal piece attached to the skull. Neural activities elicited by sound stimuli were observed in the auditory cortex. The responses elicited by monotonous stimuli were rapidly attenuated after repeated exposure to the stimuli, while the responses elicited by mixed sound stimuli or by sound stimuli paired with an air puff on the face were maintained in the cortical area including the posterior association area. These results indicate the importance of the meaning or novelty of stimuli for activating the posterior association area, and the usefulness of the present method in awaking mice.

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P2-343 Validity of source localization of interictal epileptic spike potentials estimated by the dipole tracing method.

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Calculation of dipole tracing method is performed taking account for the conductivities of the brain, skull and scalp, and it must be performed in the own head model created from the each subject's MRI or CT. However, the artifact and noise could be affect on the estimation of dipole location. In this study, we investigated the convergence of dipole localization if noise were artificially added to the single and the averaged potentials. Noise were added to the single interictal epileptic spike and the averaged interictal epileptic spikes, and we compared the dipole locations estimated from these two potentials. Dipoles estimated from the averaged potentials were converged more smaller extent than those from the single potential, but the centers of dipole localization were both corresponded.

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P2-344 Evaluation of a shield for extra-FOV activity for a rat brain study with microPET P4

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MicroPET is a PET scanner for small animal study, which has 2 mm FWHM and performs 3D acquisition only. Although 3D acquisition is essential for high sensitivity, this acquisition mode is influenced by the radioactivity from extra filed of view (FOV). The lead shield box of 7 x 8 x 9 cm with 9 mm wall thickness was designed to reduce the influence. The effects of this shield on noise equivalent count rate (NECR), scatter fraction (SF) and ROI value were investigated with a rat phantom set, which consists of a head and a body part, both parts were filled with FDG solution and total activity ratio of the head and body was ca. 1:20. NECR was improved clearly for >150 kBq/ml. SF was reduced from 30% to 10% and non-uniformities of SF and ROI value along with the axial direction were also reduced. The linearity of ROI value against activity was confirmed for <500 kBq/ml. Thus, the shield can improve accuracy of rat brain studies and allows high injection dose to improve image quality.

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P2-345 Concurrent imaging of multiple radioactive tracers with a Compton camera

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A practicable prototype of a collimator-less γ ray emission imaging (GREI) system has been developed. The system is a Compton camera that is composed of a pair of planar double-sided orthogonal strip high purity germanium detectors for three-dimensional detection of the interaction points. Currently, the effective FOV is approximately 100 cm^2 and the resolution is 4 mm FWHM. The efficiency is 3 x 10^{-5} at 1115 keV. As the system can detect a broad range of γ rays between 200 keV and 2500 keV at a high energy resolution, it is suitable for imaging the multi-tracer that contains multiple radioactive nuclides, each of which can tag a separate biochemical process, concurrently under an identical experimental condition. In addition to the conventional tracers such as ^{15}O and ^{18}F, metals such as ^{65}Zn and ^{54}Mn can be used, which makes GREI a unique imaging modality.

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P2-346 Development of the simple analysis system for neuronal molecular function with spatial and temporal resolutions

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Inactivation of molecule with temporal and spatial resolutions without special equipment is a powerful tool for analysis of molecular function in living cells. For the establishment of this approach, we developed a method that the target molecule can be inactivated by employing fluorophore-associated light inactivation (FALI). We first constructed an assay system using COS-7 cells expressing Neuropilin-2 (Npn2) and Plexin-A2. These cells shrank in response to Semaphorin 3F (Sema3F). In this system, the cells were irradiated with conventional light (60w bulb), after incubation with FITC-labelled monoclonal antibody against Npn2. This manipulation, FALI of Npn2, resulted in remarkable inhibition of Sema3F binding to its receptor, Npn2 and eventual cell shrinkage. We try to apply to this simple method to the cell and organotypic cultures of the nervous system.

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P2-347 Optical monitoring of membrane potential changes of neurons using protein-based voltage-sensitive probe and neuron specific vectors

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We developed a novel probe system for monitoring membrane potential changes using fluorescence resonance energy transfer (FRET) between modified GFP and oxonol. In this study, we evaluated the performance of this probe-system and summarized as follows: 1. The size of signal change of this probe system associated with membrane potential change was 10 to 100 times larger than that of the conventional dyes, 2. The change in the size of FRET signal had a highly linear relationship with the membrane potential change, 3. The time constant of the FRET signal change was approximately 0.5ms. We introduced this probe system selectively into neurons using recombinant rabies virus vectors we developed, and succeeded in recording the membrane potential change of individual neurons. The probe-vector system developed in this study is expected to work as a powerful tool for analyzing the higher function of the brain.

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P2-348 Recombinant rabies virus vectors for neuroanatomical tracers.

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Rabies virus (RV) is characterized by its neuron specific infection and transneuronal transport via synapses. These features make this virus a unique candidate for a novel neuroanatomical tracer. Recently, we developed new recombinant RV vectors with enhanced expression of foreign genes and neuron specificity by adding designed mutations to the RV genome. In the present study, we evaluated the recombinant vectors as a neuroanatomical tracer. We microinjected the recombinant vectors expressing GFP or β-Gal into the rat hippocampus and examined distribution of protein-labeled neurons. We confirmed that the vectors infected only from axon terminals. Furthermore, they were transported only retrogradely with a time-dependent manner. Expression of the marker proteins were strong enough to identify infected neurons. These results demonstrated that the recombinant RV vectors are potential neuroanatomical tools to visualize functional neuronal chains in the central nervous system.

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P2-349 Cell cycle regulation of by Amida

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Amida was isolated as a protein that associates with Arc, which is a neuron specific immediate early gene product. Previous studies have shown that Amida is developmentally regulated in rat testis and overexpression induces apoptosis. In this study, flow cytometry analysis showed that Amida caused cell cycle inhibition by blocking cell cycle from entry into mitosis. We also found that Amida interacts with Cdc2 and causes decrease in Cdc2 kinase activity. A region (aa, 76-189) was found to be responsible for DNA-binding activity and cell growth inhibition. This region also contains Ser-180, the phosphorylation site by Cdc2 kinase. PACAP treatment of Neuro2A neuroblastoma cells is known to down-regulate cell proliferation. We found that Amida expression was induced by PACAP, suggesting the function of Amida to regulate cell cycle.Taken together, these results suggest that Amida plays an important role in cell cycle interacting with Cdc2 kinase.

P2-350 A novel culture method for rat hippocampal slice using a serum-free culture medium

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Brain slice culture preparations are advantageous to examine the physiological as well as pathological effects of soluble factors, including growth factors and cytokines on cells in neural tissue, however most of the previous methods require culture medium containing serum, which is supposed to cause unidentified effects. Therefore, we considered culture methods using serum-free medium is important to obtain definite and reproducible results, and then screened culture conditions for viable neurons even in the absence of serum. As results, αMEM containing B27 supplement was found to be useful for neuronal survival in rat hippocampal slices, and lowering incubation temperature to 33°C further improved the viability. Under this condition, total and neuronal cell numbers were not significantly altered, but GFAP immunoreactivity reduced.

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P3-001 OPTICAL IMAGING OF NEURAL NETWORK ACTIVITY IN THE VESTIBULO-OCULOMOTOR REFLEX OF THE CHICK EMBRYO

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In comparison with molecular/anatomical approaches to brainstem neurons, developmental studies of functional connections have lagged behind because electrophysiological techniques are limited in the number of cells recorded simultaneously. Here we use an optical technique to visualize activity in emerging vestibular/auditory circuits in the embryonic chick brainstem. Dissected brainstems at E5-9 were labeled with a voltage-sensitive dye, NK2761. Vestibular/cochlear afferents were stimulated and a 1020-element photodiode array recorded membrane potential changes in large areas of the brainstem. Signals related to synaptic potentials in 1st- and 2nd-order vestibular/cochlear nuclei, and in ocular motoneurons involved in vestibulo-oculomotor reflexes, were visualized at appropriate latencies representative of conduction and synaptic delays. Inhibitory and excitatory potentials could be discriminated by pharmacological blockade of GABA and glutamate receptors.

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P3-002 OPTICAL MAPPING OF NEURAL CIRCUIT FORMATION IN THE CHICK VAGAL PATHWAY DURING EARLY EMBRYOGENESIS

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The optical imaging with a voltage-sensitive dye was used to survey extensively the early functional organization of neural circuits related to the vagus nerve in the embryonic chick brainstem. In addition to the response area on the stimulated side, including the vagal sensory and motor nuclei (NTS, DMNV), vagal stimulation induced an additional response area at the level of the cerebellum on the contralateral side. This response appeared to come through the 2nd synaptic pathway from the NTS to the contralateral side. Blockade of non-NMDA receptors in the NTS abolished the responses on the contralateral side, while blockade of NMDA receptors did not abolish the responses. The responses on the contralateral side were first detected from the E7 stage, *when the glutamatergic EPSP in the NTS was first* observed. This result suggests that the 2nd synaptic pathway from the NTS to the contralateral side is already generated when the primary vagal afferents make functional synapses on the NTS neurons.

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P3-003 Postnatal development of vesicular glutamate transporter 1 (VGLUT1) and VGLUT2 in the mouse forebrain

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Vesicular glutamate transporter 1 (VGLUT1) and VGLUT2 are distributed in a complementary fashion in the adult neocortex; VGLUT1 and VGLUT2 are localized in the nerve terminals which derived from the cortex and the thalamus, respectively. To date, however, it has not been well understood how VGLUT1 and 2 are involved in the development of glutamatergic circuitry. Thus, we studied the development by immunohistochemistry for VGLUT1 *and VGLUT2 in the developing murine forebrain from postnatal* day (P) 0 to P22. Even at P0, VGLUT1 and VGLUT2 immunoreactivities (IRs) showed almost no colocalization in single axon terminals, like in the adult brain. In the primary somatosensory cortex at P0, VGLUT1-positive terminals were distributed in layers 1 and 6, and the subplate, whereas VGLUT2-positive terminals were observed in layer 1, layers 4-6, and the subplate. During postnatal ontogeny, these patterns of VGLUT IRs shifted gradually and differentially into their mature forms.

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P3-004 Homeobox genes, eyeless and homothorax, control differentiation of projection and local-inter neurons in the Drosophila antennal lobes.

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Antennal lobes (AL) are the primary association centers for odor recognition in the Drosophila brain. The neural network of ALs consists of three cell types: olfactory receptor neurons, local inter neurons (LNs) and projection neurons (PNs). Although both LNs and PNs are generated by AL neuroblasts, mechanisms of cell type differentiation are poorly understood. *Here we show that a Drosophila Pax-6 homolog, eyeless (ey), is expressed in* a group of LNs whereas another homeobox gene, homothorax (hth), is expressed in PNs. Ectopic expression of hth in LNs partially transformed LNs into PNs with dorsal axonal projections. This phenotypes was further pronounced with concomitant suppression of ey. Currently, we are investigating developmental lineages of the PNs and LNs as well as cell autonomous functions of ey and hth by MARCM mosaic analyses.

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P3-005 Neuronal expression of chondroitin sulfate proteoglycan RPTPβ/phosphacan

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Receptor-type protein-tyrosine phosphatase β (RPTPβ)/phosphacan is a brain specific chondroitin sulfate proteoglycan. The present study examined the expression and localization of RPTPβ/phosphacan in developing neurons *in vivo* and *vitro*. RPTPβ/phosphacan immunoreactivity was observed at Golgi complex as well as neuronal surface in cultured cortical neurons and neuronal membrane in vivo developing cortex. At earlier stages of culture, the immunoreactivity of RPTPβ/phosphacan was observed entirely on neuronal surface such as somata, axons, and dendrites, while after longer culture it was detected at dendrites and somata and never detected at synapses. The addition of 6B4 RPTPβ/phosphacan antibody into neuronal culture promoted the axonal elongation. These results suggest that RPTPβ/phosphacan is produced by neurons and concerned with neuronal development.

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P3-006 Generation and analysis of Semaphorin 3F knockout mice

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Axon guidance is essential not only in the formation of neuronal circuitry but also in its refinement in the adult central nervous system(CNS). Semaphorin 3F (Sema3F) is a secreted type of the Semaphorin family of axon guidance molecules. In order to examine the physiological roles of Sema3F, we generated Sema3F knockout mice. The targeting vector contained the lacZ gene knocked into the mouse Sema3F locus and the neomycin resistance gene flanked by loxP. Chimeric mice obtained from the resultant homologous recombinant ES clones were bred to C57BL/6 mice and yielded Sema3F (loxP-neo/+) mice. By breeding Sema3F (loxP-neo/+) mice to EIIa-Cre transgenic mice, we obatained Sema3F (loxP/loxP) mice. In this presentation, we report our initial characterization of Sema3F knockout mice.

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P3-007 Three-dimensional computerized distribution of pontocerebellar projection neurons in the normal and reeler mice

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The reeler (rl/rl) mouse displays a disruption of laminar structures such as cerebral and cerebellar cortix. The non-laminated structures in the reeler brain had long been considered to be normal. We injected HRP enzyme into various areas of the cerebellar cortex of the normal and reeler mice to examine distribution pattern of pontocerebellar projection neurons and to make a spatial 3-D computer reconstruction of labeled neurons in pontine nuclei and reticulotegmental nucleus of pons. HRP-labeled cells were found in the inferior olivary complex, cuneate nucleus, lateral reticular nucleus, pontine nuclei and reticulotegmental nucleus of pons. In the reeler mouse, pontocerebellar projection neurons innervating the cerebellar cortex were distributed widely both in the rostrocaudal axis and in the mediolateral axis in comparison with those of the normal mouse. These data elucidate that the Reelin protein corded by reelin gene may play a key role in the cell positioning or migration of pontocerebellar neurons.

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P3-008 Identification of Splicing Variants of Rapostlin, a Rnd2 Effector that Interacts with N-WASP and Induces Neurite Branching

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Rho family GTPases regulate neuronal morphology. Of them, Rnd2 is specifically expressed in brain. We recently identified Rapostlin (Rapo) as a novel Rnd2 effector. Rapo induces neurite branching in response to Rnd2 in PC12 cells. We have found two splicing variants of Rapo (renamed as RapoL), RapoM and S with deletion within the unique insert region. RapoL is predominantly expressed in brain, whereas RapoS is expressed ubiquitously. All variants bind to Rnd2. However, RapoM and S induce less neurite branching when coexpressed with Rnd2 in PC12 cells, indicating that the insert region is important for branching activity. All variants bind to N-WASP through the SH3 domain, and this domain is essential for branching activity. Rnd2 reduces RapoL-N-WASP interaction *in vivo*, whereas it has little effect on the interaction of RapoM or S with N-WASP. Therefore, we found that functionally different splicing variants of Rapo have different responses to Rnd2 in association with N-WASP.

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P3-009 Small GTPase Rnd1 is essential for the semaphorin 4D-induced growth cone collapse in hippocampal neurons

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Plexins function as receptors for the repulsive factor semaphorins and regulate axon guidance in the developing nervous system. We previously showed that the Rho family small GTPase Rnd1, which is highly expressed in hippocampal neurons in the developing nervous system, directly interacts with the semaphorin 4D (Sema4D) receptor Plexin-B1. To investigate the functional significance of Rnd1 in the Sema4D signaling in neurons, we constructed an expression vector encoding an Rnd1-specific short interfering RNA (siRNA) to knock down Rnd1 protein in rat primary hippocampal neurons. The Rnd1 siRNA effectively reduced the amount of Rnd1 protein in hippocampal neurons. Co-expression of GFP and the Rnd1 siRNA in hippocampal neurons significantly suppressed the Sema4D-induced growth cone collapse. We conclude that Rnd1 is necessary for the Sema4D-induced growth cone collapse in hippocampal neurons. We are now further investigating the pathway of the Sema4D signaling involving Rnd1.

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P3-010 The role of NADPH oxidase to neurite outgrowth induced by NGF in PC12

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The role of NADPH oxidase in neurite outgrowth was investigated in PC12 cells. Along with the outgrowth of neurite induced by NGF, the expression of NOX1, a catalytic subunit of NADPH oxidase, was significantly increased in a time-dependent manner. On the other hand, the expression of NOX2, another isoform of NOX1, was progressively decreased up to 5 days after NGF treatment. When inhibitors of NADPH oxidase were included in the medium, both diphenyleneiodonium and apocynin significantly enhanced the neurite outgrowth induced by NGF in 96 hours. Furthermore, addition of EUK-8 that possesses activity of SOD and catalase at 48 hours after the exposure to NGF significantly enhanced the neurite outgrowth. Finally, the NGF-induced neurite outgrowth was augmented in the cells depleted with NOX1 transcript by stable expression of ribozymes targeted for NOX1 mRNA sequence. Taken together, increased NADPH activity elicited by up-regulation of NOX1 may modulate neurite outgrowth in PC12 cells.

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P3-011 Role of Sema3A in dendritic branching and spine formation of mouse cortical neurons

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An anxonal guidance cue, Semapholin-3A (Sema3A) is known to activate a tyrosine kinease, Fyn through its receptor complex. Aberrant phenotypes were found in dendritic growth in the layer V cortical neurons in sema3A and fyn deficient mice. We observed significant decrease of spine density of the layer V cortical neurons in their deficient mice. To examine roles of Sema3A in spine formation, cultured cortical neurons was exposed to Sema3A and immunostained with anti-PSD-95 or anti-MAP2 antibodies. We found that Sema3A significantly increased the PSD-95 immunopositive deposit and the number of MAP-2 positive dendritic branch. These Sema3A effects were blocked by a tyrosine kinase inhibitor, lavendustin-A. Our date suggest that Sema3A plays an important role in spine formation and branching in developing cortical neurons.

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P3-012 Axon and dendrite branching patterns in GABAergic nonpyramidal cells

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GABAergic nonpyramidal (NP) cells are composed of many subtypes in terms of firing property, neurochemical expression, and morphology. We analyzed quantitative morphological properties of each subtype in the rat frontal cortex. NP cells were divided into three physiological subgroups: fast spiking (FS), late spiking (LS), and non-FS cells. Histogram of axon and dendrite internode intervals was well fitted by single exponential with characteristic decay constant for each subtype. It suggests branching occurs according to Poisson process. The number of primary dendrites was higher in FS and LS cells than non-FS cells. The proximal dendrite elongated vertically in some non-FS cell groups. The spatial compactness around the parent soma was distinct among subtypes, both on axon and dendrite. Somatostatin-positive Martinotti cells had more spines than other types. These subtype-specific axonal and dendritic phenotypes are possibly related to the distinct input and output organization for individual subtypes.

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P3-013 Neuroglycan C, a brain-specific chondroitin sulfate protenglycan, promotes neurite outgrowth from rat neocortical neurons.

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Neuroglycan C (NGC/CALEB) is a transmembrane-type chondroitin sulfate proteoglycan that is exclusively expressed in the central nervous system (CNS). To investigate the function of NGC, we examined effects of recombinant NGCs on fetal rat neocortical neurons in culture. The ectodomain of NGC enhanced neurite outgrowth from neurons. The active site for promotion of neurite outgrowth was localized within the EGF-domain and acidic domain of NGC. The EGF-domain preferably elongated one neurite from the soma, while the acidic domain tended to elongate several neurites. The EGF-domain also enhanced neurite outgrowth from GABA-positive neurons. These results suggest that the EGF-domain and acidic domain exert a distinct effect on neocortical neurons, and that NGC is involved in the neuritogenesis in the developing CNS.

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P3-014 Stathmin regulates dendritic microtubule stability of developing Purkinje cells that might be important for the olivocerebellar synapse elimination

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Each Purkinje cell (PC) is innervated by multiple climbing fibers (cf) during early postnatal period. Selective synapse elimination confers a mono-innervation of cf to a PC in adulthood. We previously identified a microtubule (MT)-destabilizing factor, Stathmin, as one of the candidates that are involved in the olivocerebellar synapse elimination. During P12-18 (critical period of the elimination), *stathmin* expression was gradually reduced in PC, which stabilized dendritic MT concomitantly extended with the dendrites growth. Stathmin overexpression dramatically limited the extension of dendrite, but not axon, of primarily cultured PC. Transgenic mice, in which L7 promoter drives Stathmin expression in a PC-specific manner, showed a decline in MAP2 signal in PC dendrites, and a motor discoordination. Our results suggests that Stathmin-caused MT-destabilization attenuates the elimination of olivocerebellar synapses.

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P3-015 Development of cerebellar network connection from inferior olive and pontine nucleus in neonatal rats

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The cerebellum-pons-medulla preparation from neonatal rat including inferior olive nucleus (IO), pontine nuclei (PN) and cerebellar nuclei is useful for analyzing activities in the cerebellar neuronal network. We examined the activity pattern induced by stimulation of IO, parallel fibers (PF) and PN, and the trace of projection from IO, PF and PN by DiI/DiD in the cerebellum-pons-medulla preparation dissected from 1-8-day-old rats. Optical imaging using voltage-sensitive dye and DiI tracing indicated that axons of inferior olive nucleus (principal olive) projected to flocculus, paraflocculus and Crus I /II as early as P1 rat. A drastic change was observed in 6-8 days-old rats with appearance of a parasagital band pattern. Moreover, axons from pontine nucleus projected to lateral edge of cerebellum and vermis area at P4/P5 postnatal days. Results suggest that PN-mossy fiber and granule cell-PF connections, and IO projection were built up during early postnatal period in the lateral edge of the cerebellum.

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P3-016 Calcium-Regulated Local Protein Synthesis Within Nerve Growth Cones

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Nerve growth cones are known to contain many kinds of mRNA. The functions of local protein synthesis within growth cones, however, remains unclear. We examined the role of eukaryotic elongation factor-2 (eEF2), a factor required for protein translation, and known to be regulated by calcium signaling. Immunocytochemistry revealed high expression of dephosphorylated eEF2 in the extending growth cones of cultured DRG neurons. In response to an artificial influx in calcium (known to induce neurite retraction), the phosphorylation of eEF2 was increased within growth cones. Since dephosphorylated eEF2 promotes protein translation, it is suggested that local protein synthesis within growth cones via eEF2 phosphorylation may be involved in calcium-regulated neurite outgrowth.

P3-017 Identification of novel members of semaphorin family

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Semaphorin gene family contains a large number of secreted and transmembrane proteins, and some of them are functioning as the repulsive and attractive cues of the axon guidance during development. Here we report murine orthologues of a novel member of class 6 semaphorin gene, semaphorin 6D (Sema6D) and class 3 semaphorin gene, semaphorin 3G (Sema3G). Sema6D is mainly expressed in the brain and lung, and the ubiquitous expression in the brain continues from embryonic late stage to adulthood, as determined by Northern blot and in situ hybridization. We also found that Sema6D has five different splicing variants, and the expression patterns of individual isoforms differ depending on the tissues. Thus, Sema6D may play important roles in various functions including the axon guidance during development and neuronal plasticity. Sema3G is mainly expressed in the lung, but the interesting specific expression is detected in the brain. Sema3G can bind Neuropilin-2, not Neuropilin-1.

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P3-018 Development and Organization of Tectobulbar Tracts involved in Visuomotor Transformation in Zebrafish

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The optic tectum is a visual center in vertebrates. It is known that startle responses are evoked by visual stimuli as well as by sound, and the activation of efferent neurons within the deep layers of the tectum can generate orienting or escape movements. However, it has not yet been well understood, how the tectum sends the motor commands to the hindbrain. Zebrafish is a vertebrate model system for a genetic analysis of neural network formation and behaviors. Using *brn3a-hsp70:GFP* construct, we established stable transgenic lines and visualized neural networks involved in visuomotor transformation in zebrafish, and we showed that the tectobulbar tracts made highly dense contact with ventral dendrites of reticulospinal neurons in the hindbrain. We also examined the development of the tectobulbar tracts. We found that the extension of dorsal branches of the tectobulbar tract was correlated with the development of visuomotor behaviors.

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P3-019 Analysis of a chick homologue of LINGO-1, a novel component of the Nogo-66 receptor/p75 signaling complex

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We previously reported a novel gene, named clone 10B4, encoding a transmembrane protein containing 12 leucine-rich repeats and an Ig domain by signal sequence trap from a cDNA library of chick embryonic motor neurons. Recently we found that 10B4 is a chick homologue of LINGO-1, a component of the Nogo-66 receptor/p75 signaling complex (Mi *et al. Nat. Neurosci.* **7**, 221-228, 2004). In the central nervous system, chick LINGO-1 expression is seen in forebrain, hindbrain and ventral part of spinal cord from early developmental stages. Chick LINGO-1 is also expressed in the dorsal root ganglion cells, especially in the ventrolateral region. Furthermore, we found that LINGO-1 is also expressed in the muscle, a target of motor neurons. These results suggest that LINGO-1 may play some roles in the development of the nervous and the muscular system.

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P3-020 The Ras GAP-homologous domain of Plexin-B1 is functionally regulated by the small GTPase Rnd1

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Plexins are receptors for the axon guidance molecules semaphorins. We recently reported that Rnd1 directly interacts with the cytoplasmic domain of Plexin-B1 and this interaction is necessary to induce Sema4D-mediated cell contraction. B-plexins interact with PDZ-containing GEFs at the C-terminus PDZ-binding motif and this interaction is of functional importance in mediating growth cone collapse/neurite retraction. Apart from this motif, the cytoplasmic domain of plexins is highly conserved among the family and shows sequence similarity to Ras GTPase activating proteins (Ras GAPs); however no functional role has been revealed yet. We revealed here that Rnd1 binding to Plexin-B1 is required for exhibiting GAP activity and resultant repulsive responses mediated by Sema4D. We are currently investigating molecular mechanisms for regulating GAP activity of Plexin-B1 by Rnd1.

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P3-021 Multiple defects in the pathway formation of rat cerebral cortex after prenatal X-irradiation

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In the present study, we examined the formation of cortical afferent and efferent pathways in rats which had received X-irradiation (1.0 Gy) on embryonic day 14 (E14) and obtained the following results. 1. Subplate neurons, a transient target of thalamic afferent axons, were partially destroyed by X-irradiation and the thalamocortical axons misrouted in the cortical anlage. 2. Subcortical heterotopia was formed after X-irradiation and obstructed the pathway formation of cortical efferent axons. 3. Cystic cavities which were formed around the cortex-striatum boundary region between E15 and E17 interrupted the normal formation of the internal capsule containing both cortical afferent and efferent axons. These results indicate that X-irradiation during initial cortical development causes multiple defects in the formation of cortical afferent and efferent pathways.

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P3-022 Large-scale identification and characterization of genes with asymmetric expression patterns in the developing chick retina

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To understand the molecular basis of topographic retinotectal projection, an overall view of the asymmetrically expressed molecules in the developing retina is needed. We performed a large-scale screening using restriction landmark cDNA scanning (RLCS) in the embryonic day 8 (E8) chick retina, and finally identified 33 asymmetric molecules along the naso-temporal (N-T) axis and 20 along the dorso-ventral (D-V) axis were identified. These included transcription factors, secretory factors, transmembrane proteins, and intracellular proteins with various putative functions. Their expression profiles revealed by in situ hybridization are highly diverse and individual. The molecular repertoire revealed by this work will provide candidates for future studies to elucidate the molecular mechanisms of topographic retinotectal map formation.

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P3-023 Kinesin superfamily protein 2A (KIF2A) functions in suppression of collateral branch extension

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During neuronal development, postmitotic neurons develop primary axons extending toward targets, while other collateral branches remain short. Although the process of collateral branching is important for correct wiring of the brain, the mechanisms involved are not well understood. In this study, we analyzed *kif2a*$^{-/-}$ mice, whose brains showed multiple phenotypes, including aberrant axonal branching due to overextension of collateral branches. In *kif2a*$^{-/-}$ growth cones, microtubule-depolymerizing activity decreased. Moreover, many individual microtubules showed abnormal behavior at the *kif2a*$^{-/-}$ cell edge. Based on these results, we propose that KIF2A regulates microtubule dynamics at the growth cone edge by depolymerizing microtubules and that it plays an important role in the suppression of collateral branch extension.

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P3-024 An essential role of Vav2/Vav3 in NGF-induced Rac1/Cdc42 activation and neuritogenesis in PC12 cells

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It has been widely accepted that Rac1 and Cdc42, members of Rho-family GTPase, promote neurite outgrowth through reorganization of actin cytoskeleton. By use of GFP-based FRET probes, we have shown that Rac1 and Cdc42 are diffusely activated at the periphery of PC12 cells just after NGF addition, followed by localized and repetitive activation at neurite tips. Further analyses have suggested that NGF-induced activation of Rac1/Cdc42 appears to be mediated by common GEFs (Aoki et al., JBC 279: 713, 2004). Candidates for such GEFs are Vav2 and Vav3, which are expressed ubiquitously and activate a broad range of Rho GTPases. To test this possibility, we employed RNAi techniques. In PC12 cells depleted of both Vav2 and Vav3, NGF-induced Rac1/Cdc42 activation and neuritogenesis were both inhibited markedly. In contrast, in PC12 cells depleted of both Sos1 and Sos2, NGF-induced Rac1/Cdc42 activation was not inhibited. These results revealed an essential role of Vav2/Vav3 in neuronal morphogenesis.

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P3-025 Visualization of visual pathways in ascidian larvae using wheat germ agglutinin (WGA) transgene (II)

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Ascidian larvae share a basic body plan with vertebrates. The larva has a central nervous system with about 100 neurons, which is simple enough to allow every neural pathway to be known. The visual pathways of the larva were visualized by expressing a WGA transgene in the photoreceptor cells under the control of the arrestin gene (*Ci-arr*) promoter. In order to discriminate the first-order neurons from the higher-order neurons a transgene construct containing both sequences of *Ci-arr-WGA* and *Ci-arr-EGFP* was introduced into fertilized eggs by electroporation. Immunofluorescence labeling revealed that WGA and GFP transgene products were expressed in the photoreceptor cells and WGA transgene products were transported along their axons and transsynaptically to the second neurons in the brain vesicle and then the motor neuron in the visceral ganglion in ascidian larvae.

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P3-026 Expression of aryl hydrocarbon receptor induces neurite outgrowth in Neuro-2a cells

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The intracellular aryl hydrocarbon receptor, AhR, is a ligand-activated transcription factor that mediates the response to environmental endocrine disruptors, EDs, such as 2,3,7,8-tetrachlorodibenzo-p-dioxin, i.e. TCDD, and structurally related congeners. EDs can induce many toxic affects to various organs, including neurotoxicity. They are causal factors of various developmental disorders of brain. Since AhR has been detected in regions of the rat brain, AhR can play some roles in ED-induced neurotoxicity. However the relationship between the AhR action and neurotoxicity is not understood well.To determine the function of AhR in neurotoxicity, we have established a stable transfected Neuro-2a over-expressing AhR nemed N2a-R. N2a-R shows morphological and functional changes like neuron, such as the marked increase in the number of neuritis and in AchE activity ligand-independently. It provides a suitable model to investigate the role of AhR in neurotoxicity.

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P3-027 Role of Neuro-p24 in neurite extension and its distribution in the mouse brain

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Neuro-p24 is a novel membranous protein with a molecular size of 24kDa, specifically localized on small vesicles in neurites. Based on previous transfection studies, we showed that Neuro-p24 plays an essential role in neurite extension. To investigate its physiological role, we made in situ hybridization in the adult mouse brain. Intense Neuro-p24 mRNA expression was detected in the pyramidal cells of the cerebral cortex and hippocampus and also cells in the diagonal band. Neurons in these regions are important components to relay inputs for memory, suggesting that Neuro-p24 plays significant roles in the memory function. Neuro-p28, a Neuro-p24 homologous gene, that also encodes a neuron-secific membranous protein showed a similar distributuion pattern to that of Neuro-p24. These results indicate that Nuero-p24 and p28 collaborate each other on the same physiological functions.

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P3-028 Motor nervous system of the ascidian larva

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Ascidians are primitive chordates, and their tadpole-like larvae share a basic body plan with vertebrates. The larva has a remarkably simple central nervous system with about 100 neurons. To identify the motor neurons controlling the larval swimming behavior, we prepared two types of antibodies: one against vesicular acetylcholine transporter for identifying cholinergic neurons and the other vesicular GABA transporter for identifying GABAergic neurons. Cholinergic neurons were detected in the brain vesicle and the visceral ganglion. In the visceral ganglion, the neurons extended their axons posterioly and innervated muscle cells in the anterior part of the tail. GABAergic neurons were detected in the brain vesicle, the visceral ganglion, and the anterior nerve cord. In the anterior nerve cord, the neurons extended their axons anteriorly and connected to the contralateral cholinergic neurons. These results suggest that cholinergic and GABAergic neurons regulate the larval swimming behavior.

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P3-029 Role of phospholipase A_2 and lipoxygenase pathway in formation of dendrites and spines of Purkinje cells

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Cytosolic phospholipase A_2 (cPLA$_2$) is the rate-limiting enzyme of arachidonic acid cascade. We studied a novel function of cPLA$_2$ in mouse cerebellar Purkinje cells. Immunohistochemistry revealed that cPLA$_2$ was selectively expressed in Purkinje cells in mice at the ages of postnatal day 1 and older. In cerebellar cell cultures, cPLA$_2$ was expressed in Purkinje cells on the 5th day *in vitro* and after. The *in vivo* and *in vitro* expression of cPLA$_2$ appeared to correlate with the dendritic extension in developing Purkinje cells. A lipoxygenase inhibitor treatment of cultured Purkinje cells impaired the regulation of shape of individual dendritic spines. These results indicate that cPLA$_2$ and lipoxygenase pathway may play a role in developing and maintaining dendrites and spines in Purkinje cells.

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P3-030 The functional evolution of neural Ig superfamily cell adhesion molecules (IgCAMs): The analysis of the most primary IgCAMs in planarian brain

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The IgCAMs play crucial roles in the formation of neural network. We identified the most primitive orthologs for mammalian NCAM, DSCAM and Tag-1/Contactin from planarian. The phylogenetic studies show that IgCAMs of planarian are ancestors of those in vertebrates and insects. These molecules show different expression patterns during the formation of central nervous system. To analyze functions of Planarian IgCAMs in vitro, all molecules were expressed on the cells by the vaculo virus system. Furthermore, to elucidate the roles of these IgCAMs in vivo, we tried knockdown analysis by RNAi methods and behavioral assays. These analysis revealed that planarian IgCAMs bind in homophilic manner and this association may play the coordinate aspects of morphogenesis in the most primitive brain.

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P3-031 Development of morphological diversity of dendrites of Drosophila sensory neurons by the BTB-zinc finger protein Abrupt

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Morphological diversity of dendrites contributes to specialized functions of individual neurons. We approached a molecular basis that generates distinct morphological classes of Drosophila dendritic arborization (da) neurons. da neurons are classified into classes I to IV in order of increasing territory size and/or branching complexity, and we found that Abrupt (Ab), a BTB-zinc finger protein, is expressed selectively in class I cells. Misexpression of ab directed neurons of other classes to take the appearance of smaller and/or less elaborated arbors. Loss of ab functions in class I neurons malformed their typical comb-like arbor patterns and generated supernumerary branch terminals. All of our data supported that Ab controls dendritic development that is characteristic of the class I neurons.

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P3-032 Visualization of dorsolateral commissural neurons and their connections in zebrafish hindbrain

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Developing dorsal neural tube generates classes of interneurons defined mostly by expression of certain transcription factors. Little is known, however, about their connections and association with specific sensory modalities. We address these issues by generating transgenic zebrafish that express green fluorescent protein (GFP) in dorsolateral hindbrain under the control of a 120kb *zic1* genomic fragment. GFP-positive neurons form extensive commissures that are subdivided into two populations according to their destinations. One population turns rostrally after crossing the midline and projects to the midbrain ventral to the optic tectum. The other population projects straight and terminates on the contralateral side, and thus forms a reciprocal projection. Neuropiles of these neurons interact with the central axon terminals of lateral line ganglion. The role of this circuit in sensory information processing and similarity to the cochlear system of higher vertebrates will be discussed.

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P3-033 Microarray analysis of the axon branching factor genes

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During neural development, growth cones direct growing axons into appropriate targets. In some brain regions, target innervation occurs through the development of collateral branches that extend interstitially from the axon shaft. Although several molecules that guide the primary axonal growth have been identified, little is known about the molecules that induce the collateral branch formation. Here we attempted to identify the genes that code axon branching factors. For this end, we selected corticospinal tract as a model, because extensive studies have revealed the existence of a diffusible factor(s) that is secreted by the basilar pons and induces axon branch formation on layer 5 cortical neurons. By using microarray analysis, we compared the gene expression profiles between the tissues that are thought to have axon branching factor(s) and the ones that are not. We have thus obtained several candidate genes that are expressed in the regions where axon branching factor(s) are expected to be present.

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P3-034 δ-catenin-induced filopodial elaboration is mediated by interactions with its PDZ domain targets

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δ-catenin is a neuronal protein that binds to a juxta-membrane region of classical cadherins and to several PDZ domain containing post-synaptic proteins. Transfection of δ-catenin into young hippocampal cultures increased filopodial number. Deletion of its PDZ binding motif further amplified filopodia induction in mature neurons. δ-catenin has a role in metabotropic-induced dendritic protrusions. Treatment with DHPG induced the elaboration of filopodial, the tyrosine phosphorylation of δ-catenin and the transient disassociation from its PDZ domain interaction with PSD-95. Increasing the amount of endogenous δ-catenin not bound to a PDZ domain triggered filopodial elaboration. From these data we hypothesize that the displacement of δ-catenin from its PDZ targets induces filopodia.

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P3-035 Compartmented chamber for interface type brain slice co-culture and its use for studying corticospinal projection

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Gas-liquid interface culturing method is widely used for co-culture of brain slices. A method to separately manipulate medium composition for each co-cultured slice would be of great use for analysis of developmental processes. To obtain such a method, we newly developed a compartmented chamber that consists of a culture insert and a liquid phase divider. We applied it to our corticospinal slice co-culture system in which we demonstrated corticospinal synapse formation and its developmental plasticity (Takuma et al. 2002, Ohno et al. 2004). Orthograde labeling with biocytin showed the corticospinal axons projected across the border between compartments and their terminals distributed in the dorsal spinal cord. This is consistent with our previous results obtained from conventional culturing chamber. Further characterization of this compartmented culture and its application for pharmacological analysis of the projection pattern formation is now under way.

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P3-036 Suramin mediates neurite outgrowth via MAP kinase-signaling pathways in a neuronal cell line

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Suramin is a well-known antitrypanosomal drug and a novel experimental agent for the treatment of several cancers, yet the molecular mechanisms through which suramin exerts its effects on cell functions, especially on neuronal cells, are not completely clear. In this study, we studied the effect of suramin on the neurite outgrowth in the cultured neuroblastoma cells (NB2a). Suramin was found to stimulate mitogen-activated protein kinase (ERK1/2) activity of the cells in a dose- and time-dependent manner, and the stimulation was suppressed by a MEK inhibitor PD98059. Suramin was also found to induce neurite outgrowth that was significantly inhibited by PD98059. These results suggest that suramin regulates the cellular functions in the neuronal cells by regulating the neurite outgrowth via MAP kinase-signaling pathways.

P3-037 BDNF induces dentate neural network reorganization.

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In the brains of temporal lobe epilepsy patients, the hippocampal mossy fibers (MFs) abnormally sprout out and reorganize dentate neural network. Because brain-derived neurotrophic factor (BDNF) is upregulated in epileptic hippocampus, we hypothesized that BDNF mediates MF sprouting. In organotypic slice cultures, chronic treatment with picrotoxin induced an increase in BDNF protein levels in the MF pathway, an effect that depends on L-type Ca2+ channels. The TrkB inhibitor K252a or function-blocking anti-BDNF antibody prevented the MF sprouting. Placement of a BDNF-including bead onto the dentate hilus efficiently caused MF sprouting. In isolated granule cell cultures, BDNF-induced formation of axonal branches was blocked by transfection with dominant negative TrkB receptors. Thus, BDNF is likely causal for MF sprouting.

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P3-038 Sema3F-mediated growth cone collapse: differential actions of cAMP on axons and dendrites of dentate granule cells

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Sema3F is widely expressed in the developing hippocampus, but its role in the hippocampal network formation remains unclear. Using cultured dentate granule cells, we examined how cAMP modulates the responses of growth cones to Sema3F. Sema3F efficiently collapsed growth cone, the EC50s were almost equal between axons and dendrites. Sema3F increased the cAMP level in axonal growth cones, and its action on axons was prevented by cAMP signaling inhibitors, but neither effect was observed in dendrites. Increased levels of cAMP alone collapsed axonal growth cones, whereas it increased the number of dendritic growth cones. These results suggest that in dentate granule cells, cAMP differentially regulates the actions of Sema3F on axons and dendrites and may shed right on the cellular mechanisms of neurite morphogenesis and axo-dendritic polarization.

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P3-039 Significance of the phosphorylation sites in SCG10, a growth cone protein depolymerizing microbutules

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SCG10, a membrane-bound member of the stathmin family, is one of the growth-associated proteins enriched and localized in the growth cone. SCG10 has four phosphorylation sites (S50, S62, S73, and S97), however, the relationship between the biochemical and the biological significance of each phosphoryation site is not clear. We have evaluated quantitatively the tubulin-binding activity of SCG10 using BIAcore. We also chased the phosphorylation site mutants of SCG10 in the growth cone, and found that some mutants were differently localized within the growth cone from the wild type. We are now investigating the involvement of phosphorylation in the localization of SCG10.

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P3-040 Effects of strucural diversity in chondroitin sulfates on retinal axon guidance

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Understanding the formation of neuronal circuits has long been one of the basic problems in developmental neurobiology. Projection from the retina to their higher center, the optic tectum in chick, is most amenable to experimental approaches; thus, a lot of information has been accumulated about the mechanisms of axonal guidance. The retinal axons navigate along the appropriate pathway with the help of a series of guidance cues. While much of the work has focused on proteinaceous factors, proteoglycans have been identified as playing important roles in retinal axon guidance. Chondroitin sulfate proteoglycans exhibit structural diversity in their carbohydorate chains. However, it has not been determined whether diversity of the carbohydrate chains results in differential effects and how their diversity is recognized by growth cones. The effects of the different structural motif in carbohydrate chains in culture are reported.

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P3-041 Identification of cis-acting elements regulating tissue-specific expression of *neuropilin-1 (nrp-1)* in zebrafish

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Our previous studies have demonstrated that *nrp-1*, a receptor component of class 3 secreted semaphorins, is expressed in a spinal motor neuron, caudal primary (CaP), and its dynamic pattern is consistent with the axonogenesis. To understand mechanism regulating *nrp-1* expression, we analyzed its enhancer/promoter region, and found that 7.5kbp upstream of *nrp-1* gene is enough to arise expression on CaPs and the other tissues. Next, to identify cis-elements regulating its expression, we performed reporter gene expression assay by injecting various upstream DNA fragments into fertiled eggs, and isolated the -5893/-5529 region to induce high-frequency expression on CaPs. Furthermore, by analyzing this region in detail, we identified two short critical portions, both of which contain consensus sequences of homeo-box type transcription factors. We are now trying to identify regulatory molecules of *nrp-1* expression on CaPs.

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P3-042 Fgf8-induced Sema3F is involved in axon guidance of the midbrain dopaminergic neurons

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Previously, we showed that downstream molecules of Fgf8 repelled axons of the midbrain dopaminergic neurons (MDANs) and that Fgf8 induced an expression of a chemorepulsive factor Semaphorin3F (Sema3F). Here we investigated whether Sema3F was involved in axon guidance of MDANs. In situ hybridization studies revealed that mRNAs of Sema3F and Neuropiln-2(NP2), a receptor for Sema3F, were expressed complementary manner in the midbrain. Double staining with antibodies against tyrosine hydroxylase, a specific marker of MDANs, and anti-NP2 antibody showed the expression of NP2 in most rostral MDANs axons. In *Np2* mutant mice some MDANs axons invaded aberrantly dorsal midbrain region. These data indicate that Sema3F induced by Fgf8 regulats axon guidance of MDANs.

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P3-043 Phenotypic analyses of the mutants that have defects in the axonal projection of the olfactory sensory neurons in *Drosophila*

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The olfactory sensory system has become an attractive model to reveal molecular mechanisms of the axonal targeting in a complex neural circuitry. The olfactory sensory neurons (OSNs) expressing a given odorant receptor (OR) project their axons to only a few specific glomeruli in the olfactory bulb in a mammalian brain. This targeted projection is also seen in the olfactory sensory system in *Drosophila*, where the numbers of the OSNs, ORs and the target glomeruli are much smaller than in mammals. To identify genes involved in the targeted axonal projection of the OSNs in this animal, we conducted a mutant screen for mosaic flies with defects in the axonal projection of the OSNs. Among 1200 independent lines mutagenized with EMS, we have isolated a number of mutants showing various defects in the axonal projections. In this presentation, we will show our recent data from the phenotypic analyses of the mutants.

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P3-044 Live imaging of synaptic matchmaking and synaptogenesis: the role of target recognition molecule Capricious

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During neuromuscular synaptogenesis, muscle cells extend motile protrusions named myopodia. We had previously reported that the target recognition molecule Capricious (Caps) clusters at the tip of myopodia. The observation suggested that neuromuscular recognition begins at the tip of myopodia as they contact with the motoneuronal filopodia. To further pursue this possibility, in this study, we examined the effect of deleting Caps on the initial phase of neuromuscular interaction. By genetically expressing myristylated GFP both in pre- and post-synaptic cells, we visualized dynamic morphological changes of myopodia and filopodia during synaptic matchmaking. In wild type, myopodia and filopodia merged to form synapses soon after they touch each other. On the other hand, in caps mutant, they often failed to undergo these morphological changes. These observations support the idea that Caps plays a role at the tips of myopodia to stabilize pre- and post-synaptic interaction.

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P3-045 Analysis of the function of MBR-1, a C. elegans transcription factor involved in olfactory plasticity, in axon pruning

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We previously identified a novel C. elegans transcription factor, MBR-1, and demonstrated its roles in the early phase of olfactory adaptation. In the present report, we analyzed the function of MBR-1 in axon outgrowth. We introduced an mbr-1 promoter::gfp construct into the wild-type and mbr-1 mutant nematode to compare the morphology of the mbr-1-expressing neurons. In mbr-1 mutant adults, an extra connection was found between a pair of AIM interneurons, but not in the wild-type. However, this connection was observed in both the wild-type and the mbr-1 mutant at the larval stages. These results indicate that the AIM neurons undergo axon pruning and that MBR-1 is involved in this process. Furthermore, we found that the expression of zig-3, which encodes a neurosecretory protein, is attenuated in the AIM neurons of the mbr-1 mutant, suggesting that MBR-1 is involved in the expression of zig-3.

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P3-046 The effects of K252a to cell cycle and Purkinje cell development

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Immature Purkinje cells mostly degenerate rapidly in cultures. Recently we reported massive cell death in culture could be successfully rescued by the administration of K252a. K252a is known as the blocker to many kinds of kinases, including Cdc2 and Cdc25. We examine it has the effect of G2 synchronization on immature granule cells. Further surviving Purkinje cells treated with K252a, elongate their dendrites to a molecular layer within 3 days in culture. We observe glutamate released from cultures optically. In cultures treated with both colcemid with K252a, and high concentration of K252a alone, glutamate release is observed in an oscillatory manner, whereas in cultures treated with colcemid alone, it is released transiently. In oscillatory glutamate-released cultures, many GABA(A) α6 positive cells are observed in the deep layer. We suggest that oscillatory glutamate release involved by K252a, modulates granular cell differentiation, and has effects on Purkinje cell survival and development.

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P3-047 Distribution of the RhoG effector Elmo and its binding protein Dock180 in the developing mouse brain

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The small GTPase Rac has a central role in regulating the actin cytoskeleton during axonal outgrowth and guidance. We recently reported that RhoG, another member of Rho family small GTPases, activates Rac and regulates neurite outgrowth through Elmo and Dock180. In this study, we examined the distribution of Elmo and Dock180 in the developing mouse brain. In situ hybridization shows that at postnatal day 20, expressions of Elmo and Dock180 mRNA were similar throughout the brain, particularly in the cerebral cortex, hippocampus, and cerebellum. On the other hand, Dock180 mRNA was observed in the ventricular zone at embryonic day 14 mouse brain. These results suggest distinct pre- and postnatal roles of Elmo and Dock180 in the developing brain. We are currently investigating the expression of Elmo and Dock180 proteins in the developing mouse brain by immunohistochemical studies.

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P3-048 Axonal netrin-G proteins regulate local postsynaptic properties

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Netrin-G1 and netrin-G2 compose a vertebrate-specific subfamily within the UNC-6/netrin family, which is characterized by glycosyl phosphatidyl-inositol linkage onto cells. Previous cell culture experiments indicated that netrin-G1 and -G2 act as bi-directional signaling molecules in neurite outgrowth. Here, we report their physiologic functions in mice. Netrin-G1 and -G2 proteins distributed to distinct populations of axons. Loss of these proteins by gene targeting induced characteristic behavioral deficits, without any gross abnormalities in brain cytoarchitecture, including axonal development. Electrophysiologic studies of the mutants also revealed deficits in NMDA-receptor mediated postsynaptic responses in a pathway-specific manner. The results suggest that presynaptic netrin-G proteins modify local postsynaptic properties (forward signaling via putative netrin-G receptors) and have a crucial role in the formation of functional neural connectivity.

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P3-049 Thalamic regions symmetrically and asymmetrically connected to chick Wulst

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Bird's visual pathway consists of two different projections; tectofugal and thalamofugal ones. In chick, the latter is developmentally instructed to be asymmetrical by light just before hatch. Anatomical, electrophysiological, and behavioral studies have suggested an important role of the pathway not simply for vision itself, but rather as a part of integration system in multimodal sensory-motor behavior. It is uncertain, however, how and what extent this circuit contributes in visually guided behavior. In order to address this question, we need precise identification of thalamic nuclei connected to Wulst. Then, we simultaneously injected several fluorescent tracers in Wulst and examined the pattern of retrogradely labeled thalamic neurons. Using the same brain slices, we further carried out immunohistochemical staining by antibodies to GABAergic cell marker-calcium binding proteins, ligand/recepto-bodies for Eph/ephrin as well. Thus, we could demarcate new dorsolateral thalamic regions symmetrically and asymmetrically connected to Wulst.

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P3-050 5-HT enhances NGF-induced neurite outgrowth via intracellular Ca^{2+} increase in PC12 cells

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Serotonin (5-HT) is known as a neurotransmitter in the central and peripheral nervous systems, and recent studies indicate that 5-HT is involved in neural development. PC12 cell is a useful model of neurite outgrowth and neural differentiation, and it is suggested that 5-HT have an effects on NGF-differentiation in PC12 cells. However, it is unclear how 5-HT regulates NGF signal. We hypothesized that 5-HT-induced Ca^{2+} increase could enhance this differentiation. In this study, we investigated 5-HT-induced Ca^{2+} increase and actin dynamics with YFP-labeled actin in PC12 cells. When 5-HT was applied, NGF-treated PC12 cells induced Ca^{2+} increase via 5-HT_3 receptors. In accordance with this Ca^{2+} increase, 5-HT enhanced neurite outgrowth. And, we observed neurite sprouting had been stimulated by 5-HT application. This effect could contribute to NGF differentiation.

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P3-051 synaptic transmission between thalamic axons and subplate neurons during embryonic periods

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Rat thalamic axons seem to form active synapses with subplate neurons before its birth (Higashi et al 2002 in Neurosci.). The time course of the optical response during embryonic ages, however, was longer than that of matured one. These results suggest that transmission properties during embryonic ages are quite different from those of matured ones or polysynaptic responses are included. I performed the whole cell recording from subplate neurons in the embryonic slices. At embryonic day 19, synaptic current was recorded and AMPA antagonist perfusion almost abolished it at -70mV but outward current still remained at +50mV with the same latency. In addition, the synaptic currents having much longer latency were also observed. These results suggest that active synapses are already formed between thalamic axons and subplate neurons before brith and polysynaptic connections are formed among cortical neurons at same time. Synaptic activity during embryonic days might have an important role for neural circuit refinements and formations.

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P3-052 Characterization of expression of brain-specific transmembrane semaphorin, Sema4C

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Semaphorins provide crucial attractive and repulsive cues involved in various physiological events. We have identified Sema4C (previously called M-SemaF) from mouse brain, which is predicted to encode a transmembrane semaphorin. Sema4C is specifically expressed in both embryonic and adult neuronal tissue, suggesting that Sema4C functions such as directing axon pathfinding, synapse formation or synapse maintenance, but the physiological role was poorly understood. In this study, we report that Sema4C forms a disulfide-linked homodimer and the changes in Sema4C monomer and dimer protein levels during brain development. Western blots analysis of mouse brain protein under nonreduced condition showed that Sema4C predominantly forms dimer in embryonic stages but the monomer level dramatically increase during brain development. These results suggest that the two forms of Sema4C may play a different roles in neuronal function.

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P3-053 The behavior of multipolar cells in the developing cortex

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During the coritcal plate development, neurons are generated in the ventricular zone (VZ) and migrate toward the pial surface. Two distinct modes of radial neuronal migration, locomotion and somal translocation, were reported, and we have recently found the third mode of migration, multipolar migration. Multipolar migration does not seem to be a rare mode of migration, because the multipolar cells account for the major population of the migrating cells in the intermediate zone/subventricular zone in the developing cortex. These cells express the neuronal markers and a marker for the cells that derived from the cortical VZ, suggesting that multipolar cells are radially migrating neurons. Time-lapse study revealed that multipolar cells frequently change migration direction and rate, and the cell position move very slowly toward the pial surface. We also observed that the multipolar cells transformed into locomotion cells before entering the CP.

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P3-054 Polymer Hydrogel Electrodes for the Study of Neural Network Formation

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We have evaluated conducting polymer hydrogel electrodes (PHEs) for neural interfacing. Neural interfacing puts high demands on the biocompatibility and the electrical parameters of the materials to create desired neural networks as well as good electrical coupling to neurons. An interesting category of materials for neural interfacing is the conducting polymer hydrogel family. These materials allow for the fabrication of 3-dimensional biomimetic scaffoldings for cell attachment in addition to having parameters suited for electrical interfacing. Neuronal cells were cultivated on PHE substrates and PHE microelectrode arrays fabricated using electropolymerization from a solution of the monomer ethylenedioxythiophene and a dispersion of poly (3,4-ethylenedioxythiophene) - poly (styrenesulfonate). Neurons grown on PHE substrates exhibited axonal sprouting and interconnections between cells. The versatile characteristics of the PHE make it an interesting novel tool for the study of neuronal networks.

P3-055 Selective inhibition of chondroitin sulfate to neurite growth from mouse retina

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In order to determine signaling mechanisms associated with chondroitin sulfate (CS) induced inhibition, we grew explants from ventral temporal (VT) and dorsal nasal (DN) retina of E14 mouse embryos on laminin, and observed responses of neurites to a CS coated spot. We found CS selectively inhibited neurite growth from VT but not nasal retina at a critical concentration of the coating solution (10 mg/ml). This inhibition could be abolished by 0.2U/ml chondroitinase ABC. Further experiment was done to investigate whether protein kinase C (PKC) is involved in this inhibitory response. We first characterized expression of various PKC isoforms in VT and DN retinal growth cones. Within the growth cone, PKC-α was predominantly expressed in the periphery and in filopodia, whilst PKC-β1 was localized at the core. PKC-β2 was found at a lower level and showed a punctuate appearance. Work is still undergoing to examine whether this selective inhibition can be altered by manipulating PKC levels within the growth cone.RGC Grant: CUHK4417/03M

P3-056 Expression of Nogo-A in the chiasm of mouse embryos

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Nogo-A is known to be a myelin-associated protein that inhibits regeneration of central axons. Its roles on development of central pathways, however, are largely unknown. We have investigated Nogo-A expression in the mouse retinofugal pathway, during period of major axon growth at the chiasm. In the retina, Nogo-A was found on the membrane and processes of the radial glial-like cells and not in the ganglion cells and their axons. In the diencephalon, Nogo-A was observed exclusively on the radial glial cells, which are immunopositive to RC2, at the midline regions of the chiasm. The staining was dramatically reduced at the stalk-chiasm junction, and at the threshold of the optic tract. Labels at the midline revealed a dense ramification of Nogo-A rich glial processes that interact with ingrowing optic axons, which was lacked in those at the optic tract. We concluded that Nogo-A is expressed on the radial glia particularly at the chiasmatic midline, and is probably involved in controls of axon routing, including divergence of optic axons, at the mouse chiasm. RGC Grant: CUHK4417/03M

P3-057 Passage culture of undifferentiated PC12 cells in serum-free medium

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PC12 cells are a widely used model system for neurons. However, hormonally defined serum-free medium (HD), suitable for maintaining PC12 cells, has not yet been described. Serum contains many unknown factors whose effects remain for a few passages. Thus, cells must be maintained in a HD in order to exclude the effects of the unknown factors.
We developed a HD for PC12 cells and succeeded in maintaining the cells for more than 2 months (12 passages). Unlike N2 supplements, the HD does not contain progesterone. Both the plating efficiency and doubling time of the cells maintained in the HD were comparable to those of the cells maintained in the serum-supplemented medium. The responses to the NGF of cells maintained in the HD were normal: prevention of cell death; inhibition of proliferation; and extension of neurites. The supplements cost less than the serums. Therefore, the HD is suitable for PC12 cell cultures which are free from unknown factors.

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P3-058 PEDF induces pro-inflammatory genes in neonatal astrocytes through activation of NFκB and CREB

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Pigment epithelium-derived factor (PEDF) is a 50kDa glycoprotein and a member of the serine protease inhibitor (SERPIN) gene family. Some of the effects of PEDF reflect specific changes in gene expression, mediated via activation of NFκB in neurons. To investigate whether PEDF also modulates the transcriptional activities in astrocytes, we analyzed the transcription factor-DNA binding activity using EMSA. RT-PCR analysis revealed that PEDF induced NFκB and AP-1-DNA binding in neonatal astrocytes. Western blot analysis revealed that phosphorylation of CREB protein was increased in PEDF treated astrocytes. Overexpression of IκBαM or dominant negative CREB inhibited PEDF-induced pro-inflammatory genes and AP-1-related genes in astrocytes. These results suggest that the induction of pro-inflammatory genes and AP-1-related genes by PEDF is mediated via activation of NFκB and CREB in neonatal astrocytes.

P3-059 Negative Function of EGF/ErbB1 Signaling in Synaptic Development and Maturation

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In developing central nervous system, positive and negative regulation of synaptic formation and maturation is an important neuronal process for organization of neural network, although responsible molecular mechanisms remains to be illustrated. Here, we tested the possibility that epidermal growth factor receptor, ErbB1 signaling negatively regulates the maturation of synapse in developing cortical neurons. In cultured neocortical neurons as well as in the neocortex of rat neonates, EGF administration induced ErbB1 phosphorylation and down-regulated protein levels of a presynaptic protein, synaptophysin and a postsynaptic molecule, PSD95. The reduction in levels of synaptic markers represented the inhibition of synaptic maturation. These results suggest that EGF/ErbB1 signaling has negative impact on synaptic maturation in developing cortical neurons.

P3-060 Activity-dependent transcriptional and post-transcriptional regulation of pituitary adenylate cyclase-activating polypeptide (PACAP) coordinated by calcium and cAMP signals in neurons

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Activity-dependent gene expression in CNS contributes to a variety of neuronal functions including neuronal survival and synaptic plasticity. In this study, we particularly focused on the activity-dependent expression of the PACAP gene in cultured rat cortical neurons. The PACAP gene promoter was synergistically activated in the presence of calcium and cAMP signals in neurons. On the other hand, the level of PACAP mRNA markedly decreased by cAMP signals but increased by a co-administration of calcium signals. In support of this phenomenon, the calcium signals reduced the degradation rate of PACAP mRNA, indicating that PACAP mRNA can be stabilized by the calcium signals. These results indicated that both the transcription and post-transcription of PACAP gene were coordinately controlled in an activity-dependent manner in neurons.

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P3-061 NF-kB-dependent Activation of Glutamate Uptake by Interleukin-1 in Rat Primary Astrocytes

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Interleukin-1 (IL-1), which is induced by brain insult and psychological stress, activates HPA axis and transmitter turnover and is implicated in psychiatric diseases. As astrocytes carry high amounts of IL-1 receptors, IL-1 activity might influence astroglial and brain functions. Here we prepared cultured astrocytes from rat cerebral cortex to assess their response to IL-1. IL-1α treatment (5ng/ml, 48h) increased uptake of ^{3}H-glutamate. Immunoblotting revealed that IL-1α increased the expression of glial glutamate transporter, GLT-1, in parallel. An inhibitor of phosphorylation of IkB (APDC) blocked the glutamate uptake whereas the p38 MAP kinase inhibitor (SB203580), the COX inhibitor (NS-398) and the NOS inhibitor (L-NAME) did not. In agreement, IL-1α challenge enhanced IkBα degradation and NF-kB nuclear translocation. These results demonstrate that the IL-1-triggered increase in glial glutamate uptake results from the activation of the NF-kB and subsequent gene transcription of GLT-1.

P3-062 Decreased EGF/ErbB1 receptor signal in the brain of patients with Parkinsons disease

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Epidermal growth factor (EGF), tumor growth factor alpha (TGFa) and heparin-binding EGF (HB-EGF) act as a trophic factor for dopaminergic neurons and their dysregulation might contribute to neurodegeneration of Parkinsons disease. Using two-site ELISA, we quantified immunoreactivities of EGF family in postmortem brains of control subjects and patients with Parkinsons disease. EGF immunoreactivity was decreased in the striatum of Parkisonian patients. A similar decrease in EGF immunoreactivity was mimicked in the striatum of the rats, in which midbrain dopaminergic neurons were depleted with 6OHDA. Expression of EGF receptors (ErbB1) also declined in the striatum of both Parkisonian patients and the 6OHDA-treated rats. These findings suggest that the neurotrophic EGF activity in the striatum depends on the innervating midbrain dopaminergic neurons and is impaired in patients with Parkinsons Disease.

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P3-063 The secretory granule-associated protein CAPS2 regulates neurotrophin release and cell survival

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We isolated a clone that is predominantly expressed in granule cells of mouse cerebellum, which turned out to be a paralog of CAPS (Ca2+-dependent Activator Protein for Secretion), and named CAPS2. CAPS2 is enriched on vesicular structures of presynaptic terminals of granule cells. Vesicle factions affinity-purified by the CAPS2 antibody from cerebella contained significant amounts of NT-3, BDNF. In cerebellar primary cultures, punctate CAPS2 immunoreactivities are largely colocalized with those of NT-3 and BDNF. Exogenously expressed CAPS2 enhanced release of exogenous NT-3 and BDNF from PC12 cells and of endogenous NT-3 from cultured granule cells in a depolarization-dependent manner.

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P3-064 Mechanisms of BDNF synthesis in the rat hippocampus: intracellular signals activated in the downstream of p38 MAPK and/or ERK1

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The signaling pathway of brain-derived neurotrophic factor (BDNF) synthesis in the rat hippocampus remains still unclear although the synthesis is known to be activity-dependent. We found that riluzole, an inhibitor of voltage-dependent sodium channels, markedly elevated BDNF levels in the hippocampus. Therefore, we used riluzole as a tool to examine the pathway of BDNF synthesis and found increased phosphorylation of p38 MAPK and ERK1 after riluzole injection, indicating the activation of these kinases. Such an activation was also observed in cases of other BDNF-enhancing reagents. Then, we investigated intracellular signals activated in the downstream of p38 MAPK and ERK1. The activation of p70 S6 kinase, S6 kinase, elf4E and elF-4G was observed, suggesting "translation on". A transcription factor, ATF2, was found also activated.

P3-065 Secretion of glial cell line-derived neurotrophic factor (GDNF) from rat cultured microglia

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Since microglia in culture have been suggested to produce neurotrophic factors specific for dopaminergic neurons, we investigated the ability of microglia to produce and secrete glial cell line-derived neurotrophic factor (GDNF) in vitro. The analysis of microglial conditioned medium (MCM) revealed that non-stimulated microglia secrete relatively high amount of GDNF into the medium. However, the activation of microglia with lipopolysaccharide (LPS) led to the significant decrease of the secretion. Quantitative analysis showed that LPS stimulation decreases the secretion to approximately one third. On the other hand, the intracellular amount of GDNF in the LPS-stimulated cells was sustained at higher level than that in non-stimulated microglia. GDNF mRNA was detected in both non-stimulated and LPS-stimulated microglia. Thus, microglia in culture were demonstrated to have the ability to produce and secrete GDNF.

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P3-066 Expression and localization of LECT2 in mouse brain

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It has been suggested that the neutrophil chemotaxis factor LECT2 is related to the regulation of proliferation in the liver regeneration. In the present study, we have examined the expression and localization of LECT2 in the mouse brain by *in situ* hybridization and immunostaining using anti MAP2 and anti GFAP to find the novel physiological function of LECT2. LECT2 mRNA was remarkably expressed in neurons of glomerular circumference and granule cell layer of the olfactory bulb, neurons in cerebral cortex and pointine nuclei, Purkinje cells and granule cells of the cerebellum. The neuronal form of KO mice was small and different in comparison with neuronal form of normal mice. These results suggest the possibility that LECT2 has potential actions on the neuronal cells in cooperation with the other growth factor and cytokine to keep the normal neuronal function.

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P3-067 Developmental changes in concentrations and distributions of neurotrophins in the cerebellum of macaque monkeys

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We determined the developmental changes in concentrations and distributions of BDNF, NT-4/5, NT-3 and TrkC in the cerebellum of macaque monkey using ELISAs and immunohistochemical method. We found that the concentrations of neurotrophins were different from each other, suggesting that the gene expressions of neurotrophins are regulated differently and they play distinct roles in the monkey cerebellum during development. All kinds of cerebellar neurons were BDNF immunopositive throughout postnatal stages examined, indicating that BDNF may be the essential factor for the maintenance of the cerebellum. The Bergmann glial fibers were strongly NT-3 immunopositive at the early postnatal stages, and TrkC immunoreactivities were observed in the premigratory granule cells, suggesting that NT-3 in the Bergmann glial fibers may be involved in the migration of the premigratory granule cells.

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P3-068 Perinatal EGF/ErbB1 activity potentiates excitatory synaptic transmission to midbrain dopaminergic neurons

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Subchronic administration of epidermal growth factor (EGF) to rat pups (s.c.) promotes tyrosine hydroxylase expression and dopamine metabolism during brain development. Here, we analyzed effects of the EGF challenge on synaptic sensitivity of dopaminergic neurons in the ventral tegmental area (VTA), monitoring fiber volley and synaptic field potentials. Neonatal EGF administration increased amplitudes of the field EPSP at juvenile stages (PND 16-18), compared with control mice. Mini analysis indicated that EGF increased mEPSC amplitudes, but not their frequency in the dopaminergic neurons. There was no significant influence on mEPSCs of neighboring GABAergic neurons, however. In contrast, immunoblotting revealed no differences in AMPA receptor levels in the VTA after the treatment. Perinatal EGF/ErbB1 activity in the brain persistently alters postsynaptic plasticity of VTA dopaminergic neurons to influence behavioral development.

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P3-069 Localization of TROY/TNFRSF19 in the postnatal and adult CNS

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Previously, we reported that TROY, a member of TNF receptor superfamily, was expressed in the develoing CNS, including the olfactory system. Northern blot and western blot analyses using various regions of adult CNS revealed that TROY was widely distributed in most CNS regions. Using in situ hybridization and immunohistochemistry, we showed that TROY was expressed in the subventricular zone (SVZ) and rostral migratory stream at high levels and in the cortex and hippocampus at low levels. Double-immunofluorescence method showed that TROY-expressing cells in the cerebral cortex and SVZ were colocalized with GFAP, indicating that TROY was expressed in the astrocyte lineage cells, from SVZ precursor cells to differentiated astrocytes in the postnatal and adult CNS. These results suggest that TROY may be involved in the postnatal and adult gliogenesis.

P3-070 Autoregulation of Brain-derived neurotrophic factor gene expression in rat cortical neurons

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Brain-derived neurotrophic factor (BDNF) plays an improtant role in neuronal survival, neurite extension and synaptic plasticity. BDNF gene expression is mediated by intracellular Ca^{2+} elevation in an activity-dependent manner. We are assuming that BDNF regulates its own gene expression and maintains its own expression elevated, possibly resulting in maintaining synaptic functions. In this study, we demonstrate that BDNF induces biphasic expression of BDNF exon III mRNA and continuous activation of BDNF promoter III in rat cortical neurons. Moreover, short-term treatment of cortical cells with BDNF induces early and late phase expressions of BDNF exon III. Since, in this culture condition, BDNF increases the frequency of Ca^{2+} oscillations, corresponding well with the increase in synaptic density, this autoregulation of BDNF gene expression may play an important role in promoting synaptogenesis.

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P3-071 Effect of (-)-1-(benzofuran-2-yl)-2-propylaminopentane on the expression of neurotrophins and superoxide dismutase activity in mesencephalic slice cultures

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(-)-1-(Benzofuran-2-yl)-2-propylaminopentane [(-)-BPAP] enhances electric field stimulation-induced release of catecholamines from isolated rat brainstem, and improves motor deficits in rats. (-)-BPAP also upregulates the synthesis of neurotrophins in cultured astrocytes. We evaluated the effects of (-)-BPAP on expression of brain-derived neurotrophic factor (BDNF) and glial cell line-derived neurotrophic factor (GDNF), as well as on superoxide dismutase (SOD) activity in mesencephalic slice cultures. (-)-BPAP caused a significant increase in expression of BDNF and GDNF, and in SOD activity. Moreover, (-)-BPAP increased trkB mRNA but not p75 mRNA. These actions of (-)-BPAP may contribute to neuroprotection against dopaminergic cell death in Parkinsonian substantia nigra.

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P3-072 Mouse 3-phosphoglycerate dehydrogenase is essential for brain morphogenesis

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D-3-Phosphoglycerate dehydrogenase (Phgdh) is the first committed enzyme of L-serine biosynthesis. To determine the physiological importance of Phgdh-dependent L-serine biosynthesis in vivo, we generated Phgdh-deficient mice. The absence of Phgdh led to a drastic reduction of L-serine metabolites such as phosphatidyl-L-serine and sphingolipids. Phgdh null embryos have small bodies and died after days post-coitum 13.5. Phgdh null embryos showed hypoplasia of brain; in particular, the olfactory bulbs, ganglionic eminence, and cerebellum appeared as indistinct structures. These observations demonstrate that the Phgdh-dependent phosphorylated pathway is essential for normal embryonic development and brain morphogenesis.

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P3-073 Interplay of perinatal interleukin-1 and mouse genetic backgrounds in neurobehavioral consequences

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Genetic factors interact with environmental factors and influence the risk of psychiatric diseases. Abnormal interleukin (IL)-1 often signal is implicated in various psychiatric diseases including schizophrenia. IL-1α activity in neonates can induce abnormal behaviors at adult stage. Here, we evaluated the influence of genetic backgrounds on activation of IL-1 signaling pathways as well as behavioral consequences in mice. Different mouse strains (C3H/He, DBA/2, C57BL/6, ddY) were treated with IL-1α (0.1mg/kg weight, s.c.) on postnatal days 2-10. In parallel, Iκ-B degradation and p38 MAPK phosphorylation were monitored in several brain areas. DBA mice exhibited most severe influences on behaviors of locomotor and prepulse inhibition. The activation of IL-1 signal transeduction was most marked in DBA strains. These results suggest that mouse genetic backgrounds have crucial impact on behavioral development, determining IL-1 responsiveness in the brain.

P3-074 Transcriptional mechanisms of neurotrophin-3 (NT-3) gene promoter IB by Sp3 and Sp4 in cultured rat cortical neuron

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It has already been reported that neurotrophin-3 (NT-3) acts on cerebellar granule and Purkinje cells to promote their differentiation and proliferation, resulting in the development of cerebellum. Using RT-PCR analysis, we previously demonstrated that transcriptional activity of NT-3 promoter IB decreased in response to the calcium influx into neurons. Using a DNA transfection in rat cortical neuronal cells in culture, in this study, we further characterized transcriptional activity of the NT-3 promoter IB. As the results, we found that two GC-boxes were critical for the transcriptional activation of NT-3 promoter IB. Using a gel-shift assay, we have demonstrated that the transcriptional factors, Sp3 and Sp4, bound these two GC-boxes.

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P3-075 Regulation of brain-derived neurotrophic factor gene transcription mediated by neuron-restrictive silencer factor (NRSF)

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A large number of neuron-specific genes are under the control of negative transcriptional regulation in neuronal and non-neuronal cells, which is mediated by the neuron-restrictive silencing *cis*-element, NRSE. The NRSF, a specific binding protein to NRSE, is required to repress a subset of neuron-specific genes in non-neural tissues and undifferentiated neural precursors during early embryogenesis. We found that NRSE-like element, which is located in the proximal region of the BDNF promoter II, repressed the activities of BDNF promoters through NRSF. Partial deletions of N- and/or C-terminals of NRSF reduced the repressive activity of NRSF. We are now investigating the Ca^{2+} signal-dependent activation of BDNF promoter I, which is mediated *via* CRE/UBE located in the proximal region of the promoter, in relation to the repressive activity of NRSF.

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P3-076 Involvement of neuronal activity in intracellular signaling, which is important for BDNF-potentiated glutamate release.

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Studies showed that BDNF regulates synaptic transmission. However, it is unknown about mechanisms through which BDNF modulates synaptic efficacy. Here, we showed that BDNF treatment for 24 hr potentiated exocytotic glutamate release in cultured cortical neurons. It was suggested that the up-regulation of synaptic vesicle-associated proteins, but not plasma membrane-associated synaptic proteins, was involved in the BDNF-potentiated release. Further, we found that the effects of BDNF required the activation of both MAPK and PLC-γ pathways and also depended on neuronal activity. Interestingly, TTX attenuated long-lasting activation of MAPK stimulated by BDNF, suggesting that neuronal activity was necessary for the maintenance of MAPK activation, which might be required for the BDNF-potentiated release.

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P3-077 Glial cell line-derived neurotrophic factor influences the activation of peritoneal macrophages in autocrine/paracrine manners

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Glial cell line-derived neurotrophic factor (GDNF) is produced by activated microglia/macrophages in injured central nervous system (CNS), but such physiological roles of GDNF have not been fully elucidated. We cultured macrophages from the heterozygotes of the GDNF gene-disrupted mice or from the normal littermates, and compared the effects of lipopolysaccharide or exogenous GDNF on the phagocytosis and the mRNA expression of monocyte chemoattractant protein (MCP)-1 of the macrophages. The phagocytic activity, not MCP-1 mRNA level, was significantly lower in the heterozygous cells than the normal cells. GDNF mRNA was expressed in heterozygous cells, but was substantially lower than that of normal cells. These results demonstrate that endogenous GDNF is involved in the activation of macrophages, and suggest that the GDNF produced by macrophages themselves plays important roles for activation of macrophages in the injured CNS tissues.

P3-078 The developmental shift of the BDNF actions on $GABA_A$ receptor responses by the rapid up- or down-regulation of cell surface functional $GABA_A$ receptors in rat visual cortex

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In visual cortex of P14 rats, we have recently reported that BDNF rapidly increases the total number of cell surface $GABA_A$ receptors (J Biol Chem 2003;278:44097-102). In visual cortex of P40 rats, muscimol radioligand binding assays revealed that BDNF rapidly downregulated the cell surface $GABA_A$ receptors. BDNF rapidly induced a sustained suppression of $GABA_A$ receptor mediated currents in visual cortical pyramidal neurons isolated from P40 rats. By contrast, in visual cortex of P40 rats reared in the complete darkness from birth, BDNF rapidly upregulated the surface $GABA_A$ receptors and induced a sustained potentiation of $GABA_A$ receptor currents. Thus, visual experience could influence the developmental shift of the BDNF actions on $GABA_A$ receptor responses by the rapid regulation of cell surface $GABA_A$ receptors.

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P3-079 Glucocorticoid suppresses brain-derived neurotrophic factor (BDNF)-regulated glutamatergic transmission in cultured cortical neurons.

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Neuronal damage due to glucocorticoid and alterations of BDNF-dependent neuronal function have been implicated in the pathogenesis of stress-induced psychiatric disorders such as depression. We investigated the possible effect of glucocorticoid on BDNF-dependent glutamatergic neurotransmission in cultured cortical neurons. We found that BDNF-induced release of glutamate was significantly suppressed after pretreatment with glucocorticoid (dexamethasone, DEX) for 24-48 hours. BDNF-increased intracellular Ca also decreased after DEX treatment. DEX depressed BDNF-stimulated activation of PLC which is in line with previous findings (Numakawa et al., 2002, 2003) that BDNF-induced glutamate release is through PLC/IP3/Ca pathway. These results suggest that neuronal response to BDNF is down-regulated after intense exposure to glucocorticoid.

P3-080 BDNF Val68Met Mutant Increases an Inhibitory Synaptic Machinery

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There is a well-known amino acid substitution of BDNF ($BDNF^{V66M}$) resulted from a SNP in human being. The individuals who are homozygous of this SNP shows milder phenotype than had been assumed, suggesting that $BDNF^{V66M}$ would share some roles of wild-type BDNF ($BDNF^{wt}$). To evaluate possible remaining function of rat $BDNF^{V68M}$, we expressed the cDNA of $BDNF^{wt}$ or $BDNF^{V68M}$ in cultured hippocampal neurons. Over expression of both $BDNF^{wt}$ and $BDNF^{V68M}$ significantly increased the GABA synthetic enzyme, GAD65, in presynaptic terminals around the expressed neurons in comparison with control neurons. This result suggests that the effect of BDNF on inhibitory neurotransmission is one of the common functions. Furthermore, the increase of GAD65 around $BDNF^{wt}$ transfected neurons was not attenuated by applying tetrodotoxin to the cultures. Since depolarization-induced secretion of $BDNF^{V68M}$ has been reported to be low, these results suggest that activity independent secretion of BDNF acts on the maturation of inhibitory synapses.

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P3-081 Induction of chemokine receptor CCR7 mRNA in activated Microglia

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Microglia/brain macrophages are known to display activated phenotypes in response to injury or inflammation in the CNS. Using cerebellar cultures, we have searched for microglial genes up-regulated during granule neuron apoptosis, and found that the expression of the chemokine receptor CCR7 mRNA, but not of CXCR3 mRNA, is induced upon microglial activation during cell death as well as following treatment with lipopolysaccharide. CCR7 and its ligand CCL19 are known to be involved in migration and homing of lymphocyte and dendritic cells. However, it has been shown that CCR7 mRNA is not expressed, whereas CXCR3 mRNA is highly expressed in the mammalian CNS. Here we show that CCR7 mRNA is barely expressed in microglia under normal conditions, but markedly up-regulated upon their activation. Indeed, its ligand CCL19 augmented phagocytosis in activated microglia, suggesting that under activated conditions the CCL19-CCR7 interaction plays a major role in regulating migration, cytokine production and phagocytosis.

P3-082 Growth hormone receptor modulates adult neural stem cell proliferation.

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Reports of growth hormone (GH) effects on progenitor cells during development and the presence of GH binding sites in the adult CNS prompted us to investigate the role of GH receptor (GHR) in adult neural stem cell (NSC) proliferation. Subventricular zone (SVZ) cells were isolated, plated with EGF in the presence or absence of hrGH and the number of spheres counted at 7 DIV. GH produced a dose dependent reduction in number of spheres indicating an attenuation of EGF-induced proliferation of adult precursors. We tested the hypothesis that GHR is expressed on NSCs by sorting GHR+ve cells from primary tissue and characterized their proliferative potential. Overall, ~2% of viable cells were GHR+ve and10% of these formed spheres. Long-term passaging of spheres from adult GHR-/- mice resulted in a reduced proliferative potential based on rate of doubling over time. These results support the hypothesis that GHR plays a role in NSC proliferation.

P3-083 Perineuronal net of chondroitin sulfate proteoglycan phosphacan and plasticity in the central nervous system

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Perineuronal nets are lattice-like accumulation of extracellular matrix components of chondroitin sulfate proteoglycans (CSPGs). Phosphacan is one of main componets of CSPGs in the perineuronal nets. In the present study, the localization of phosphacan was examined at perineuronal net in the rat brains. Phosphacan was densely observed at the perineuronal nets of inhibitory interneuron in the cerebrum, pyramidal somata of hippocampal CA2 neurons, and hypothalamic supraoptic nucleus (SON). Electron microscopic observation reveals that phosphacan was localized mainly at axonal membrane and astrocytic processes. The levels of phosphacan in the hypothalamic SON are downregulated with chronic stimulation, salt-loading. Thus, it is concluded that CSPGs are regulated with activity-dependent manner and thereby may contribute to the plasticity of neural circuits.

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P3-084 Internalization of cell adhesion molecule OBCAM at dendritic spines

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OBCAM belongs to the immunoglobulin superfamily cell adhesion molecule and shows a polarized distribution in cortical and hippocampal dendritic spines. In the present study, the cellular localization and dynamics of OBCAM were monitored in cultured cortical neurons. Double labeling immunofluorescence study showed strong OBCAM immunoreactivity in postsynaptic density even after removal of cell surface OBCAM by trypsin, indicating that OBCAM localizes intracellularly and extracellularly at spines. Moreover, it was shown that OBCAM was internalized via a clathrin-independent pathway, and its internalization was enhanced under conditions of increased excitability with the K^+ channel blocker, 4-aminopyridine. The present study suggests that spine surface OBCAM is dynamically regulated in activity-dependent manner and may be implicated in synaptic plasticity.

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P3-085 Stability of dendritic spines and synaptic contacts is controlled by αN-catenin

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Morphological plasticity of dendritic spines and synapses is thought to be crucial for their physiological functions. We show that αN-catenin, a linker between cadherin adhesion receptors and the actin cytoskeleton, is essential for stabilizing dendritic spines in hippocampal neurons in culture. We found that in the absence of αN-catenin, spines showed elongated shape, and abnormally motile, actively protruding filopodia from their synaptic contact sites. Conversely, αN-catenin overexpression in wild-type neurons showed reduced spine turnover, causing an increase in spine and synapse density. Furthermore, we found that the synaptic accumulation of αN-catenin is regulated by neural activity, and excess αN-catenin rendered spines resistant to the morphological chage induced by neural activity blockage. These results suggest that αN-catenin is a key regulator for the stability of synaptic contacts.

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P3-086 Zebrafish Pcdh family genes: genomic organization and expression during brain development

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Protocadherin family is a highly diversified receptor-type molecule in the nervous system. We have examined the Protocadherin genes of a zebrafish (DrPcdh). There were DrPcdh1 and DrPcdh2 gene clusters. The DrPcdh1 gene cluster contained DrPcdh1α and DrPcdh1γ gene clusters. On the other hands, DrPcdh2 gene cluster contained DrPcdh2o, DrPcdh2αa and DrPcdh2αb clusters. These constant regions were orthologs to that of the mammalian Pcdhs. Orthologous relationships, however, are few between zebrafish and mammalian variable exons. Their transcripts analysis revealed that the DrPcdh has alternatively spliced variants in the constant region that were not found in mammals. Expression of DrPcdh2α genes began from 16 hr stages in the bilaterally clusters in the brain. Hence, molecular diversity of DrPcdh family genes provides key insight towards the analysis of the diversity and complexity of vertebrate brains during evolution.

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P3-087 Splicing products of RA175/TSLC1/SynCAM and regulation of their cell adhesion activity by processing

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RA175 is a cell adhesion molecule of a new member of immunoglobulin superfamily with trans interaction activity. TSLC1, a tumor suppressor gene, and SynCAM, a cell adhesion molecule forming synapse, are isoforms of RA175. A 11 amino acid sequence is inserted in C-terminal region of RA175, but not in TSLC1/SynCAM. To understand the relationship between the biological functions and isoforms of the RA175/TSLC1/SynCAM, we examined their expression in mouse tissues by in situ hybridization and RT-PCR. A new exon (exon 8b; 33bp) was located between exon 8 (exon 8A) and exon 9; i.e., RA175 contained exon 8b, while TSLC1/SynCAM did not. Both RA175 and TSLC1/SynCAM mRNA were expressed in the developing nervous system and in the epithelium of the various tissues and were processed at extracellular region in the vicinity of the transmembrane domain. Thus, RA175 and TLC1/SynCAM may play roles as cell adhesion molecules in the nervous system and epithelium and their activity is regulated by processing enzyme.

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P3-088 The expression pattern of CNR/Pcdhα isoforms correlates with genomic methylation status.

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Diversified isoforms of CNR/Pcdhα family make a gene cluster in vertebrate genome. The isoforms are expressed in each neuron with different combination. It is possible that their combination gives individuality to each neuron and makes independent function of neural circuits. To explore the mechanism for the specific expression of CNR/Pcdhα isoforms in each neuron, we compared the mRNA levels of the isoforms and their genomic methylation status.
In neuroblastoma C1300, CNR/Pcdhα isoforms transcripted at low level were methylated in their genome. In contrast, the isoforms transcripted at high level were unmethylated in their genome. Furthermore, in C1300 cells treated with 5-aza-Cytidine (DNA methyltransferase inhibitor), CNR/Pcdhα genomic locus became hypomethylated, and an isoform transcripted at low level in untreated C1300 cells was upregulated. These data suggest that the expression pattern of CNR/Pcdhα isoforms correlates with genomic methylation status.

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P3-089 Distribution of L1-cam mRNA in the adult mouse brain.

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Immunohistochemical analysis revealed a wide distribution of L1-cam-positive neural and non-neural structures in adult mouse brain. Although there were numerous immunoreactive nerve terminals, only a few immunoreactive neuronal cell somata were detectable. To explore the distribution of L1-cam-producing cells, we performed in situ hybridization histochemistry using antisense L1-cam cRNA probe. L1-cam mRNA containing cells were distributed widely in adult brain. All positive cell somata were neuronal in the present study. A high density of neuronal L1-cam mRNA was found in the thalamus, mammillary body, and hippocampus. In addition, marked condensation of strong hybridization signals was localized in various neurons such as the pyramidal, mitral, principal neurons of some cranial nuclei, and presumably monoaminergic cells. Collectivelly, it is suggested that L1-cam has wide and significant roles in the excitatory neural networks of the adult brain.

P3-090 Structural and functional characterization of CNR/Pcdhα family protein

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CNR/Pcdhα family is the diverse set of synaptic cadherins. This family protein is composed of six extra cellular (EC1-6) domains, a trans-membrane domain and an intracellular domain. Especially, EC1 domain is well conserved among mouse CNR/Pcdhα family members. It is already reported that EC1 domain of classical cadherins has crucial roles in cell adhesion, however, current knowledge of CNR/Pcdhα's function is still insufficient. Here, we report structural and functional characterization of CNR1/Pcdhα4 EC1. Structural analysis was examined using NMR and mass spectroscopy. Besides, we established functional assay system to reveal its functional significance. Variously truncated CNR/Pcdhα fusion proteins were developed and assessed its adhesive property by visualizing binding sites in the several ages of mouse brain. We show our current trials for elucidation of CNR/Pcdhα protein structure-function relationship.

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P3-091 Variation of *CNR /Pcdh* α transcripts in a single neuron

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The *CNR /Pcdh* α genes, diverse cadherins in the brain, make a gene cluster analogous to immunoglobulin and T cell receptor genes. Expression study have demonstrated that individual neurons can express an overlapping but distinct subset of *CNR /Pcdh* α genes. To further investigate the variation of the *CNR /Pcdh* α transcripts in single neuron, we did RT-PCR and we did direct sequence methods. We found differential expression pattern of each variable exon in individual neuron. It was possible that RT-PCR amplified from only a single cDNA molecule. To address this concern, we confirm the expression pattern of *CNR /Pcdh* α transcripts in single neuron without PCR methods.

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P3-092 CNR/Pcdhα family proteins in the developing cortical connectivity

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The CNR/Pcdhα family is one of the diverse protocadherin families identified as candidate determinants of neuronal connection specificity. However, their roles during neural circuit formation have never been examined. Here, we performed immunohistochemical analysis in vivo and in vitro function assay of CNR/Pcdhα family proteins. Immunohistochemistry for the conserved portion of the proteins revealed that CNR/Pcdhα proteins consistently appear in the subplate neurons, and the developing main input and output axonal fiber trajectories between the cortex and the rest of the central nervous system, while down-regulated after forming functional neural circuits. Next, using in vitro functional assay system, we found that the extra-cellular domain of CNR1/Pcdhα4 protein induce neurite outgrowth in embryonic cortical cells. Our in vivo and in vitro studies indicate their novel role in pioneering and establishing axonal fiber trajectories in developing cortical connectivity.

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P3-093 Numerical and morphological abnormalities of L1-immunoreactive synapse in neuropsin-deficient mice

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Neuropsin, a serine protease, is a candidate molecule to regulate synaptic adhesion depending on neural activity. Here we demonstrate that neuropsin-deficient mice (NP-/-) induced synaptic abnormality in hippocampal CA1 subfield using quantitative electronmicroscopic analysis. NP-/- mice showed a decrease in total synaptic number whereas increase in spine number. Surprisingly, the number of cell adhesion molecule L1-immunoreactive synapses was increased in NP-/- mice. In addition, NP-/- mice showed the L1 accumulation in small varicosity-like structure which contains synaptic vesicle lacking postsynaptic specialization. Additional experiment showed that many L1-immunoractive presynapses formed filopodial extension which is a characteristic motile structure of mossy fiver presynaptic terminal. Together, these in NP-/- mice finding predict that neuropsin could act on presynaptic L1 to change synaptic formation.

P3-094 Screening of possible postsynaptic binding partners for presynaptic L1CAM in the hippocampus

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In adult hippocampus, L1CAM, a neural cell adhesion molecule of the immunoglobulin super family, is considered to play important roles in neural plasticity. Previous studies have shown that L1CAM-dependent adhesion modulates establishment and stabilization of long-term potentiation (LTP). This fact strongly suggests that synaptic strength is modulated by the interactions of pre- and postsynaptic cell adhesion molecules. However, it is unclear what sort of molecules interacts with L1CAM in the hippocampus. Here, we screened postsynaptic binding partners of L1CAM by immunoprecipitation using antiL1CAM antibodies. The immunoprecipitates contain three unidentified proteins of 110, 130, and 160kDa in addition to L1CAM. The immunoprecipitates does not contain classic cadherin, NCAM, contactin, TAG-1, NB-2, NB-3, KILON, OBCAM, or glutamate receptors (NR1, 2A, 2B, GluR1, 2, 3, 4).

P3-095 Role of complex N-linked oligosaccharides on embryonic neural stem cells

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The cell surface carbohydrate moieties are important element of intercellular communications such as differentiation and development in the immune system. However, in the nervous system, the role of oligosaccharides on neural stem cells remains unknown.In this study, we performed immunostaining with phaseolus vulgaris agglutinins (PHA) which is specific for complex type oligosaccharides. At E12 brain, PHA-immunoreactivity was present on the cell surface of the lateral region of ventricular zone (VZ) and subventricular zone (SVZ) of lateral ventcle, as well as ependymal cells layer. PHA showed partial overlap with nestin in the lateral SVZ. Next, we investigated the influence of PHA-binding to E12 telencephalic cells. On PHA-coated dish, many nestin-positive attached cells were detected 0.5h after plating. These results suggest that the some type of complex N-linked oligosaccharaide on neural stem cells could play important roles on cell adhesion.

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P3-096 Differential expression of CNR/Pcdhα family proteins in the olfactory and vomeronasal nerves

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CNR/Protocadherin (Pcdh) α family consists of 14 members in mice. It has been demonstrated CNR/Pcdhα family mRNAs are highly expressed in the olfactory and vomeronasal neurons. To investigate the distribution pattern of each CNR/Pcdhα protein in these neurons, we raised CNR/Pcdhα isoform-specific antibodies against CNR/Pcdhα v4, v9 and v11, and then examined the localization of CNR/Pcdhα isoforms by immunostainig. In the olfactory neurons, we observed CNR/Pcdhα v4, v9 and v11 immunoreactivities in their axons. In the vomeronasal nerves, however, we could not detect CNR/Pcdhα v9 and v11 whereas CNR/Pcdhα v4 immunoreactivity was clearly observed. Moreover, a part of the olfactory glomeruli showed considerably weak immunoreactivities for CNR/Pcdhα v9 and v11. Our results demonstrate the differential expression of CNR/Pcdhα proteins in the olfactory and vomeronasal nerves and it may be involved in the formation of the topographical projections of these nerves to the olfactory bulb.

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P3-097 Phenotype analysis of CNR/Pcdhα splicing variant-specific gene-targeted mice

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CNR/Pcdhα family is a subfamily of the clustered protocadherin family that consists of three subfamilies and over fifty protocadherins. It has been proposed that the diversity of the clustered protocadherins potentially contributes to the neuronal recognition during the brain development. Little is known, however, about the roles of the clustered protocadherins. To examine in vivo function of the CNR/Pcdhα family, we generated several CNR/Pcdhα gene-targeted mice and subjected them to morphological and behavioral examinations. Among these mutants, CNR-B$^{-/-}$mice, which lack B-type alternative splicing form of CNR/Pcdhα, displayed enhanced contextual conditioning, whereas cued conditioning remained same as that of wild-mice in the contextual and cued fear conditioning test. No significant impairment in physical characteristic, open field test, home-cage activity and motor coordination were found in CNR-B$^{-/-}$mice. Our results indicate that B-type splicing variant of CNR/Pcdhα is required for normal brain function.

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P3-098 Activation of the signal cascade by the activity-regulated adhesion molecule Arcadlin in brain

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Arcadlin is rapidly and transiently induced in hippocampal neurons by synaptic activity. The extracellular (EC) domain of Arcadlin consists of six cadherin repeats, and its cytoplasmic region interacts with a splice variant form of TAO2 kinase that is the most upstream kinase of p38 MAPK pathway. We reported the homophilic interaction of EC domains induced p38 MAPK phosphorylation and internalization of Arcadlin in HEK293T cells.
In this study, we investigated the functional coupling among Arcadlin, TAO2 and p38 MAPK in brain. We confirmed that Arcadlin was co-immunoprecipitated with TAO2 from the rat brain. We observed that the homophilic binding of Arcadlin activated p38 MAPK in *arcadlin-EGFP*-overexpressing neurons. We are currently examining whether the homophilic interaction of Arcadlin induces the internalization of Arcadlin in synapses. This study will give us a clue to understand an activity-induced synaptic reorganization.

P3-099 In vitro ischemia regulates proliferation and differentiation of murine neural stem cells.

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We examined the effect of hypoglycemia or hypoxia on the proliferation and the differentiation of NSCs in vitro. NSCs derived from the ganglionic eminence of E15.5 mice were cultured by neurosphere method, and treated with low glucose or low oxygen medium. We found that under continuous hypoglycemic conditions, the proliferative activity was significantly diminished and more neurospheres with a smaller diameter were observed compared with in control medium condition. Furthermore, hypoglycemia during the differentiative period significantly increased the differentiation of NSCs into both neurons and astroglial cells. On the other hand, hypoxia enhanced the proliferative activity and differentiative activity to both neurons and astroglias. These results suggest that hypoglycemia and hypoxia differentially regulates NSCs functions via distinct mechanisms.

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P3-100 Low-dose NO promotes axonal regeneration of cat retinal ganglion cells

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Anti-glaucoma drug, nipradilol (NPR), promotes axonal regeneration of retinal ganglion cells (RGCs). We examined either NO supply or the blocking adrenoreceptors promotes the regeneration of RGCs when a peripheral nerve is transplanted to the optic nerve. An injection of NPR 0.1 μM increased mean numbers of regenerated (R-) RGCs into 13,884 6 weeks after the transplantation, comparing 3,470 in no-injected control retinas. An injection of sodium nitroprusside 0.1 mM increased the numbers of R-RGCs by 1.9 fold, and DETA/NONOate 10 μM did them by 1.7 fold. None of adrenoreceptor blockers increased the numbers of R-RGCs or axonal regeneration. Numbers of R-RGCs were not increased when NO-scavenger was injected prior to the NPR injection. Injections of NPR and then combination of BDNF, CNTF, forskolin had no additive effect. The results suggest that axonal regeneration of cat RGCs is promoted by NO provided by NPR at low concentration. The mechanism is not understood why BDNF, CNTF, forskolin injection did not increased the numbers additively.

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P3-101 Effect of pleiotrophin, up-regulating in dopamine-depleted striatum, on neural transplantation

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Pleiotrophin (PTN) and GDNF expressions are enhanced in dopamine (DA)-depleted striatum, and PTN as well as GDNF promotes survival of DAergic neurons in vitro. To investigate the effect of PTN on grafted DAergic neurons, ventral mesencephalic donor cells were treated with PTN (100 ng/ml) and/or GDNF (500 ng/ml) during cell preparation, and then they (3.0×10^4) were grafted into the striatum of hemi-parkinson model rats. In PTN-treated group, improvement of methamphetamine-induced rotations was found earlier and it was significantly greater at 5 weeks after transplant, and there were more TH-positive cells in the striatum compared to those in control. In co-treated (PTN and GDNF) group, more prominent motor recovery and cell survival were found, showing that effects of PTN and GDNF on grafted DAergic neurons were additive. Data suggest that PTN treatment during cell preparation promoted survival of grafted DAergic neurons.

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P3-102 Comparison of the Nogo binding specificity between Nogo receptor and its homolog

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The axonal re-growth after injury is difficult in the mature CNS neuron. Currently, it is thought tat this problem is partly due to inhibition of growth by the molecules in the surrounding cells of the neuron. There are several myelin-derived inhibitory molecules characterized, i.e., Nogo, myelin-associated glycoprotein, and oligodenderocyte-myelin glycoprotein. The receptors for these three molecules are curiously the same as each other, identified as Nogo receptor (NgR). NgR, has two other homologs, i.e., NgRH1 and NgRH2. Although it was reported that both homologs do not show the binding activity to those growth-inhibitory ligands, which part of the NgR molecule is the most important for the ligand recognition is not clearly shown. We have succeeded in semi-quantitation of the binding between Nogo and NgR or NgRH2, and NgRH2 has a weak binding activity to Nogo. We are now examining the deteremination of its precise binding site.

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P3-103 Establishment of LacZ-labeled mouse and rat Schwann cell lines for neural transplantation

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Although Schwann cell transplantation has been extensively studied as a therapeutic strategy against neurotrauma and demyelinating disorders, clear identification of the transplanted cells has been difficult. In this study, immortalized Schwann cells derived from mouse (IMS32) and rat (IRS1) were transfected with pcDNA3.1/lacZ, and X-Gal-positive stable transformants IMS32L1 and IRS1L11 were established. Their Schwann cell properties were confirmed by immunofluorescence and RT-PCR. The mouse IMS32L1 cells were identified as X-Gal-positive cells after transplantation into fetal and adult striatum and lateral ventricle without tumor formation. Similarly, the rat IRS1L11 cells survived after transplantation into adult striatum and facial nerve avulsion injury site. These results indicate that the LacZ-labeled Schwann cell lines can be transplantable to various experimental models for neurotrauma and neurodegeneration.

P3-104 Decrease of larger retinal ganglion cells regenerating axons into a peripheral nerve graft of rats

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Rat retinal ganglion cells (RGCs) regenerate their axons into a grafted peripheral nerve segment (PN). When RGCs with regenerated axons were labeled with rhodamine-dextran, numbers of larger RGCs (L & LL) reduce after 6 wk while medium-sized and small cells do not for 8 wk. The causes for L & LL decrease can be attributed to somal shrinkage, cell degeneration, or axon retract of L & LL. To clarify whether shrinkage occurs, we grafted a PN of varying length. Results that reduction of L & LL numbers was prolonged may exclude somal shrinkage. When RGCs were labeled with DiI put at the PN end at wk 3-6 then the retina was fixed at wk 9, less reduction of numbers of L & LL occurred. Labeling with DiA at wk 8-10 showed similar results. These results suggest that few regenerated RGCs degenerated or retracted axons. We conclude that there may be other reason(s) for the decrease of numbers of regenerated L & LL.

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P3-105 The role of Benzodiazepine receptor in the neural stem cells proliferation

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Benzodiazepine receptors (BZR) are classified into two types (ω1 and ω2). In this study, we examined the effects of diazepam (agonist for ω1 and ω2 receptor) or zolpidem (agonist for ω1 receptor) on the proliferation of the cultured neural stem cells (NSCs). The NSCs were derived from E15 mice striatum and their proliferation was evaluated by WST-8 assay The treatment with diazepam, but not zolpidem, suppressed EGF-induced proliferation of the NSCs in a concentration-dependent manner.When we examined the expression of BZRsubtypes by RT-PCR, we found that NSCs expressed dominantly α2, α3, β2, β3 and γ1 subunit, which are known to form ω2 type of BZR. These results indicate that NSCs proliferation is regulated by ω2 type of BZR and raise a possibility that agents for ω2 types of BZR diminished the self-renewal activity of the NSC in the CNS.

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P3-106 Putative precursor cells for microglia in vitro; microglioblasts and promicroglioblasts.

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We characterized three stages of microglial differentiation; microglia (MG), MG-precursors microglioblasts (MGBs), and MGB-precursors promicroglioblasts (ProMGBs). Any of the cells expressed a microglia marker CD11b. Among the three types of cells, only MG expressed inducible nitric oxide synthase (iNOS) in response to lipopolysaccharide treatment. MGBs were characterized by large somata, high-level expression of nestin, and high proliferative activity. ProMGBs turned into cells with neuroectodermal phenotypes by incubation in serum-free medium. Although nestin has been considered as a marker for neural stem cells, it is also useful to determine the differentiation stage of MG-lineage cells. The high expression of nestin in MGBs may be related to the multipotential of MG-lineage cells.

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P3-107 Migrating neuronal precursors in adult rat brain express drebrin E but not drebrin A

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We have previously shown that the cells, which cell somata are intensely immunostained with anti-drebrin antibody (M2F6), made a stream from the anterior horn of lateral ventricle to the olfactory bulb (RMS). This suggests that migrating neuronal precursors express drebrin, but it has not been clarified which drebrin isoform is expressed in them. In this study, we carried out immunohistochemical analysis using a newly generated antibody that selectively recognizes drebrin A (RDA3). The packed cells immunostained with M2F6 in RMS had morphological characters of elongated bipolar shape, and a high nuclear/cytoplasmic ratio. These cells were GFAP-negative and subsets of them are Ki-67-positive. On the other hand, RDA3 did not immunostain these migrating cells, although it intensely immunostained neuropils. These findings indicate that migrating neuronal precursors in adult rat brain express drebrin E but not drebrin A.

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P3-108 Cytoplasmic $p21^{Cip1/WAF1}$ enhances axonal regeneration and functional recovery after spinal cord injury in rats

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$p21^{Cip1/WAF1}$, known as a cell-cycle inhibitory protein, facilitates neurite outgrowth from neurons when present in the cytoplasm. The molecular mechanism of this action is that $p21^{Cip1/WAF1}$ forms a complex with Rho-kinase and inhibits its activity. As myelin-derived inhibitors of axonal outgrowth act on neurons by activating Rho, that is responsible for the lack of spontaneous regeneration of the injured central nervous system (CNS), Rho-kinase may be a good molecular target against injuries in the CNS. In this study, we delivered TAT-fusion protein of cytoplasmic $p21^{Cip1/WAF1}$ locally after dorsal hemisection of the thoracic spinal cord in rats. The treatment significantly stimulated axonal regeneration and recovery of hindlimb function, and inhibited the cavity formation in the spinal cord after the injury. Cytoplasmic $p21^{Cip1/WAF1}$ may provide a potential therapeutic agent that produces functional regeneration following CNS injuries.

P3-109 Generation of motor neurons from mouse embryonic stem cells: analysis of ES-cell-derived neural cells

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ES cells are pluripotent stem cells which are able to differentiate into various cell types including neural cells. In this study, we investigated the precise expression profiles of neural and regional specific genes by embryoid bodies (EBs) exposed to retinoic acid (RA). The results indicated that the concentration of RA strictly and simultaneously regulates the temporal and positional identity during neural differentiation of ES cells. Next, we have established the strategy of inducing regionally specified neurospheres from EBs exposed to low-concentration RA, which efficiently generated HB9-positive motor neurons. These neurons showed functional property as neurons in the electrophysiological analysis, and formed neuro-muscular junctions *in vitro*. This strategy may provide an in vitro model for investigating motor neurons, and may be applied to the treatment of ALS, if it is applicable to human ES cells and if these motor neurons can function *in vivo*.

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P3-110 Expressions of Reg/PAP family members are differentially regulated after nerve injury

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Reg-2/PAPI is a member of Reg family and has been identified as a Schwann cell mitogen as well as a neurotrophic factor. In this study, we examined the expression of rat Reg/PAP family members both in injured nucleus and nerves after rat hypoglossal nerve injury model. In situ hybridization and RT-PCR analysis demonstrated that expression for Reg-2/PAPI mRNA was remarkably induced in injured motoneurons and substantial increase of PAPIII mRNA expression was observed in the distal part of injured nerves. The immunohistochemical analysis using anti-PAPIII antibody confirmed the increased level of PAPIII protein in Schwann cells of injured nerve. These results suggest that Reg/PAP family members act as potent mediators for Schwann cell and neurons to serve appropriate functions during nerve regeneration.

P3-111 Effects of granulocyte colony-stimulating factor (G-CSF) on spinal cord injury in mice

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The aim of the present study is to elucidate the efficacy of granulocyte-colony stimulating factor (G-SCF) to treat spinal cord injury. Bone marrow cells were isolated from GFP Tg mice and transplanted intravenously into irradiated C57BL/6 mice. Four weeks after bone marrow transplantation, spinal cord injury was produced by a static load at T8 level. G-CSF was injected for 5 days. Motor recovery and histology were assessed. G-CSF treated mice showed significant recovery. Immunohistochemistry revealed that GFP-positive cells were detected in the spinal cord sections. In conclusion, G-CSF may have beneficial effect for spinal cord repair.

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P3-112 Oligodendroglia differentiation of neural stem cells in a demyelination model

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Mammalian neural stem cells reside not only in developing brains but also in adult brains, and contribute to continuous neurogenesis in particular regions of the adult brain throughout lifespan. However, little is known about the behavior of adult neural stem cells in a damaged brain. To investigate the roles of the neural stem cells in pathology, we utilize a mouse model for demyelination; proteolipid protein transgenic (PLP tg) mice. PLP tg mice, which pathogenically mimic a human hypomyelination disease of Pelizaeus-Merzbacher disease, show normal myelination but later present demyelination after 4 months. We isolated neural stem cells as neurospheres from E14.5 GFP mouse brains, then transplanted them into 4 to 8 month-old PLP tg mice. The transplanted cells survived and differentiated into oligodendrocytes. The present system was proved to be useful in studying the neural stem cell kinetics in pathologic conditions and will provide information for therapeutic use of the neural stem cell.

P3-113 Lamina-specific Morphogenesis of Dentate Granule Neurons

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Dentate granule cells (DGCs) and their elaborating microcircuits have been implicated in hippocampus-dependent memory encoding and epileptogenesis, but little is known about how the proper maturation of DGCs is determined by their intrinsic programs or external factors during development. In order to explore this, we dispersed premature DGCs on living hippocampal slices. The density of surviving DGCs, which were engrafted on living hippocampal slices was the highest on the stratum granulosum. The mossy fibers growing on this substratum were properly guided towards CA3, whereas other misplaced DGCs exhibited heterotopic axon projection. In particular, about half of the axons originating from the hilus were misguided into the molecular layer, which resembles supragranular mossy fiber sprouting seen in epileptic disorders. These results suggest that local environmental factors influence the cell adhesion, neurite polarization and axon guidance of DGCs.

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P3-114 A new cDNA clone of which expression increased after optic nerve transection in goldfish retina

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The mammalian central nervous system, CNS, fails to regenerate after lesions, while the CNS neurons of fish successfully recover their axons after lesion. We have investigated this recovery mechanism of CNS axons by using goldfish optic nerve. To study which type of genes involves in regeneration, we have cloned several genes of which expression level increase after optic nerve transection, by a differential hybridization method. The clone 18 was one of cDNA clones obtained, and we found its mRNA amount increased 2 to 5 days after axotomy to about 2 times in retina. The clone 18 was expressed in ganglion cell, inter nuclear, and outer nuclear layers. Because the clone 18 obtained did not contain protein coding region, we are re-cloning the complete cDNA of clone 18 now.

P3-115 FGF-2 facilitates axonal regeneration of the injured spinal cord via inducing CNS-derived fibroblasts proliferation

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We have already reported that FGF-2 facilitates axonal regeneration and improves locomotion activity in the completely transected spinal cord. In this study, we examined how FGF-2 affects to axonal regeneration in the injured spinal cord. The rat spinal cords were completely transected at the position of T10, and 1 ml each of FGF-2 (1 mg/ml) or vehicle solution was injected five times into rostral and caudal sites of the injury. FGF-2 increased in the number of the cells in the lesion site, compared with control. The majority of them are fibronectin-positive fibroblasts. To characterize FGF-2 induced fibroblasts (FIF), we tested whether FIF promotes the cortical neuritic extension compared with meninx-derived fibroblasts (MDF) invading the lesion site. We also investigated their property by using RT-PCR and immunocytochemical technique. The results suggested that FIF is involved in the therapeutic effect of FGF-2.

P3-116 Does retinoic acid trigger axonal regeneration of fish optic nerve after injury ?

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We cloned a goldfish homolog to chicken purpurin, a retinol-binding protein in the goldfish retina 2-5 days after optic nerve transection. We further prepared the recombinant purpurin from E.coli inserted by purpurin encoding cDNA. In retinal explant culture system, purpurin alone did not affect any change of neurite outgrowth, but a concomitant addition of purpurin with retinol induced a dramatic enhancement of neurite outgrowth in the adult goldfish retina for 5 days of culture. Retinoic acid, a metabolite of retinol also induced a comparable neurite outgrowth to the purpurin with retinol. Furthermore, the neurite promoting effect of purpurin with retinol was completely blocked by presence of disulphiram, a specific inhibitor of retinoic acid synthesizing enzyme. The data all together suggest that retinoic acid triggers neurite outgrowth of optic nerve in the adult goldfish retina after lesion.

P3-117 In vitro and in vivo characterization of novel semaphorin 3A inhibitors

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Semaphorins are thought to act as inhibitory factors for regeneration of injured axons as well as neural development. However, direct evidence to support the hypothesis has not been available. We isolated novel semaphorin 3A (Sema3A) inhibitors from the fungus *Penicillium* sp. The inhibitors completely abolished collapsing and repulsive activity of Sema3A *in vitro* at less than several μM. Analysis for mechanism of action suggested that the inhibitors interact with Sema3A directly and inhibited the binding of Sema3A to its receptor. When a Sema3A inhibitor was administrated in the lesion site of rats following olfactory nerve axotomy, the regeneration of the olfactory nerve was significantly accelerated. The result suggested the involvement of Sema3A in neural regeneration as an inhibitory factor. The inhibitors should be excellent molecular probes to investigate the function of Sema3A both *in vitro* and *in vivo*.

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P3-118 Possible neurogenesis in the hippocampal CA3 subfield after trimethyltin-induced neural damage

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Our previous studies have indicated that trimethyltin chloride (TMT) leads to enhancement of neurogenesis in the dentate gyrus following neurodegeneration in the dentate granule cells selectively. Here we find possible neurogenesis in the hippocampal CA3 subfield after TMT treatment in mice. Positive cells immunoreactive to 5-Bromo-2'-deoxyuridine-5'-monophosphate and proliferating cell nuclear antigen were markedly increased in the hippocampal CA3 subfield and dentate gyrus 2 to 7 days after TMT treatment. Furthermore, Nestin-positive cells were increased transiently in the CA3 subfield 2 to 5 days after treatment. Double staining of NeuN with BrdU revealed that NeuN co-localized with most of BrdU-positive cells in the CA3 subfield. These results suggest that the hippocampal CA3 subfield have the ability of generating neurons following the dentate neurodegeneration induced by TMT in mice.

P3-119 Analysis of progenitor cells in the striatum and midbrain by MPTP administration

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We examined the progenitor cells in the striatum and midbrain by MPTP administration. To explore the repair processes mediated by brain progenitor cells, we focused on a selective lesion of the nigrostriatal dopaminergic pathway by MPTP administration. To detect the progenitor cells in the brain, we used transgenic mice expressing green fluorescent protein (GFP) under the nestin promoter. A single dose of MPTP was administered i.p. to 10 week old transgenic mice. Immunocytochemistry was performed. Abundant nestin and S100 beta immunoreactivities were predominately found in the striatum of MPTP-treated transgenic mice expressing GFP under the nestin promoter. Profound astrogenesis in the striatum of young adult mice in response to toxic dopaminergic insult was found.

P3-120 Characterization of immature bone marrow cells migrating into the brain parenchyma after bone marrow transplantation

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We have reported that some immature bone marrow cells (BMC) migrating into the brain parenchyma in an early phase after bone marrow transplantation (BMT) express early hematopoietic markers. The present study further characterizes GFP-positive BMC in the brain after BMT. Immuno-double labeling confirmed that BMC in the brain expressed hematopoietic, but not neural markers and some of them preserved immaturity at even four and eighteen weeks after BMT. Analysis of the DNA content indicated that most of the migrated BMC were arrested at the G0/G1 phase. When cells from the transplanted brain were cultivated, GFP-positive BMC became proliferative. About 40% of them changed morphology to ramified form like microglia and expressed microglial markers. Furthermore, a part of them expressed immature hematopoietic markers. Thus a part of BMC can transform to the cells indistinguishable from microglia or microglial precursor cells when migrated into the brain.

P3-121 Epibranchial placodes have a developmental potential of the olfactory placode.

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The olfactory placodes (OP) give rise to the sensory receptor cells of the olfactory epithelia (OE), whose axons project to the olfactory bulb (OB). Ablation of OP resulted in the failure of development of OE and disturbed the development of OB in chick embryos. By transplanting the quail cranial placodes on ablated chick OP, we examined whether quail placodes repair the development of chick olfactory system. The quail OP developed a normal appearing OE and chick OB developed comparable to the intact OB. The explanted quail trigeminal placodes did not form OE but developed ganglions whose axons directed to the OB. In several cases of transplantation of the quail epibrachial placodes, there appeared OE constituting with host cells in addition to quail-derived ganglia and OB developed comparable size to the intact OB. We will also discuss the influence of olfactory nerve on the histogenesis of the OB.

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P3-122 RGM is a neurite outgrowth inhibitor upregulated in response to central nervous system injury

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RGM (repulsive guidance molecule) has been previously reported to play a role in the formation of the topography of retino-tectal projections of embryonic chicken. Here we report RGM as a potent inhibitor of axon regeneration in the adult central nervous system (CNS). RGM inhibits mammalian CNS neurite outgrowth by a mechanism dependent on Rho/ROCK. Expression of RGM is found in oligodendrocytes and neurons in adult rat spinal cord and is induced around the injury site following spinal cord injury. These data suggest that RGM is a potent inhibitor of axon regeneration in the adult CNS. In addition, an antibody to RGM efficiently blocks the effect, providing a possible therapeutic agent in clinical conditions characterized by a failure of CNS regeneration.

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P3-123 Characterization of EGF and FGF responsive human embryonic neural stem/progenitor cells

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Individual effect of EGF and FGF on human cultured embryonic neurospheres proliferation, phenotype expression and differentiation were studied. We found that EGF responsive cells grow slowly than FGF responsive cells and for this higher number of FGF responsive cells were present in the neurospheres than EGF responsive cells. During neurospheres culture EGF generated neurospheres easily attached to the surface of the non-coated flask and begun to differentiate easily whereas FGF generated neurospheres remained as floating. The most important finding was that FGF responsive cells express higher level of progenitor cells as well as progenitor markers than EGF responsive cells, which led FGF responsive cells more differentiation than EGF responsive cells.

P3-124 Role of Keap1/Nrf2/Antioxidant-Responsive Element/Hemeoxygenase-1 System in the Neuroprotective Effects by Cyclopentenone Prostaglandin Derivatives

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Here we report Keap1/Nrf2/antioxidant-responsive element (ARE)-mediated neuroprotective effects by NEPP compounds. The bindings of NEPP to proteins were abolished by N-ethylmaleimide. NEPP11 stimulated the expression of a luciferase under the controlof HO-1 enhancer in an ARE-dependent manner and activated an ARE core element. NEPP11 increased levels of Nrf2 protein and enhanced its translocation from cytoplasm into nucleus. A biotinylated NEPP bound to Keap1. In primary cortical cultures, NEPP11 activated HO-1 enhancer in an ARE-dependent manner and protected neurons against excitotoxity. HO-1 protein was induced in neurons and a biotynylated NEPP accumulated mainly in neurons. Keap1/Nrf2/HO-1 pathway is a novel thrapeutic taret for drug development.

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P3-125 The regulation of unfolded protein response by OASIS, a transmembrane bZIP transcription factor, in astrocytes.

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We identified a novel endoplasmic reticulum (ER) stress transducer; OASIS, a basic leucine zipper (bZIP) transcription factor of the CREB/ATF family with a transmembrane domain that allows it to associate with ER. This molecule is cleaved at the membrane in response to ER stress and its cleaved N-terminal cytoplasmic domain, which contains the bZIP domain, translocates into the nucleus; and then activates transcription of target genes via acting to the ER stress responsive element and the cyclic AMP responsive element. Intriguingly, OASIS is induced at the transcriptional level during ER stress in astrocytes of the central nervous system but not is induced in other types of cells we examined, and its overexpression in astrocytes results in suppression of ER stress induced cell death. We are now trying to identify other OASIS target genes, that are transcriptionally induced by OASIS, by using microarray analysis.

P3-126 Overexpression of dE2F1 and dDP induces apoptosis in Drosophila postmitotic neurons

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Neurons exit from the cell cycle just after differentiation from their precursors. E2F1 is a transcription factor to promote cell cycle progression and must be silenced in neurons. Drosophila melanogaster has two E2F factors, dE2F1 and dE2F2, both of which dimerize with dDP and bind to the promoters of E2F-regulated genes. dE2F1 is a potent transcription activator, whereas dE2F2 serves as a transcription repressor. Here we show that dE2F1/dDP overexpression induces apoptosis in postmitotic neurons. The GAL4/UAS system was adopted for the targeted expression of dE2F1/dDP, and nuclear DNA fragmentation, a characteristic of apoptosis, was detected by TUNEL reactions. Targeted expression of dE2F1/dDP to neurons using the elav-GAL4 driver resulted in apoptosis in the larval brain. Similarly, dE2F1/dDP expression by Cha-GAL4 caused apoptosis of cholinergic neurons in the lamina of the fly optic lobe. These findings suggest that dE2F1 pathway activates the apoptotic machinery in postmitotic neurons.

P3-127 Purification of protein that interacts with procaspase-3 from rat neonatal cortex

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Recent studies have provided the evidence that neuronal susceptibility to cell death decreases during brain development. Using in vitro caspase-3 activation system (PC12 cell extract), we have searched for the age-related factors that enhance apoptosis. Addition of extract from neonatal rat cortex markedly potentiated the cytochrome c-induced caspase-3 activation. During the purification, we found using chemical cross-linking experiments with disuccinimidyl suberate that there was a complex consisting of dimer of procaspase-3 and an unidentified 20 kDa protein in the extract from neonatal cortex. Here, we report the purification of the 20 kDa protein that interacts with procaspase-3 and promotes the activation of procaspase-3.

P3-128 Calcineurin stablizes hypoxia-inducible factor (HIF-1α).

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Hypoxia-inducible factor (HIF-1) is a heterodimeric transcription factor consisting of an α- and a β-subunit. The α-subunit of HIF-1 (HIF-1α) expresses when cells sense low level of oxygen and starts transcription although HIF-1α is rapidly degraded through the ubiquitin-proteasome system under normoxia. When SH-SY5Y cells were incubated under hypoxia for 24h, a majority of SH-SY5Y cells became AnnexinV-Fluorescein positive, indicating apoptosis of the cells. ω-conotoxin, a N-type calcium channel blocker, suppressed HIF-1α expression under hypoxia so did cyclosporinA (CsA), a calcineurin inhibitor. Further western blot analysis and immunofluorescence staining revealed that calcineurin increased expression of HIF-1α under normoxia. This effect of calcineurin was inhibited by addition of CsA and expression of dominant negative calcineurin (H101Q). These results suggest that calcineurin stabilizes expression of HIF-1α and may be involved in hypoxia-induced neuronal cell death.

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P3-129 Kainic acid-induced oxidative DNA damage in mouse brain and neuro-protection by MTH1 encoding an oxidized purine nucleoside triphosphatase

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It has been suggested that neuronal excito-toxicity caused by glutamate involves cellular damage caused by free radicals produced through multiple mechanisms during excessive excitation of neurons. In the present study, we examined accumulation of 8-oxoguanine (8-oxoG), an oxidized form of guanine, in mouse brains after systemic administration of kainic acid. We observed an increased accumulation of 8-oxoG in CA3 neurons of hippocampi from animals which suffered severe seizure. Expression level of Mth1 gene, which encodes an oxidized purine nucleoside triphosphatase hydrolyzing 8-oxo-dGTP or 2-OH-dATP to the monophosphate, was found to be altered in hippocampi from such animals. Now, we are comparing degrees of neuronal death in hippocampi of MTH1-null and wild-type mice after systemic administration of kainic acid, in order to examine whether MTH1 can protect neurons from the excito-toxicity.

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P3-130 Survival of developing motor neurons mediated by Rho/Rho-kinase signaling pathway

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Rho/Rho-kinase signaling pathway is essential for diverse cellular processes. In the present study, we report the in vivo role of this signaling pathway during development of motor neurons (MNs) in the spinal cord. Suppression of Rho/Rho-kinase signaling pathway in developing spinal MNs induced enhanced apoptosis of MNs at the early embryonic stages and moderately increased apoptosis at the late embryonic stages. Our findings indicate that this signaling pathway plays a crucial role in survival of spinal MNs during embryogenesis, particularly at the early developmental stages.

P3-131 Glycogen synthase kinase-3 inhibitors prevent caspase-dependent apoptosis induced by ethanol in cultured rat cortical neurons

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The effect of ethanol on cell viability was examined in rat cultured cortical neurons. Ethanol induced apoptosis which was characterized by cell shrinkage, nuclear condensation or fragmentation, and internucleosomal DNA fragmentation. Ethanol-induced apoptosis was prevented by N-methyl-D-aspartate, an agonist of NMDA receptor which is one of subtype of the ionotropic glutamate receptors. Incubation with glycogen synthase kinase-3 inhibitors SB216763 and alsteropaullone, but not a cyclin-dependent protein kinase 5 inhibitor roscovitine, completely protected the neurons from the apoptosis induced by ethanol. The apoptosis was accompanied by activation of caspase-3 and prevented by a caspase-3 inhibitor.These results suggest that ethanol induces caspase-dependent apoptosis mediated glycogen synthase kinase-3 activation in cultured rat cortical neurons.

P3-132 Manserin, a secretogranin II-derived peptide, is a novel neuropeptide

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We have isolated a novel 40-amino-acid neuropeptide, designated as manserin, from the rat. Manserin is derived from secretogranin II (SgII), a member of granin acidic secretory protein family by proteolytic processing, as previously reported secretoneurin and EM66. Immunohistochemical analysis using anti-manserin antibody revealed that manserin localized in the endocrine cells of the pituitary anterior lobe, but not in the posterior lobe. Interestingly, manserin never co-localized with ACTH in the anterior pituitary, which is in contrast with SgII, suggesting specific immunoreactivity of the antiserum against manserin. Manserin immunostaining was also observed in the neuronal cells of several hypothalamic nuclei and the neurons in the median eminence. These results suggest that manserin exerts a specific role in the neuroendocrine system.

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P3-133 Reciprocal regulation of caspase-3 by two splice variants of caspase-3 binding protein

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Caspase-3 plays a critical role in the process of apoptosis. To clarify this mechanism in neuron, we have purified novel caspase-3 binding proteins (C3BPs) from rat brain using affinity chromatography on an immobilized active-form of caspase-3. One of C3BPs (C3BP2) is supposed to have two alternative splice variants, full-length (C3BP2-F) and short form (C3BP2-S). Therefore, these two splice variants were expressed in each in mammalian cultured cells together with another active-form of caspase-3 (Csp). Csp showed caspase-3 activities and induced cell apoptosis, both of which were further enhanced by C3BP2-S, or inversely suppressed by C3BP2-F. Neither caspase-3 activities nor cell apoptosis were detectable with C3BP2-S or C3BP2-F, unless Csp was co-expressed. These results indicate that caspase-3 was reciprocally regulated by two splice variants of C3BP2.

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P3-134 Survival-promoting effect of transcorneal electrical stimulation on axotomized RGCs *in vivo*

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Previously, we demonstrated that electrical stimulation (ES) to the optic nerve (ON) enhances the survival of axotomized retinal ganglion cells (RGCs) in adult rats. In this study, we introduced transcorneal ES (TcES) and evaluated the neuroprotective effect of TcES on the axotomized RGCs. Seven days after injection of Fluorogold (FG) into the superior colliculi for retrograde labeling, the left ON was transected, then TcES was commenced immediately and lasted for 1 hour. One week after ON transection, mean surviving RGC density of ON transectioned retinas without TcES decreased to 54% of that of intact retinas. On the other hand, TcES increased the density of surviving RGCs up to 84% of intact retinas. In conclusion, TcES after ON cut enhanced the survival of RGCs *in vivo*.

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P3-135 The prevention of H_2O_2-induced necrosis by coactivation of PKA and PKC is mediated by ERK and p38 MAP kinase activation in PC12 cells.

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We previously reported that the coactivation of protein kinase A (PKA) and protein kinase C (PKC) prevented H_2O_2-induced death in PC12 cells. In this study, we investigated characteristic feature of H_2O_2-induced cell death (apoptosis or necrosis) and involvement of mitogen-activated protein (MAP) kinase in the cytoprotective effect of treatment with a combination of phorbol 12-myristate 13-acetate (PMA) and forskolin. H_2O_2 induced both apoptotic and necrotic cell death in PC12 cells. Pretreatment with a combination of 100 nM PMA and 300 nM forskolin for 24 hr inhibited H_2O_2-induced necrosis, but did not affect H_2O_2-induced apoptosis. PD98059 (a MEK inhibitor) and SB203580 (a p38 MAP kinase inhibitor) prevented the cytoprotective effect of the pretreatment. These results indicate that coactivation of PKA and PKC protects PC12 cells from H_2O_2-induced necrosis through ERK and p38 MAP kinase activation.

P3-136 Neuronal glutamate transporter, EAAC1/EAAT3, prevents neuronal death with novel anti-apoptotic function

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Nerve-injured motor neurons of the adult rat can survive, whereas similar axotomy causes gradual cell death of injured motor neurons in the adult mouse. Here, we identified that the expression of a neuronal glutamate transporter, EAAC1/EAAT3, in response to nerve injury was up-regulated in the rat, but down-regulated in the mouse, although most of the other molecules associated with cell survival responded similarly. Axotomy-induced neuronal death was abolished in the EAAC1 transgenic mouse, and EAAC1 over-expression rescued NGF-deprived PC12 cells. The EAAC1-mediated rescue activity was independent of glutamate uptake. The N-terminus of EAAC1 was essential for suppressing caspase-3 activation through the interaction with the newly identified EAAC1 N-terminal binding protein. This novel function of EAAC1 may shed light on a new therapeutic intervention for neurodegenerative disease.

P3-137 Activation of ATF6 in response to peroxynitrite-induced endoplasmic reticulum stress in human neuroblastoma SH-SY5Y cells

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It is widely accepted that the toxicity of peroxynitrite is mediated by mitochondrial dysfunction. We have reported that peroxynitrite induced expression of GRP78/Bip, which is an endoplasmic reticulum (ER) lumen protein induced during ER stress. ATF6 is a member of the leucine zipper protein family, can constitutively induce the promoter of GRP genes through activation of ER stress element. In the present study, we tested whether ATF6 is activated in response to peroxynitrite-induced ER stress. Treatment with a peroxynitrite generator N-morpholinosydnonimine (SIN-1) for 12 h induced cell death and a significant increase in the levels of high molecular ubiquitin-conjugated proteins in human neuroblastoma SH-SY5Y cells. SIN-1 increased the levels of GRP78/Bip and GRP94, and decreased the level of ATF6. These results indicate that peroxynitrite induces ER stress mediated by activation of ATF6.

P3-138 TRPM2 channels are involved in the H_2O_2-induced cell death in cultured cerebral cortical neurons

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A rat cDNA encoding TRPM2 was cloned from rat brain mRNA, which was characterized as Ca^{2+}-peameable channel opened by extracellular H_2O_2. In cultured cerebral cortical neurons from fetal rat, TRPM2 proteins are detected by immunocytochemistry. In Fura2-loaded neurons, application of H_2O_2 elicited a transient and a delayed-onset increases in intracellular Ca^{2+} concentration, which were caused by Ca^{2+} release and Ca^{2+} influx, respectively. Both TRPM2 immunoreactivity and H_2O_2-induced Ca^{2+} influx were decreased by knockdown of TRPM2 protein by siRNA treatment. Moreover, neuronal death induced by H_2O_2 was significantly suppressed by the knockdown of TRPM2. These results suggest that TRPM2 channels are involved in the redox state-dependent Ca^{2+} influx and neuronal death in immature cerebral cortical neurons.

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P3-139 Dysregulated protein expression of CRMP-2 in neurite degeneration following trophic factor withdrawal

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To elucidate the mechanisms of neurite degeneration, we have employed a mutant C57BL/Wld mouse possessing a unique phenotype of resistance to Wallerian degeneration. Cultured sympathetic neurons isolated from mutant mice are resistant to neurite degeneration after NGF withdrawal. By comparing two dimensional electrophoresis patterns from wild-type and mutant mice, we found that two spots were markedly up-regulated 24 hrs following NGF deprivation when neurite degeneration began to occur, whereas the corresponding spots were barely detected in mutant mice. MALDI-TOF-MS analysis revealed that the two spots correspond to CRMP (collapsin response mediator protein)-2 and 4. Western blotting using anti-CRMP-2 antibody (kindly donated from Prof. Ihara) showed that the band corresponding to 58kDa(±1kDa) was specifically up-regulated during neurite degeneration. It is currently under investigation as to whether it is due to different splicings or different posttranslational modifications.

P3-140 Activation of NMDA receptors suppresses kainate-induced neurotoxicity in murine hippocampus

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Our previous studies have showed that a prior treatment with NMDA protects the hippocampal pyramidal neurons against excitotoxicity of kainate in vivo. We show that pretreatment with the K+ channel blocker 4-aminipyridine (4-AP) can protect kainate-induced neuronal cell death in murine hippocampus. The systemic injection of kainate alone induced severe damages in pyramidal neurons of hippocampal CA1 and CA3 subfields 3-7 days later, with producing positive cells for an anti-single-stranded DNA antibody in the pyramidal neurons 1 to 3 days after the injection. The systemic administration of 4-AP 1-24 h before protects kainate-induced neuronal cell death in the hippocampal pyramidal neurons. MK-801 was effective in abolishing this neuroprotective effect of 4-AP against the neuronal damage induced by kainate. Taken together, in vivo activation of NMDA receptors is capable of protecting kainate-induced neuronal damage in murine hippocampus.

P3-141 Mitochondrial DNA mutation induced by trimethyltin in murine hippocampus

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Trimethyltin (TMT) induced neuronal damage specific for the dentate gyrus of mouse hippocampus. However, the mechanisms underling neuronal damage induced by TMT is not well understood. We evaluated if treatment with TMT induces damage in the mitochondrial DNA of the hippocampus. Mitochondrial DNA damage was detected by mean of PCR using primers specific for the mitochondrial DNA and templates of mitochondrial DNA prepared from the hippocampus at various time points after the systemic administration of TMT. TMT was effective in decreasing the products when the primers for PCR reactions were used for amplifying regions encoding NADH-ubiquinone oxidoreductase subunits, ND1 and DN6, 16 and 24 h after treatment. Direct sequencing analysis revealed that TMT treatment induces mutation in ND1 and ND6 16 h after TMT treatment. Taken together, TMT is capable of inducing mutation in mitochondrial DNA as one of mechanisms underling neuronal damage in murine hippocampus.

P3-142 Altered levels of glucocorticoid receptors in the mitochondrial fractions by dexamethasone in murine brain

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Recent studies have shown that Glucocorticoid receptors (GR) are present within mitochondria in the neuronal and muscle tissues. By means of the immunoblot analysis, in this study, we evaluated if GR are translocated into mitochondria in the discrete brain structures of mouse systemically injected with dexamethasone. Dexamethasone administration induced an increase in the level of GR in mitochondrial fractions, as well as nuclear fractions, of the frontal cortex and hippocampus 30 min up to at least 2 h later. However, both cerebellum and medulla-pons were insensitive to increasing the level of GR in mitochondrial fractions 1 h after the administration. In the frontal cortex of untreated animals, GR in the mitochondrial fractions was resistant to trypsin digestion. However, the mitochondrial GR increased by dexamethasone administration were digested by the treatment with trypsin. Taken together, glucocorticoid may be capable to affecting GR level in mitochondria in particular brain structures.

P3-143 Possible protective effect of endogenous glutathione against trimethyltin-induced neuronal cell damage

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Trimethyltin (TMT) is a kind of organotin derivatives that are known to induce behavioral changes and neuronal damage in human and rodent. To elucidate roles of glutathione (GSH) in TMT-induced neural damage, we examined the effects of GSH depletion on TMT-induced granule cell death in mice. 2-cyclohexen-1-one (CHO, 150 mg/kg) was i.p. injected into mice to prepare GSH-depleted animals. In untreated animals, TMT at the dose of 2 mg/kg led to expression of a few ssDNA-positive cells, which are a maker of DNA fragmentation, in the dentate gyrus 2 days after injection. Marked increase in the positive cells was seen in the dentate gyrus in GSH-depleted animals. In addition, TMT-induced activation of Jun N-terminal kinase pathway was enhanced in the dentate gyrus by GSH depletion. Taken together, endogenous GSH was capable of protecting neural damage induced by TMT in murine hippocampus.

P3-144 Gene expression profile change by transcriptional suppression in primary culture neurons: a model of polyglutamine diseases.

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Various molecular mechanisms are implicated in polyglutamine diseases. Among the hypotheses, a number of results have suggested that nuclear transport of abnormal proteins and resultant perturbation of nuclear functions are essential for the pathology. Especially, transcriptional dysregulation seems to be a critical component causing neuronal dysfunction. Meanwhile, it is not clear which types of genes are affected. We reported general transcription is disturbed in polyglutamine diseases. Therefore, we observed changes of gene expression profile in primary culture neurons treated with αamanitin, which is known to inhibit transcription by direct interaction with RNA polymerase II. We will discuss about some regulated genes and their roles.

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P3-145 Enhanced expression of the high-molecular-weigth heat shock proteins in murine hippocampus after trimethyltin treatment

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We tested changes in expression of high-molecular-weight HSPs in the hippocampus after trimethyltin (TMT) treatment in vivo. TMT was systemically injected into ddY male mice to cause neural damage in the dentate gyrus selectively. TMT was effective in increasing in any levels of HSP105, APG-1, and APG-2 in granule cell layer of the dentate gyrus, but not in CA1-CA3 pyramidal cell layer, 16 h to 2 days later. Double staining of NeuN and these HSPs revealed that these HSPs expressed by TMT almost co-localized with NeuN in the granule cells of the dentate gyrus. However, none of these HSPs was co-localized with GFAP in the dentate gyrus of TMT-treated animals. These results suggest that expression of high-molecular-weight HSPs is enhanced in the dentate granule cells following TMT treatment in vivo.

P3-146 Effects of nicotine on neural development and plasticity in the hippocampus

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From our previous studies, we confirmed that nicotine had beneficial effects on the hippocampal neurons partly by inducing expression of IGF-1 mRNA. Nevertheless, the situation may be somewhat different as far as the nicotine's effects on the neuronal progenitor cell, which is still on the way of differentiation are concerned.To clarify this theme, nicotine was intraperitoneally injected into rats in dose of 0.1mg/kg, 0.5mg/kg or 1mg/kg, and effects of nicotine on plasticity-related process in the dentate gyrus of the hippocampal formation were examined by immunohistochemical staining using specific antibodies against PSA-NCAM, NeuN and GFAP. As the results, PSA-NCAM and NeuN immunoreactivities were decreased by nicotine injection in a dose-dependent manner, while nicotine had no effect on GFAP expression. It is concluded that nicotine inhibits plasticity-related process including neurogenesis in the hippocampus.

P3-147 Characterization of inhibitory effects of brain-specific, SemaF (Sema4C)-associating protein, SFAP75, on serum-deprivation-induced apoptosis in CHO cells

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We have identified SemaF (Sema4C)-associating protein with an Mr of 75 KDa (SFAP75), but the physiological role was poorly understood. In this study, we have established SFAP75 stable expressing CHO cell line and the functional activities of SFAP75-overexpressing CHO cells and wild type CHO cells were compared. There were no differences in their growth and adhesion activities in both of CHO cells, but serum deprivation-induced cell death, characterized by DNA fragmentation and cell lysis, was markedly inhibited in SFAP75-overexpressing CHO cells. Furthermore, the caspase-3-like activity induced by serum deprivation also decreased in SFAP75-overexpressing CHO cells. These observations suggest that SFAP75 may play a role in regulation of caspase activity in neuronal cell death.

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P3-148 The CA2-CA3a is one of the most vulnerable regions to bicuculline stimulation in the rat hippocampal slice cultures

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It is known that the CA2 region of the hippocampus is more resistant to the principal cell loss seen in CA1 and CA3 in both animal models of temporal lobe epilepsy (TLE) and in human TLE. In order to understand the characteristics of the CA2 region, rat hippocampal slices from postnatal days 7-8 were cultivated for 6-7 days and then exposed to 6 μM bicuculline (BIC), a GABAA receptor antagonist, for 12-72h. Viability marker propidium iodide (PI) was used to monitor the cell loss. Significantly PI uptake was observed in the CA2-CA3a, but not in CA1 and CA3b-c regions after exposure to BIC for 12h. The PI uptake was suppressed by co-exposure with ω-agatoxin IVA, a P/Q-type voltage dependent calcium channel (VDCC) antagonist. These results indicate that the CA2-CA3a is one of the most vulnerable regions to bicuculline stimulation in rat hippocampal slice cultures and that the cell loss may be related P/Q-type VDCC.

P3-149 Glutamate toxicity in short-term cultures of rat motoneurons

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Glutamate-mediated excitotoxicity might contribute to the loss of motoneurons in most cases of sporadic ALS patients. However, there is no report on short-term in vitro assay systems for glutamate toxicity to motoneurons. Therefore, we aimed to establish the bioassay system to evaluate the glutamate toxicity using the pure culture of rat motoneurons. We found that glutamate toxicity was more remarkable when glutamate was added to the culture on day 0 and day 4 than on day 1 through day 3. The toxicity was evident with as less as 30μM of glutamate added on day 0, while 300μM added on day 4. These cell deaths were blocked by MK801 and NBQX, showing the involvement of both NMDA receptors and AMPA receptors in the cell death. This culture system may be useful for the analysis of selective glutamate toxicity to motoneurons.

P3-150 Action of a neuroprotective factor derived from cerebral hemispheres

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We previously reported that removal of meninges *in vitro* induced the massive neuronal death in the developing cerebral cortex. In the present study, we found that insulin is a causative agent of the cell death. The neuronal death in the cerebral cortex *in vitro* was completely rescued by the addition of an antioxidant, Trolox. Because Trolox is a non-selective scavenger for free radicals, we then used a selective scavenger of nitric oxide, carboxy-PTIO. Again carboxy-PTIO was could completely rescued the neuronal death. To investigate whether the removal of meninges results in the generation of free radicals, we monitored the fluorescence of dichlorofluorescein (DCF). The removal of meninges from cerebral hemispheres increased DCF fluorescence, which was inhibited by coculturing with other hemispheres. These results suggest that cerebral hemispheres secret a strong radical scavenger, which completely rescues the neuronal death induced by insulin *in vitro*.

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P3-151 Hydrogen sulfide protects neurons from oxidatieve stress

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Hydrogen sulfide (H_2S), which is a well-known toxic gas, is found in relatively high concentrations in the brain. Although a neuromodulatory role of H_2S has been demonstrated, there is little knowledge of its other biological functions. Here we show that H_2S protects primary cultures of neurons from death in a well-studied model of oxidative stress caused by glutamate, a process called oxidative glutamate toxicity or oxytosis. We found that H_2S increases the glutathione levels, which normally decrease during the cell death cascade, by enhancing the activity of γ-glutamylcysteine synthetase and up regulating cystine transport. Reduced cystine (cysteine) is the rate-limiting substrate of glutathione synthesis. These observations show that H_2S protects neurons from oxytosis by increasing the production of the antioxidant glutathione. (FASEB J., in press)

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P3-152 ER stress induced caspase-7 activation is regulated by Bcl-2 family at endoplasmic reticulum

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The Bcl-2 protein plays a ctitical role in apoptosis by antagonizing Bax and Bak at mitochondria and regulating activation of initiator caspase independently of the "apoptosome". However, it remains to be unknown about the contribution of endoplasmic reticulum (ER) for apoptosome independent caspase activation. We generated PC12 cells expressed by ER targeting (ER-) Bcl-xL, which was specifically localized at ER by substituting transmembrane region for cytochrome b5. We found that ER-Bax and ER-Bak, and ER stress caused caspase-7 activation, which was specifically inhibited by ER-Bcl-xL. These data suggested that the Bcl-2/Bcl-xL proteins regulate the release of proapoptotic molecules from mitochondria as well as the caspase-7 activation at ER by competing with Bax and Bak. Thus, translocation of BH3 protein, such as Bax and Bak, may drive apoptotic cascades at distinct subcellular compartments.

P3-153 Age-dependent mitochondria-mediated neuronal apoptosis in transgenic mice overexpressing cellular prion protein

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We demonstrate the mitochondria-mediated apoptosis in aged transgenic mice overexpressing wild type cellular prion protein (PrP^C). Although these mice remained healty and no pathological evidence was observed in their brains, surprisingly that exhibited an aberrant mitochondrial localization of PrP^Cin accordance with decreased proteasome activity, whereas younger littermate did not. These processes seems to result from the overflow of PrP^C over a long period as proteasomal function gradually decreases with age. Our observation may explain the age-dependent neurological dysfunction in overexpressing wild-type PrP^C.

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P3-154 Functional innervation of the interstitial cells of Cajal in the mouse small intestine

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Recent studies have demonstrated that intramuscular interstitial cells of Cajal (ICC) are preferential targets for neurotransmission in the stomach. ICC at the deep muscular plexus (ICC-DMP) of the small intestine form tight contacts with nerve terminals, but little is known about the role of these cells. ICC-DMP express neurokinin 1 receptors (NK1R) and internalize NK1R in response to exogenous substance P. We used NK1R internalization as an assay of functional innervation of ICC-DMP. In unstimulated muscles, NK1R was distributed around the peripheries. After electrical field stimulation (EFS), NK1R was internalized within the cell. NK1R was internalized in 80% of ICC-DMP, as compared to 10% of cells before EFS. Internalization was blocked by the NK1R antagonist WIN 62577 and by tetrodotoxin, suggesting that internalization resulted from stimulation of receptors with neurally-released neurokinins. These data suggest that ICC-DMP are primary targets for neurokinins released from enteric motor neurons.

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P3-155 Abdominal expiratory motor activity in the anesthetized neonatal rat

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We examined pattern of the spontaneous abdominal expiratory activity in the isoflurane-anesthetized (2.0 %) neonatal rat at postnatal days 1-3. Fine enamel-coated copper wires (50 μm) were inserted to diaphragm and abdominal muscles to get their electromyograms (EMG). When anesthesia became gradually light, the respiratory frequency and amplitude of EMG in the diaphragm increased. Furthermore, the expiratory abdominal activity also increased or appeared. The abdominal muscles showed two patterns: (1) discharge starts at the later part of the expiratory phase and peaked just before the onset of the inspiratory activity; (2) discharge exists during the whole expiratory phase. As for the former type, small additional burst often occurred just after the termination of the inspiratory activity (especially, in the vagotomized rats). Since this post-inspiratory burst was rarely observed in the adult rat, the abdominal respiratory pattern could change during the postnatal development, while the basic patterns were similar.

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P3-156 Feedback and feed-forward regulation of micturition contraction by Barrington's nucleus

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Seventy-six spinal-projecting neurones, which increase their firing rates during micturition contraction, were recorded from Barrington's nucleus of cats. During micturition contraction, three different classes of firing patterns were identified: constant, decrement, and rise. Neurones with constant-type firing displayed relatively constant firing. Together with the fact that firing increase tended to be delayed in relation to onset of micturition contraction, this type of neurones were suggested to have a role in maintaining high bladder pressure via a positive feedback loop. On the other hand, neurones with decrement-type firing displayed a firing increase prior to micturition contraction and maximal discharge during the rising phase of micturition contraction: the discharge began to decrease even though bladder pressure had not yet reached a maximum. This type of neurones were suggested to contribute to raising bladder pressure rapidly and forcefully from the base pressure to high levels via feed-forward regulation.

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P3-157 Ultrastructual evidence of the synaptic contact between VGluT3 positive terminals and sympathetic preganglionic neurons in rat thoracic spinal cord.

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Vesicular glutamate transporters (VGluTs) are a marker of glutamatergic terminals. Recently, Nakamura et al. (2003) showed that VGluT3 positive terminals exhibit close apposition with choline acetyl transferase positive neurons in intermediolateral (IML) nucleus of the rat spinal cord.To establish the synaptic contact between VGluT3 positive terminals and sympathetic preganglionic neurons, we injected WGA-HRP, a retrograde tracer, into rat superior cervical ganglion, and immunohistochemically stained the spinal cord (C8-T5) with VGluT3. After observing with light microscopy, the sections were processed for electron microscopic observation. At light microscopic level, we found the close apposition between VGluT3 positive terminals and WGA-HRP positive neurons. At electric microscopic level, we confirmed the synapse between VGluT3 positive terminals and WGA-HRP positive neurons.

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P3-158 Electrophysiological identification of the parvicellular reticular formation (RFp) neurons that project to RVLM in anaesthetized rats

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The rostral part of parvicellular reticular formation (RFp) is involved in the cardiovascular function and has efferent projections to the rostral ventrolateral medulla (RVLM). However, a neuronal activity of RFp neurons that project to RVLM is not well known. In urethan-chloralose anaesthetized rats, we explored electrophysiologically RFp for the neurons that have projections to the RVLM. One-third of the antidromically identified RFp neurons were spontaneously active (about half of them had cardiac related discharge) with the axonal conduction velocities ranged between 0.16-4.3 m/s. Remaining two-thirds of the antidromically identified neurons were silent with the axonal conduction velocities ranged 0.2-4.0 m/s. Some of these identified neurons exhibited excitatory and inhibitory responses by lingual nerve stimulation. These results suggest that RFp neurons can modulate RVLM neuronal activity.

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P3-159 Responses of spinal blood flow to cutaneous brushing in rats

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The present study investigated whether brushing of various cutaneous areas influenced spinal cord blood flow in anesthetized rats. Regional spinal blood flow was measured with a laser Doppler flowmeter probe placed on the dorsal surface of the spinal cord at either the T12-L1 or L4-6 segments. Brushing of the abdomen increased spinal blood flow as recorded at the T12-L1 segment, but not at the L4-6 segment. Conversely, brushing of the hindpaw increased blood flow at the L4-6 segment, but not at the T12-L1 segment. Brushing of the proximal hindlimb caused a small increase in blood flow at both the L4-6 and T12-L1 segments. None of these brushing stimuli produced significant changes in systemic arterial blood pressure. These results indicate that innocuous cutaneous mechanical input can produce a segmentally-organized increase in regional spinal cord blood flow.

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P3-160 Involvement of T-type Ca^{2+} channels and Ca^{2+}-activated K^+ channels in the α_1-adrenoceptor-mediated contraction of the guinea-pig vas deferens

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Electrical stimulation of 50 pulses at 40 Hz elicited a TTX-sensitive monophasic contraction in the guinea-pig vas deferens. The P2X antagonists, suramin and PPADS produced a biphasic contraction consisting of early and late α_1 components. The early α_1 component was suppressed by mibefradil and amiloride, T-type Ca^{2+} channel antagonists. The late α_1 component was depressed by L-type Ca^{2+} channel antagonists, nifedipine, nimodipine and nicardipine (DHPs). Apamin, a SK channel antagonist amplified both components. In contrast, BK channel antagonists CTX and IBTX augmented only the late α_1 component. The evoked outflow of [^{3}H]noradrenaline was increased by CTX and IBTX but not by DHPs and apamin. These results indicate that SK channels participate with both T- and L-type Ca^{2+} channels, and BK channels with only L-type Ca^{2+} channels. Both K channels greatly modify the α_1 adrenoceptor-mediated contraction.

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P3-161 Convergent inputs from the cortical and amygdaloid fibers to the lateral hypothalamic neurons in the rat

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We examined the synaptic organization between and among the fibers originating from the insular cortex (IC) and central nucleus of the amygdala (ACe), and the neurons in the lateral hypothalamus (LH) using the double anterograde labeling. Prominent overlapping distributions of the IC and ACe fibers were found in the lateralmost part of the posterior LH. In this area, the IC terminals formed asymmetrical synapses with dendrites of the LH neurons, whereas the ACe terminals made symmetrical synapses with dendrites and cell bodies of the LH neurons. Some of single dendritic profiles received convergent synaptic inputs from both sets of terminals. We also revealed that the ACe terminals were GABAergic using anterograde labeling combined with immunohistochemistry for GABA. These results suggest that the excitatory inputs from the IC and inhibitory inputs from the ACe are convergent onto single LH neurons.

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P3-162 Neural substrates inducing trigeminal depressor response

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Following electrical stimulation of the inferior alveolar nerve (IAN), trigeminal depressor response (TDR) was induced when the prestimulus mean arterial blood pressure (MABP) and heart rate (HR) were above 95 mmHg and 140 beats/min, respectively. Vasodepressor responses were induced when various fields around trigeminal sensory nucleus were stimulated. However, these were similar to the cardiovascular response induced by vagal afferent but not IAN stimulation. After spinalization or lesion of the rostral ventrolateral medulla (RVLM), MABP and HR decreased and TDR completely abolished. Neurons immunopositive for inhibitory substances were distributed along the sympatho-reflexive route including the RVLM. TDR could be induced as negative feedback to sympathetic hyperactivity whenever MABP and HR are high, because of the inhibitory control of the RVLM.

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P3-163 Participation of serotonergic systems in the lateral parabrachial nucleus in sodium and water intake in rats

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We examined whether salt and water intake alters serotonin (5-hydoxytryptamine, 5-HT) release in the lateral parabrachial nucleus (LPBN) in freely moving rats, using microdialysis methods. The ingestion of 0.3 M NaCl and water induced by subcutaneous injections of furosemide (FURO) and captopril (CAP) significantly elevated extracellular concentrations of 5-HT and its metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the LPBN area. The treatment with FURO and CAP significantly reduced the 5-HT and 5-HIAA levels under the condition that 0.3 M NaCl and water are not available for drinking. These results imply that the serotonergic system in the LPBN may participate in the control of sodium appetite and thirst.

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P3-164 Pre-Botzinger complex neurons expressing neurokinin-1 receptors receive GABAergic inputs from the nucleus of the solitary tract in the rat

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We examined afferent projections to the neurokinin-1 receptor (NK1R)-immunoreactive (ir) neurons in the pre-Botzinger complex (pre-BotC) from the nucleus of the solitary tract (NST), using tract-tracing and immunohistchemical methods. In result, 1) pre-BotC-projecting neurons were observed in a subregion of the caudal two-thirds of the NST, such as the dorsomedial, intermediate, interstitial, vetrolateral and commissural subnuclei, 2) some of these neurons were immunoreactive for glutamic acid decarboxylase (GAD), 3) GAD-ir axons originating from the NST were often in contiguity with NK1R-ir pre-BotC neurons, 4) axon terminals from the NST made symmetrical synaptic contacts with somata and dendrites of the NK1R-ir neurons. These results suggest that a population of NST neurons may exert inhibitory influences on NK1R-expressing pre-BotC neurons to control respiratory pattern generation.

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P3-165 GABAergic control of serotonin release in the rat subfornical organ area

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Experiments were done to examine whether γ-aminobutyric acid (GABA) receptor mechanisms modulate the release of serotonin (5-hydroxytryptamine, 5-HT) in the subfornical organ (SFO) in freely moving rats, using microdialysis methods. Perfusion of bicuculline (10 and 50 μM), but not phaclofen (10 and 50 μM), through a microdialysis probe enhanced extracellular concentrations of 5-HT and its metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the SFO area. Higher perfusion of muscimol (50 μM) or baclofen (250 μM) reduced the 5-HT and 5-HIAA levels. Hypovolemia induced by s.c. injection of polyethylene glycol (PEG, 30%, 5 ml) enhanced the 5-HT and 5-HIAA levels in the SFO area, and the PEG-induced increases were attenuated by perfusion of muscimol (10 μM), but not by baclofen (50 μM). These results suggest that the $GABA_A$ receptor mechanism in the SFO area may be importance for the 5-HTergic regulatory system of body fluid balance.

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P3-166 Dopaminergic effects on respiratory rhythm generation in rat brainstem-spinal cord preparation

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Dopamine (DA) is known to regulate both higher order and autonomic brain functions. Although several experiments have implicated dopamine's involvement in respiratory control, no direct evidence has yet been found. To elucidate the effects of DA on respiratory rhythm generation, isolated brainstem-spinal cord were treated with DA and each DA receptor agonist individually. DA application facilitated respiratory rhythm in pons medulla spinal cord preparations, but suppressed it in medulla spinal cord preparations. Furthermore, we found that mainly Pre-I neurons caused this suppression, not other respiratory neurons, and the application of D4 receptor agonist, PD168077, also induced respiratory suppression. Therefore, the primary target of DA action in modulating respiratory rhythm generation are the Pre-I neurons in the medulla. Our findings shed light on the deeper connections between respiration and higher brain functions, such as emotion.

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P3-167 L-aspartate immunoreactive neurons in the rat enteric nervous system

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L-aspartate (L-Asp) is an excitatory neurotransmitter in the CNS. In the present study, we demonstrate for the first time the presence of L-Asp in a particular neuronal cell class in the enteric nervous system (ENS). Although the frequency of L-Asp-immunoreactive(IR) neuronal cell bodies showed conspicuous regional differences, scattered L-Asp-IR neuronal cell bodies and nerve fibers were found in both the myenteric and submucosal plexi extensively throughout the small and large intestines. Many L-Asp-IR nerve fibers, which originated from intrinsic nerve cell bodies, were found in the ganglia and interconnecting nerve bundles. Electron microscopy revealed that these L-Asp-IR terminals frequently formed synaptic contacts with intrinsic nerve cells, suggesting that L-Asp-IR neurons might function as interneurons in the ENS. These results suggest that L-Asp-IR neurons probably play a significant neuronal role within the ENS to control gastrointestinal functions.

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P3-168 Reduced serotonin release in the lateral parabrachial nucleus area elicited by angotensin II activation of the subfornical organ in rats

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The present study was designed to investigate whether angiotensinergic activation of the subfornical organ (SFO) alters the release of serotonin (5-hydroxytryptamine, 5-HT) in the lateral parabrachial nucleus (LPBN) in freely moving rats. Extracellular concentrations of 5-HT and its metabolite 5-hydroxyindoleacetic acid (5-HIAA) were measured using microdialysis techniques. Microinjection of angiotensin II (ANG II) (50 pmol), but not vehicle, into the SFO significantly decreased the 5-HT and 5-HIAA concentrations in the region of the LPBN. The data suggest that circulating ANG II acting at the SFO may serve to reduce the 5-HT release in the LPBN area.

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P3-169 State space representation of ECG in freely moving crayfish

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State space representation is a method that gives intuitive images of dynamical behavior of data as a visible figure. We adopted this method to ECG data recorded from freely-moving-crayfish, Procambarus clarkii. Temperature was linearly shifted from low temperature (5°C) to lethal temperature (30°C) and ECG was recorded under the circumstances. At a specific temperature point, about 13°C, the trajectory in the state space showed a drastic change in configuration. We also noticed that cardio-inhibitory CNS control started at this temperature point. We consider that the change of trajectory shape reflects potential change of intrinsic burst of the cardiac ganglion and the change of discharge pattern in the cardio-regulatory brain center. This drastic changes implicate that uncharacterized neuronal mechanisms may be involved in switching the active/dormant condition of the animal.

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P3-170 Biomedical effects evoked by a random-dot image composed of the global motion vector

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As an effort to analyze causative relationship between motion pictures and their biomedical influences, motion components of a video image, corresponding to the camera work, were extracted as the global motion vector. Then a CG motion picture was composed by giving values of the motion vector to randomly arranged dots in each division of the frame corresponding to the original real image. Four volunteer subjects watched the original and corresponding CG motion pictures, which were rear-projected in an 80-inch screen. An infrared CCD eye-pupil analyzer measured eye movement. Slow components of eye movements evoked either by the composed moving random-dots or by the original real image were well comparable, while the fast components were quite different.

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P3-171 Effects of sympathetically induced vasomotion on capillary-tissue fluid exchange

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Vasomotion, rhythmic contraction of arterioles, has been observed in many tissues in various species. The physiological role of vasomotion, however, has not been fully understood because appearance of vasomotion could not be controlled *in vivo* preparations. We developed a method for induction of vasomotion of the anesthetized rabbit ear *in vivo*. We could reproducibly evoke vasomotion by electrical stimulation of the cervical sympathetic nerve. Induction of vasomotion was able to control through the change of ambient temperature. Using this method, we have examined the effects of vasomotion on capillary-tissue fluid exchange. A tracer, Cr-EDTA, was injected into the skin of rabbit ear, then a decrease in concentration of the tracer was observed. When vasomotion was induced the rate of decrease was larger, compared to that without vasomotion. It was suggested that vasomotion might enhance the capillary-tissue fluid exchange.

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P3-172 Allopregnanolone inhibits the chemoreceptor reflex by acting on $GABA_A$ recetors in the commissural subnucleus of the nucleus tractus solitarius in rats

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The neurosteroid allopregnanolone (AP) is a potent endogenous positive modulator of the $GABA_A$ receptor. The purpose of this study is to determine whether AP modulates the chemoreceptor reflex by acting on $GABA_A$ receptors in the commissural subnucleus of the nucleus tractus solitarius (commNTS). Male Wistar rats were anesthetized, immobilized and bilaterally vagotomized. AP (i.v.) decreased resting phrenic nerve activity and attenuated the increases in phrenic nerve activity and blood pressure elicited by carotid chemoreceptor stimulation. The effects of AP on the chemoreceptor reflex were antagonized by microinjection of bicuculline into the commNTS. Muscimol microinjected into the commNTS attenuated the chemoreceptor reflex. This inhibitory effect was potentiated by AP. These results suggest that AP induces inhibitory modulation on the chemoreceptor reflex by acting on $GABA_A$ receptors in the commNTS.

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P3-173 Origins of neuropeptide Y-immunoreacive projections to the rat subfornical organ

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The subfornical organ (SFO) plays a role in osmoregulation. A moderate distribution of neuropeptide Y (NPY)-immunoreactive axon varicosities is seen in the SFO. NPY is a neuropeptide colocalizing with some catecholamine neurons in the medulla oblongata. We investigated origins of NPY-immunoreactive projections to the SFO using a retrograde tracing method combined with immunohistochemistry. A retrograde tracer, WGA-HRP-colloidal gold, was injected into the SFO. After 2-5 days, colchicine was poured into the lateral ventricle to block the axonal flow. The rats were fixed on the next day, and free floating sections were silver intensified to detect retrograde label and immunostained for NPY. Retrogradely labeled neurons were found in the nucleus tractus solitarius (NTS), caudal ventrolateral medulla (CVLM), locus coeruleus, raphe nuclei, hypothalamic and preoptic nuclei. Of these, retrograde neurons in the NTS and CVLM showed NPY immunoreactivity. Exclusive NPY-immunoreactive catecholamine projections from the medullary regions are suggestive.

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P3-174 The neural circuit to induce vomiting; homology between the ferret and dog

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Our studies in dogs have revealed the neural circuit for induction of vomiting. Although ferrets have been mainly used in recent studies of vomiting, the neural circuit has not yet been clarified. This study was performed to ascertain whether the neural circuit in ferrets is similar to that in dogs. In decerebrate ferrets, the vagally mediated vomiting was blocked by applications of nonNMDA-R antagonist, NBQX, to the medial solitary nucleus (mNTS), and NK-1R antagonist to the reticular area near the semi-compact part of the nucleus ambiguus (scAMB), similarly to the results in dogs. In the ferrets suffered repetitive vomiting, c-fos immuno-stained cells were observed in the mNTS, and reticular areas near the scAMB and the compact-part. Moreover, the immunostaining with an antibody against NK-1R showed immunopositive puncta in the scAMB area. These results suggest that vomiting is produced through the homologous pathway in dogs and ferrets.

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P3-175 Firing pattern and projection to spinal cord of respiratory neurons in rabbit medullary raphe nuclei

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Several studies suggest that the medullary raphe nuclei may be involved in the regulation of breathing. The present study was undertaken to investigate the firing pattern and spinal projection of respiratory neurons in the medullary raphe nuclei of rabbits. The extracellular spikes of single respiratory neurons in the midline medullary tegmentum of urethane-anesthetized and spontaneously breathing rabbits were explored. Expiratory (E), inspiratory (I), late-I, I-frequency modulated, I-E phase-spanning and E-I phase-spanning neurons, based on the relation of their firing to the phases of respiration were recorded in the raphe magnus, obscurus and pallidus. Some of E neurons were antidromically activated from the ventrolateral quadrant of the C4-C5 spinal cord.These results suggest that medullary raphe nuclei may take part in control of respiration in rabbits.

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P3-176 MAJOR PORTIONS OF THE DORSAL TELENCEPHALON PROJECTING TO THE HYPOTHALAMUS IN A TELEOST, *OREOCHROMIS NILOTICUS*.

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The dorsal telencephalon of teleosts, recognized as homologue of the mammalian pallium, sends axons directly to the hypothalamus. The major projection areas reported are the dorsal part of area dorsalis telencephali pars medialis (dDm), the pars dorsalis (Dd), the pars posterioris (Dp), and the pars centralis (Dc). Terminal areas of the projections are a ventromedial portion of the nucleus diffusus lobi inferioris (NDLI) and the nucleus posterioris periventricularis (NPP). Because the descending tracts from the dDm, Dd, and Dc pass through the Dp, a possibility of affecting the passing fibers could not be excluded. In order to clarify and detect cells of origin of these projections, tracer (biocytin or DBA) was injected into the hypothalamic areas. As the results, we have confirmed that the main projections to the NDLI were from the dDm, Dd, and Dc. While, refered to projection to the NPP, an unknown subdivision of the Dp, originating symmetrically from the medline, was detected as the origin.

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P3-177 A characteristic distributional pattern of NOS-containing and ENK-containing nerve fibers in the sacral sympathetic ganglia.

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To investigate preganglionic regulation to sympathetic ganglia of the sacral segments (S2-S3) of rats, the distribution of enkephalin (ENK), nitric oxide synthase (NOS) and vesicular acetylcholine transporter (VAChT) immunoreactivities in the sympathetic ganglia of lumbo-sacral segments was examined. In control animals, ENK- and VAChT-positive (ENK/VAChT) nerve fibers made a basket-like network around the cell bodies in the ganglia of lumbar segments, while only a few ENK/VAChT varicose fibers were found in the sacral segments. NOS- and VAChT- positive (NOS/VAChT) fibers were more numerous in the ganglia of sacral segments than those of lumbar segments, and occasionally made basket-like network. After transection of the lumbar sympathetic trunk between the L5 and L6 ganglia, both ENK/VAChT and NOS/VAChT varicose fibers disappeared in the sacral ganglia. These results suggest that sacral sympathetic ganglia are regulated exclusively by NOS/VAChT preganglionic fibers.

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P3-178 Effects of serotonin in the dorsomedial medulla oblongata on ventilatory responses to CO_2 in mice

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We have reported that serotonin (5-HT) in the dorsomedial medulla oblongata (DMM) affects airway resistance. In the present study we examined the effects of 5-HT in the DMM on respiratory responses to CO_2. Male mice (C57BL/6N) were anesthetized with pentobarbital sodium i.p. in order to insert a microdialysis probe in the DMM and then placed in a double chamber plethysmograph. After the mouse recovered from the anesthesia and acclimatized to the chamber, 5-HT concentration and respiratory responses were assessed at baseline (inspired 100% O_2 gas) and during 25 min hypercapnia (inspired 5%, 7%, 9% CO_2 balanced with O_2). The 5-HT concentration in the DMM during fluoxetine perfusion, as well as frequency, tidal volume and minute ventilation, were increased by hypercapnia. The increases of tidal volume and minute ventilation tended to be diminished with perfusion of LY-53857 (a 5-HT_2 receptor antagonist). 5-HT may affect ventilatory responses to CO_2 via 5-HT_2 receptors in the DMM.

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P3-179 Effect of rostral pontine reticular formation stimulation on bladder activity and the spinal glycine level in rats with or without cerebral infarction

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We examined the effect of the rostral pontine reticular formation (RPRF) stimulation on bladder activity and the spinal glycine level in rats with or without cerebral infarction (CI). Rats were anesthetized with urethane. In 20 rats, isovolumetric cystometry was performed before and after injection of flavoxate into the RPRF. In 30 rats, flavoxate was injected and the spinal glycine level was measured. In intact rats, injection of 0.3μ flavoxate abolished bladder contraction and increased the spinal glycine level. In CI rats, injection of 3μ flavoxate abolished bladder contractions, but the spinal glycine level did not change. Therefore, in intact rats, activation of the RPRF may inhibit the micturition reflex by activation of spinal glycinergic neurons. However, in CI rats, activity of the RPRF may be weak by the decrease of excitatory projection from the upper CNS to the RPRF.

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P3-180 Involvement of suprachiasmatic nucleus and histaminergic sysytem in effect of L-carnosine autonomic nervous and cardiovascular systems

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In the present study, using urethane-anesthetized rats, we examined whether blocks of the histaminergic receptors eliminate sympathetic and cardiovascular effect of L-carnosine, and found that pre-treatment of thioperamide, an antagonist of histamine H3 receptor, blocks low dose of L-carnosine-induced suppression of the RSNA and BP, and diphenhydramine, an antagonist of histamine H1 receptor, blocks high dose of L-carnosine-induced elevation of the RSNA and BP. Furthermore, we found that in SCN-lesioned rats, RSNA and BP were not affected by injections of low and high doses of L-carnosine. These findings suggest that the SCN is implicated in the mechanism of the effects of L-carnosine on the RSNA and BP via histaminergic system.

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P3-181 The spinal sympathetic ganglia of the chelonian *Trachemys scripta elegans*

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In a macroscopic study, the chelonian thoracic spinal ganglia were located in the space was bordered anteriorly and posteriorly by the ribs, medially by the vertebral body, and superiorly by the neural plate. The dorsal and the medial root were fused in the medial side of the spinal ganglion and two branches of unequal thickness, the ventral and dorsal rami of the spinal nerves, exited on its lateral side. There was a protuberance on the postero-lateral surface of the ganglion from which a single branch communicated with the sympathetic trunks. The tyrosine hydroxylase (TH) immunohistochemical positive perikarya were located only in the protuberance, and its processes were distributed only in a distal direction. The TH immunohistochemical positive perikarya were identical to perikarya of the frog sympathetic ganglion without central processes in the spinal ganglion. We conclude that the chelonian thoracic spinal ganglia are spinal sympathetic ganglia.

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P3-182 Microinjection of 8-OH-DPAT into dorsal raphe nucleus produces respiratory inhibition and faster rhythms on electrocorticogram in rat.

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Our previous study demonstrated that microinjection of serotonin (5-HT) into dosal raphe nucleus (DRN) caused respiratory inhibition and faster rhythms on electrocorticogram (ECoG). These changes may be caused either by activation of 5-HT autoreceptor or inhibition of 5-HT neurons by GABA interneurons. To evaluate these two possibilities, we examined effects of microinjection of 5-HT$_{1A}$ receptor agonist, 8-OH-DPAT into DRN on respiration (intercostals EMG) and cortical activity (ECoG) in an anesthetized, spontaneously breathing rat. Microinjection of 8-OH-DPAT into DRN produced the changes in respiration and corical activity similar to those of 5-HT microinjection, suggesting that 5-HT$_{1A}$ receptor plays an important role in respiratory and cortical modulations induced by 5-HT microinjection in DRN.

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P3-183 Non-uniform regional sympathetic nerve responses to 2-deoxy-D-gulcose in conscious rats

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The present study aimed to investigate a regional difference of the response between renal and lumber sympathetic nerve activity to acute glucoprivation, which was induced by intravenous administration of 2-deoxy-D-glucose (2DG). Wistar rats were instrumented chronically with electrodes for the measurements of renal and lumber sympathetic activities, electroencephalogram, electromyogram and electrocardiogram and catheters for the measurements of systemic arterial and central venous pressures. Following at least 2 days recovery periods, 750mg/kg of 2DG was administered intravenously. Both renal and lumber sympathetic nerve activities suppressed immediately after the 2DG administration. Then, renal sympathetic nerve activity increased significantly while lumber sympathetic nerve activity remained suppressed. *It was therefore clearly shown that the 2DG administration resulted in non-*uniform regional sympathetic nerve responses.

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P3-184 *Effects of intravenous or intrathecal noradrenalin* on the micturition reflex and the spinal glycine level in rats

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We examined the effects of intravenous (IV) or intrathecal (IT) injections of noradrenalin (NA) on the micturition reflex and the spinal glycine level in rats. When the bladder was filled with saline under the threshold volume inducing rhythmic bladder contractions (BCs), IV- or IT-NA induced BCs. When the bladder contracted rhythmically, IV-NA shortened the interval and decreased the amplitude of BCs. However, IT-NA transiently abolished BCs. After recovery of BCs, the interval of BCs was shortened, but the amplitude did not change. Simultaneous IV-NA and IT naftopidil (an adrenergic alpha-1d receptor antagonist) did not change the interval of BCs, but the amplitude was decreased. IV- or IT-NA decreased the spinal glycine level. These results suggest that the increase of the intravenous NA level facilitates *afferent activity of the micturition reflex*, and induces urinary frequency by inhibiting glycinergic neuronal activity.

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P3-185 Changes of bladder activity and the glycine level in the lumbosacral cord and serum after partial bladder outlet obstruction in rats

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We examined whether the glutamate and glycine levels in the lumbosacral cord (LC) and serum were related to bladder activity after partial bladder outlet obstruction (BOO) in rats. Female rats were anesthetized with halothane, and the urethra was loosely ligated to create BOO. Bladder activity and the glutamate and glycine levels in the LC and serum were measured at 3 days to 4 weeks after BOO. At 2-4 weeks after BOO, residual urine volume increased, and isovolumetric cystometry showed detrusor hyperreflexia. The glycine level in the LC was decreased at 2-4 weeks, while the serum glycine level was decreased at 4 weeks after BOO. The glutamate level in the LC and serum did not change after BOO. These results suggest that detrusor hyperreflexia after BOO is caused by the decrease of glycinergic neuronal activity in the lumbosacral cord.

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P3-186 An improved method of recording a single-unit activity from medullary cardiovascular neurons in baroreflex experiments

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Analyzing a single-unit response of cardiovascular neurons to baroreceptor stimuli is one of the most direct approaches to unravel the central baroreflex mechanism. In medium- or large-size animals, however, it is not easy to maintain a stable recording over a wide-range manipulation of blood pressure, because the change in blood pressure causes a mechanical movement of the medulla. Therefore, we have developed an automatic tracking-control system of the electrode to follow-up the brain movement. The movement was optically detected, and the position of the electrode was automatically adjusted to cancel the movement. Effectiveness of the system was evaluated in a series of baroreflex experiments in urethane-anesthetized rabbits. The brain surface was dislocated up to 0.5 mm during a manipulation of blood pressure (40-160 mmHg in mean arterial pressure), while the unit recordings were stable enough to perform the baroreflex analysis.

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P3-187 Centrally administered CRH-induced elevation of plasma corticosterone is mediated by activation of the brain stem noradrenergic neurons in rats

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The present study was undertaken to clarify the central mechanisms involved in the CRH-induced elevation of plasma corticosterone (CORT) in urethane- and α-chloralose-anesthetized rats. Intracerebroventricularly (i.c.v.) administered CRH (1.5 nmol/animal) elevated plasma CORT with a concomitant increase of noradrenaline (NA) release in the hypothalamic paraventricular nucleus (PVN). The CRH-induced elevation of CORT was abolished by phentolamine (0.66 μmol/animal, i.c.v.). The CRH-induced hypothalamic NA release and increase of plasma CORT were abolished either by hexamethonium (1.8 μmol/animal, i.c.v.), α - conotoxin MII (α3β2 nicotinic receptor antagonist) (30 nmol/animal, i.c.v.) or indomethacin (1.2 μmol/animal, i.c.v.). These results suggest that the centrally administered CRH elevates plasma CORT by brain α3β2 nicotinic receptor- and prostaglandin-mediated NA release in the PVN in rats.

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P3-188 Centrally administered bombesin elicits adrenal noradrenaline release by brain thromboxane A2-mediated mechanisms in rats

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Plasma noradrenaline (NA) reflects the release from adrenal medulla and sympathetic neurons. We want to characterize the source of plasma NA induced by intracerebroventricularly (i.c.v.) administered bombesin (BOM) and prostaglandin E2 (PGE2) in urethane-anesthetized rats. BOM elevated NA and adrenaline (Ad), while PGE2 elevated only NA. BOM-induced elevations of NA and Ad were attenuated by i.c.v. administered furegrelate (thromboxane A2 synthase inhibitor) and S-145 (prostanoid TP receptor antagonist), and were abolished by bilateral adrenalectomy. PGE2-induced elevation of NA was not influenced by adrenalectomy. These results suggest that centrally administered BOM releases NA (in addition to Ad) from adrenal medulla by brain prostanoid TP receptor-mediated mechanisms, while centrally administered PGE2 releases NA from sympathetic neurons by brain prostanoid EP receptor-mediated mechanisms in rats.

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P3-189 Control of central fatigue by using a combination of injected L-tryptophan and β-endorphin

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For fatigue control, there are two important factors, tryptophan and β-endorphin. When L-tryptophan of 62.5 mg/Kg and 125 mg/Kg were injected, the treadmill running time, was shortened dose-dependently. To create an animal model of central fatigue chronically, the treadmill running of rats for five days were given to exhaustive fatigue. As an activity index of CNS and autonomic nerve system, we measured urinary noradrenaline and 3-Methyoxy-4-hydroxyphenylglycol (MHPG) before and after the injection of β-endorphin for the rat model of fatigue. When β-endorphin of 50 ng/body and 100 ng/body were injected, the MHPG concentration, recovered to its basal level from its fatigue, dose-dependently. The β-endorphin caused diminished central fatigue. In conclusion, the index of running time and urinary MHPG (not noradrenaline) concentration has a direct relationship with the level of tryptophan and β-endorphin.

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P3-190 mRNA analysis of enzymes and receptors for neurosteroids in an adult male rat hippocampus

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To investigate neurosteroidogenic pathway and their effects in the central nervous system, we analyzed the expression of steroidogenic enzyme and steroid receptor transcripts in an adult male rat hippocampus by semi-quantitative RT-PCR-Southern blotting assay. mRNAs of major steroidogenic enzymes from P450scc to P450arom were detected, and we found receptors for androgen and estrogen in the hippocampus. Those findings indicate that the hippocampus synthesizes testosterone and estradiol from cholesterol, and imply that these brain neurosteroids may serve as the autocrine and/or paracrine modulators.

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P3-191 17-beta estradiol promotes the survival of cultured male rat cerebellar granule neurons.

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Steroids are present widely in the brain and thus assumed to play important roles. It is long known that the sexual dimorphism of the brain is determined by sex steroids in fetal period. Recent studies using cultured neurons have demonstrated that steroids can exert a neuroprotective effect. However, in the rat cerebellar granule neurons (CGN), an often-used model in the study of neuronal survival, the neuroprotective effect is under debate. We report here that previous unstable results in the assays of steroids'neuroprotection neuroprotection in CGN should be due to the neglect of the sex of the source animal. When we cultured CGNs isolated from male pups, the survival was promoted by 17-beta estradiol (E2). CGN prepared from female pups showed no survival promotion by E2. This survival promotion took form as the CGN's requirement of glutamate in the medium for survival was lowered by E2 in male CGN. The cerebellum, usually not regarded sexually dimorphic, might be actually so.

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P3-192 Injection of corticotropin-releasing hormone (CRH) receptor antagonist blocks the suppression of pulsatile LH secretion induced by neuromedin U (NMU) in ovariectomized (OVX) rats after 48 hours of fasting

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We recently reported that NMU and fasting synergistically suppressed the pulsatile LH secretion. In the present study, blood samples were taken under freely-moving conditions at 6-min intervals for 180 min from 0900 h to 1200 h in adult OVX rats that had been fasted for 48 h. After the first 60 min of blood sampling, astressin (2 nmol/3 ml), a CRH receptor antagonist or vehicle as a control was injected into the third ventricle. Thirty min after the first injection, the rats were injected with NMU (1 nmol/3 ml) into the third ventricle. We found that pre-treatment with astressin completely blocked the prolongation of the interpulse interval, which should be induced by NMU. The present study suggests that synergistic inhibitory effect of NMU and fasting on the pulsatile LH secretion is at least in part mediated via CRH neurons.

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P3-193 Glucose transporter 4 (GLUT4) translocation system in the hypothalamic arcuate nucleus of ob/ob mice

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Insulin signaling in the hypothalamus of mice with insulin resistance remains unclear, whereas insulin receptor (IR) and GLUT4 are expressed in such region of the brain as well as in skeletal muscle and adipose tissue. In this study, we investigated the insulin signaling and the translocation of GLUT4 in the arcuate nucleus of ob/ob mice with insulin resistance and hyperinsulinemia. Although there were no differences between lean and ob/ob mice in the phosphorylation of IR and insulin receptor substrate (IRS)-1, serine phosphorylation of Akt and IRS-1 were increased in the hypothalamus of ob/ob mice compared to lean mice. Interestingly, GLUT4 was significantly increased in the hypothalamus of ob/ob mice, which was localized on the plasma membrane of the neurons in the arcuate nucleus. These results show that the translocation of GLUT4 is not impaired in the arcuate nucleus of ob/ob mice.

P3-194 Sexual difference in the orexin neurons in rat brain.

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Orexin neurons are distributed in the perifornical and lateral hypothalamic area, a region classically implicated in the control of feeding behavior, and project their axons within the hypothalamus and throughout the central nervous system to be involved in the control of feeding. In the present study, we investigated the sexual difference of orexin neurons using specific orexin-A and orexin-B antibodies. The density of both orexin-A and orexin-B immunopositive neurons in male was preferentially higher than those in female rats. On the other hand, ovariectomy in female rats induced the change to the male pattern. Colocalization of orexin and estrogen receptor was not observed. These results suggested that orexin neurons were indirectly regulated by estrogen through the other estrogen dependent neurons.

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P3-195 Neurosteroid Synthesis in the Rat Hippocampus.

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The synthesis of 17β-estradiol in the hippocampus of adult male Wistar rats was demonstrated with HPLC analysis. The conversion of [^{3}H]pregnenolone to [^{3}H]estradiol through dehydroepiandrosterone and testosterone was observed. Immunohistochemical staining and immunoelectron microscopy demonstrated the localization of cytochrome P45017α and aromatase in pre- and postsynaptic compartments in principal neurons. Pregnenolone production from endogeneous cholesterol was demonstrated with radioimmunoassay. Taken together, these results suggest that 17β-estradiol is synthesized by P45017α and aromatase localized in the synapses of hippocampal neurons from endogenous cholesterol.

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P3-196 Identification of a novel E3 ubiquitin ligase specifically exists in hypothalamic magnocellular neurons

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We performed an immunoscreening using endocrine tissue-specific antibodies on rat hypothalamus cDNA libraries, and obtained a cDNA fragment encoding a novel Hect domain-containing E3 ubiquitin ligase. The predicted protein product consisted of about 2600 amino acids. In order to characterize the localization of this protein in the adult rat tissues, we prepared specific polyclonal antibodies. The intense immunoreactivity was observed in posterior pituitary gland and hypothalamic paraventricular and supraoptic nuclei. Double immunostaining revealed that the signals were colocalized with neurophysin II immunoreactivity. Signals were not detected in another regions of brain and other tissues.

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P3-197 Temporal changes in fos expression of GnRH surge-generating system and possibility of asymmetrical GnRH-secreting activity in female rats

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Preovulatory GnRH surge is induced by positive feedback effects of estrogen. The anteroventral periventricular nucleus (AVPvN) and suprachiasmatic nucleus (SCN) are involved in GnRH surge generation. The present immunohistochemical study examined fos expression of GnRH neurons, AVPvN and SCN in estrogen-treated ovariectomized rats. Plasma LH was also determined by ELISA. The expression of fos in GnRH neurons was temporally changed, and fos was highly expressed during estrogen-induced LH surge. Temporal change in fos expression of the AVPvN was parallel to that of GnRH neurons. In the SCN, peaks of fos expression preceded those in GnRH neurons and AVPvN. In addition, total number of GnRH neurons on the left side became smaller than that on the right side during LH surge. These results indicate the dynamic process of neuronal activation in the GnRH surge-generating system. Also, asymmetry of GnRH-secreting activity is presumably existed.

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P3-198 Imaging of Glucocorticoid Receptor (GR) in the Brain of Green Fluorescent Protein-GR Knockin Mouse

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To investigate the distribution and function of glucocorticoid receptor (GR), we examined the brains of green fluorescent protein (GFP)-GR knockin mice. Green fluorescence was consistent with GR immunoreactivity in the cerebral cortex, olfactory bulb, hippocampus, amygdala, thalamus, and hypothalamus, where GR immunoreactivity showed strong intensity and high density. To examine the subcellular trafficking, we performed adrenalectomy (ADX) and found nucleus-to-cytoplasmic translocation of GFP-GR after ADX as observed in endogenous GR. In living hippocampal slice, GR-containing nuclei were clearly visualized. GFP-GR knockin mice may open new ways for analyzing GR functions in vivo and in vitro.

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P3-199 Expression of the steroid receptor coactivator during differentiation of neural stem cells

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The steroid receptor coactivator (SRC) family contains three members that can enhance transcriptional activities of nuclear hormone receptors and other transcription factors. SRCs are involved in the precise gene regulation through brain development. We recently demonstrated that SRC-1 was highly expressed at embryonic stages and the disruption of SRC-1 caused the delayed Purkinje cell development in the mouse cerebellum. To further examine the SRC-1 function in the brain development, we investigated the localization of SRC-1 in neural stem cells derived from ganglionic eminences of E15.5 mice and the expression profiles after 1% FBS-induced differentiation. Initially, SRC-1 expression was detected in nestin positive neural stem cells and then SRC-1 was clearly localized in the nucleus of both neuron and astrocyte lineage cells during differentiation. This study provides SRC-1 might participate in the differentiation of neural stem cells.

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P3-200 Regulation of hippocampal neuropsin (KLK8) mRNA by steroid hormones

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Neuropsin (KLK8), one of the kallikrein family serine proteinase, is expressed specifically in the limbic system and plays an important role in hippocampal plasticity. It was demonstrated that many kallikrein family genes are regulated by steroid hormones such as estradiol and corticosterone. In addition, steroid hormones have been known to influence hippocampal plasticity; i.e. neurite outgrowth, spinogenesis, and long-term potentiation. Therefore, it is assumed that neuropsin is involved in at least partly steroid-dependent plasticity. To test the hypothesis, we examined effects on the expression of neuropsin mRNA in cultured hippocampal neurons. As a result, we found marked increases of neuropsin mRNA by administered estradiol and corticosterone in a dose-dependent manner. The increases of neuropsin mRNA were apparent at least after two hours of the treatment.

P3-201 Analysis of Neurosteroidogenic Enzymes in Stress Steroid Synthesis in Adult Rat Hippocampus

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Neurosteroids are novel modulators of the neural activities in the brain, such as learning and memory in the hippocampus. To investigate the synthesis of stress related neurosteroids and its effect on neural transmisson, we analyzed the expression of steroidogenic enzyme and receptor transcripts in adult male rat hippocampus by semi-quantitative RT-PCR and southern blotting assay. We found that both stress steroidogenic enzyme and stress steroid receptor transcripts are present in the hippocampus, although the hippocampal enzymes are different from those in adrenal steroidogenesis. This finding demonstrates that stress steroids are synthesized in adult male rat hippocampus.

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P3-202 Expression of the melanocortin receptor (MC-R) family genes in tyrosine hydroxylase (TH)-producing neurons in the mouse brain

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The brain MC-system and catecholaminergic system both participate in higher brain functions including the stress-responses. Since the TH and MC-R genes are expressed in discrete brain stem areas, green fluorescent protein (GFP)-tagged TH neurons are employed in order to examine whether the MC-R's are expressed in the TH neurons. Mice expressing GFP under the control of the TH promoter were used. Neurons obtained from fetal mice, heterozygous for the GFP gene, were dispersed, and GFP-positive cells were collected using a FACS machine. MC3-R and MC4-R mRNA's, among the five MC receptor mRNA's examined by RT-PCR, were expressed in the sorted GFP-positive (TH-producing) neurons. These results suggest the involvement of the MC-system in the regulation of catecholaminergic neurons in the mouse brain.

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P3-203 Estrogen and ovariectomy regulate expression of glutamic acid decarboxylases and cation-chloride cotransporters in adult female rat hippocampus.

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Estrogen (E) exerts a number of effects on adult rat hippocampus. E has time-dependent or region-specific effects on expression of synthesizing enzyme for GABA (GAD) in CA1 interneurons, where cell nuclear estrogen receptors were found. Recent studies have demonstrated that GABA can induce both inhibitory and excitatory actions in adult CNS neurons. The actions could predominately depend on intracellular chloride gradient across neurons which cation-chloride cotransporters NKCC1 and KCC2 have been identified to control. We have studied E effects on expression of GADs, NKCC1, and KCC2 using immunohistochemistry and *in situ* hybridization. We found that E differentially regulates GAD65 and GAD67 in CA1 pyramidal cell layer. We hypothesize that E regulates GABA synthesis and a ratio of excitatory/inhibitory GABA actions to maintain a balance in excitatory and inhibitory synaptic transmission onto CA1 pyramidal cells in adult hippocampus.

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P3-204 Enhanced PGE_2 production in brain blood vessels of LPS-treated rat and its enzymatic mechanism

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Brain endothelial cells are thought to produce Prostaglandin E_2 (PGE_2), a mediator of fever, as they express PGE_2-synthesizing enzymes such as cyclooxygenase-2 (COX-2) under the inflammatory state. We established an *ex vivo* protocol to directly assess the PGE_2-synthesizing ability of brain blood vessels. Subarachnoidal blood vessels together with arachnoidal membrane were isolated from rat brain 4 h after intraperitoneal injection of either saline or or lipopolysaccharide (LPS, 400 μg/kg), and incubated for an hour at 37°C. Several prostanoids (PGs) in the incubation medium were measured using EIA. Among the PGs, PGE_2 release was most significantly augmented by the LPS treatment. A COX-2 inhibitor added to the medium suppressed PGE_2 production, while none of PLA_2 inhibitors ever tested was effective. This study directly proved the specific enhancement PGE_2 synthesis in brain blood vessels by LPS.

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P3-205 Peripheral lipopolysaccharide injection induces apoptosis in the murine olfactory bulb

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Olfactory bulb (OB) is known to be continuously supplied with newly generated neurons from subventricular zone. In this study, the effect of the propagation of innate immune reaction triggered by peripheral lipopolysaccharide (LPS) on OB of C3H/HeN mice was examined, because LPS-induced tumor necrosis factor (TNF)-α is known to trigger the apoptotic cascade mediated by TNF/TNF receptor (TNFR) pathway. Within 2 h after the 50 mg LPS injection, mRNA expression levels in OB of the genes encoding IκB, TNF-α and TNFR-1 were significantly enhanced. Double immunofluorescence microscopy confirmed that almost all TNF-α-immunopositive cells were in the subependymal zone and overlapped with anti-GFAP positive cells. The number of TUNEL-positive cells exclusively identified in granule cell layer was significantly increased in LPS-injected mice. These results suggest that peripheral LPS injection may cause a disequilibrium between the supply and the disappearance of the cells in OB.

P3-206 Expression of an independent, non-neuronal cholinergic system in lymphocytes and its roles in regulation of immune activity

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Acetylcholine (ACh) has been considered as a classical neurotransmitter in vertebrates. However, lymphocytes possess most of the components to constitute an independent cholinergic system. These include ACh, choline acetyltransferase (ChAT), high-affinity choline transporter and acetylcholinesterase as well as both muscarinic and nicotinic ACh receptors (mAChRs and nAChRs, respectively). T cell activation via T cell receptor/CD3 complex, or interaction of T cells with antigen presenting cells modulates lymphocytic cholinergic activity, as evidenced by up-regulation of ChAT and M5 mAChR mRNA expression. Furthermore, activation of mAChRs and nAChRs on T and B cells causes various functional effects. Abnormalities in lymphocytic cholinergic system have been detected in animal models of immune disorders. These findings provide evidence for the first time that immune function is, at least partly, under the control of an independent, non-neuronal cholinergic system in lymphocytes.

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P3-207 Effect of lipopolysaccharide and norepinephrine on the expression of proinflammatory cytokines

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It has been reported that norepinephrine (NE) regulates proinflammatory cytokine expression, which could contribute to the observed anti-inflammatory properties of NE. We examined the alteration of NE and inflammatory cytokine expression levels at locus coeruleus (LC) of C3H/HeN mice (male, 8W) after intraperitoneal lipopolysaccharide (LPS) injection. Since we have already reported the change of NE at the early time course, we measured the subsequent NE content. The content of NE in the LC decreased at 24 hr after LPS injection. The mRNA expression levels of genes encoding *IL-1β*, *TNF-α* in the LC increased within 2 hr, reached a maximum level at 4 hr, and again increased at 15 hr after the LPS injection. The research with microglia from mouse neonate brain is currently in progress in order to elucidate the relationship between the change of NE content and inflammatory cytokine upregulation at the LC.

P3-208 Characterization of CC chemokine CCL6 in microglia

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Chemokines were originally identified as chemoattractants for leukocytes under inflammatory conditions. There is growing evidence that chemokines play important roles in the immune surveillance of central nervous system (CNS). In the CNS, microglia (MG) are primary immune effector cells and secrete various chemokines in response to the circumstances. Therefore, we are challenging the profiling of chemokines and its receptors expressed by MG. Expression of CC chemokine CCL6 was detected in rat MG but not in astrocytes (AS). Recombinant CCL6 acted as a chemoattractant for both MG and AS, which expressed CCR1 (a receptor for CCL6). LY294002 (a PI3-kinase inhibitor) blocked CCL6-mediated migration of MG, suggesting that the activation of PI3K/Akt pathway plays a role in CCL6-mediated chemotaxis. Treatment of IL-6 and interferon-g enhanced CCL6 gene expression in MG, while TGF-β decreased CCL6 mRNA. Thus, CCL6 expressed by MG might act on glial network in the CNS.

P3-209 Analysis of antibodies to peripheral myelin P0 in sera from human peripheral inflammatory neuropathies

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P0 glycoprotein is a major component in the PNS myelin, and plays crucial role for myelin formation as well as maintenance. We have previously reported that some of sera from patients with acute and chronic inflammatory neuropathy reacted to the peptide portion of P0. In present study, We examined the binding activities of the patients sera to rat teased nerve fibers. Immunofluorescence appeared to co-localize with anti-P0 monoclonal antibody. To determine antigenic epitopes, we prepared three types of P0 peptides, full length of P0 (aa 1-248), an extracellular domain of P0 (aa 1-153) and a cytoplasmic domain of P0 (aa 180-248). Immunoblot analysis using patients sera indicated that these antibodies may be subdivided into different groups depending on their antigenic epitopes. Such difference may influence their pathogenic roles on demyelination.

P3-210 Attempt to map the honeybee brain regions involved in the dance language by using the immediate early gene

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The honeybee can communicate the location of food to their nest mates using the dance language. To identify the brain regions involved in the dance language, we have been trying to develop an experimental system to map the active regions in the honeybee brain, using an immediate early gene, which is expressed in response to neural excitation. In the present study, we have identified a novel immediate early gene, termed *kakusei*, and analyzed its expression and structure. The expression of *kakusei* is restricted to the brain and is transiently upregulated, with peak at 15 mins after the induction. In addition, the expression of *kakusei* in the mushroom bodies, the higher-center of the insect brain, is stronger in workers which show behaviors related to the dance language than in those which do not. These results suggest that *kakusei* can be used as a neural excitation maker in the honeybee brain.

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P3-211 Role of prolactin-releasing peptide in the control of anxiety behaviour in rats

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Prolactin-releasing peptide (PrRP) is synthesized in neurones of the medulla oblongata and of the dorsomedial hypothalamus. These PrRP neurones are activated after emotional stimuli. PrRP nerve fibers are distributed in the hypothalamus, the amygdale and the bed nucleus of stria terminalis. These brain areas play an important role in emotional behaviour. It is thus possible that PrRP has a role in the control of emotional behaviour. Here, we examined effect of icv administration of PrRP or anti-PrRP antibodies (Ab) upon anxiety behaviour in rats. Rats were injected icv with PrRP or anti-PrRP Ab and tested with an elevated plus maze test. PrRP administration increased total length of time spent in the open arm and the frequency of entry into the open arm. On the contrary, anti-PrRP Ab administration decreased time spent in the open arm and the frequency of entry into the open arm. These data suggest that PrRP plays an anxiolytic role in rats.

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P3-212 Involvement of brain polyamines in stressful conditions: effects of putrescine and α-difluoromethylornithine

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We examined the roles of brain polyamines in stressful conditions in mice using behavioral pharmacology methods. In an open-field test, treatment with putrescine (400 mg/kg, i.p.) decreased the amount of rearing and locomotor activity, and increased the amount of grooming. In addition, putrescine treatment (800 mg/kg, i.p.) prolonged the hypnotic activity of pentobarbital. In contrast, daily treatment with α-difluoromethylornithine (500 mg/kg/day i.p. for 14-days), a specific inhibitor of polyamine synthesis, enhanced the seizure susceptibility to pentylenetetrazol, although this drug has no effect on open-field behavior or pentobarbital-induced sleep. These results suggest that brain putrescine acts as an inhibitory neuromodulator in stressful conditions.

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P3-213 A role of the histaminergic system for the regulatory mechanism of feeding by orexigenic peptides: orexin-A, neuropeptide Y, and ghrelin

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We investigated the effect of orexigenic peptides on the rat hypothalamic histamine release and on food intake (FI) and locomotor activity (LMA) in H_1-receptor knockout (H1R-KO) mice, to clarify the interactions among these peptides and the histaminergic (HA) system.
Orexin-A increased histamine release and LMA but not FI, suggesting that the HA system participates in arousal by orexin-A. NPY also increased histamine release but its effect was not immediate. NPY-injected H1R-KO mice consumed more food than wild type mice, thus the HA system may act a feedback factor downstream of NPY. Ghrelin did not affect histamine release, although it increased FI and decreased LMA even in H1R-KO mice. Thus, ghrelin expresses its action in a histamine-independent manner.

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P3-214 Activin is involved in anxiety-related brain function

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We previously isolated activinβA as one of the genes whose expression was regulated by neural activity in the hippocampus. ActivinβA, a member of transforming growth factor-β superfamily, plays an important part in the development. However, the functional role of activin in the adult brain is still unknown. To address the functional role of activin in the adult brain, we generated transgenic mice in which activin or follistatin, which antagonizes activin signal, was over-expressed in the forebrain under the control of αCaMKII promoter. Follistatin over-expression mice showed decrease in general locomotor activity and enhanced anxiety. By contrast, activin over-expression mice showed more aggressive behavior than the wild type littermates and reduced anxiety-related behavior. Our finding was the first report that activin modulated anxiety-related behaviors. Thus, these transgenic mice may be a new and useful animal model for the bipolar disorder.

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P3-215 A novel neuronal activator: function and mode of action of 7α-hydroxypregnenolone

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We have recently demonstrated that vertebrate brains produce actively a novel neurosteroid. This neurosteroid was identified as 7α-hydroxypregnenolone in the newt using biochemical techniques combined with HPLC, TLC and GC-MS analyses. The production of 7α-hydroxypregnenolone in the brain was high unlike peripheral steroidogenic glands. Behavioral analysis demonstrated that this neurosteroid specifically increased, in a dose-related way, locomotor activity. Furthermore, this neurosteroid increased dopamine release in the rostral brain region. The effect on locomotion was abolished by dopamine D_2 receptor antagonists. These findings suggest that the identified novel neurosteroid, 7α-hydroxypregnenolone, acts as a novel neuronal activator to stimulate locomotor activity through the dopaminergic system.

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P3-216 Increased motor function and reduced prepulse inhibition in Wilson disease model rats

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The LEC rat shows abnormal Cu metabolism and is a model of Wilson disease (WD). Although LEC rats have no visible neurological abnormalities in contrast to patient with WD, we have noticed that LEC rats have some behavioral deficiencies. We assessed motor, cognitive and memory functions of LEC rats by Open-field, Acoustic Startle reflex and Prepulse Inhibition, and Shuttle-box avoidance learning tests. LEC rats showed hyperactivity in Open-field test and less attention in Prepulse inhibition test than control Wistar rats, but no significant differences observed in avoidance learning. Moreover, those effects shown in Cu group were attenuated by D-penicillamin. Therefore our data suggest that Cu metabolism in the brain is involved in motor and cognitive functions.

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P3-217 Analysis of transgenic mice overexpressing estrogen receptor in forebrain

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Estrogen receptors (ERs) have been known to function as a ligand-dependent transcription factor. Although estrogen receptors express in adult brain, the function of ERs in brain remains unknown. As shown previously, we found that the injection of ligands activating ER into mice causes significant increase in aggression and anxiety. To further understand roles of ERs in emotional behavior, we derived transgenic mice overexpressing ERalpha in forebrain under the control of alphaCaMKII promoter, and performed open field test, elevated zero maze test, social investigation and social dominance tube test using these transgenic mice. The results showed that overexpression of ER in forebrain region leads to significant increase in aggression and anxiety. Interestingly, these features of mutant mice is similar with them of mice injected ER-agonists. More importantly, transgenic mice injected ER-antagonist showed normal emotional behavior, strongly suggesting that ER signaling pathway in forebrain regulates aggression and anxiety.

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P3-218 Effects of testosterone (T) or estrogen (E) replacement on sex differences in the response of melanin-concentrating hormone (MCH) neurons to glucose in the LHA

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Using the expression of phosphorylated cyclic AMP response element-binding protein (pCREB) as a marker of neural activity, we have shown that fasting for 48 h increases the ratio of MCH neurons expressing pCREB (double-stained neurons) in the LHA of both male and female rats, and glucose decreases the ratio of double-stained neurons in female rats 5 min after injection, but not in male rats. In a time-course study, glucose significantly decreased the ratio of double-stained neurons in male fasted rats 15-30 min after injection, indicating that the response of MCH neurons is more rapid in female rats than in male rats. In T and E replacement studies in gonadectomized rats, T attenuated and E enhanced the response of MCH neurons in male and female rats, respectively. The demonstrated sex difference in the response of MCH neurons to glucose probably explain the sex difference in feeding behavior.

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P3-219 Increase in histamine H_1 receptor mRNA level in hypothalamus of starved rats

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[Aim] Histamine H_1 receptor-mediated H_1 receptor gene expression was observed. The mechanism of H_1 receptor gene expression and H_1 receptor gene expression in vivo using starved rats were studied. [Methods] HeLa cells and male Wister rats weighing 100-120 g were used for studies. H_1 receptor mRNA was determined by real-time PCR. [Results] Histamine induced increase in H_1 receptor mRNA level, and following H_1 receptor up-regulation. Histamine-induced activation of H_1 receptor promoter was also observed. Stability of H_1 receptor mRNA was not changed by the stimulation of histamine. Increase in H_1 receptor mRNA level was completely suppressed in the presence of Ro 31-8220, a PKC inhibitor. Increase in H_1 receptor mRNA level was observed in hypothalamus of both ciproxifan (a H_3 receptor antagonist)-treated and starved rats. The increase in starved rats was reversed in the presence of diphenhydramine, a H_1 receptor antagonist.

P3-220 Roles of the noradrenergic neurotransmission at the amygdala in pain-related aversion

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Roles of noradrenergic neurotransmission at the amygdaloid nuclei in pain-related aversion were examined in rats. Place-conditioning paradigm revealed that both somatic (i.pl. formalin) and visceral (i.p. acetic acid) pain induced aversion. Visceral, but not somatic, pain increased the noradrenaline (NA) release in the central amygdaloid nucleus (CeA). On the other hand, somatic, but not visceral, pain increased the NA release in the basolateral amygdaloid nucleus (BLA). Bilateral injection of α2-adrenoceptor agonist, clonidine into the CeA suppressed the aversion induced by visceral pain, while this agonist did not affect the aversion induced by somatic pain when bilaterally injected into the BLA. These results suggest the involvement of the noradrenergic transmission at the CeA in the visceral pain-induced aversion. On the other hand, the increment of NA release in the BLA does not seem to be involved in the somatic pain-induced aversion.

P3-221 Vesicular zinc distribution in the monkey amygdaloid complex

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Vesicular zinc (VZ), a co-factor of glutamate, is implicated in plasticity, epileptogenesis, excitotoxic cell death, and Alzheimer's disease. We found that the monkey amygdala is VZ-enriched in all nuclear groups, but there are differences in density. The highest level of VZ occurs in the ventral part of the deep nuclear group, the posterior part of the superficial group, and in the central nucleus. Interestingly, regions with higher levels of VZ in the deep nuclei correspond to regions vulnerable to human pharmacoresistant temporal lobe epilepsy. We further found that regions high in VZ tend to correspond to regions of low parvalbumin (PV), a calcium binding protein, probably related to PV-ir GABAergic terminals. This complementary distribution may correlate with activity-related responses. Some reports have suggested the PV expression levels are directly activity dependent, whereas VZ levels seem inversely correlated. The VZ-enriched circuitry in the amygdala may be involved in neuronal plasticity in normal and pathological conditions.

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P3-222 Fasting induced changes in GABA and glutamate release in the ventromedial hypothalamus using in vivo microdialysis

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In the ventromedial hypothalamus (VMH), it is thought to exist a satiety center. Especially GABAergic system in the VMH is known to play an important role in the regulation of feeding behavior. In this study we examined the effect of prolonged fasting on the secretion of GABA and glutamate in the VMH using in vivo microdialysis in the rats. We also investigated the effect of leptin treatment on the amino acids secretion in the VMH in the fasted rats. In the fasted rats, an increase in GABA release and a decrease in glutamate release was observed. Though leptin treatment in the fasted rats inhibited the increase in GABA release in the VMH, the decrease in the glutamate release did not influenced. These results suggest that GABAergic and glutamatergic system in the VMH may participate in the regulation of feeding behavior. Furthermore it is possible that GABAergic system in the VMH may play an important role in mediating the effect of leptin in the central nervous system.

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P3-223 CENTRAL ACTION OF PROLACTIN (PRL) ON THE INDUCTION OF COURTSHIP BEHAVIOR IN THE MALE NEWT

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The effects of intracerebroventricular (ICV) and intraperitoneal (IP) injections of PRL or antiserum against newt PRL on the courtship behavior of male newts, *Cynops pyrrhogaster*, were studied to see whether PRL acts centrally or peripherally to induce the behavior. Both of ICV and IP injections of PRL to gonadotoropin-primed males enhanced the expression of the behavior dose-dependently. The minimum effective dose of PRL was 100 ng for ICV injection, whereas it was 100 μg for IP injection when injected intraperitoneally. ICV injection of antiPRL serum (0.5 μl) blocked the spontaneously occurring behavior in sexually developed males, whereas IP injection of the same volume of the antiserum did not affect the behavior. These results suggest that PRL induces the behavior by acting centrally. Furthermore, PRL prolonged the time spent for the courtship behavior.

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P3-224 ACTIVITY OF VTA NEURONS IN SINGING BIRDS IS MODULATED BY SOCIAL CONTEXT.

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Male zebra finches sing a similar song in two distinct social contexts. "Directed song" is used during courtship of females, while "undirected song" is not targeted to any particular recipient. Previous studies have shown that singing-related neural activity (Hessler & Doupe, 1999) and gene expression (Jarvis, et al. 1998) in the anterior forebrain pathway nuclei LMAN and Area X depend on the social context in which a bird sings. As a likely source of such modulation, we recorded single-unit activity in the midbrain dopaminergic nucleus VTA while birds sang both directed and undirected songs. We found 2 types of VTA neurons whose activity depended on social context. "Type-1" neurons fired phasically during directed, but not undirected singing. "Type-2" neurons ceased firing during directed singing, but did not alter their tonic firing during undirected singing. These results suggest that VTA may directly modulate forebrain activity, dependent on the social context. This system should be useful for investigating the role of dopamine in modulation of behavior and neural activity.

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P3-225 Investigation of behavioral and anatomical lateralization in courtshipsinging.

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While previous studies have examined songbird brain lateralization inrelation to sensory and motor processes, we are examining its relationship to motivational function. We have previously shown that activity in medial Habenula (MHb) and interpeduncular nuclei (IPN) is strongly modulated by thesocial context in which a zebra finch sings, with increased egr-1 expression specifically during courtship singing. As expression is stronger in limited subregions of IPN, it is possible that MHb-IPN inputs may be lateralized. Weare combining anterograde tracer injection into left and right MHb with egr-1 expression after courtship singing to test this possiblility. Further, as a possible behavioral source of such lateralization, in initialbehavioral results, 3 of 4 birds tested viewed the female during initialcourtship preferentially with the right eye.

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P3-226 Intake of palatable tastants is mediated by neuromodulation in the ventral pallidum

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It is suggested that the ventral pallidum (VP) is involved in feeding behavior through projections from the nucleus accumbens shell (AcbSh). We previously reported that microinjections of bicuculline, a $GABA_A$ antagonist, into the VP increased the intake of a sweet solution (5 mM sodium saccharin), but not distilled water or a bitter solution (0.3 mM quinine hydrochloride). However, it is still unclear how the VP controls palatability-induced ingestion. To further clarify the function of the VP we examined the effects of microinjections of SCH23390, a D_1 dopamine receptor antagonist, into the VP on the intake of taste solution. Injections of SCH23390 decreased the intake of 5 mM sodium saccharin, but not distilled water. These results suggest that both GABAergic and dopaminergic neuromodulation in the VP mediate the palatability-induced facilitation of feeding.

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P3-227 Organization of Sexually Dimorphic Nucleus and the Distribution of ERαReceptor Are Affected by the Intrauterine Position

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The sexually dimorphic nucleus of the preoptic area (SDN-POA) plays a key role in the activation of several sexually differentiated behaviors in adulthood. To determine whether intrauterine position modifies the region development, we examined the SDN-POA in adult rats after identifying fetal positions between two male fetuses (2M) and between two female fetuses (2F). Brain sections were analyzed by immunohistochemistry using an antibody for calbindin-D28K. Three-dimensional image analysis of SDN-POA showed that 2M adult males had greater SDN-POA volume than 2F males, while 2M and 2F females had no significantly difference. In addition, we also investigated the effect of intrauterine position on distribution of ERα in SDN-POA in adulthood.

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P3-228 Membrane properties as interneurons of the extra-ocular photoreceptors in the *Onchidium* ganglia

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Four extra-ocular photoreceptors, the photoresponsive neurons, A-P-1, Es-1, Ip-1 and Ip-2 which respond directly to light, exist in the abdominal ganglion of the mollusc *Onchidium*. A-P-1/Es-1 of these neurons respond to light with a depolarizing receptor potential, whereas light hyperpolarizes the other Ip-1/Ip-2. The present study was conducted to examine the functional significance of the above Ip-1/Ip-2, having a peak sensitivity at 510 nm light. The body wall of the animal transmitted enough 510 nm to hyperpolarize Ip-1/Ip-2 in the ganglion covered by the body wall. Ip-1/Ip-2 were coupled with weak electrical synapses and involved in a spontaneous beating or bursting spike activity. Ip-1/Ip-2 were not coupled to A-P-1/Es-1 one another. Finally, Ip-1/Ip-2, the first order photosensory cells were also the second order interneurons or motoneurons relaying various sensory inputs and innervating the pneumostome and viscera.

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P3-229 Light-induced c-Fos expression and phosphorylation of ERK and CREB in the SCN in mice lacking PACAP

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PACAP is expressed in RHT, a direct pathway from retina to the SCN. We recently demonstrated that PACAP is important for light-induced phase shift, especially for phase advance at late night. In this study, to clarify the molecular mechanism in PACAP-mediated phase shift, we examined induction and phosphorylation of proteins in the SCN in mice lacking PACAP (PACAP-KO).
Light-induced c-Fos was significantly blunted in PACAP-KO only at early night. Phosphorylation of ERK and CREB was not changed at early night, whereas it was lower in PACAP-KO at a higher irradiance compared with wild types. These data show the dissociations between phase shift and c-Fos induction as well as protein phosphorylation, and the involvement of PACAP in photoentrainment in an irradiance-dependent manner, providing new insights into the mechanism of the circadian clock.

P3-230 Instantaneous acceleration of hippocampal theta wave associated with pontine phasic activity during REM sleep in rats

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Pontine phasic potential, which is called pontine wave (P-wave), is recorded during REM sleep. This potential occurs concomitantly with the burst firing of pontine reticular formation (PRF) neurons. Ascending pathway from PRF to the hippocampus is known to be involved in frequency modulation of the hippocampal theta wave. In this study, we observed the frequency of theta wave and P-wave during REM sleep in rats. A scalogram of hippocampal EEG was calculated based on the Gabor wavelet in order to see a temporal change in the frequency of the theta wave. The scalogram showed that the theta wave was accelerated just after the P-wave occurrence. This finding suggests that a P-wave generator modulates the frequency of hippocampal theta wave.

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P3-231 mPER1 acts as a circadian adaptor to entrain theoscillator to environmental light-dark cycles by regulating mPER2 protein

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mPer1 and *mPer2* genes are essential for the mammalian circadian clock. Gene targeting studies have demonstrated that *mPer2* plays a dominant function for rhythm generation, while the role of *mPer1* has not been fully clarified. Phase-shifting by light is an essential feature of circadian rhythms. Here we report that prolongation of the lighting period induces a larger delay-phase shift of behavioral rhythm in *mPer1*-deficient(*mPer1*$^{-/-}$) mice. Prolongation of the light exposure in *mPer1*$^{-/-}$ mice suppresses mPER2 protein decay in the suprachiasmatic nucleus, the mammalian master clock. *mPer1*$^{-/-}$ mice could not adapt to environmental light-dark cycles in long photoperiods which mimic natural environmental changes present at high latitudes for nocturnal mice.

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P3-232 Different sensitivity of the suprachiasmatic nucleus neurons and optic fibers to light stimulation in mice

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Mammalian circadian rhythm is entrained to environmental light/dark cycles by the suprachiasmatic nucleus (SCN) receiving photic information from the retina. Electrophysiological studies suggest that the light response sensitivity of the SCN neuron is characteristically inferior to the retinal response. However, the SCN neuronal light response has never been compared with response of the optic fiber in the animals in vivo. Therefore, we investigated the light response sensitivity in the firing activity of the SCN neurons and the optic fibers in mice in the same experimental system in the same time zone (Zeitgeber time 14-16). As the result, the light intensity yielding half maximum response was about 30-50 times higher in the SCN neuron than in the optic fiber. This finding suggests that SCN neurons tune light response sensitivity to dawn and dusk.

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P3-233 Spontaneous neuronal oscillation suppresses the evoked pEPSP in guinea-pig hippocampal slices

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Spontaneous hippocampal neuronal oscillation (SHNO) was observed in the CA3 region of hippocampal slices. In this study, the effect of SHNO on pEPSP was studied. *Hippocampus was cut into 4-500μm-thick slices.* pEPSP and SHNO were measured by the same glass microelectrode containing 2-3M NaCl (3-5 MΩ). The electrode was located at stratum pyramidale. Schaffer collateral and commissural fibers were stimulated with bipolar stimulating electrode. SHNO had the peak frequencies of 15-20, 30-40, 50-55Hz, and the mean frequency was 35.4±1.2Hz (mean±SE, n=23 slices). It could be observed even in the isolated CA3 mini slices. SHNO was reversibly blocked by 1μM bicuculline and blocked by 10μM tetrodotoxin. The blockade of SHNO also facilitated pEPSP. SHNO occurred for at least one hour. When SHNO became smaller, the evoked pEPSP was bigger than pEPSP induced at normal SHNO. These results suggested that SHNO will come from the spontaneous activity of the inhibitory neurons in CA3 and it may modulate the excitatory synaptic transmission.

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P3-234 Identification and functional analysis of new interacting proteins with transcription factor CLOCK and BMAL1

Function of NSP-C (Neuroendocrine specific protein-C) in CLOCK/BMAL1-mediated transcription

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bHLH-PAS transcription factor CLOCK and BMAL1 play essential roles in regulation of circadian rhythm. The CLOCK/BMAL1 binds to E-box sequence and activates transcription of core clock genes. The activity of CLOCK/BMAL1 has been shown to be inhibited by PERIODs and CRYs. However, the molecular mechanisms of transcription activation by CLOCK/BMAL1 remain unknown. Therefore, we performed yeast two-hybrid screening using brain cDNA library to identify new interacting proteins with CLOCK. Here we show that NSP-C, highly expressing in brain, interacts with the bHLH-PAS domain of CLOCK in yeast. We also detected this interaction of NSP-C with CLOCK in COS-1 cells using IP-Western analysis. Importantly, co-expression of NSP-C with CLOCK/BMAL1 enhances the transcription activation by CLOCK/BMAL1, suggesting that NSP-C functions as positive regulators for CLOCK/BMAL1-mediated transcription.

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P3-235 Cell-to-cell interaction on the rat suprachiasmatic nucleus

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In mammals, circadian rhythms are driven by a pacemaker located in the suprachiasmatic nuclei (SCN) of the hypothalamus. The pacemaker is composed of an ensemble of multiple, single-cell oscillators in the SCN. We measured vasopressin (AVP) release in SCN slices. The SCN culture showed circadian oscillation of AVP release with a period length (± S.E.M) of 23.83 ± 0.03 h . This period is very similar to the one we previously reported in dispersed SCN cultures and is also close to that of behavioral rhythms. When the ventral part was removed by surgical cut across the slice in the horizontal plane, however, the period became shorter (23.17 h ± 0.11 h). On the other hand, the removal of the dorsal part did not affect period length. These results suggest that the oscillators in ventral and dorsal cells contribute differently to period length and that the dorsal oscillators are regulated by ventral one to form a single integrated oscillator.

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P3-236 Accumulation of Peripheral Glycine in the Pineal Body

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The pineal is believed as one of the regulator of the circadian rhythm. Melatonin has been ascribed to a possible mediator of this function, but much has been remained to be elucidated. Our autoradiographic study indicated specific accumulation of peripherally administered glycine in the pineal. This finding prompted us to carry out a series of experiments focusing on how peripheral glycine was incorporated in the pineal. We found: 1) concentration of glycine in the pineal body was much higher than that in either plasma or cerebrospinal fluid, 2) glycine transporter (GlyT1) was highly expressed in the pineal body. This glycine transporter may be responsible for active transport of peripheral glycine into pinealocytes. Thus, the pineal may work as a "pump" to pump up glycine from the peripheral circulation and possibly be involved in the circadian rhythm regulatory mechanism.

P3-237 Enhancement of sleep by IL-1β is not mediated by prostaglandin D2 in mice

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Infectious disease is associated with an increase in sleep, and production of cytokines and prostaglandins (PGs). Both IL-1β and PGD2 induce sleep, however, little is known about the molecular interaction between them in the sleep regulation. Here we used PGD2 receptor knockout (DPR KO) mice to examine the involvement of PGD2 in the somnogenic effect of IL-1β. Intraperitoneal injection of IL-1β (1.7 and 5 μg/kg) significantly increased non-rapid eye movement sleep by 1.8 and 2.3 fold in wild-type mice, and by 1.6 and 2.2 fold in DPR KO mice, respectively, and concomitantly reduced body temperature and locomotor activity for 6 h after injection, as compared with the value for baseline day. In addition, pretreatment of NS-398, a COX-2 inhibitor, did not inhibit IL-1β-induced sleep in mice. These results indicate that PGD2 is not involved in the somnogenic effect of IL-1β in mice.

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P3-238 Neural Network Model for Oscillatory Phenomena in Thalamus with Neuromodulation Due to Acetylcholine

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We propose a neural network model consisting of a layer of the thalamic reticular neurons and that of the cholinergic neurons, and investigate an effect of neuromodulation due to the acetylcholine on oscillatory phenomena in the thalamus by numerical simulations. We introduce a dynamics of concentration of the acetylcholine, which depends on a state of the cholinergic neurons, and assume that the conductance of the thalamic reticular neurons is dynamically regulated by the acetylcholine. From results by numerical simulations, we find that a dynamical transition between a bursting state and a resting state occurs in the layer of the thalamic reticular neurons by the effect of neuromodulation due to the acetylcholine. It turns out that the effect of neuromodulation due to the acetylcholine is important for the dynamical transition between the oscillatory phenomena in the thalamus.

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P3-239 *Caenorhabditis elegans* as a tool opening up new insights into the circadian clock mechanism.

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C. elegans carries homologues of *per* and *tim* constituting the core of circadian clocks, *lin-42* and *tim-1*, which appeared to be involved in functions other than the circadian clock. *C. elegans* lacks *cry*, another core component of rodent circadian clock. Nevertheless, *C. elegans* exhibits convincing circadian rhythms. Then, uncovering the core of circadian clock in *C. elegans* is crucial to understand a common basic mechanism of the circadian clock in animal kingdom. *C. elegans* provides various convenient molecular biological techniques. We have devised a new system to measure the behavioral rhythms in a number of isolated nematodes. We are examining the behavioral rhythms in isolated nematodes with genes knocked-out or rescued. We report tentative results obtained with certain genes knocked out.

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P3-240 Intracellular signaling involved in the crucial time-delay of the mammalian clock-oscillating system

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We previously reported circadian phosphorylation and nucleocytoplasmic shuttling of BMAL1:CLOCK in the cellular clock model. There is a significant time-delay between BMAL1 transcription and protein accumulation into the nucleus. In CRY-deficient fibroblasts, BMAL1 is only present as hyperphosphorylated forms and their circadian nucleocytoplasmic shuttling is absent. We examined roles of the putative modulators, such as Periodically fluctuating kinase (PFK) which phosphorylate BMAL1 and CRY1/CRY2 which affect on BMAL1 phosphorylation, in nucleocytoplasmic shuttling of BMAL1 and generating Per1- and Bmal1-reporting luciferase circadian rhythms, using RNAi-knock down and ectopic expression strategies. Here, we propose that regulation of nucleocytoplasmic shuttling and phosphorylation states of BMAL1 by the circadian intracellular signaling have crucial roles in the clock-oscillating system.

P3-241 Postnatal development of rPer2, rBmal1 and Arg-vasopressin mRNA circadian rhythms in the suprachiasmatic nucleus of rat pups under the light-dark cycle and constant dim light.

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Developmental mechanism of the clock related genes in the suprachiasmatic nucleus (SCN) remains unclear. Circadian rhythm of arg-vasopressin (AVP) peptide, already starts to oscillate before the postnatal day 5. To clarify the entraining ability of dam on the clock related genes of pups, we measured the circadian rhythm of rPer2, rBmla1 and rAVP mRNAs in the SCN of sighted rat-pups nursed under the light-dark (LD) cycle and the pups nursed under the constant dim light (DD). The mRNAs were determined using RT-PCR and normalized by beta-actin mRNA in each sample. The rPer2, rBmal1 and rAVP mRNA rhythmicity were free run during the nursing period even under the LD entrained condition. The rBmal1 transcriptional activation by PER2 might be functioning after the maturation of clock mechanisms. The results indicate that the nursing dam partially entrains pup-circadian-rhythm.

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P3-242 Altered behavior of sleep-wake cycle and histaminergic systems in mice lacking histamine H_1 receptor

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Histaminergic system is considered to play a crucial role in mediating arousal via histamine H_1 receptors (H_1R). We characterized the sleep-wake behavior of H_1R knockout (KO) mice to clarify the contribution of H_1R in the arousal control. As compared with wild type (WT) mice, H_1R KO mice exhibited decreases in the histamine content and the mRNA expression of histidine decarboxylase and H_3 receptor (H_3R) in the brain. The KO mice showed identical sleep-wake cycles to those of WT mice but with fewer number of brief awakening (<16 sec epoch) and prolonged duration of non-rapid eye movement (NREM) sleep. Although ciproxifan, an H_3R antagonist, increased histamine release in both genotypes, it increased wakefulness in WT mice but not at all in H_1R KO mice. These results indicate that H_1R is important in the regulation of state transitions from NREM sleep to wakefulness and is essential for the arousal effect of H_3R antagonist.

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P3-243 Circadian fluctuations in the activity of repairing DNA damaged in Paramecium nuclear and mitochondria.

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We previously demonstrated that *Paramecium* mitochondrial activity fluctuates in a circadian manner. Recent studies show that mitochondria are both the target and the source of oxidative stress. Oxidative damage in DNA could occur in a circadian manner. Then, we measured the circadian rhythm in the accumulation of 8-OHdG, a bi-product of repairing damaged DNA, in *Paramecium*. Concentrations of 8-OHdG appeared to fluctuate in a circadian manner with a phase delay of about 90° to the circadian fluctuation of the rate of *Paramecium* O_2 consumption, suggesting that the repair activity of damaged DNA fluctuates in a circadian manner. We argue a possibility that circadian homeostasis is inherited in eukaryotic cells to minimize errors accumulated in DNA as possible in the environment cycling with 24-h period, which absolutely governs dynamics of various cellular molecules including even clock genes.

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P3-244 Brain oxidation is an initial process in sleep induction

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An endogenous antioxidant, glutathione, is a sleep-promoting substance, yet the relationship between sleep and cerebral oxidation remains unclear. Here we report that a low concentration of the organic peroxide (TBHP) in the preoptic/anterior hypothalamus (POAH) triggers the release of nitric oxide and adenosine, and promotes sleep in rats. Also, Ca^{2+} release from mitochondria and delayed-onset Ca^{2+} influx via NMDA receptors was visualized during TBHP exposure using Ca^{2+} indicator proteins expressed in POAH neurons. High levels of TBHP caused neuronal degeneration and reduced sleep. Glutathione inhibited intracellular Ca^{2+} mobilization and protected POAH neurons. Therefore, low-level cerebral oxidation under the control of an antioxidant system may trigger sleep via the Ca^{2+}-dependent release of sleep-inducing neuromodulators in the POAH.

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P3-245 Sleep and body temperature profiles in FMR1 knockout mice

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Fragile X syndrome is a inherited mental retardation caused by loss-of-function mutations in the fragile X mental retardation 1 (fmr1) gene. In order to observe the function of mammalian fmr1 gene, we recorded body temperature (Tb), spontaneous activity (ACT), EEG and EMG under 12-12h light-dark (LD) cycle and constant darkness (DD) conditions in fmr1 knockout mice. The diurnal rhythms of sleep, ACT and Tb in the knockout mice were not significantly different from that in the wild-type mice. Fmr1 knockout mice showed a mild learning deficit in radial maze. Under LD condition, REM sleep was significantly increased in the fmr1 knockout mice. Power of hippocampal theta rhythm during REM sleep was larger in fmr1 knockout mice. Fmr1 gene is suggested to be involved in REM sleep-related brain function.

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P3-246 CCK-A receptors as Photic Entrainable Signal Transducers in the Mouse Suprachiasmatic Nucleus

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We examined photic response of the circadian clock system in CCK-A receptor knockout mice. We found that the mutant mice exhibited attenuated phase-shifts in response to a light pulse. The photic induction of mPer1/2 mRNAs and c-Fos proteins in the SCN was also attenuated in the mutant mice. Therefore, CCK-A receptors may positively modulate the photic entrainment pathways in the murine clock system.

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P3-247 Effects of 30 minutes oral reading on human EEG

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We previously examined the effect of voluntary abdominal breathing (VAB) related to Zen Meditation on EEG, and found that the spectral power in high-frequency alpha band increased during VAB. In the present study, we tested the effects of 30 minutes oral reading (OR) of a classic text such as Buddhist scriptures on EEG, because the breathing pattern of OR was similar to that of VAB. The alpha power began to increase several minutes after OR and its topographic distribution extended from the occipital to the frontal area. Since OR consisted of rhyme and rhymeless period, we divided into two periods. As a result, we found that the alpha power during rhyme period was higher than that during rhymeless period. The urinary and/or blood serotonin (5-HT) levels tended to increase after OR. These results suggest that the activation of 5-HT neurons in the brain stem may be involved in the appearance of alpha band during OR.

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P3-248 Aging of cholinergic, monoaminergic and GABAeregic neurons in the brainstem of old rats

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Sleep pattern in animals changes in old age. The cholinergic (ACh) neurons in the laterodorsal tegmental nucleus (LDT) play a main role in the regulation of paradoxical sleep (PS) or waking. Monoaminergic neurons and GABAergic neurons participate in the regulation of PS. To know the roles of these neurons in sleep of old animals, we examined cholinergic natures of the ACh neurons in the LDT and the immunostainings of monoaminergic and GABAergic neurons in the brainstem of rats aged from 15 to 24 months, and compared them with those in young adults. The specific activity of choline acetyltransferase (ChAT) was lower than that in young adults, while the immunostainings by ChAT, tyrosine hydroxylase and glutamate decarboxylase in the brainstem were similar to those in young adults. These results suggest that the ACh neurons in the LDT may partly relate to the change of sleep pattern.

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P3-249 Spatial cognition deficit in a water maze during both weaning and adult periods in experimentally-induced microencephalic rats with an abnormal morphogenesis of the CA1 hippocampus

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We examined spatial cognition in a Morris water maze during weaning and adult periods in experimentally-induced microencephalic rats with a morphogenetical abnormality of the hippocampus. Jcl:Wistar pregnant rats were injected with a 25 mg/kg methylazoxymethanol (MAM) or saline on the 15th day of gestation. Offspring were trained to reach a hidden escape platform which was placed in the center of a quadrant in a circular pool for 10 days (4 trials per day) during weaning or adult period. Histologically, the MAM-treated rat had abnormal hippocampus (disruption of the CA1 pyramidal layer and ectopic neuron mass) as well as a marked decrease in cortical thickness. Behaviorally, the MAM-treated rat showed significantly longer escape latency through the training during both weaning and adult periods. These results suggest that abnormal hippocampal morphogenesis produces a long-term spatial cognition deficit.

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P3-250 Theoretical implications for stable cortical dopamine release: contribution of the fronto-mesofrontal system

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This work investigates computationally the mechanisms by which dopamine (DA) level in the prefrontal cortex (PFC) is controlled. In our model, D1 receptor activation assumes to increase both the excitatory synaptic weight and the time constant of inhibitory interneurons. These effects provide an inverted U-shape profile of the PFC neurons' delay-period activity. The DA neuron is modeled by a first-order system, whose input is derived from the cortical feedback, forming a model fronto-mesofrontal closed-loop. The result indicates that the fronto-mesofrontal closed-loop circuit can produce the optimal DA release, thus enabling the PFC neurons to evoke persistent activity during the delay period. Furthermore, the control theoretic analysis makes it clear that this optimized DA level is at one of the stable equilibrium points. It is also noteworthy that the stability range enlarges as the cortical feedback gain decreases.

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P3-251 Common properties of aversive olfactory learning and LTP in the OB

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Rat pups show aversion to the odor that was exposed with foot shock. The mechanism underlying this aversive olfactory learning is considered to involve disinhibition of mitral cells in the olfactory bulb (OB) by the noxious stimulation-induced noradrenergic activation. It was reported that NMDA receptors and L-type voltage-gated calcium channels (VGCCs) are involved in LTP induction in the hippocampus. We examined the effects of AP5 or nifedipine infused into the OB on olfactory learning. Infusion of AP5 failed to prevent olfactory learning, but facilitated olfactory learning without foot shock. Nifedipine had a biphasic effect on olfactory learning, which was faciliatory at a high dose and inhibitory at a low dose. We already reported that NA-LTP which is induced with noradrenaline (NA) application in the OB is partially reduced by AP5, and almost completely blocked by nifedipine. These results suggest a possible link between aversive olfactory learning and LTP.

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P3-252 Afferent and efferent connections of the area corticoidea dorsolateralis in the pigeon

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The present study was examined afferent and efferent connections of the area corticoidea dorsolateralis (CDL) in the pigeon by a tract-tracing method using two types of tracers, BDA and CTB. In particular, fiber connections between CDL and hippocampal formation was noticed. Major distribution of CTB-labeled neurons was found in CDL, CPi, ventral NCm, TPO, HD, HL, ventrolateral edge of rostral AV and hippocampal formation. In the hippocampal formation, a large number of labeled neurons were seen in medial DL, lateral DM, Ma, Tr and a part of medial layer. In the diencephalon, DLM contained many CTB-labeled neurons. Distribution pattern of BDA-labeled fibers showed similar to those of CTB-labeled neurons in the telencephalon. However, in the hippocampal formation, dense labeling was seen in lateral DM, Po and V-shaped layer. The olfactory bulb contained numerous BDA-labeled fibers. These findings indicate strong fiber connections between CDL and hippocampal formation and suggest that the avian CDL is comparable to mammalian cingulate cortex.

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P3-253 Action evaluation by secondary rewards in the medial prefrontal cortex

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To clarify neural mechanisms by which actions are evaluated by secondary rewards, we trained two monkeys on a task with secondary rewards and recorded cell activity of the medial prefrontal cortex (MPFC). In a stimulus-block (S-blc), a same visual stimulus was paired with a liquid reward 3 times to make it a visual secondary reward (V-rew). In a following action-block (A-blc), the monkey searched correct action (left- or right-lever press) based only on the V-rew. A wrong action was followed by another visual stimulus (V-pun). When the monkey made 3-4 successive corrects, a new S-blc started. The monkeys showed correct rates >90 % even in the 2nd trials of A-blcs. Many MPFC cells selectively responded to either V-rew or V-pun. The responses to V-rew were significantly stronger in the 1st correct trials in A-blcs than in the 3rd correct trials, and they were also stronger than the responses to the same stimuli in S-blcs. These suggest that MPFC contributes to the action evaluation by secondary rewards.

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P3-254 A novel kruppel-like factor induced by neuronal activity in slug brain

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The terrestrial slug *Limax* has an excellent ability to learn to avoid the odor of food once it was presented in combination with the aversive stimulus (bitter taste or electrical shock). Input and output nerve tracts which are likely involved in the odor-taste associative learning were identified recently using *in vitro* recording system (Inoue et al, 2004). But it remains be uncovered how and where the information is processed and memory is stored in the slug CNS. Here we screened genes induced in an activity-dependent manner in slug CNS to isolate a neuronal activity marker to visualize the trace of information processing. We identified a novel kruppel-like factor (limKLF) as an immediate-early gene up-regulated in CNS including procerebrum. We also compared the expression profiles of limKLF and that of C/EBP. The threshold of neuronal activity for the induction of limKLF is higher than that of C/EBP. These two genes showed induction in different ganglia by aversive, uncomfortable stimuli to the body of slug.

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P3-255 Role of cerebellar LTD in the long-term adaptation of horizontal optokinetic response in mice

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The role of cerebellar LTD in the adaptation of ocular reflexes induced by short-term training is well characterized, however, that induced by long-term training is not known. We recently developed a paradigm for long-term adaptation of mouse horizontal optokinetic response (HOKR). Reversible shutdown experiments indicate that the memory traces of short- and long-term adaptations are located differently. Here, we evaluated the role of LTD in the long-term adaptation of the HOKR. Mice treated with the inhibitor of NO syntase (NOS) during training sessions exhibited significantly reduced adaptation compared with mice treated with the control drug. Moreover, long-term adaptation was markedly depressed in mice devoid of NOS compared with wild-type mice. These results suggest that LTD is essential for both short- and long-term adaptations of HOKR.

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P3-256 Role of hippocampal NMDA receptors in trace eyeblink conditioning

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We examined the effects of acute injections of competitive NMDA receptors antagonist APV into the dorsal hippocampus on classical eyeblink conditioning, delay, trace, and pseudo paradigms. During the delay conditioning APV-injected mice acquired conditioned responses (CR) as well as aCSF-injected control mice. However, in the trace conditioning, in which the conditioned stimulus (CS) and unconditioned stimulus (US) are separated by a stimulus-free trace interval (500 msec), APV-injected mice showed impairment in acquisition of CR. There is no difference on eyeblink responses in the pseudoconditining between the APV- and aCSF-injected mice. These results provide evidence that NMDA receptors in the hippocampus are critical for the acquisition of CR in long trace eyeblink conditioning.

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P3-257 A novel cellular mechanism for graded persistent activity in a model neuron and its implications in working memory

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Working memory is believed mediated by persistent neuronal firing. However, the mechanism of such persistent activity remains elusive. In this study, we propose a novel mechanism to generate persistent firing of multiple firing rates in single neurons. In the proposed mechanism, graded stable concentrations of inositol 1,4,5-trisphosphate (IP3) and Ca2+ can be achieved by IP3 formation and IP3-induced Ca2+ release from stores. Our model is consistent with several experimental findings, including demonstration of graded persistent activity of the rat enthorhinal neurons. We suggest that the shortest input duration necessary for inducing graded rate changes, which was of the order of several seconds in previous experiments in a slice preparation, can be significantly shortened to some hundred of milliseconds, which is compatible with the typical duration of transient stimuli in working memory.

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P3-258 Changes of electrical responsiveness of vestibular nuclei correlated with the long-term adaptation of horizontal optokinetic responses in mice

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Recently, importance of cerebellum is suggested for long (4-5 days) (LTA) as well as short (1 hour) -term adaptation of optokinetic response (OKR) eye movement. Because vestibular nuclei mediate optokinetic signals via flocculus and prepositus hypoglossal complex, we investigated changes of the vestibular nuclei during LTA. One hour optokinetic trainings were performed for 5 successive days in mice. Evoked field potentials by electrical stimulation of lateral and posterior semicircular canals were examined in vestibular nuclei under chloralose anesthesia. Responsiveness of monosynaptic field-EPSPs are increased in lateral and medial vestibular nuclei after LTA, suggesting that vestibular nuclei are related to memory of flocculus-dependent LTA.

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P3-259 Reversion of mecamylamine-induced acetylcholine decrease in rat hippocampus and learning and memory impairment by nociceptin

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The effects of nociceptin on learning and memory impairment, and the decrease in acetylcholine release induced by mecamylamine were investigated in rats. Mecamylamine (49 μmol /kg, s.c.), a nicotinic acetylcholine receptor antagonist, impaired learning and memory in the step-through type passive avoidance test, and decreased in acetylcholine release in the hippocampus as deterimined by in vivo microdialysis. Administration of nociceptin (10 and 100 fmol/rat, i.c.v.) reversed the impairment of learning and memory and blocked the decrease in acetylcholine release induced by mecamylamine. These effects were blocked by [Nphe[1]]nociceptin (1-13) NH_2, an ORL-1 receptor antagonist. These results suggest that nociceptin improves the impairment of learning and memory and the decrease in acetylcholine release induced by mecamylamine via ORL-1 receptors.

P3-260 Behavioral performance and brain nestin distribution in the C57BL old mice.

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We investigated the behavioral performance and neuronal stem cell which were stained by nestin antibody, in male, female, mature and or old C57BL mice. Behavioral test were performed particuler in memory, feeding and moving performance. Several difference were cleared in behavioral performance between male, female and old mice. The distribution of nestin positive cell in the SVZ (subventricular zone) and DG (dentate gyrus) were discussed comparisons of gender and age.

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P3-261 Acquisition and performance of the reaction time task in mice

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The reaction time (RT) paradigm in rodent models provides a useful task for examining the neural mechanisms underlying operant behaviors. In the paradigm, animals have to start or stop an operant behavior according to a cue stimulus to gain a certain reward. The acquisition and performance of this paradigm require the function of the dorsal striatum dependent on dopamine transmission. In this study, we tested the acquisition and performance in RT task in mice. C57BL/6J male mice were trained to press a lever and release it as quickly as possible after a light-tone cue stimulus. RT was measured from the cue stimulus to the release of the lever for each trial. We found that 8 of 11 mice performed the bar-releasing behavior within 600 msec of the cue stimulus in 80% of the trials on average. The results suggest that RT paradigm is useful for research on the central mechanism of dopamine-mediated operant behaviors in mice.

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P3-262 The rat dorsal cingulate cortex is subdivided into two distinct areas

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The rat cingulate cortex has been divided into two areas: dorsal cingulate (Cg1) and ventral cingulate (Cg2) cortices. Although behavioral and physiological studies demonstrated that the anterior part of the Cg1 might be involved in the higher brain functions, anatomical substrate of this region has not been fully elucidated. The present study characterized the associational connections within the medial wall of the rat cerebral cortex by using the anterograde tracer, BDA. The results indicate that most areas in the rat midline cortex possess extensive interconnection, which shows two patterns: one is the dorso-ventral connection, and the other is the rostro-caudal connection. Moreover, the rat Cg1 has two distinct subareas: one is the anterior part (Cg1a), which is anterior to corpus callosum, and the other is the posterior part (Cg1p), which lies on the corpus callosum. Cg1a might be the nodal point to converge the information of medial wall of the rat cerebral cortex.

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P3-263 Birdsong plasticity is correlated with cytoskeletal F-actin content within the dendritic spine in zebra finch song control nuclei.

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Young songbirds learn their song during a sensitive period. Their initial vocalizations become stereotyped and stable through auditory feedback. In parallel with song stabilization, the dendritic spines with the eccitatory synapses mature gradually. In the motor pathway of songbird brain, the HVC neurons project direct to the RA. The HVC generates sequences of song elements, and the RA produces motor commonds for each element, suggesting that the synaptic connections among HVC-RA neurons form the song structure. The dendritic spine contains the highest concentration of F-actin. Here, dendritic spines in zebra finch RA were visualized using phalloidin-TRITC. Two groups with different song stereotypy index were compared: normal bird with high song stereotypy and birds reared under solo isolation with lower stereotypy. Consequently, song stereotypy was correlated with the contents of F-actin in the dendritic spine.

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P3-264 Antidromic activation of rat ventral hippocampal neurons from prefrontal cortex and amygdala

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Neurons in the ventral hippocampus are known to project directly to the medial prefrontal cortex (mPFC) and basolateral nucleus of amygdala (BLA). However, there is no evidence whether the same hippocampal neurons project to both the mPFC and BLA. In sodium pentobarbitone-anesthetized rats, single unit activity was recorded in the ventral hippocampus to obtain antidromic response to mPFC and BLA stimulation. Twenty-five cells were activated antidromically from the mPFC and/or BLA. These neurons were located in the CA1 and subicular regions of the ventral hippocampus. Antidromic latencies to mPFC stimulation ranged from 6 to 20 msec (mean, 11.7 msec, n=19) and those to BLA stimulation ranged from 4 to 17 msec (mean, 9.4 msec, n=19). Approximately 52% of the neurons recorded revealed antidromic responses simultaneously from the mPFC and BLA, and they were found to bifurcate near the soma. These results suggest that the same neurons in the ventral hippocampus project to both the mPFC and BLA.

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P3-265 Effects of hippocampus-prefrontal LTP/LTD induction on cross correlation between hippocampal and prefrontal spontaneous local field potential powers in anesthetized rats

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To investigate whether synaptic plasticity alters the relationship between hippocampus (HP) and prefrontal cortex (PFC) spontaneous local field potentials, we analyzed cross-correlation (CC) of theses local field potential powers (8-100 Hz) before and after the posterior dorsal HP-PFC LTP/LTD induction in vivo. Results showed different changes of the CC in some frequency powers between LTP and LTD inductions. LTP but not LTD increased the ratio of period observed significantly higher CC (significant CC ratio) in 8-15 Hz and lower gamma-band (about 30 Hz) power. LTD but not LTP increased the significant CC ratio in higher gamma-band (about 60 Hz) power. These results indicate LTP/LTD alters the relationship between spontaneous local field potentials. Different direction of synaptic plasticity may be reflected to different frequency-band activity in the spontaneous local field potential.

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P3-266 Sexual Difference of Song Motor Connections in Bengalese Finch Telencepalon

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Song of Bengalese finches is learned motor behavior, which is observed only in adult males. One of the song nuclei, RA is the output site of the telencephalic song control system, and this nucleus sends descending projections that terminate directly on vocal motor neurons and premotor respiratory nuclei in the medulla. The remarkable feature of RA in Bengalese finches is that it is sexually dimorphic, being much larger in males than in females. In this study, BDA retrograde tracing technique was applied in RA of both sexes. In HVC, the higher song nucleus, retrogradely labeled neurons were seen only in males whereas in LMAN, a song learning nuclei, labeled neurons were seen in both sexes. Results suggest that sexual difference in song motor system contributes to sexually dimorphic vocal behavior. (Work supported by JST).

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P3-267 Effect of NO-cGMP signaling in non-associative learning of the earthworm

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Effect of NO-cGMP signaling in non-associative learning of escape reflex of the earthworm was investigated. Repeated tactile stimulus to the body wall (every 5 min) decreased number of action potentials (APs) of the giant fiber, and number of APs was hardly observed within 40-50 min from the onset of tactile stimulus. Relatively excess NO and cGMP treatment using an NO donor NOR3 and a cGMP analogue 8-Br-cGMP, also decreased the number of APs by single tactile stimulus compared with control. Number of APs did not recover within at least 30 min after removing NOR3 or 8-Br-cGMP. These results indicate that cGMP accumulation by NO reduced the responsibility to tactile stimulus, and thus non-associative learning such as habituation in the earthworm is assumedly induced via NO-cGMP signaling.

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P3-268 On-going sequence learning: evidence from event-related potentials study

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To investigate the neural correlates of online sequence learning, we recorded event-related potentials (ERPs) during a serial reaction time task (SRT). Participants responded to a visual stimulus occurring at one of the four locations by pressing buttons that were spatially corresponding. Unbeknownst to them, a recurring loop of 12-item sequence for ten successive repetitions was presented per block. Results show that reaction times (RT) gradually decreased as blocks of repeated sequences progressed and increased when it was switched to a random sequence. ERPs showed larger P2 amplitudes with more learning, and were highly correlated with RT. This study provided firstly neurophysiological evidence for online sequence learning processes. Supported by: PRESTO, Japan Sci & Tech Agency and by a Grant in aid for Scientific Research #16011208.

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P3-269 Noradrenaline transmission in the amygdala enhances memory retrieval of conditioned taste aversion

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Noradrenaline (NA) transmission plays an important role in long-term memory formation. Mice heterozygous for the mutation of the gene encoding tyrosine hydroxylase (TH) impair long-term memory of conditioned learning because of reduced NA metabolism. Here we report the role of NA transmission in modulation of the retrieval process of conditioned taste aversion memory. Administration of a NA uptake inhibitor before the retention test restored memory deficits in the TH mutant mice. Intra-amygdala infusion of the NA uptake inhibitor also restored the memory deficits in the mutants. This restoration was mediated by β- or α1-adrenergic receptors in the amygdala. These results suggest that NA transmission in the amygdala enhances the retrieval process of taste aversion memory.

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P3-270 acetylcholine-dopamine interaction in the ventral hippocampus on memory in rats.

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We previously demonstrated possible involvement of hippocampal dopamine D2 receptor, D_2R, in mnemonic function through interaction with acethylcholine (ACh).To study ACh dopamine interaction in the hippocampus more extensively we examined the effect of hippocampal dopaminergic manipulation on cognitive performance in rats, whose cholinergic systems are compromised by dietary restriction of choline or intrapenitreal injection of scopolamine (cholinergic antagonist). The memory performance of rats fed with diet deficient in choline was deteriorated by the bilateral hippocampal direct injection of raclopride (D_2R antagonist) in passive avoidance task. To the contrary, the bilateral hippocampal direct injection of quipirole reversed the scopolamine induced memory impairment in normally fed rats in eight arm radial maze. These observations suggest that ACh-dopamine interaction in the hippocampus may play a crucial role in mnemonic function.

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P3-271 Systems consolidation requires the post-learning activation of cortical NMDA receptors

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Declarative memory is believed to be reorganized from a hippocampus-dependent to independent one and finally stored somewhere in the neocortex. By using trace eyeblink conditioning, we have revealed that the initial hippocampus-dependent memory becomes medial prefrontal cortex (mPFC)-dependent within 6 weeks after the learning. Therefore, it is possible that a part of the memory trace is consolidated in the mPFC. Here we investigated the involvement of the NMDA receptors in the mPFC in such reorganization by chronic microinfusion of APV with an osmotic pump. Rats that received two-week-blockade of the NMDA receptors in the mPFC showed a marked impairment in 6-week-later memory retention, but relearned with subsequent conditioning. These results suggest that successful formation of remote memory requires activation of the NMDA receptors in the mPFC during the first two weeks of the post-learning period.

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P3-272 A novel gene, *Mahya*, is shared between Hymenoptera and vertebrates

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We identified a novel gene, *Mahya*, which encodes the large secreted protein with Kazal type serine/threonine protease inhibitor domain, EF-hand calcium binding domain, and two immunoglobulin domains from an eusocial insect, honeybee. Intriguingly, *Mahya* homologs are present in the vertebrates but not in two Dipteran species (*Drosophila melanogaster* and *Anopheles gambie*), nematode, and ascidiacea. Two genes homologous to *Mahya* are present in zebrafish, mouse, and human. In mouse brain, *mMahya-1* is highly expressed in the olfactory bulb, cerebellum, and CA3 region of hippocampus, meanwhile *mMahya-2* is expressed in the cortex and CA3 region and dentate gyrus of hippocampus. The relationship between the evolution of *Mahya* and the structures and functions of brains will be discussed.

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P3-273 Effect of reward schedules on monkey dorsolateral prefrontal and orbitofrontal activities

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We examined an effect of reward schedules on delay activity observed in orbitofrontal (OF) and dorsolateral prefrontal (DLPF) neurons. We used oculomotor delayed-response tasks under two reward schedules; in one condition, reward was delivered after every correct performance, whereas in another condition, reward was delivered only after successful completion of 4 consecutive trials. In OF neurons, 82% did not exhibit spatial selectivity in delay activity. But, 92% showed the magnitude modulation of delay activity depending on the proximity to the reward delivery. In DLPF neurons, delay activity showed spatial selectivity and its magnitude was modulated depending on the proximity to the reward delivery. These results indicate that DLPF participates in both cognitive and motivational aspects of the behavioral control whereas OF participates more in motivational aspects of the behavioral control.

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P3-274 Characterization of the relationship between memory reconsolidation and extinction

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Reconsolidation acts to stabilize, whereas extinction tends to weaken, the expression of the original memory. Therefore, memory retrieval may initiate two potentially dissociable but opposite these processes. To understand the relationship between reconsolidation and extinction, we compared biochemical signatures of these processes using contextual fear conditioning and Morris water maze. We found that the presence of three distinct time-dependent phases of memory processing following memory retrieval: While retrieved memory is stable, extending retrieval triggers a reconsolidation process required for the stability of the memory trace and further prolonged retrieval induces the formation of a new "extinction" trace. Importantly, we showed that the temporal dynamics of reconsolidation are dependent on the strength and age of memory. We will also report results showing the roles of CB1 or LVGCCs in these processes.

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P3-275 Neuronal dynamics activated by local glutamate application in CA3 region of hippocampal slices

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The hippocampal CA3 region has been expected to function as an associative memory organized by unsupervised learning. In this study to evaluate this hypothesis, multiple neuronal spikes in the CA3 region of hippocampal slices were recorded with two tetrodes inserted in the stratum pyramidale. Two glass pipettes filled with glutamate were also located neighboring to the tetrodes for the iontophoretic application. Current pulses were applied to the pipettes alternately with the interval of 5s for the test stimuli. For the conditioning stimuli, current pulses were applied to both the pipettes simultaneously with the interval of 10s for 20 times. After the conditioning stimuli, it was found that a part of recorded neurons increased their firing rates, and the other part decreased or unchanged. In addition, some neurons changed firing patters. These results could suggest the change in pattern of synaptic connectivity in the CA3 network after the conditioning stimuli.

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P3-276 Phenotype analysis of HPC-1/syntaxin 1A knock-out mice

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HPC-1/syntaxin 1A is known to regulate the exocytosis of synaptic vesicles. We have produced HPC-1/syntaxin 1A knock-out mice to analyze the phenotype in vivo. Surprisingly, the null mutant mice revealed normal development and basal synaptic transmission in cultured hippocampal neurons appeared to be normal. The brain structures were morphologically normal in null mutant mice compared with control littermate. In order to examine if the mice exhibits behavioral impairment, several behavioral test were carried out. Motility in open field and special memory acquisition in water maze were normal in null mutant mice. Interestingly, the extinction in fear conditioning test was impaired with unaffected fear memory acquisition.

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P3-277 Different contribution of NMDA receptors to the eyeblink conditioning between GluRδ2 mutant mice and wild-type mice

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The mutant mice lacking the glutamate receptor δ2 subunit (GluRδ2) require the hippocampus in short-trace eyeblink conditioning (ST), whereas wild-type mice do not in ST but do in long-trace conditioning (LT). Here we investigated the role of N-methyl-D-aspartate (NMDA) receptors in ST in GluRδ2-/- mice and compared it with that in ST and LT in wild-type mice. Daily injection of the NMDA receptor antagonist (+)MK-801 impaired acquisition of the conditioned response (CR) in ST in GluRδ2-/- mice more severely than in LT in wild-type mice. MK-801 also impaired expression of the pre-acquired CR in ST in GluRδ2-/- mice, but not in wild-type mice. These results suggest that contribution of NMDA receptors to ST in GluRδ2-/- mice is different from that to LT in wild-type mice, although both of them largely depend on the hippocampus. Supported by CREST and SORST, JST.

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P3-278 Neural mechanism of long-term motor learning revealed by different effects of floccular lesion and shutdown on the long-term adaptation of mouse ocular reflex

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Adaptation paradigm of the mouse horizontal optokinetic response (HOKR) eye movement provides an experimental model of cerebellum-dependent motor learning. Our previous study revealed that memory trace of the long-term adaptation (LTA) of HOKR resides outside the flocculus, although flocculus is necessary for the formation of memory trace. In order to further evaluate the role of the flocculus in LTA, we examined the effects of flocculus lesion on the LTA immediately after LTA occurred. Five days of optokinetic training, 1h for each day, induced long-term increase of HOKR gain, which was nearly completely abolished 1h after bilateral acute flocculus thermal lesions. These results were contrasting to the effects of flocculus shutdown which only abolished the gain increase by short-term (1h) training. We propose possible neural mechanisms underlying the memory formation of LTA.

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P3-279 Enhancement of memory consolidation in aged mutant mice overexpressing CaMKIV in forebrain

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Ca^{2+} signaling pathway plays a central role in memory consolidation. CaMKIV has been shown to phospshorylate transcription factor CREB and activate transcription, which is required for memory consolidation. Indeed, CaMKIV KO mice show an impairment of long-term memory. To further understand the roles of CaMKIV in memory consolidation, we have generated and analyzed transgenic mice overexpressing wild type CaMKIV in forebrain region. These transgenic mice show higher expression of 14-3-3 mRNA than wild type mice, suggesting that overexpression of CaMKIV causes the increase in the expression of CREB-target genes. Interestingly, the results of contextual fear conditioning indicated that aged mutant mice shows the significant enhancement of long-term memory while adult mutant mice showed normal memory consolidation. These results suggest that the age dependent impairment of memory consolidation may be rescued by increase in the activity of CaMKIV in forebrain.

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P3-280 Analysis of a role of transcription factor SRF in learning and memory

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Ca^{2+} signaling pathway regulates gene expression that required for memory consolidation. We have reported that CREB functions as a downstream transcription factor of Ca^{2+} signaling and plays an essential role in memory consolidation. To further understand the molecular mechanisms of memory consolidation, we are investigating the roles of Serum Response Factor (SRF) in learning and memory, that is also a major downstream transcription factor of Ca^{2+} signaling. SRF has been known to regulate transcription of immediate early genes, whose expressions are increased after learning. Because mice with a null mutation of SRF gene show embryonic lethal, we generated several transgenic lines that show forebrain-specific expression of dominant negative SRF mutant fused with mutated ligand binding domain of estrogen receptor. In these transgenic mice, the activity of this repressor is regulated by Tamoxifen. We will present the results of analysis of these transgenic mice.

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P3-281 Soft diet feeding after weaning affects sex difference in the acetylcholine release in the hippocampus

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We previously found that acetylcholine (ACh) release in the prefrontal cortex showed a sex difference and soft diet feeding after weaning affected it. In the present study, to examine the sex difference in the ACh release in the hippocampus and the effect of soft diet feeding after weaning, in vivo microdialysis study was performed in male and female rats under the freely moving condition. Both male and female rats showed a daily rhythm of ACh release, which was high during the dark phase but low during the light phase. In the standard dietary condition, male rats showed significantly greater ACh release than female rats in both light and dark phases. In the soft dietary condition, both sexes of rats showed ACh release which was significantly increased, showing no sex difference. We found a marked sex difference in the ACh release in the hippocampus, but soft diet feeding affected this difference in rats.

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P3-282 Single neuronal responses related to reward expectancy in insular cortex

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We previously showed that single neurons in rhesus monkey insular cortex have response selectively in multi-trial reward schedules, a task in which monkeys must complete schedules of 1, 2, 3 or 4 visual color discriminations to earn a juice reward. Here we summarize selectivity patterns in 147/286 neurons with task-related responses. 102/147 responded at reward delivery; 54/102 started responding before the reward, but late in the trial. 90/147 neurons had one of three reward contingent response patterns in the schedule: responses in (1) rewarded trials only, (2) non-rewarded trials, (3) all trials with the response strength differing categorically between rewarded and non-rewarded trials. Thus insular neurons appear sensitive to expectancy of immediate reward, a result consistent with imaging results suggesting that insular cortex plays a role in predicting immediate reward.

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P3-283 CREB-dependent memory localizes in the mushroom bodies in *Drosophila* Larvae

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The fruit fly *Drosophila melanogaster* has been used successfully as a model animal for the study of learning and memory because of its powerful genetics. Apart from the adult brain, the larval brain has recently attracted much interest because of its simple organization, which would allow us to identify neural networks involved in complex brain functions with better resolution. Here, we show that larval memory localizes in the mushroom bodies (MBs). With appetitive olfactory learning paradigm, wild type larvae are able to associate a given odor with reward of sucrose. We found that larval appetitive memory persists beyond two hours and depends on CREB function. By expressing shi^{ts1} in MBs, we found that output activity of MB neurons is required for the retrieval of both CREB-dependent and independent memory in the larval brain.

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P3-284 NMDA and muscarinic blockade in the perirhinal cortex impairs object discrimination and recognition in rats

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To determine the possible involvement of *N*-methyl-D-aspartate (NMDA) and muscarinic activation of the perirhinal cortex in object discrimination and recognition, an NMDA antagonist, DL-2-amino-5-phosphonopentanoic acid (AP5), and a muscarinic antagonist, scopolamine (SCP) were injected into the perirhinal cortex of rats. In object discrimination tasks, each drug at the higher dose (AP5 60 mM, SCP 80 mM) significantly decreased correct choices on the retention test. In spontaneous object recognition tasks, a high dose of AP5 (60 mM) and two doses of scopolamine (20 and 80 mM), but not a low dose of AP5 (30 mM) alone, significantly impaired discrimination between novel and familiar objects. These results suggest that activation of both NMDA and muscarinic receptors in the perirhinal cortex contributes to object discrimination and recognition.

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P3-285 The neural projection from the amygdala to the hippocampus studied with an optical recording method

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The amygdala, which plays an important role in emotional behaviors, is known to form neural connections with the hippocampus and the surrounding cortices. This indicates that the neural activity of the amygdala has an influence on one of the main paths that plays a crucial role in memory process. The aim of this study is to investigate the manner of the neural projection from the amygdala to the neural circuit in the hippocampal formation, and also to investigate the physiological effect on the neural processing in the circuit. In this study the rat brain slices were used. The lateral nucleus of the amygdala was stimulated electrically, and the following propagation of neural activity in the brain slice was recorded with a high-speed optical recording technique. We also investigated the change in the activation pattern of the try-synaptic circuit in the hippocampal formation when a part of the circuit receives the amygdala input.

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P3-286 Testosterone maintains daily acetylcholine release in the hippocampus in male rats

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We reported a marked sex difference in the daily acetylcholine (ACh) release in the hippocampus. In the present study, to examine the role of testosterone in the daily ACh release in the hippocampus, in vivo microdialysis study was performed for more than 24 hours in intact, orchidectomized, and orchidectomized testosterone-primed male rats. Spontaneous locomotor activity was simultaneously monitored throughout the experiment. ACh release during the dark phase was significantly greater than during the light phase in intact male rats, but orchidectomy severely attenuated the ACh release duing the dark phase. Testosterone replacement in orchidectomized rats successfully ameliorated the ACh release during the dark phase. Spontaneous locomotor activity showed a distinct daily rhythm, but no significant difference was observed among groups. Present results suggest an essential role of circulating testosterone to maintain the ACh release in the hippocampus in male rats.

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P3-287 Neuronal activities of rat nucleus accumbens during eight-arms maze working memory task

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We measured neuronal activities of rat nucleus accumbens using multi-unit chronical recording technique in eight-arms maze working memory task to clarify difference of its contribution for memory guided and non-guided behaviors. This task consists of 2 sub-sessions. In the first sub-session, training session, each rat has to memorize the position of arms gated randomly. In the next sub-session, test session, it visits selectively the arms which have been gated in the prior session. We found neurons which show activities especially related to the sub-phase in each sessions such as "pre food-seeking period" or "food-seeking period". Furthermore, some neurons of nucleus accumbens changed the pattern of neuronal activities between the training session and the test session which requires calling guide memories. Based on these results, we will discuss difference between the shell and core of nucleus accumbens in these behaviors.

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P3-288 Taste aversive conditioning in *Lymnaea*

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A sucrose stimulus applied to the lips of *Lymnaea* results in a feeding response; while a tactile stimulus to the head results in the cessation of feeding. We asked whether following pairing of a sucrose stimulus to the lips as the conditional stimulus (CS) with a tactile stimulus to the head as the unconditional stimulus (UCS) if sucrose was still effective in eliciting feeding. Control cohorts received either backward conditioning (i.e. the tactile stimulus to the head was preceded the sucrose to the lips) or repeated application of water or sucrose to the lips. We found that following conditioning sucrose (the CS) was significantly less likely to elicit a feeding response compared to its ability before conditioning and compared to the ability of sucrose to elicit feeding before and after training in the control groups. Thus snails learned that sucrose signaled an upcoming aversive stimulus and it no longer activated feeding. We further found that this learning could be consolidated into a memory that persisted for at least 24h.

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P3-289 Cortical terminations of hippocampal projections from area CA1 in macaque monkeys

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Recent neuroanatomical studies have demonstrated that temporo-frontal neocortex receives hippocampal inputs originating from area CA1 as well as from the subiculum. In order to establish the areal and laminar distribution of axon terminals projecting from the CA1, the anterograde tracers Biotinylated Dextran Amine and Fluoro-Ruby were injected iontophoretically into area CA1 of macaque monkeys. Cortical recipient zones were areas 28, TF, TH, 35, 36, TEv and TG of the temporal cortex and areas 13,14, 25 and 32 of the prefrontal cortex. Labeled terminals in area 28 were distributed in layer V with some in layers II-III. In contrast, in other cortical areas, they were located mainly in layers II-III and V with some few in layer I. These patterns of termination suggest that CA1 cortical projections might be considered similar to 'feedback' connections, but that these contribute to the microcircuitry in an area-specific fashion.

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P3-290 Population activities related to contextual learning and coding in rat hippoocampal formation

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We visualized population activity in hippocampal formation of animals exposed to a novel environment for 5 minutes, 10 minutes and 24 hours by immunostaining of activated-form of MAP kinase. Among components of hippocampal formation, CA1, subiculum and lateral entorhinal cortex (LEC) showed intensive population activity with the temporal-portion-biased distribution along their septo-temporal axes. Although this spatial distribution of activity did not differ in any time point, the intensity of the population activity showed time-dependent changes. The intensity of population activity in CA1, subiculum and LEC superficial layers was rapidly enhanced by exposure to a novel environment, and in 10 minutes decreased to the level that was maintained for at least 24 hours. These results suggest that the rapidly decaying population supports formation of an internal representation of context, and the maintained population relates coding of context.

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P3-291 Effects of SKF38393 (D1 receptor agonist) injection on inhibition of Ubiquitin-activating enzyme (Ube1x) mRNA induced by methamphetamine (MAP) administration in rat vermis

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Administeration of MAP (1 mg/kg, every 3 days 5 times, rMAP group) induced hyperlocomotion. Daily injections of the SKF (1 mg/kg, 1 week, rMAP+SKF group) reversed the hyperlocomotion. We examined if the SKF reversed the MAP effects on Ube1x mRNA expression by in situ hybridization (35S labelled oligo probe). Golgi cells were covered with silver grains. There were small patches of Ube1x mRNA in rMAP group. The O.D.measurement of autoradiographic film showed that the mRNA level in rMAP group was lower and the number of the patch was higher than in control or rMAP+SKF group. We conclude that Ube1x mRNA level decreased with increase its number of patch after the MAP injections, which were reversed by the SKF injections.

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P3-292 Memory of object-place association and object relocation formed as a hierarchical associative network with theta phase coding

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Our memory on objects and their places in a natural environment can be obtained instantaneously and allows frequent changes in objects' locations. The neural mechanism to enable memory storage of such an environment is an open question. We hypothesized that the neural dynamics of "theta phase precession" observed in rat hippocampus should contribute to the memory for object-place associations owing to its robustness in one-trial memory encoding. Our computer experiments demonstrated that the object-place associations can be instantaneously stored in the hippocampus as a hierarchical associative network, where object-relocation is stored by partial rewiring of the associative network. Our results indicate that the ability to transform spatial scales of inputs to temporal scales of theta phase coding gives an advantage in the memory of natural environment.

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P3-293 Dopaminergic inputs to the rat auditory cortex required for sound sequence discrimination learning

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We have reported that sound sequence discrimination learning, which is driven by reward, is dependent on D2-like dopamine receptors in the rat. In the present study, we investigated the responsible targets of dopaminergic inputs in the brain. Rats were trained to discriminate the sequences of two sound components, and licking a spout during a particular sequence was rewarded with water. Percentage of trials in which rats licked the spout was calculated separately for the rewarded and unrewarded sequence, and test performance was estimated as the difference. 6-hydroxydopamine (6-OHDA) lesions of the auditory cortex specifically attenuated the sound sequence discrimination learning, while it has no clear effect on the discrimination between the two sound components. These findings indicate that dopaminergic inputs to the auditory cortex are critical for sound sequence discrimination learning with reward.

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P3-294 A double dissociation within prefrontal-striatal circuits between win-shift/stay radial maze performance and retention of intra-maze cue in rats

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Recent studies have reported that lesions with the prefrontal cortex and the striatum induce working memory deficits in radial arm maze, in which normal rats usually use win-shift strategy more dominantly than win-stay strategy. The present study investigated the role of prefrontal-striatal circuits during performance of additional intra-maze cue version of the delayed win-shift/stay task using asymmetrical lidocaine-induced reversible disconnection. In win-shift task, this disconnection impaired the rank of choice of the cued arm but not correct performance. On the other hand, in win-stay task, the disconnection impaired both correct performance and the rank of choice of the cued arm. Results suggest that the prefrontal-striatal circuits are involved in efficient navigation through retention of additional cue, behavioral flexibility, or more cognitive aspects.

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P3-295 Conditioned stimulus and amygdalic amino acids and dopamine

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Neurons in the amygdala, considered an important area for memory storage in fear learning, are a site of interaction between several monoaminergic and aminergic neurotransmitters in the memory process of Pavlovian conditioned fear. This study was designed to test whether, in the amygdala, conditioned fear stress changes extracellular levels of glutamate, glycine, taurine and dopamine, using an in vivo microdialysis technique at each instance of memory acquisition and memory retention, in the same rats. Extracellular DA levels increased each time a memory was acquired and retained. Extracellular glutamate levels increased in the amygdala upon acquisition of the fear memory, but not with retention of the memory. There were no significant changes in other amino acids with either acquisition or retention. These findings indicate that dopaminergic and aminergic neurons in the amygdala may play different roles in the neuronal responses of Pavlovian fear stress.

P3-296 The microinjections of GABA$_A$ receptor antagonist into the ventral pallidum attenuate the expression of conditioned taste aversion in rats

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To elucidate a possible role of the ventral pallidum (VP) in the expression of conditioned taste aversion (CTA), we examined the effects of microinfusions of a GABA$_A$ receptor antagonist bicuculline into the VP in rats. Initially, rats received a pairing of 0.1% saccharin with an i.p. injection of 0.15 M lithium chloride. After this conditioning, vehicle or 200ng bicuculline was bilaterally infused into the VP immediately before the presentation of saccharin as an extinction test. The infusions of bicuculline significantly increased the saccharin intake. On the other hand, the same infusions significantly decreased the intake of aversive 0.3mM quinine hydrochloride (QHCl). These results suggest that the GABA$_A$ receptors in the VP are involved in the intake of taste solutions. However, the neural mechanisms that regulate the CS intake seem to be different from those that regulate the intake of normally aversive QHCl.

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P3-297 Neuronal property in the hippocampal CA1 area during the same behavioral context by memory retrieved and visual guided performances in the rat

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Neuronal activities of dorsal hippocampal CA1 region were examined by twelve chronically implanted tetrodes during the spatial simple alternation and the visual guided behaviors whose behavioral contexts were the same. From the firing rate analysis, some CA1 pyramidal neurons showed task-related differential activity between around the left and right nose poking only in the simple alternation task. However, neurons which showed such task-related activity in the visual guided task were always task-related in the simple alternation. These result suggested that there were two kinds of task-related neurons in the simple alternation task, one might response to right-left alternative behavioral context itself, the other might concern to a short-term memory retrieve process.

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P3-298 Functional differences between primate prefrontal and frontal eye field neurons in a memory-guided sensorimotor transformation

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Transformation of sensory memory to a motor command is a critical process in cognitive motor control. We have demonstrated that the lateral prefrontal cortex (lPFC) is involved in this process for goal-directed saccades. Here, we hypothesized that the frontal eye field (FEF) receives memory-guided saccade command from the lPFC. To approach this issue, we compared neuronal activities in both areas of monkeys using a delayed anti-saccade paradigm, in which a saccade-rule was presented during a delay period. 90 lPFC and 46 FEF neurons, which showed directional delay-period activities, shifted their representation from sensation to motion after the rule onset. The shifting was faster in the lPFC (466 ms) than the FEF (1434 ms), and the shift-related activity was prominent in the lPFC. These results suggest that the FEF neurons receive directly/indirectly the memory-guided motor command generated in the lPFC based on short-term and long-term memories, thereby executing memory-guided saccade.

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P3-299 Phase Shift of Theta Oscillation in Hippocampal CA1 Pyramidal Cell Membrane with the Stimuli to the Shaffer Collaterals

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Phase precession is thought to represent one of the examples of temporal coding. It, however, remains to be determined what mechanisms induce the phenomenon. We focus on the oscillation at theta frequency in hippocampal pyramidal cell membrane during theta activity in vivo. In this study, we investigate the effect of synaptic inputs to the phase shift of theta oscillation in cell membrane. Whole-cell patch clamp recordings from the CA1 pyramidal cells located in hippocampal slices of rats were used to examine the potential mechanisms of phase precession. To mimic a membrane potential oscillation, hyperpolarizing 5-Hz sine wave current ware injected into the CA1 pyramidal cells. An electrode was placed at the stratum radiatum to stimulate excitatory synaptic inputs. We show that the synaptic inputs induce the phase shift of the membrane oscillation, whereas the increases of inputs dose not induce gradual phase advancement.

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P3-300 Effects of local injection of AP5 and SCH23390 into the prefrontal cortex on radial arm maze performance in rats

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In order to investigate the role of rat medial prefrontal cortex (mPFC) in spatial working memory, we used standard and delay-interposed radial maze tasks. We examined effects of N-methyl-D-aspartate (NMDA) and dopamine D$_1$ receptor blockade in mPFC on performance of these tasks once acquired. Neither NMDA antagonist AP5 nor D$_1$ antagonist SCH23390 impaired performance of the standard task. In the delay-interposed task, one trial was divided into two phases by 2 h delay. Drugs were injected at one of three timings to assess the involvement in memory processes: before the first-half performance (timing I), after the first-half performance (timing II), or before the second-half performance (timing III). The second-half performance was disrupted by AP5 treatment in timing III, while it was not affected by SCH23390 in all injection timings. These results suggest that activation of NMDA but not D$_1$ receptor in mPFC is involved in retrieval process of spatial working memory.

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P3-301 Backward sensory preconditioning in the terrestrial mollusc, *Limax marginatus*

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It has been believed that learning process includes integration of old memories and a new experience. Since many species of animals can learn not only value of objects but also relationships between objects, it is interesting to study how a new value-learning experience is integrated with related memories. Backward sensory preconditioning (BSPC) is suitable to research such problem in classical conditioning paradigm. Here we studied BSPC in the terrestrial slug, *Limax marginatus*, with vegetable odors as the conditioned stimuli (CSs) and quinidine sulfate as the unconditioned stimulus (US). When slugs experienced CS2-CS1 and CS2-US training pairs, their odor preference for CS1 was reduced as well as that for CS2, showing BSPC. This result indicates that the value of retrieved CS1 has changed during CS2-US training, suggesting even a slug has an ability to make consistent memory network.

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P3-302 Change in brain activity during arbitrary auditory-visual association learning as revealed by functional MRI

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To examine the plasticity of neural representation during learning, we conducted functional MRI with auditory-visual stimulus-stimulus paired association learning of delayed-matching-to-sample type task (with 1st stimulus followed by delay period, 2nd stimulus, and feedback to response). As learning proceeded, enhanced was the cross-modal responses of the unimodal areas in the delay period, suggesting that the representation of the paired associates include unimodal visual and auditory areas. In contrast, the polymodal STS was activated by the 2nd stimulus and feedback, maximally in early phase, decreased as learning proceeded. This finding implies that the STS may play a key role in generating the neural networks representing the audio-visual paired association.

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P3-303 Effect of pseudo-microgravity environment on acquisition and retention of mouse conditioned taste memory

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We examined the effect of gravitational force on acquisition and retention of the conditioned taste aversion (CTA) paradigm as a model of emotional learning and memory. The pseudo-microgravity (PMG) environment was applied between 0.1% sodium saccharin as a conditioned stimulus (CS) and an i.p. injection of 0.15 M LiCl as an unconditioned stimulus (US) using 3D clinostat to cancel the gravitational force. Although PMG condition did not serve as US, it suppressed both acquisition and retention of CTA memory. Furthermore, after CTA acquisition with the PMG environment, expression of an immediately early gene, c-Fos was greatly suppressed in the external lateral subnucleus of parabrachial nucleus, which strongly appeared under control condition. These results suggest that the PMG force impairs CTA memory formation and consolidation through suppression of neuronal plasticity in the limbic system as well as in the gustatory pathways of the brain stem.

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P3-304 Dopamine release time course of the mesocortical system during multiple spatial working memory processing

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It is suggested that dopamine (DA) controls fundamental cognitive operations (Tanaka, 2002). In addition, Yamashita and Tanaka (2003) have shown that the cortico-mesocortical system forms a 'regulator' of DA release. However, there is a window in which this regulator works properly (Tanaka, 2003). We investigate, with a newly developed cortico-mesocortical loop model, how DA is released during the processing of multiple spatial working memories. The simulation shows that the DA release level changes widely when the locations of multiple targets are memorized. Nevertheless, it is possible to stabilize the DA release. Our simulation suggests that the gain of cortical feedback should be low to attain the stabilization. The cortico-mesocortical long loop thus works as a reliable regulator for the DA release in the prefrontal cortex during spatial working memory processing.

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P3-305 Difference in firing synchronicity of task-related neurons between primary and secondary gustatory cortices in monkey

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Multiunit activities were recorded from both area G, the primary gustatory area, and the orbitofrontal cortex (OFA), the secondary gustatory area, during a taste discrimination GO/NOGO task in monkeys to examine firing synchronicity of cue-related neuron pairs. A larger fraction of neurons also responded to other events, e.g., LED, reward, or lever pressing in the OFA than in area G. Many OFA neurons produced inhibitory responses. Correlation of synchronicity with discharge rate revealed that, in area G, when both components of neuron pairs had excitatory cue responses, firing synchronicity changed in a high rate after cue compared to that at a pre-cue period. Whereas, in the OFA, the rate of synchronicity changes was low with neuron pairs having excitatory cue responses, but high when only one component produced excitatory responses or both components yielded inhibitory ones. It is suggested that the OFA receives various inputs and has complex processing mechanism.

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P3-306 Cortical Magnetic Responses to Musical Tones: Comparison between Musicians and Non-Musicians

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We recorded neuromagnetic (MEG) responses from 10 musicians who had started piano lessons before age of 7 and 10 non-musicians who did not have any experience of music training. Stimuli were 19 single piano tones and 12 major chords (duration 625 ms). Five identical tones/chords were successively generated (total duration 3125 ms) and these sequences were presented at offset-to-onset intervals of 1000-1050 ms. MEG responses were recorded using a 204-channel whole head system. Two magnetic components (N1m and P2m) were observed following each stimulus tone/chord in both hemispheres. For the 2nd to the 5th sounds of both piano tone and chord, the N1m was largely reduced in a way that the P2m was larger than the N1m in amplitude, where the P2m amplitude was significantly larger in musicians than non-musicians. This finding provides evidence that early music training affects neural activities for musical sounds.

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P3-307 Brain activation during the sentence comprehension: an fMRI study

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The purpose of this study was to compare activated regions related to SOV sentence with OSV sentence by fMRI. In this study, we divided each Japanese SOV and OSV sentence into the *subject*, the *object* and the *transitive verb* and presented Japanese participants with each part one by one at regular intervals. The participants read the presented sentence silently. As a result, *subject* of SOV sentence activated the same region as *object* of OSV sentence. *Object* of SOV sentence activated the same region as *subject* of OSV sentence. SOV sentence and OSV sentence were different in regions activated when *transitive verb* was presented. Therefore, brain activation was defined depending on the order of recognition, but grammatical functions like *subject* did not affect brain activation very much until the *transitive verb* was presented.

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P3-308 Predictive action planning in the striatum based on the history of rewarding but not aversive events.

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Animals plan behaviors by predicting future rewards or penalty based on the history of these reinforcements. To study how the striatum is involved in reward- and aversion-based action planning, we recorded activity of projection neurons in the striatum of 2 monkeys performing reaction time task for lever release with outcomes of either reward water or aversive airpuff on their face or beep sound. Monkeys initiated the task trials by pressing a lever more swiftly when reward outcome was expected at higher probabilities based on an outcome history in the past several trials. Of 89 task-related neurons, 43 showed activity depending on the occurrence of reward in the preceding trial, and activity of some of them was either positively or negatively correlated with reward probability estimated from previous trials. Only 10 showed activity depending on the aversion history in the previous trials. These results suggested that the dorsal striatum participate in predictive action planning based on the reward history.

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P3-309 Representation of immediate and final behavior goals in the prefrontal cortex

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To investigate the involvement of the prefrontal cortex (PFC) in multi-step behavioral planning, we recorded neuronal activity from the PFC while monkeys were performing a path-planning task in a maze. In this task, the animals were required to move a cursor on the screen stepwise from a start to a goal, avoiding obstacles, by controlling two manipulanda with each arm. We found that a majority of neurons showed activity-changes related to final or immediate goal. In contrast, neurons rarely showed significant relation to arm movements. We also found that, during a delay preceding the first step of actions, neuronal activity representing the final goal appeared first, followed by the representation of the immediate goal. Our data demonstrated that the PFC was involved in processing information of the final goal to generate information for the immediate goal. This functional property of subgoal formation is entirely different from sensory-motor transformation.

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P3-310 Purposeful meanings of behaviors coded by parietal neurons

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We investigated activities of neurons in area 7b of parietal cortex to find out if they could code meanings of behaviors which were purposefully linked to the visual stimuli and monkeys' hand movements. We trained monkeys to several serial processes consisted of monkeys' hand manipulations and experimenter's actions. In each process, the monkey was required to execute several kinds of hand manipulations and to observe experimenter's actions. We found a group of neurons in area 7b, which code meanings of behaviors linked to the monkey's hand manipulation and experimenter's action. For example, some of these neurons discharged when the monkey could expect to gain foods by observing the experimenter's action, and when they execute the hand manipulation aiming at gains. Present observation would indicate that neurons in area 7b code particular purposeful meanings of behavior and the selectivity is fixed to a function of the required behavior.

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P3-311 Changes of functional connectivity between the superior parietal and prefrontal cortex during a spatial coding task: a fMRI study

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The spatial coding of an object location can be represented in the human brain with respect to different types of reference frames, either relative to or independent of the body center. We used MRI to identify cortical regions of the human brain subserving mainly body-centered and object-centered reference frame by asking subjects to judge the location of a visual stimulus with respect to either their body or object center. We identified the common activation to body- and object-centered task in right superior parietal lobule and middle frontal gyrus. We estimated the functional connectivity between these areas; the parietal and frontal signal changes were more strongly correlated during the body-centered task condition. The results suggest that body-centered and object-centered spatial coding process share common neural basis in the parieto-frontal network; the connectivity of this network can be modulated by the types of reference frame.

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P3-312 Noradrenergic control of multi-target spatial working memory

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Previous studies have found that the locus coeruleus (LC)-noradrenergic system plays a critical role in the modulation of the working memory-related activity of the prefrontal cortex (PFC). We developed a computational model of the LC and PFC network to examine the influence of LC activity (firing mode) on spatial working memory. The model network is composed of single-compartment leaky integrate-and-fire neurons. The model assumes that norepinephrine(NE)-induced modulatory effects predominantly appear on the inhibitory interneurons, providing the regulation of the memory fields of PFC neurons. Our simulation shows the synergetic control of multi-target spatial working memory by the PFC-LC network. Dysfunctional model LC-NE system implicates aspects of psychiatric disorders such as attention-deficit hyperactivity disorder (ADHD) and stress disorder.

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P3-313 Neuronal activity for duration discrimination in monkey prefrontal cortex

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We have studied single-neuron activity in prefrontal cortex (PFC) while a macaque monkey performs a duration-discrimination task. In the task, two visual cues (C1 and C2; a blue- or red-colored square for each) were successively displayed for different durations from each other. Each cue (C1 or C2) was followed by a delay period (D1 or D2, respectively). After D2, two cues C1 and C2 were simultaneously appeared. Then, the animals were rewarded if they chose a longer one. We found neurons that showed significantly different activity during C1, D1 and /or C2 between C1-longer trials and C2-longer trials. For example, a group of neurons showed larger activity after a nearly constant time-span from an onset of C1 during C1 in C1-longer trials, and also showed larger activity during D1 in C2-longer trials. The results suggests that the neurons in the PFC are involved in estimating whether the cue is longer or not during the cue period, and also in storing whether the cue is longer or shorter during the delay period.

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P3-314 Retrospective monitoring of behavioral context in monkey prefrontal neurons

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For purposeful behavior, it is important to monitor and manipulate prior events / context such as preceding behavioral response, its outcome, and information that guided the response. Dorsolateral prefrontal cortex (DLPFC) is likely to be involved in such a "retrospective monitoring" of prior events. To test this hypothesis, we examined neuronal activity in the DLPFC of monkeys performing memory- and sensory-guided saccade tasks with two reward conditions. We found that post-response activity of a subset of DLPFC neurons was modulated by three factors of earlier events: direction of immediately preceding response, its outcome (reward / nonreward), and the information (memory / sensory) that guided the response. Such neuronal coding should play a role in associating response-outcome with information or neural systems that guided the response, that is, "retrospective monitoring" of behavioral context or neural systems, thereby contributing to context-dependent flexible control of behaviors.

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P3-315 Activation of the left anterior insula in speech

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Brain activity was measured from 17 subjects during speech using fMRI to investigate task-depending activities of the insula. In speech condition the subjects produced CVCVCV syllables in a gentle voice. In inner speech condition they performed the task with no articulation. The subjects performed two sessions; in "Repeating" session, they repeated the same syllable sequence, while in "Random" session they performed three-syllable sequence in different orders in the both conditions. In the result, activation in the left anterior insula was detected only in Random session. The tasks in the two sessions differ in the process before the motor execution. In Repeating session, phonetic encoding and motor programming are set in only at the first utterance, while in Random session encoding and motor programming are done at every execution. This infers that the left anterior insula is involved in both encoding phonetic plans and motor programming before speech production. This research was supported by the National Institute of Information and Communications Technology.

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P3-316 Cognitive functions of thalamocortical neurons with nigral input projecting to prefrontal cortex in monkeys

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Two Japanese monkeys were trained by a go/no-go discrimination task; Cue (S0), green or red LED, was illuminated and, after delay period, either of the two colors was presented as an imperative stimulus (S1). If S1 was the same color as S0, the monkeys were required to lift a lever (go trial). If S1 was a different color from S0, they were required to withhold the response (no-go trial). After training session, thalamic relay neurons with nigral input projecting to prefrontal cortex were identified, and then unit activities of the thalamic neurons were examined during task performance. A high proportion of MD neurons with inhibitory input from SNr projecting to prefrontal area ventral to PS (PSv) changed the activity related to S1 rather than to S0, most of which responded differently to go and no-go trials. The results suggest that the SNr-MD-PSv pathway plays an important role in recognition of the behavioral meaning of visual signal.

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P3-317 The role of GABAergic inhibiton in suppressing perseverative responses in the monkey prefrontal cortex

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To examine the role of GABAergic inhibition in the dorsolateral prefrontal cortex (dlPFC) in suppressing perseverative responses, we locally injected a $GABA_A$ receptor antagonist, BMI, into the dlPFC of monkeys performing an oculomotor delayed-response task. BMI injection induced misdirected saccades for contralateral targets. Some of misdirected saccades went towards the direction which had been cued and/or to which the saccade had been made in the immediately preceding trial. This "perseverative" saccade occurred more frequently than would be expected by chance. It was followed by a second saccade towards current target, indicating that the monkey could remember correct target location. These findings suggest that GABAergic inhibition in the dlPFC plays an important role in suppressing perseverative responses. Thus, GABAergic inhibition in the dlPFC may be involved in attentional control, i.e., suppressing interferences from the prior trial.

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P3-318 Cognitive calibration by periodically altering static electricity stimulus

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Periodically altering static electricity stimulus may enhance human cognition. When parts of our body are electrically charged by the stimulus, respiration motor area detects its electricity and fires inspiration motor neurons, and inspiration is reinforced. This dynamical process enhances our unconscious proper oxygen supply and effective cognition. Lack of electric stimulus coherence induces droop of inspiration phase and hinders balanced native respiration and cognitive neuronal activities. We may have inherent evaluating value for respiration balance i.e. amygdala receptivity for hypoxia and hypercapnia so called homunculus. The value may be stored in long-term memory but one after another control order for inspiration-expiration switching pace may be in short-term memory. Exposure of periodic electric field plays a role for respiration pace calibration, which is hard to be manipulated with conscious. We may try to think, read, and hear something, but in vein memorization is insufficient without that calibration. Lasting of this situation may cause stress, learning deficiency, and mental disorder.

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P3-319 A neural basis for switching action plans in the thalamo-striatal system

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Facing an unexpected situation, we switch planned actions to alternatives. Previously, we showed that neurons in the centromedian (CM) nucleus of thalamus (n=30) are specifically activated when monkeys switch from reward-expected action to unexpected one. We sought mechanisms for the action switch in the striatum by recording neuron activity of putamen that receives CM inputs. A Japanese monkey was trained to perform an asymmetrically rewarded GO-NOGO button press task. Longer reaction times in small-reward GO signal than in large-reward ones indicated that monkey switched planned action. Some putamen neurons (5/13) showed activation at small-reward NOGO trials (4/5) while monkey was expecting large-reward GO request or at small-reward GO trials (1/5) while expecting large-reward NOGO request. This was in contrast to CM neurons that were activated after small-reward request independent of action type. The results suggested that unwanted-event-driven switch signal in CM is integrated into action-specific signal in the striatum.

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P3-320 Dopamine neurons encode uncertainty of reward and of decision

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Fiorillo and Schultz (2003) reported that dopamine (DA) neurons discretely code reward probability and uncertainty in the classical conditioning paradigm. We trained a Japanese monkey to perform a decision making task to find a correct target among three alternatives by trial and error. Average reward probabilities of first (N1), second (N2) and third (N3) choices were 31, 47 and 79% respectively. Once a correct target was hit, monkey got water reward by choosing the same target in a succeeding trial (R1) at 93% probability. Activity of 31 DA neurons was recorded. Neurons responded to start cue (24/31) and/or reinforcer beep (30/31) after choice in each trial. Responses to beep represented reward expectation errors. The start cue responses were small in N1 and R1, larger in N2 and largest in N3. With larger magnitude of reward, start cue responses increased in N2 and N3 but not N1 and R1. The results suggest that start cue responses of DA neurons code reward and decision uncertainty.

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P3-321 A shared network of cortical regions involved in a first and second language comprehension

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We carried out an fMRI study to investigate whether the same brain regions process first and second languages. Ten right-handed Japanese native speakers participated in this study. Subjects were instructed to read active and passive sentences in Japanese or English, and to judge the plausibility of those sentences. As a result, the comprehension of passive sentences showed greater activation in the left middle and inferior frontal gyri than that of active sentences in both Japanese and English. These results suggest that a shared network of cortical regions processes first and second languages.

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P3-322 Information processing in the primate mediodorsal thalamic nucleus: population vector analysis

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To examine how spatial information is processed in the thalamic mediodorsal nucleus (MD), we analyzed MD neurons activity recorded while monkeys performed 2 oculomotor delayed-response tasks (ODR and R-ODR). In the ODR task, monkeys were required to make a memory-guided saccade to the location where a visual cue was presented, whereas in the R-ODR task, they were required to make a memory-guided saccade 90° clockwise from the cue direction. Using a population of MD activities, we calculated population vectors (PV) during the 200 ms period along the delay period. We found that the directions of PV were maintained along the delay period in the ODR task, while the directions of PV rotated gradually from the visual cue direction to the saccade direction in the R-ODR task. This result indicates that MD participates in sensory-motor information processing.

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P3-323 Analysis and modelling of neuronal activation in the monkey cingulate cortex during decision processes

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Some neurons show trial-average activities that gradually increase with time at a nearly constant rate during decision processes. Such activities probably participate in integration of evidence, on the basis of which a decision will be made. However, little is known about the time course of single-trial activity itself. To examine this, we devised an analysis method. Spike data recorded in the anterior cingulate area of the monkey performing delayed conditional Go/No-go discrimination tasks (Isomura et al., 2003) were investigated by this method. Results are consistent with the idea that: Each neuron is bimodal, residing in the high- or low-firing state; the timing of transition from one to the other uniformly fluctuates across trials. We also put forward a computational model that accounts for such single-trial activity. Supported by CREST/JST.

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P3-324 Temporal order judgment of three stimuli delivered to unimanual fingers

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We examined temporal order judgment of three successive stimuli, delivered one to unimanual three fingers (2nd, 3rd and 4th). In judging the order of three successive taps, separated by inter-stimulus intervals of 100 ms, the probability of correct judgment depended on the order of stimuli: each subject yielded the highest probability of correct judgment to a particular order of stimuli (e.g. 2-3-4). When the hand was flipped from the palm-down to the palm-up position, the preferred order generally changed in terms of the fingers (e.g. from 2-3-4 to 4-3-2) but remained unchanged in terms of the order in space (left-middle-right). When the palm was placed in a vertical position, the preferred order was no longer evident. These results suggest that subjects cannot be basing their judgments solely on the locations of stimuli on the body surface, but rather on their locations in space.

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P3-325 Involvement of the monkey lateral prefrontal cortex in active suppression of gaze shift: a local inactivation study with muscimol

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Active suppression of gaze shift according to a given situation is important for the appropriate behavioral control. To examine a possible involvement of the lateral prefrontal cortex (LPFC) in such a function, we locally injected muscimol at various sites in the LPFC of a monkey that performed an oculomotor Go/No-Go (OGNG) task. This task required the subject to shift (Go) or suppress (No-Go) its gaze toward a target location, according to a visual instruction. The local injection of muscimol into several sites of the LPFC induced significant increase in false saccades toward a few specific target locations during No-Go trials, whereas no errors appeared in Go trials. These results suggest that the LPFC is essential for active suppression of gaze shift to a specific target location, which expands the previous idea of inhibitory control of behavior by the LPFC.

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P3-326 Prefrontal activity related to time coding

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To examine where and how we deal with time in the brain, Japanese monkeys were trained to perform a time-reproducing task. Single unit activity of prefrontal neurons were recorded from area 9 while the monkeys tried to discriminate exactly between 1 and 5 sec according to instruction. On a GO-signal, the monkeys had to wait for 1 or 5 sec with a 15% margin before leaving a hold key and pressing a button. We found that cells in area 9 were active in response to an instruction cue. In both 1-sec and 5-sec trials, a number of area 9 cells displayed the peak activity around 1 sec after the onset of the cue. The activity lasted longer in the 5-sec trial than in the 1-sec trial. The present results suggest that area 9 may be involved in time coding.

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P3-327 Involvement of dopamine and N-methyl-D-aspartate receptors in methamphetamine-induced cognitive impairment

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We investigated the role of dopamine (DA) and N-methyl-D-aspartate (NMDA) receptors in methamphethamine (METH)-induced cognitive impairment in the novel object recognition task. Repeated METH treatment (1 mg/kg, 7 days) in mice caused a significant impairment in the novel object test. Co-treatment of DA D1 receptor antagonist, SCH23390, or NR2B subunit antagonist of NMDA receptors, ifenprodil, prevented METH-induced cognitive impairment. In contrast, co-treatment of DA D2 receptor antagonist, raclopride, or noncompetitive NMDA receptor antagonist, MK-801, failed to prevent METH-induced cognitive impairment. These results suggest that DA D1 receptors and NR2B subunit of NMDA receptors play a critical role in the development of METH-induced cognitive impairment

P3-328 Spatial distribution of prefrontal cortical cells projecting to the supplementary eye field and pre-supplementary motor area in the monkey

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The supplementary eye field (SEF) and pre-supplementary motor area (pre-SMA) receive inputs from prefrontal cortex (PF). However, it is controversial whether the banks of the principal sulcus (PS) are the input source to the SEF. Furthermore, it is not known to what extent the PF cells projecting to the SEF and pre-SMA overlap. To address these questions, we applied a multiple retrograde labeling technique. The SEF and pre-SMA were physiologically identified, based on findings of intracortical microstimulation and single cell recording. Diamidino yellow and fast blue were injected into the SEF and pre-SMA, respectively. Cells projecting to the SEF and pre-SMA were distributed separately in the upper and lower banks of the PS. Cells projecting to the SEF were also observed in areas 8a, 8b, 45, and 12. These findings suggest that the SEF and pre-SMA receive different sets of information from the PF.

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P3-329 Retrospective and prospective coding of rules in the monkey prefrontal cortex during a rule-shifting task

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The dorsolateral prefrontal cortex (dlPFC) has been implicated in the shifting of behavioral rule based on response-outcome, but its neuronal mechanism is still unknown well. To address this issue, we examined neuronal activity in the dlPFC of a monkey performing an oculomotor delayed rule-shifting task in which the monkey chose red (rule A) or green target (B) from a set of targets (red and green) by his saccade. The rules A and B were arbitrarily changed, and the monkey must find the on-going rule based on the outcome (reward/non-reward). We focused on neuronal activity after outcomes to examine neuronal process for rule-alternation. Among 629 dlPFC neurons recorded, 47 and 43 neurons showed differential activity by the rule that was required in the next trial and by the rule that was used in the current trial, respectively. These neurons appear to represent retrospective and prospective coding of rules, which would be critical neuronal coding for rule-shifting mediated by the dlPFC.

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P3-330 Lack of visual responses in song nucleus HVC of Bengalese finches

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In male Bengalese finches, seeing females triggers singing behavior. We conducted electrophysiological experiment to investigate how visual information could modify neural responses in the song control system. We recorded neural activities in a higher order song control nucleus HVC by tungsten electrodes while playing back the bird's own song. With this setting we occasionally presented strobe lights to the eye to see how visual input could modulate auditory response. Although there is evidence from both anatomical and physiological studies suggesting visual input to HVC, we did not find single unit electrophysiological activities from HVC by visual stimulation. Since we obtained unit activities from the thalamic visual relay nucleus with the same setting, our data suggest the absence of functional visual input to HVC. Visual input may not directly trigger singing but may modify motivational states of the male birds. Supported by JST.

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P3-331 Prefrontal activity related to positive, but not negative, reward outcome in macaque

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We trained monkeys to perform a memory-guided saccade task in variable combination of liquid reward (J) and air-puff punishment (A), namely J+A- (APP condition), J-A+ (AVE condition), and J-A- (NEU condition). Animals performed significantly better in APP and AVE conditions than in NEU condition, presumably through positive and negative reinforcement, respectively. Among 406 neurons in the lateral prefrontal cortex (LPFC), roughly 10% changed activity depending on the reinforcer conditions. Many of them were differential between APP and AVE (48%), and between APP and NEU (54%), but few were differential between AVE and NEU (11%). Typically, activity was selectively enhanced or depressed in APP condition and unchanged between AVE and NEU conditions. The LPFC appears to be involved in positive, but not negative, reinforcement.

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P3-332 Role of the two-state membrane potential fluctuations in regulating cortical network activity: Modeling study

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Previously, we reported that spontaneous two-state membrane potential fluctuations ('Up-Down' transitions) can self-organize in networks of single-compartment neuron model expressing various ion channels (e.g., H-current). In this process, spike-timing-dependent plasticity (STDP) played an active role in regulating the network activity. To further clarify the possible interplay between the 'Up-Down' transitions and the STDP-induced activity regulation, and the role of the H-current in producing these transitions, we have studied similar networks consisting of multi-compartment neuron models. Our model incorporates the non-uniform dendritic distribution of H-current, both AMPA and NMDA receptor-mediated synaptic currents, fast-spiking interneurons, and the STDP with variety of the learning time window. We argue essential roles of the 'Up-Down' transitions in dynamic regulation of recurrent synapses.

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P3-333 Neural process for decision of saccade direction in the prefrontal cortex

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To examine prefrontal (PF) contributions in the decision of a saccade direction, neuron activity was recorded while 2 monkeys performed 2 ODR tasks. In ODR, monkeys had to make a memory-guided saccade to the direction where a visual cue was presented. In S-ODR, 4 identical visual cues were presented during the cue period and monkeys had to make a saccade to any one of these 4 directions after the delay. We found that 1) neurons having directional pre-saccadic activity in ODR showed the activity with the same magnitude and directional selectivity in S-ODR, 2) onset latencies of these pre-saccadic activities correlated with monkeys' saccade latencies, 3) the temporal change of the strength of directional selectivity during the peri-saccadic period was similar in both tasks. These results suggest that PF pre-saccadic activity represents commands for executing a saccade and that the motor preparation process could be dissociable from the decision process of movement directions in PF.

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P3-334 Neural correlates of linguistic similarity among L1, L2 and L3 : An fMRI study

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To investigate how typological differences such as those in terms of basic word order are related to activations in multilingual speakers' brains, we measured the functional organization of the human brain involved in comprehension of Korean, Japanese and English by native speakers of Korean who learned English and Japanese after puberty. The present study provides the possibility that syntactic similarity/dissimilarity among languages is reflected by a difference in brain activity, particularly, in the left inferior frontal cortex including Broca area.

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P3-335 Developmental changes of cerebral responses to a Japanese phonemic contrast measured with NIRS

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A Japanese phonemic length contrast such as long/short vowels is one of the most difficult phonemic contrasts to acquire for children. This study aims to explore neural basis of the developmental process of the phonemic length contrast. Cerebral responses to long/short vowels were measured with multi-channel near infrared spectroscopy for 65 infants (age: 4 - 27 months). The results showed a phoneme-specific neural response in a relatively younger age group, i.e. a group of 4-7 months old. However, left-dominant responses were only found for the groups older than 13-17 months. These results suggest that infants have already noticed some phonetic features of Japanese long/short vowels in their age of 4-7 months, however these phonetic features may not processed linguistically until they are 13-17 months.

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P3-336 EEG observation under thinking on questions relating to emotional affairs

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EEG has been observed as a useful tool monitoring brain activities. However, not yet recognized any reflections of intellectual working as thinking originated in the celebral cortex. Here we tried to observe any EEG change under questions that may induce emotional activities of the limbic system. EEG were observed with half open eyes in 15 male students (aged 20-22) with four electrodes at O_1, T_4, P_z, and F_z. Questions regarding anxiety, apetite, urination and ladies attraction were each provided, and the EEG signals were analysed by the FFT. Several students showed some changes on the frequency spectra, presenting broad increases in 20 to 40Hz range, remarkably different from those at relaxation. However, some artifacts probably due to eye-movement and muscle activities inevitably accompanied to thinking were found as difficult to reject perfectly their influences from the spectra.

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P3-337 Brain activation with the changes of presentation rate of stimulus during a working memory task.

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We investigated brain activation by fMRI during the Paced Visual Serial Addition Task. Subjects were seventeen young adults and asked to add the current number and last presented number. Each number was presented at the rate of 1.2 sec., 1.6 sec., 2.0 sec. and 2.4 sec.. A single regression analysis with the rate of presentation revealed negative correlation in the lt. precentral gyrus, lt. inf. parietal lobule, rt. precuneus, lt. mid. occipital gyrus, rt. fusiform gyrus, lt. parahippocampal gyrus, lt. thalamus, lt. lentiform nucleus and bilat. cerebellum (uncorrected, p>.001). These areas may well be related to the increase of the consumption of cognitive resource in this task.

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P3-338 Sensation at the tips of virtual tools

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When we touch something with a tool, we feel the touch at the tip of the tool, rather than at the hand that holds the tool. We reported that the judgment of the temporal order of two successive stimuli, delivered to the tips of sticks, was altered by crossing the sticks. The previous report provided evidence for the referral of tactile signals to the tip of a *real* tool. In this study, we examined whether the referral occur to the tip, or action point, of a *virtual* tool with which subjects could manipulate objects in a virtual reality with haptic feedback. The *virtual* tool consisted of a single spherical action point that could be moved with a stylus in hand. Temporal order judgment of two stimuli, delivered to the two styli held in each hand, was altered by changing the positions of the actions points in the virtual space without changing the positions of the hands and the styli. The results show that somatosensory signals can be referred to the action point of a *virtual* tool without any rigid body connecting the action point and the hand.

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P3-339 The neural basis of conscious and unconscious repetition priming revealed by functional MRI.

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Recent neuroimaging studies have revealed that repetition priming causes the reduction of the activity of the left infero-temporal cortex and the left inferior frontal cortex while the activity of several regions was increased by repetition priming. However, it has been unclear which region is specific to conscious and unconscious priming as these results have possibly failed to dissociate the conscious and unconscious priming effect. We employed event-related functional MRI study to investigate the neural substrates of conscious and unconscious priming effect using exclusion and inclusion task. This procedure enabled us to strictly dissociate the brain regions concerning the conscious and unconscious priming effect.

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P3-340 Location selective response in the mesial parietal cortex of monkeys during navigation task in virtual environment

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To investigate neural basis of the cognitive map, we recorded neuronal activity in the mesial part of the parietal cortex of monkeys performing a navigation task in a virtual environment. A virtual building with two floors was created stereoscopically by computer graphics. The monkeys were trained to navigate from starting point to goal room beyond the direct view by operating a joystick. A group of neurons in the mesial parietal cortex changed their activity depending on the locations in the virtual environment. In some of these neurons, however, the responses were diminished when the static image around the preferred location was presented and the monkey went through the same route passively. These results suggest that the neural activity of the location selective neurons in the mesial parietal cortex cannot be explained by the visual aspect only.

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P3-341 Visual pattern-dependent reward-predicting activity by primate prefrontal neurons

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We trained a monkey with a sequential association, which is composed of two successive oculomotor matching-to-sample tasks (pattern A->green->O or pattern B->red->X). To investigate reward-predicting activity in the associative chain by prefrontal neurons, asymmetric reward schedule was introduced. 310 neurons were recorded from the prefrontal cortex. 34% of them (104) showed significantly different activity for reward and non-reward cues during pattern-cue (first cue) period. These neurons can be divided into two types: pattern-independent reward predicting neuron (R type; 65%) and pattern-dependent reward predicting neuron (VR type; 35%). The differential activity evoked by reward and non-reward pattern-cues was observed significantly earlier in VR type than in R type, although the visual latency for both types was not different. The results suggest that the reward-predicting information might be processed hierarchically in the prefrontal cortex.

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P3-342 Pulse-coupled resonate-and-fire models

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We analyze two pulse-coupled resonate-and-fire neuron models. Numerical simulation reveals that an anti-phase state is an attractor of this model. We can analytically explain the stability of anti-phase states by means of a return map of firing times, which we propose in this paper. The resultant stability condition turns out to be quite simple. The phase diagram based on our theory shows that there are two types of anti-phase states. One of these cannot be seen in coupled integrate-and-fire models and is peculiar to resonate-and-fire models. The results of our theory coincide with those of numerical simulations.

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P3-343 Simulation analysis of signaling dynamics regulating nerve growth in computational model using E-CALI system

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We modeled signaling networks within nerve growth cones. Virtual large calcium transient caused decrease neurite extension rate and subsequent retraction in this model, indicating that the simulation results well fitted to phenomena in living cells. The model was analyzed using E-CALI system that enables acute inactivation of specific molecules in silico, so that the model can be compared and examined by the experiments employing the chromophore-assisted laser inactivation (CALI) technology that generates laser light-mediated temporal loss of function of a specific protein at precise times in living cells. The sensitivity analysis and the E-CALI simulation revealed that protein phosphatases such as protein phosphatase 1 and protein phosphatase 2A may play key roles in the regulation of microtubule dynamics.

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P3-344 Simulation analysis of cyclin-dependent kinase 5 in hippocampal long-term potentiation

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We have modeled and analyzed a signal transduction system of long-term potentiation (LTP) at hippocampal postsynapse in silico. Calcineurin played an important role in our previous simulation model. The sensitivity analysis for LTP revealed the robustness of protein phosphatase 2A dynamics as characteristics of bistability in the model. In this study, we focused on the role of cyclin-dependent kinase 5 (Cdk5) that could be eventually activated by phosphatases and involved in the phosphatase activation. Inhibitor 1 (I1) phosphorylation by Cdk5 led to its weak inactivation, indicating that protein phosphatase 1 (PP1) and Cdk5 may interact with a positive feedback loop that activated PP1 through I1 inactivation. Thus, the simulation showed an important role of Cdk5 in hippocampal LTP signaling.

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学会掲示板

東京薬科大学生命科学部分子生命科学科生体高次機能学研究室教員公募

職名および人員：教授1名

応募要件：生体高次機能学研究室では、脳の構成要素である神経細胞とグリア細胞における細胞内外の情報伝達機構や情報処理機構を、光プローブとコンピュータを使った画像処理の技術を用いて解析している。また、脳機能の損傷とその修復に関わる機構についても研究を進めている。脳や神経の機能の解明を目指して積極的に研究を進めている方で、熱意をもって私立大学における学部および大学院教育を行うことのできる方。

現在の研究室の構成：

工藤佳久教授（2005年3月退職予定）、宮川博義助教授、森田光洋助手、井上雅司助手

応募書類：(1) 履歴書（写真貼付）、(2) 研究業績リスト、(3) 主要業績の別刷またはコピー（10編以内）、(4) 現在までの研究概要（2000字以内）、(5) 今後の研究と教育に関する理念と抱負（2000字以内）、(6) 2名の方からの推薦状（作成者が封をしたものを応募書類に同封）、(7) そのほか選考の参考となる資料（学会活動、科学研究費取得状況など）。選考終了後に推薦書以外の書類の返却を希望される方は、返信用封筒（宛先記入、切手貼付）を同封してください。なお、選考過程において研究に関するセミナーや模擬講義を行っていただく場合があります。

応募期限：2004年9月30日（木）消印有効

着任時期：2005年4月1日（金）

書類送付先：〒192-0392　東京都八王子市堀之内1432-1
東京薬科大学生命科学部　事務課長　清野文子
（「生体高次機能学教授選考応募書類在中」と朱書し、簡易書留にて郵送のこと）

問い合わせ先：東京薬科大学生命科学部　多賀谷光男
Tel. 0426-77-7496
Fax. 0426-76-8866
E-mail: tagaya@ls.toyaku.ac.jp

生理研カンファレンス・未来開拓国際シンポジウム

International Symposium on
"Adult neurogenesis in normal and pathological conditions"

日　　時：平成16年11月11日（木）13:00～13日（土）15:00

場　　所：自然科学研究機構　山手3号館 2F（愛知県岡崎市明大寺町）
名鉄東岡崎駅よりタクシー利用にて約7分
バス利用にて約10分

Oversea Symposists（敬称略）

Steve A Goldman（Univ. Rochester / U.S.A.）
Magdalena Gotz（Max-Planck Inst. Neurobiol. / Germany）
Rune Hen（Columbia Univ. / U.S.A.）
John A. Kessler（Northwestern Univ. / U.S.A.）

Derek van der Kooy（Univ. Toronto / Canada）
Jeff Macklis（Harvard Univ. / U.S.A.）
Cindi Morshead（Univ. Toronto / Canada）
Stephen C Noctor（Columbia Univ. / U.S.A.）
※シンポジウムは全て英語にて行われます。

参 加 費：4,000円（ポスターパーティー、昼食代2回含む）
申し込み方法：住所・氏名・所属・Tel番号・メールアドレスを明記し、下記連絡先宛にE-mailまたはFaxにてお申し込みください。

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申込締切：9月末日

組織委員：Derek van der Kooy University of Toronto
等　　誠司　自然科学研究機構・生理学研究所
池中　一裕　自然科学研究機構・生理学研究所

連 絡 先：自然科学研究機構・生理学研究所　窓口：北山　理英（秘書）
Tel: 0564-59-5249　Fax: 0564-59-5247
E-mail: trie@nips.ac.jp
http://www.nips.ac.jp/ninfo/sympo.html

助成・褒賞案内

（財）内藤記念科学振興財団（日本神経化学会からの推薦枠 各1件）

・内藤記念科学振興賞（日本神経化学会締切日：2004年9月1日必着）

[趣旨]

人類の健康の増進に寄与し得る自然科学の基礎的領域における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。

[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上げた研究者。
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。
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・内藤記念海外学者招へい助成金
後期：2005年7月1日～2006年12月31日に外国の研究者を招聘するもの
（日本神経化学会締切日：2004年9月1日必着）

・資料請求／問合せ先
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http://www.naito-f.or.jp/　　E-mail info@naito-f.or.jp

財団法人東レ科学振興会

下記の2件については日本神経化学会からの推薦枠は各2件です。

（日本神経化学会締切日：2004年9月1日必着）

1. 第45回科学技術賞

・対　　象：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。例年、紫綬褒章クラスの方が受賞されているそうですが、既に国家的表彰を受けられた方（例えば勲一等、勲二等や学士院賞受賞者）についてはご遠慮くださいとの事です。推薦を受けた候補者はその年度および次年度にわたって選考の対象となります。従って平成15年度に推薦された候補者は平成16年度候補者としての再度推薦の必要はありません。

・褒　　賞：賞状、金メダル、賞金（500万円）　受賞者は2名前後

2. 第45回科学技術研究助成

・対　　象：国内の研究機関において自らのアイディアで萌芽的基礎研究に従事し、かつ本人が主体性をもって研究を遂行できる方で、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歳以下）。
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。

・研究助成金：総額1億3000万円。1件3000万円程度まで10件程度。

資料請求／問合せ先

（財）東レ科学振興会
〒279-8555千葉県浦安市美浜1-8-1　東レビル
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学会事務局でも何部か申請書類をお預かりしております。

各助成・褒賞におきましても申請用紙は各財団に直接請求し、学会推薦を希望されます場合は、日本神経化学会事務局まで簡易書留にてご送付ください。

〈事務局連絡先〉
113-8622　文京区本駒込5-16-9
日本学会事務センター内
日本神経化学会
TEL.03-5814-5801
FAX.03-5814-5820

Neuro 2004

東レ科学振興会　第54回科学講演会

テーマ「私たちの細胞はどのようにして生まれたか」

1. 細胞の中に入った細胞
   石川　　統（放送大学　教授）
2. ミトコンドリアと葉緑体はどこからきたか
   黒岩　常祥（立教大学理学部生命理学科　教授）

日　　時：2004年9月17日　開場15：30　開演16：00～（19：00終演）

開　　場：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11F
(JR有楽町駅前)

※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

問合せ先：財団法人東レ科学振興会
千葉県浦安市美浜1-8-1
TEL.047-350-6103
FAX.047-350-6082
E-mail：jk.tsf@nifty.com
URL http://www.toray.co.jp/tsf/index.html

第13回日本バイオイメージング学会学術集会のご案内

会　　期：平成16年11月5日（金）～7日（日）

公開シンポジウム：11月5日（金）　午後1時～5時
「光による生体機能の計測と操作」

学術集会：11月6日（土）～7日（日）
シンポジウム「光学顕微鏡によるナノバイオテクノロジー」
シンポジウム「医療とバイオイメージング」

会　　場：京都府立医科大学 図書館ホール・基礎医学学舎講義室

問合せ先：第13回日本バイオイメージング学会
〒602-8566　京都市上京区河原町広小路梶井町465
京都府立医科大学大学院医学研究科　細胞分子機能病理学部門
TEL.075-251-5322　　FAX.075-251-5353
E-mail: bioimage@koto.kpu-m.ac.jp
http://koto8.kpu-m.ac.jp/~pcr/bioimaging.htm

千里ライフサイエンス振興財団からのご案内

千里ライフサイエンスセミナー

「タンパク質のクオリティコントロールとその破綻」

日　時：平成16年9月7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

着眼点：種々のストレスや遺伝的変異によって、細胞内にミスフォールドしたタンパク質が蓄積し、凝集するといった事態に対応するため、細胞は何段階もの巧妙なタンパク質品質管理機構を備えている。本セミナーではこのような細胞内におけるタンパク質の品質管理機構の分子基盤と、その破綻としての神経変性疾患をはじめとする、いわゆるフォールディング病についての最新の知見を紹介する。

コーディネーター：京都大学　再生医科学研究所　　教授　永田　和宏
奈良先端科学技術大学院大学　　教授　河野　憲二

プログラム：

1. はじめに
2. 分子シャペロンによる蛋白質の変性・凝集の阻止と回復
   東京工業大学　資源化学研究所　教授　吉田　賢右
3. 小胞体ストレス感知システムとその可視化
   奈良先端科学技術大学院大学　遺伝子教育研究センター　教授　河野　憲二
4. 小胞体の品質管理機構－細胞内メカニズム－
   京都大学　再生医科学研究所　細胞機能調節学分野　助教授　細川　暢子
5. 異常タンパク質を処理する細胞内装置
   東京都臨床医学総合研究所　副所長　田中　啓二
6. 樹状細胞による抗原クロスプレゼンテーションの機構
   (株) 医学生物学研究所　伊那研究所　所長　矢原　一郎
7. 運動ニューロン疾患の病態と治療
   名古屋大学大学院　医学系研究科　神経内科　教授　祖父江　元
8. ミスフォールド蛋白質とパーキンソン病
   理化学研究所 脳科学総合研究センター　チームリーダー　高橋　良輔
9. おわりに

千里ライフサイエンスセミナー

「ヒト安全性予測システムの現状と今後の展開：ヒトSNP・蛋白質解析、動物モデル、in silico解析」

日　時：平成16年9月28日（火）10:00～17:15

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

着眼点：薬物療法は現代医療の中心的な治療法である。安全・安心で有効な薬物療法の確立のためには、重篤な副作用をさけることが必須である。また、薬物開発においても心臓不整脈や肝障害などの副作用の出現がその薬物にとって致命的となることが知られている。

本セミナーにおいては、ゲノム・蛋白の網羅的解析、動物モデル、in silico解析など、様々の方法によるヒトにおける薬物の安全性がどこまで予測できるか、また、今後どのように研究を進展させるべきかを考える。

コーディネーター：大阪大学大学院医学系研究科　教授　倉智　嘉久

東京大学医学部附属病院薬剤部　助教授　鈴木　洋史

プログラム：

1. 新医薬品の安全性予測と臨床試験のあり方
   東京都多摩老人医療センター　名誉病院長
   医薬品医療機器総合機構　顧問　上田　慶二
2. トキシコゲノミクスプロジェクトの特徴と進捗
   国立医薬品食品衛生研究所　所長　長尾　　拓
3. 新しいたんぱく質科学の構築を目指して
   創薬プロテオームファクトリープロジェクトリーダー　松尾　壽之
4. トランスポーターを利用した医薬品誘発副作用の回避：in vitro実験、遺伝子改変動物を用いたin vivo実験に基づく予測
   東京大学大学院薬学系研究科　教授　杉山　雄一
5. PETによる薬物動態解析－11C-phosgeneによる標識薬剤合成装置
   大阪大学大学院医学系研究科　教授　畑澤　　順
6. 薬物動態・安全性試験におけるヒト肝細胞キメラマウスの有用性
   金沢大学大学院医学系研究科　教授　横井　　毅
7. 特異体質性の薬物毒性発現機構に関する考察
   三共株式会社薬剤動態研究所　所長　池田　敏彦
8. ヒト臓器・組織を用いた安全性評価システムの開発
   大阪大学大学院医学系研究科　教授　野村　大成
9. タンパク質の表面物性の類似性にもとづく機能予測
   大阪大学蛋白質研究所　客員助教授　木下　賢吾
10. 薬物による心臓副作用in silico予測
    大阪大学大学院医学系研究科　教授　倉智　嘉久

ブレインサイエンスシリーズ第17回

「統合失調症：分子から治療まで」

日　時：平成16年10月19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

着眼点：わが国の精神科入院患者の約6割を占めると言われる統合失調症は、その臨床症状の多様性から、発症機構解明が遅れてきた。しかし、ここ数年のうちに薬理学的、分子生物学的、遺伝学的に発症機構を全く新しい視点で解明しようとする試みがなされている。本セミナーではこれら新しい話題を提供し統合失調症の本質に迫る。

コーディネーター：浜松医科大学　教授　佐藤　康二
大阪大学大学院医学系研究科　教授　遠山　正彌

プログラム：

1. 統合失調症の分子薬理学的解析：ドーパミン受容体およびNMDA受容体作用薬を用いたアプローチ
   東京医科歯科大学大学院医歯学総合研究科　教授　西川　　徹
2. PACAPノックアウトマウス－新しい精神機能障害モデル
   大阪大学大学院薬学研究科　教授　馬場　明道
3. DISC1に結合する蛋白質とその機能的修飾
   大阪大学大学院医学系研究科　助手　片山　泰一
4. 統合失調症脆弱性遺伝子ディスバインジンの関連解析と神経細胞における機能解析
   国立精神・神経センター神経研究所　室長　橋本　亮太
5. 統合失調症の神経幹細胞機能異常仮説
   浜松医科大学　教授　森　　則夫
6. 統合失調症関連表現型の遺伝子解析
   理化学研究所・脳科学総合研究センター　チームリーダー　吉川　武男

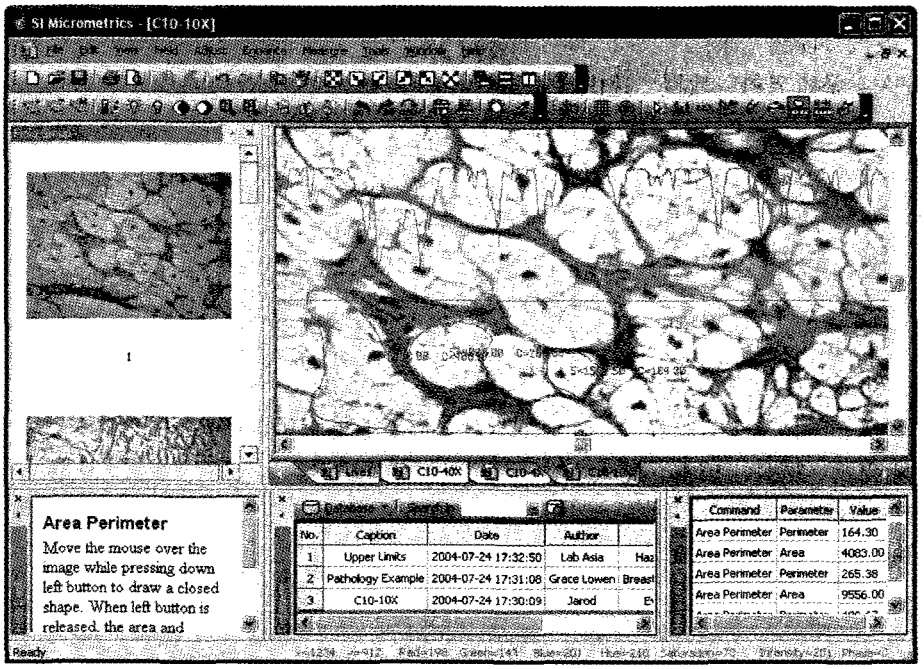
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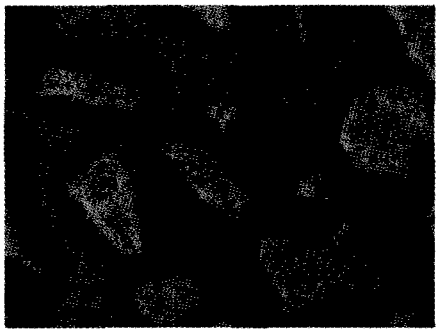
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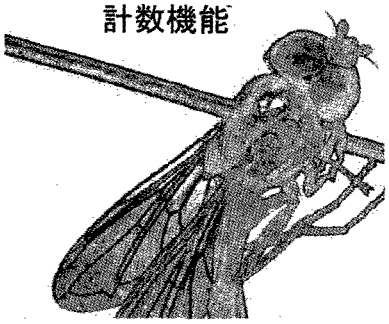
Micrometrics for Windows

画像取込み、画像データベース、画像処理・編集、画像計測・計数、レタリング、画像系列蓄積、電子透かし埋込、印刷、顕微鏡制御、顕微鏡フィルタ・シミュレーション、フュージョン、等々、高価格の画像処理ソフトウエアでしか使えなかった機能がすべて用意されています。
しかも、使い方はとても簡単です。

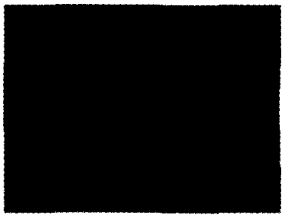




計数機能

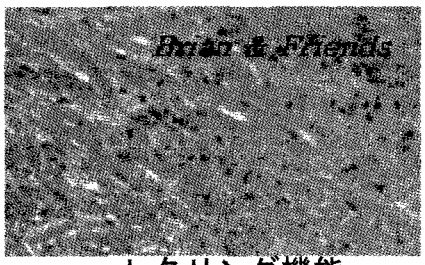


スケッチ化機能

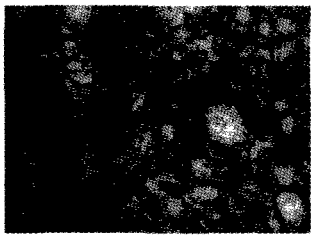


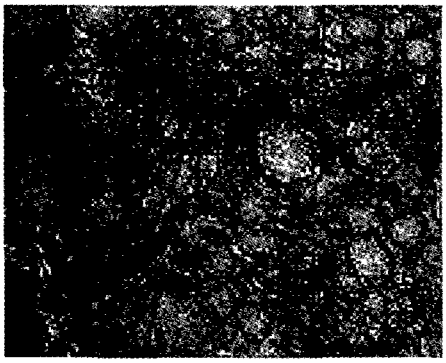


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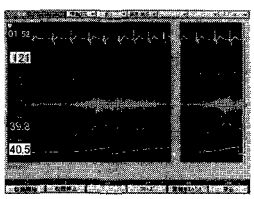
エレクトロニクスで病魔に挑戦
NIHON KOHDEN

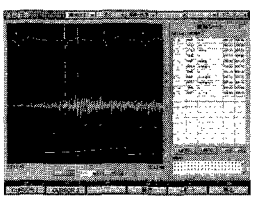
PCベースのポリグラフ
ポリグラフ
Polygraph / PEG-1000

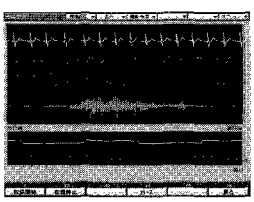
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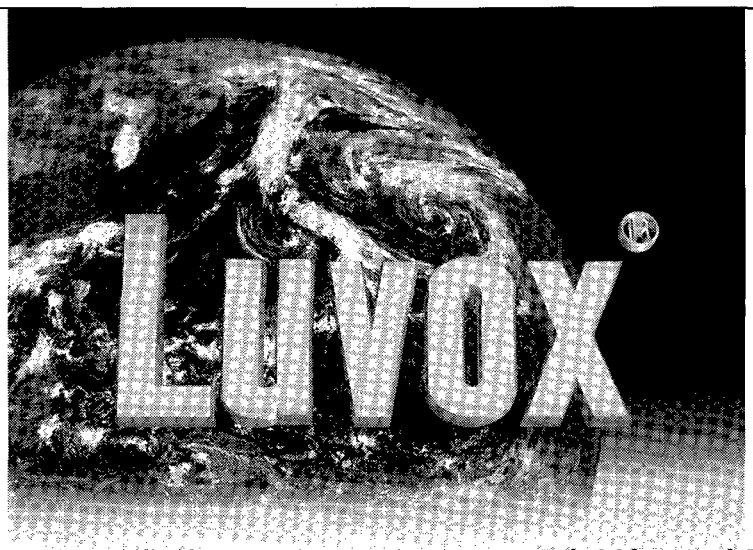
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ACD0406-4　2004年6月作成

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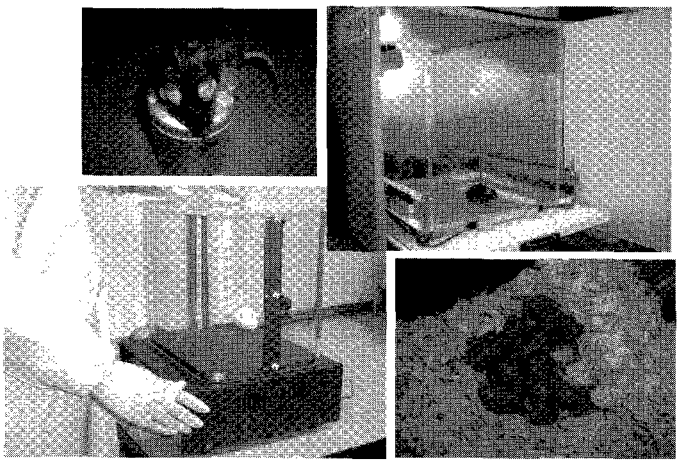
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作成年月2003年1月

A42



・オープンフィールドテスト
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・水迷路学習実験
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・ホットプレートテスト
・強制水泳テスト　他

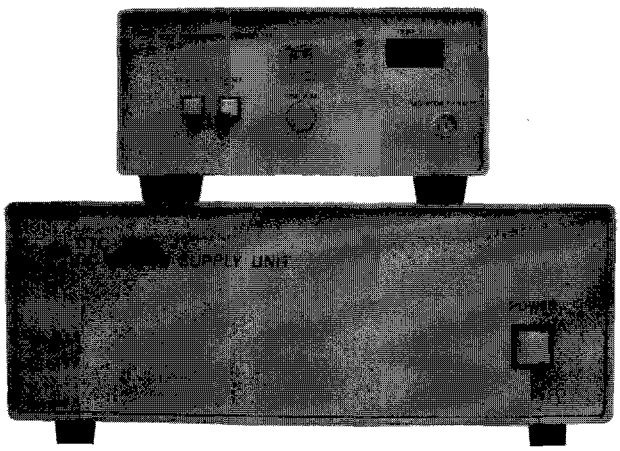
・マウスの行動解析の受託
・各種向精神薬、抗鬱薬等の薬効薬理試験
・行動科学研究に関する総合コンサルタント
・動物の行動実験機器の開発と販売

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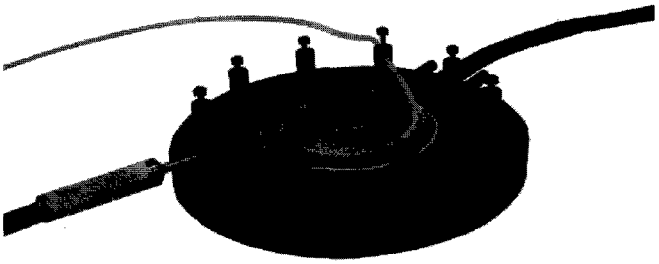
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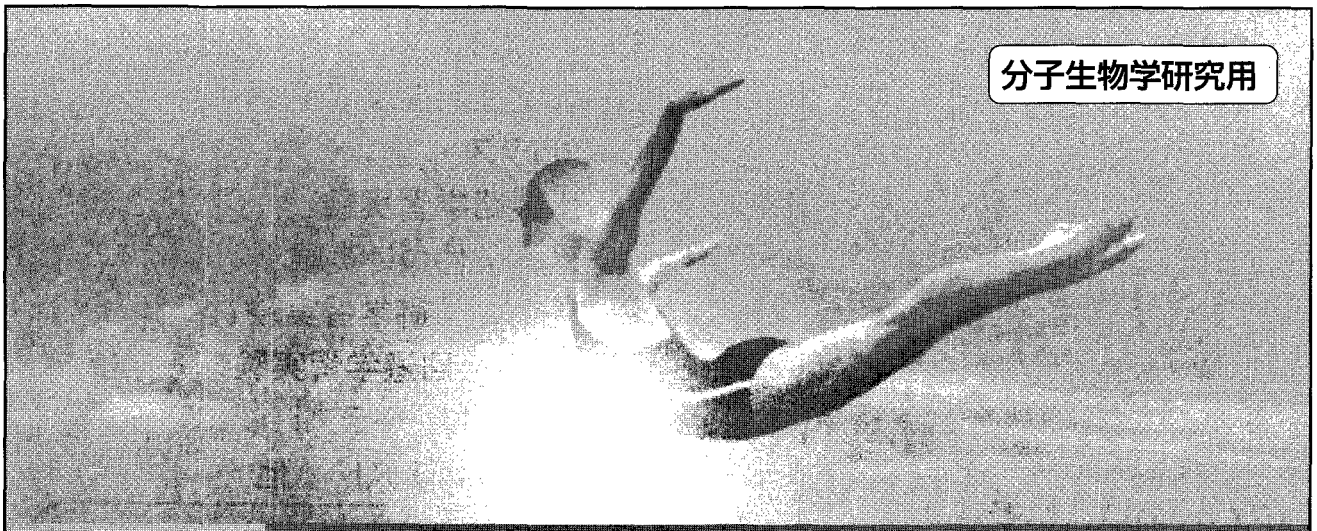
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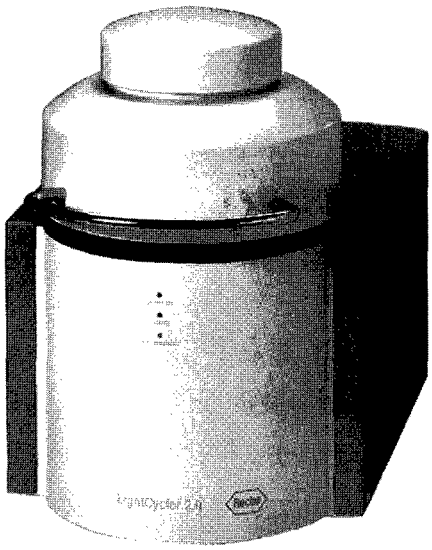
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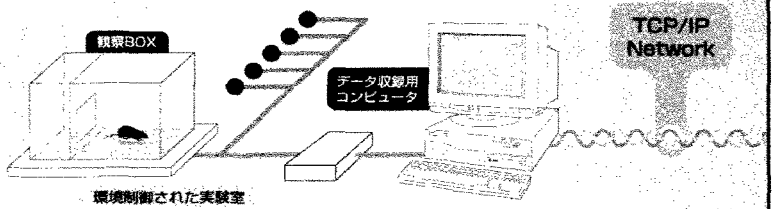
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※写真はKUROBOX6台システム設置例です。

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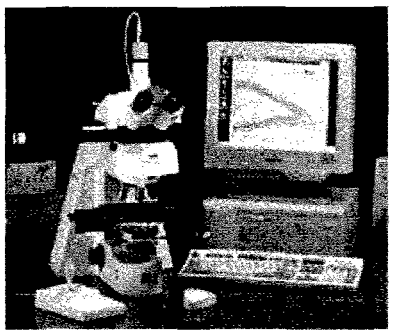
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- ◆ 3D 再構築画像の回転表示が可能
- ◆ コンフォーカル・MRIイメージにも対応

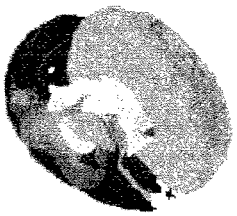


ニューロンのモデリング、構造解析

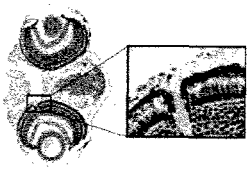


Probes
Optical fractionator /Physical disector /Schmitz Nearest Neighbor /Nucleator (2D and 3D) /Planar rotator /Optical rotator /Disector/Cavalieri estimator etc...

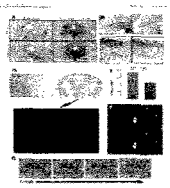
脳の 3D 再構造

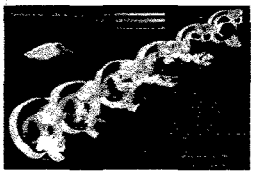


Virtual Slide の作成

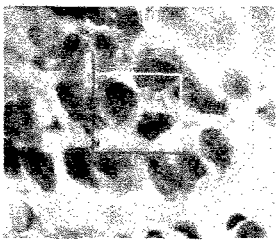


Stereo Investigator

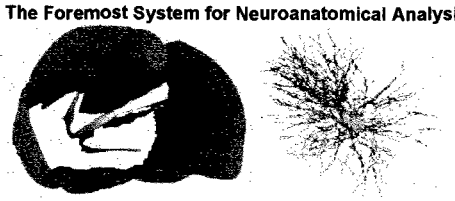




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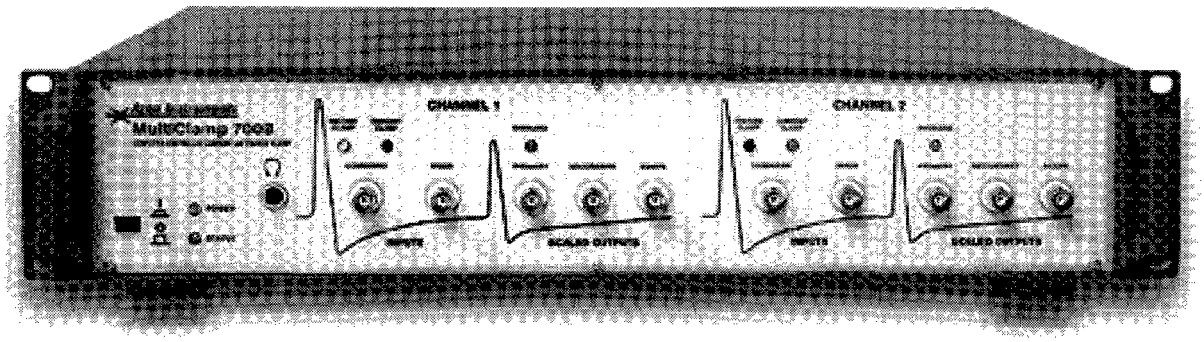
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Inter Medical co.,ltd.
株式会社 インターメディカル

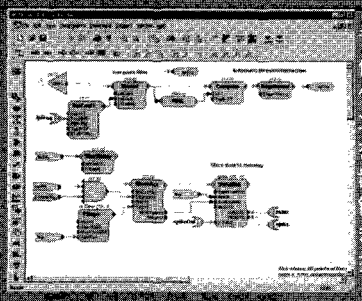
本　　社　〒464-0850 名古屋市千種区今池三丁目40番４号　TEL(052)731-8000(代)
FAX(052)731-5050
大阪営業所　〒532-0011 大阪市淀川区西中島四丁目３番４号　TEL(06)6886-4151
FAX(06)6886-4152
e-mail:int-med@mbf.sphere.ne.jp　website:http://www.intermedical.co.jp/

音刺激コントロールDSPシステム

TDTシステムIII

www.brck.co.jp/auditory.htm

TDTシステムIIIは、ABR、AEP、OAEに最適な音刺激コントロールDSPシステムです。
音刺激出力の高速処理やS/N比110dBの高品質DSPにより、世界中で高い評価を受けております。



音刺激のコントロールには標準添付のRPvds(ビジュアルデザインスタジオ)を使用します。RPvdsはアンプ、ジェネレータなどのアイコンをドラック&ドロップで画面上に配置し、個々をラインでつなぐだけで簡単に音刺激ルーチンを作成できる、優れたインターフェイスを持ったソフトウェアです。



RMモバイルプロセッサはUSBポートからの電源供給をサポート



DSP、アンプ、ミキサーなどの各ユニットは組替え可能なモジュール式となっており、用途に応じて自由に選択、追加ができます。

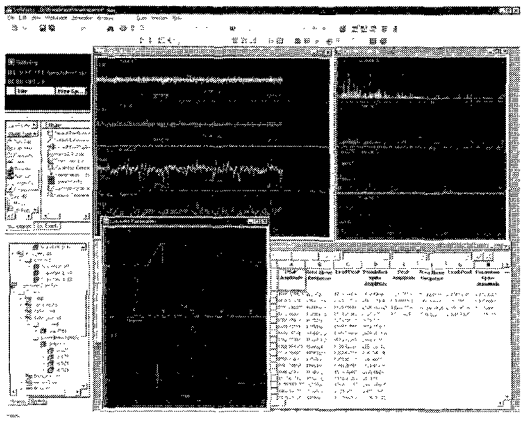
PCとの接続はUSBで行いますのでノートPCでも手軽にご利用いただけます。

プロフェッショナルワークステーション

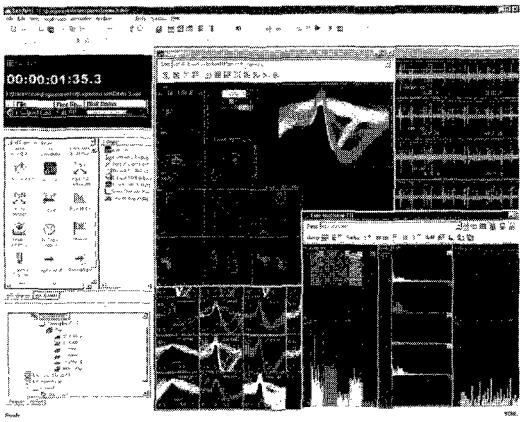
SciWorks

www.brck.co.jp/product/sciworks.htm

電気生理、神経生理学、生理学、生物学等に関する実験向けのプロフェッショナル用データ解析ソフトウェアです。
実験目的に合わせて自由に、データの収録方法・プロット方式・解析の組合せ・保存するデータの選択などをカスタマイズできます。また全ての解析項目はリアルタイムでもオフラインでも同様に行えます。詳しくはWebサイトへ！



波形から面積、高さ、傾き、潜時など80以上のパラメータを読み取り、オンラインでグラフやスプレッドシートにプロット



シングル・ステレオ・テトロード電極のマルチスパイクをリアルタイムにクラスター分別してヒストグラムをプロット

USB接続タイプも出ました。
ノートPCでもお使いいただけます。

バイオリサーチセンター株式会社

本社 名古屋市東区泉2-28-24 ヨコタビル4F TEL(052)932-6421
東京 東京都千代田区岩本町2-9-7 RECビル TEL(03)3861-7021
大阪 大阪市淀川区西中島6-8-8 花原第8ビル203 TEL(06)6305-2130
http://www.brck.co.jp mail:sales@brck.co.jp

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。**

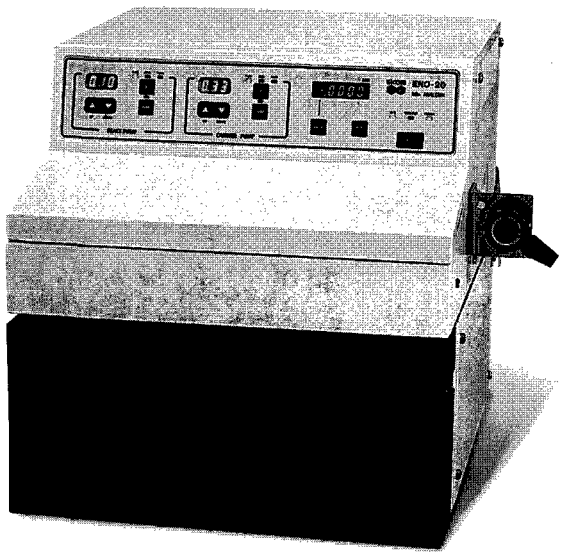
生体内神経活動をリアルタイムで観察することは、
神経科学的研究を行う上で極めて大きな目標の一つです。
エイコムでは今以上の高感度分析には一体化が
必要だと考え分析に必要なユニットを
すべてまとめてHTEC-500型を完成しました。
専用器的な高感度分析計として、限りなく
目標に近づくことを目指します。

- 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。

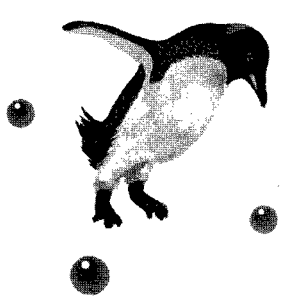
- 0.1ピコモルの他に例を見ない高感度。
- NO_2^-、NO_3^-を高精度で分離測定。
- 安定性と再現性が良い高稼働率測定器。

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。

EiCOM 株式会社 エイコム

本　　社：京都市伏見区下鳥羽円面田町24-2　〒612-8474
TEL(075)622-2112　FAX(075)622-2114
東京営業所：TEL(03)3818-5223　FAX(03)3818-4540
札幌営業所：TEL(011)813-3268　FAX(011)813-6001
http://www.eicom.co.jp　E-mail: webmaster@eicom.co.jp

高品質ペプチドがあなたの研究の信頼性を高めます！



PEPTIDE

アルツハイマー病の研究用試薬

● Amyloid β-Protein Fragments

コード	品名	容量	定価
4307-v	Amyloid β-Protein (Human, 1-40)	0.5 mg Vial	18,000
4379-v	Amyloid β-Protein (Human, 1-40) [HCl Form]	0.5 mg Vial	20,000
4349-v	Amyloid β-Protein (Human, 1-42)	0.5 mg Vial	30,000
4370-v	Amyloid β-Protein (Human, 1-43)	0.5 mg Vial	35,000
4359-v	Amyloid β-Protein (Human, 1-16)	0.5 mg Vial	10,000
4367-v	[Pyr^3]-Amyloid β-Protein (Human, 3-42)	0.5 mg Vial	30,000
4309-v	Amyloid β-Protein (Human, 25-35)	0.5 mg Vial	4,000
4358-v	β-Sheet Breaker Peptide iAβ5	5 mg Vial	16,000

● Amyloid β-Protein Control Peptides

	コード	品名	容量	定価
NEW	4413-s	Amyloid β-Protein (40-1)	0.1 mg Vial	9,000
NEW	4420-s	Amyloid β-Protein (42-1)	0.1 mg Vial	18,000

● β-Secretase Inhibitor

コード	品名	容量	定価
4378-v	Lys-Thr-Glu-Glu-Ile-Ser-Glu-Val-Asn-Sta-Val-Ala-Glu-Phe	1 mg Vial	20,000

● β-Secretase Substrate

コード	品名	容量	定価
3212-v	MOCAc-Ser-Glu-Val-Asn-Leu-Asp-Ala-Glu-Phe-Arg-Lys(Dnp)-Arg-Arg-NH_2	1 mg Vial	15,000

● γ-Secretase Inhibitors

	コード	品名	容量	定価
NEW	4394-v	L-685,458	1 mg Vial	30,000
NEW	3219-v	(3,5-Difuluorophenylacetyl)-Ala-Phg-OBu^t	5 mg Vial	10,000

● γ-Secretase Substrate

	コード	品名	容量	定価
NEW	3217-v	Nma-Gly-Gly-Val-Val-Ile-Ala-Thr-Val-Lys(Dnp)-D-Arg-D-Arg-D-Arg-NH_2	1 mg Vial	15,000

● Antisera

コード	品名	容量	定価
14359-v	Amyloid β-Protein (Human, 1-16) Antiserum	50μl Vial	25,000
14307-v	Amyloid β-Protein (Human, 1-40) Antiserum	50μl Vial	25,000
14356-v	Amyloid β-Protein (Human, 34-40) Antiserum	50μl Vial	25,000
14357-v	Amyloid β-Protein (Human, 37-42) Antiserum	50μl Vial	25,000
NEW 14414-v	Amyloid β-Protein (Human, 37-43) Antiserum	50μl Vial	25,000

PEPTIDE

PEPTIDE INSTITUTE, INC.
株式会社 ペプチド研究所
http://www.peptide.co.jp

〒 562-8686 大阪府箕面市稲 4丁目1番2号
TEL: 072-729-4121 FAX: 072-729-4124
E-mail: sales@peptide.co.jp

HAMAMATSU

50th Anniversary

浜松ホトニクスは、ビデオマイクロスコピー技術を核に蛋白質機能解析ツールを各種取り揃えています。

先端のイメージング技術で蛋白質機能を解析



浜松ホトニクスは、高感度で高解像度なイメージングデバイスを核に、ライフサイエンス分野におけるイメージプロセッシングの技術開発を推進してきました。これからも新しいアプリケーションに対応し、蛋白質機能解析ツールを開発してまいります。

蛋白質相互作用解析
AQUACOSMOS/ASHURA
FRET観察に適した冷却3CCDカラーカメラを採用。C/Y/Rチャンネルに独自の機能を持たせFRETの検出効率を向上。理化学研究所との共同開発で製品化したアクセプタブリーチング機能を搭載。

1分子挙動解析
AQUACOSMOS/1分子蛍光イメージングシステム
1分子の挙動を高感度に画像化、最長14時間のデジタルイメージングのリアルタイム録画を実現。全反射蛍光顕微鏡と組み合わせ、膜蛋白質や細胞内構造蛋白の分子レベルの挙動を記録・解析。

LIVING CELL IMAGING

FDSS/IMACS
蛍光プローブを用いた生細胞における各種イオンや蛋白質解析などのカイネティクス解析が可能。マイクロプレート中の細胞の反応を単一細胞レベルで自動解析。ヘテロジーニアスな系で威力を発揮。
ハイインフォメーションスクリーニング

AQUA-C・IMAGING
蛍光観察や生細胞を用いたダイナミクス解析まで研究用途に合わせたシステムの構築が可能。生きた細胞内における各種蛋白質挙動の時間的空間的な変化を顕微鏡下で長時間、連続的に観察。
GFPタイムラプスイメージング

蛋白質機能解析ツール ラインアップ

浜松ホトニクス株式会社

システム事業部　特機営業部　〒430-8587　浜松市砂山町325-6　TEL（053）452-2148
E-mail　salesi@sys.hpk.co.jp　　Web site　http://www.hpk.co.jp
東京支店（03）3436-0491　　大阪営業所（06）6271-0441　　仙台営業所（022）267-0121

製品Webサイト
http://www.hpk.co.jp/Jpn/products/SYS/BioJ.htm

☆製品Web siteからは、カタログのPDFファイルをダウンロードすることができます。ぜひアクセスしてください。

定評ある最新・最高の治療年鑑。過去最大規模でさらに詳しく、さらにわかりやすく

TODAY'S THERAPY 2004
今日の治療指針

私はこう治療している

総編集=山口　徹　虎の門病院院長
北原光夫　東京都済生会向島病院院長

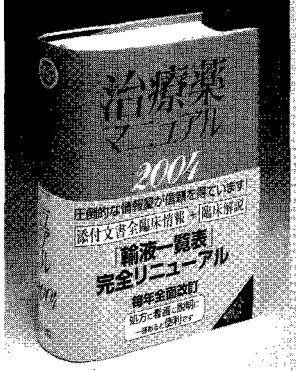


日常の診療に必携の治療年鑑

■1056専門医が1084疾患項目の治療の実際を紹介（項目数、執筆者数とも過去最多）
■本文各項目はすべて新執筆者により全面新訂（同一項目で過去3年間の執筆者の重複なし）
■増加する新薬に対応。Px処方例では薬剤を商品名で記載。健保適応外処方については及ぶかぎり明示
■カラー図譜（正常CT・MRI解剖と肺・肝の区域図、胸部・腹部リンパ節の部位、輸入感染症の世界分布）、基準値一覧、急性中毒、抗菌薬、漢方製剤、皮膚外用薬、薬物の副作用と相互作用、新薬・医薬品等安全性情報、介護保険、診療ガイドラインに関する付録を収載
■研修医、看護職、薬剤師にも役立つ情報が満載

●デスク判　B5　頁1670　2004年　定価19,950円（本体19,000円+税5%）　[ISBN4-260-10293-1]
●ポケット判　B6　頁1670　2004年　定価15,225円（本体14,500円+税5%）　[ISBN4-260-10294-X]

全医療関係者必携の薬剤データブック



治療薬マニュアル 2004

圧倒的な情報量が信頼を得ています！

監修=高久史麿　自治医科大学学長
矢崎義雄　国立国際医療センター総長
編集=関　顕　経済産業省診療所所長
北原光夫　東京都済生会向島病院院長
上野文昭　大船中央病院内科部長
越前宏俊　明治薬科大学教授

【本書の特徴】
■ 添付文書全臨床情報 ＋ 臨床解説
■「輸液一覧表」完全リニューアル
■ 毎年全面改訂
■ 処方に看護に説明に1冊あると便利です
（見出しタックインデックス付き）
■ 2003年12月12日付薬価基準収載新薬まで収録
（ただし後発品は2003年7月4日付収載分まで）

【その他の改訂点】
①「電解質製剤」の章を新設し、すっきりとした構成に
②法改正に対応し「生物由来製品」「特定生物由来製品」を表示
③〈薬効別一覧表〉臨床解説を一段と充実
④「抗菌薬」の章をリニューアルし、記述を一新
⑤巻頭付録を巻末に移し、シンプルな構成に
⑥〈服薬指導上の注意〉〈看護上の注意〉〈訪問診療上の注意〉を適宜追加
⑦〈重要薬解説〉さらに充実
⑧〈適用外使用〉に関する臨床解説拡充

●B6　頁2192　2004年　定価5,250円（本体5,000円+税5%）　[ISBN4-260-10541-8]
●CD-ROM　2004年　定価5,250円（本体5,000円+税5%）　[ISBN4-260-19856-4]

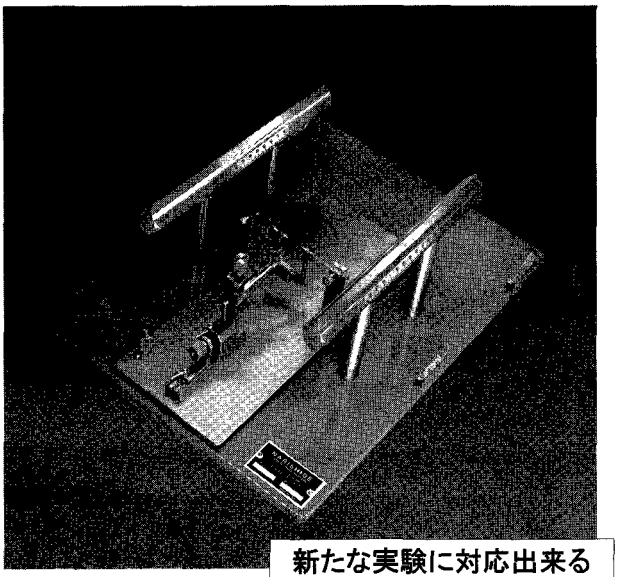
医学書院　〒113-8719 東京都文京区本郷5-24-3 （販売部） TEL 03-3817-5657 FAX 03-3815-7804
E-mail sd@igaku-shoin.co.jp http://www.igaku-shoin.co.jp 振替 00170-9-96693

消費税率変更の場合、上記定価は税率の差額分変更になります。

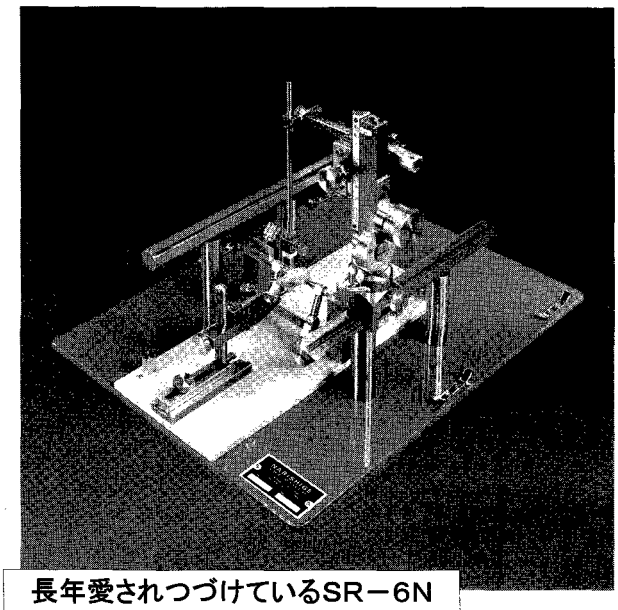
NARISHIGE JAPAN NARISHIGE

ラット・マウスなど小動物の固定はおまかせください。

小動物用固定装置



新たな実験に対応出来る
新型SRS－6



長年愛されつづけているSR－6N

最近、固定器関係の実験が見直されてきております。特にマウスやラットを使用しての実験が多く行われております。
成茂科学器械研究所は、長年愛されつづけている固定器から、多くの先生方からの要望・アドバイス・成茂科学器械研究所の技術力が生み出した、その時代の実験に合わせた最新の固定器をご用意し展示しております！
是非お気軽にお立ちより下さい。

弊社の実績・経験でお客様にあった
オリジナル品の製作も承っております。

株式会社　成茂科学器械研究所
〒157－0062　東京都世田谷区南烏山4丁目27番9号
TEL.（03）3308－8233　FAX.（03）3308－2005
e-mail：sales@narishige.co.jp
Web site：http://www.narishige.co.jp

ご質問・ご要望などございましたら、上記までお問合せ下さい。

KURABO

研究専用装置

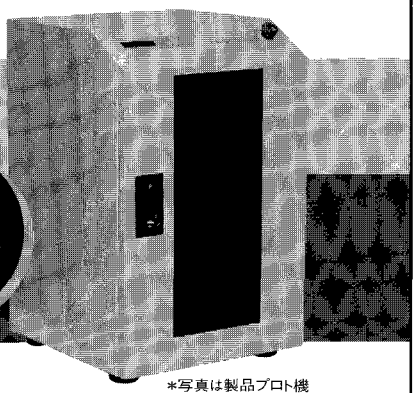
面倒なプレパラートの顕微鏡解析が省力化!!

プレパラートイメージャー

（プレパラート自動画像撮込装置）

AP-200

2004年 今秋 発売!!

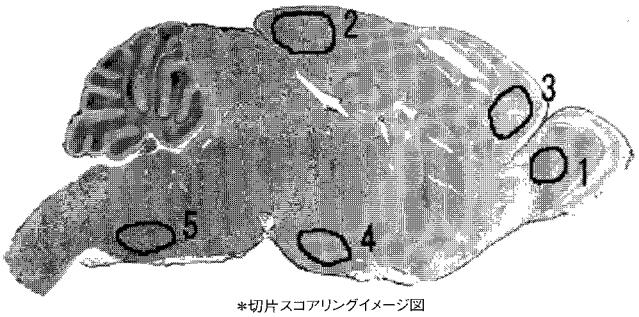


*写真は製品プロト機

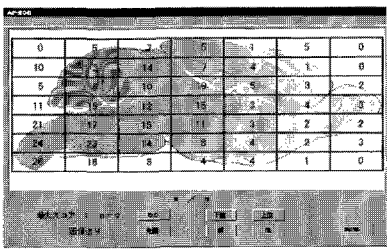
組織切片全体像の色ムラ（顕微鏡データ貼り合わせ時）が解消!

特許出願中!!

- ●高解像度のスキャナで自動的に組織切片全体を撮影・保存!
- ●各組織切片をグリッディングし、発現量を定量化!
- ●一度に組織切片全体を解析できる為、色ムラのない切片全体像を表示!
- ●プレパラートは最大200枚セット（W76×D26mm）可能!
- ●オプションで撮り込み画像の三次元化やバーコード認識が可能!



*切片スコアリングイメージ図



◆運転内容

プレパラートを収納ラックにセット（準備）

1 自動で収納ラックから スキャナ部へ搬送

2 自動でプレパラート画像 撮り込み （全体スキャン／サンプル部画像撮り込み ／画像ストレージ）

3 自動でスキャナ部から 収納ラックに返送

*1〜3の自動運転

◆仕様

モデル	AP-200
サンプルセット数	最大200枚（W76×D26mm） 最大100枚（W76×D52mm）
対応プレパラート	上記サイズに対応
画像撮り込み方式	スキャナ方式 イメージデータサイズ（約80MB／10mm角）
画像ソフト	画像のシリアル表示・ランダム表示 拡大・縮小 全画面一括画像処理（色・コントラスト補正等） 計測（長さ、体積）、マーカー、インデックス
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オプション	バーコード認識・3D構築（任意断面等）
本体外寸	約W700×D600×H700mm（卓上）
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