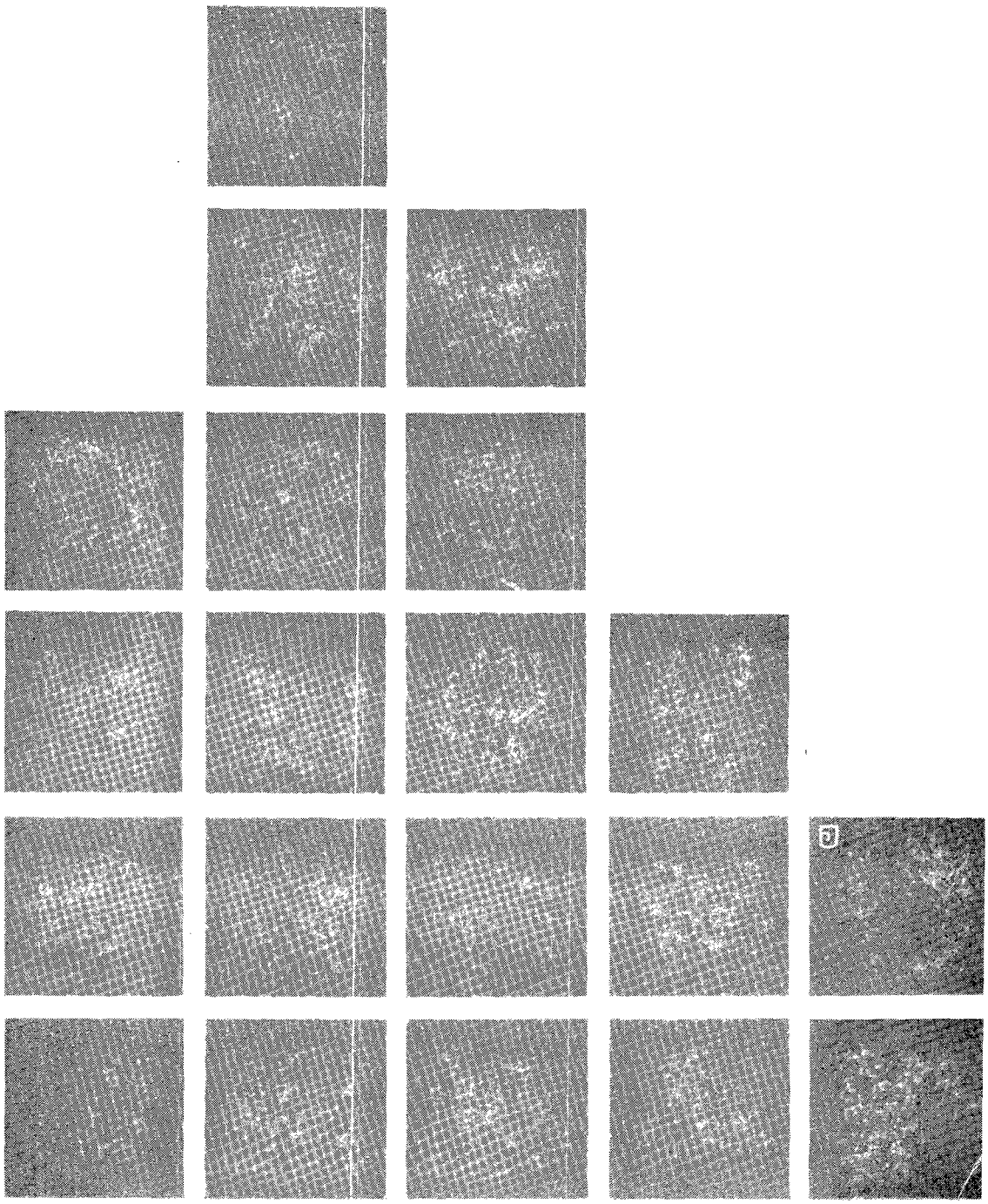
神経化学

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神 経 化 学 懇 話 会

—— シンポジウム ——

INTERNATIONAL SYMPOSIUM ON ENZYMIC ACTIVITY OF THE CENTRAL NERVOUS SYSTEM

Gothenburg, Sweden
June 17-21, 1962

Commission of Neurochemistry, World Federation of Neurology によつて organize されたこの Symposium は次の 6 部にわかれていた．提出された全論文の表題をかゝげ，特に第 3 部については抄録をそのまゝかゝげる．ある意味でまとまつたものとなつており，また新しい問題をふくんでいると思われるが故である．なお，第 3 部以外については中教授（大阪市大）が次号にくわしく紹介されることに予定されている(編集部)．

Section I : Enzymology of energetics and ionic movement
Section II : Enzymic mechanisms in amino acid and protein metabolism
Section III : Enzymic mechanisms in lipid metabolism
Section IV : Localisation and distribution of enzymic activity
Section V : Enzymic activity related to neural function
Section VI : Enzymic changes in disease

Section I : Enzymology of energetics and ionic movement

1. The metabolic pattern of the brain : A study of the participation of endogenous and exogenous metabolites in the oxidative metabolism of the brain, in brain perfusion experiment *in vivo*, *Geiger A.*, *Otsuki S.* & *Gombos G.*
2. Adenosine triphosphatase and other activities of microsomal subfractions from cerebral cortex, examined in relation to active cation transport, *Schwartz A.*, *Bachelard H.S.* & *McIlwain H.*
3. The enzymic mechanism for the active transport of sodium ions, *Järnefelt J.*
4. Inhibition and activation of sodium transport across the blood-brain barrier, *Quadbeck G.*
5. Chemical properties of brain mitochondria, *Løvtrup S.*
6. The operation of the Krebs cycle in brain mitochondrial fractions, *Balazs R.* & *Richter D.*

7. Glycolysis and ATP generation in isolated nuclei from newborn rat brain, *Rappoport D.A.* & *Fritz R.R.*
8. ATP-ases in the nerve cell, the neuronal membrane and in the glia, *Hydén H.*
9. Monovalent cations and the respiration of nervous tissue, *Hertz L.*, *Clausen T.* & *Schou M.*
10. Lipoic acid in acetate activation and transfer reactions of the brain, *Gal E.M.*
11. Effect of glutamic acid on phosphorylative activity in cerebral tissue *in vitro*, *Cohen M.M.*, *Cohen H.P.* & *Chain E.B.*
12. Active transport of amino acids in brain slices, *Tsukada Y.*, *Hirano S.* & *Matsutani T.*

Section II: Enzymic mechanisms in amino acid and protein metabolism

1. Incorporation of ^{14}C-precursors into rat brain proteolipids *in vitro*, *Mokrasch L.C.*
2. Enzymic mechanisms in protein biosynthesis of brain tissue, *Takahashi Y.*, *Mase K.*, *Satake M.* & *Ogata K.*
3. Enzymic systems converting carbohydrate into protein in brain and other organs, *Vrba R.* & *Caitonde M.K.*
4. Enzymology and function of the γ-aminobutyric acid system, *Roberts E.*
5. Metabolism of γ-aminobutyric acid *in vivo*, *Baxter C.F.*
6. Evidence for mechanisms subserving deamidation and amidation of cerebral proteins, *Tower D.B.* & *Wherrett J.R.*
7. A study of amino acid metabolism in rat brain using [^{14}C]-glucose and inhibitors, *Cremer J.E.*
8. Effect of glucose on endogenous free amino acids in rat brain tissue, *De Waart C.*
9. Activation of amino acids by "pH 5 enzymes" in the developing nervous system of guinea pigs, *Wender M.* & *Heerowski M.*
10. On the role of catechol-O-methyl transferase in brain, *Carlsson A.*
11. The methylation of normorphine in rat brain, *Clouet D.H.*
12. Reversible inhibition of brain monoamine oxidase *in vitro* and *in vivo*, *Long R.F.*

13. Effect of hydrazides and hydrazones of pyridoxal-5-phosphate on the cerebral glutamic decarboxylase and pyridoxal phosphokinase *Gonnard P.*

14. Vasopressin and protein synthesis in the hypothalamus of the dog, *Sachs H.*

Section III: Enzymic mechanisms in lipid metabolism

Chairman: J. Folch-Pi

1

Phosphatide metabolism in the developing nervous system

by

W.C. McMurray and R.J. Rossiter

Brain dispersions from rats of various ages between birth and one year were incubated *in vitro* with radioactive phosphatide precursors. The phosphatides were extracted, purified, and separated into an alkali-labile fraction (chiefly glycerophosphatides) and an alkali-stable fraction (plasmalogens and sphingomyelin). The incorporation of cytidine diphosphate [^{14}C]-choline into the alkali-labile phosphatides (lecithin) was high in the first few days after birth and decreased thereafter. Labelling of the alkali-stable phosphatide fraction was very low at birth and increased approximately 5-fold by the 20th day when myelination is maximal. The incorporation in the alkali-stable fraction during the first 3 months was approximately 10% of that in the lecithin fraction. Thereafter, the labelling of the alkali-stable phosphatides decreased markedly so that by one year it was of the same order of magnitude as in the new born. These results are consistent with the view that lecithin synthesis during early development of the nervous system is involved chiefly in the growth of unmyelinated components, while incorporation into the alkali-stable fraction is associated with the later formation of myelin.

Incorporation of cytidine diphosphate [^{14}C]-choline into the alkali-stable phosphatides was not increased by a variety of *N*-acylsphingosine compounds (Ceramides) which markedly stimulated sphingomyelin synthesis in liver dispersions (*Sribney & Kennedy*, 1958). The labelled lipid possessed a linkage which was labile to mild acid, and it migrated with lecithin during gradient elution from a silicic acid column. The results are consistent with the suggestion that the labelled lipid is a choline plasmalogen and that this phosphatide is specifically involved in the deposition of myelin during development.

[3-^{14}C]-Serine was incorported into both the glycerophosphatide fraction and the alkali-stable fraction. Labelling of the latter lipid fraction was much lower than with cytidine diphosphate [^{14}C]-choline as precursor, and remained con-

stant throughout development. On the other hand, incorporation into glycerophosphatide fraction was highest during the period of active myelination.

Sribney, M. & Kennedy, E.P. (1958). J. Biol. Chem. **233**, 1315.

2

The metabolism of brain phosphoinositides

by

R.B. Ellis and J.N. Hawthorne

At least three inositol lipids are found in brain tissue. Those recently characterized appear to have the following structures (*Dittmer & Dawson*, 1961; *Grado & Ballou*, 1961; *Ellis & Hawthorne*, 1961): (I) phosphatidylinositol, (II) phoshpatidylinositol phosphate and (III) phosphatidylinositol diphosphate. Compounds II and III differ from most phospholipids in having phosphomonoester groups. Since it seemed likely that such groups occur specifically in the lipids of nervous tissue, their turn-over has been studied with the aid of ^{32}P.

Rabbits were injected sub-arachnoidally carrier-free $Na_2H^{32}PO_4$ and killed after 6 hours. Brain phospholipids were extracted and hydrolysed under mild alkaline conditions. The resulting phosphate esters were separated by ion-exchange chromatography and the relative specific activities of the corresponding phospholipids calculated. These were as follows: phosphatidylcholine, 17; phosphatidylethanolamine, 4; inositide I, approximately 50; inositide II, 202; inositide III, 306. The figures suggested that the monoester groups in compounds II and III had a considerably higher turn-over than the diester group on the inositol 1-position. This was confirmed for compound II by treatment with acid phosphatase (*Ellis & Hawthorne*, 1961). The inorganic phosphate released had a specific activity 6.7 times greater than the diester phosphate remaining.

The turn-over rates suggest that there is a biosynthetic pathway from phosphatidylinositol via compound II to compound III. It is possible that ATP is involved in the two succesive phosphorylations, though an inositol kinase has not been found in animal tissue (*Hübscher*, 1960). Studies of the biosynthetic system are in progress.

The higher phosphoinoditides may be characteristic of nervous tissue. Further studies of their metabolism should be rewarding. The high turn-over of phosphomonoester groups could be connected with the transport of cations.

Dittmer, J.C. & Dawson, R.M.C. (1961). Biochem. J., **81**, 535.

Ellis, R.B. & Hawthorne, J.N. (1961). Biochim. Biophys. Acta, **51**, 385.

Grado, C. & Ballou, C.E. (1961). J. biol. Chem., **236**, 54.
Hübscher, G. (1960). Quoted in review, J. Lipid Research, **1**, 255.

3

Enzymatic breakdown of triphosphoinositide by water-soluble extracts of brain

by

W. Thompson and R.M.C. Dawson

The rapid hydrolysis of inositol-rich brain lipid fractions by brain homogenates with the liberation of inorganic P and inositol monophosphate was first reported by *Sloane-Stanley* (1953) and *Rodnight* (1956). Interpretation of these results is difficult because it is now known that such lipid fractions are a mixture of mono-, di- and tri-phosphoinositide (*Dittmer & Dawson*, 1961; *Dawson & Dittmer*, 1961; *Brockerhoff & Ballou*, 1961 a).

When purified preparations of triphosphoinositide were incubated with aqueous extracts of ox brain acetone-powders there was a rapid liberation of inorganic P and smaller amounts of water-soluble phosphate esters. At the same time diphosphoinositide was produced and an prolonging the incubation this was dephosphorylated to monophosphoinositide.

Addition of ether to the system reduced the formation of monophosphoinositide and inorganic P. However, there was a greatly increased liberation of water-soluble phosphate esters and a concomitant release of acetone-soluble diglyceride. The phosphate esters were identified as being predominantly inositol triphosphate with lesser amounts of inositol diphosphate.

This evidence is consistent with the following reactions:

1) Triphosphoinositide $+H_2O \rightarrow$ diphosphoinositide + inorganic P
$\downarrow +H_2O$
monophosphoinoside + inorganic P

2) Triphosphoinositide $+H_2O \rightarrow$ inositol triphosphate + diglyceride
(inositol diphosphate)

Reaction 1 is stimulated by the addition of NaCl and KCl, reaching an optimum at between 0.1 and 0.2 molar. It is apparently not catalysed by a non-specific phosphomonesterase since, under identical conditions, acid (semen) and alkaline (intestine) phosphatase caused little substrate hydrolysis.

Recent isotopic experiments by *Brockerhoff & Ballou* (1961 b) and *Ellis & Hawthorne* (1961) have suggested that triphosphoinositide can be synthesized by the stepwise phosphorylation of monophosphoinositide through diphosphoinositide. This coupled with the present evidence for the stepwide dephospho-

rylation of triphosphoinositide could constitute a phosphorylation-dephosphorylation cycle in nervous tissue. Since triphosphoinositide is knowu to be tightly bound to protein or peptide, and as such could occur as part of a membrane complex, it is conceivable that a cycle of this type might be of importance in the transport of cationic metabolites.

Brockerhoff, H. & Ballou, C.E. (1961 a). J. Biol. Chem., **236**, 1907.
Brockerhoff, H. & Ballou, C.E. (1961 b). Biochem. J., **81**, 35p.
Dawson, R.M.C. & Dittmer, J.C. (1961). Biochem. J., **81**, 540.
Dittmer, J.C. & Dawson R.M.C. (1961). Biochem. J., **81**, 535.
Ellis, R.B. & Hawthorne, J.N. (1961). 5th Int. Cong. Biochem., Moscow, Pergamon Press, p. 545.
Rodnight, R. (1956). Biochem. J., **63**, 233.
Sloane-Stanley, G.H. (1953). Biochem. J., **53**, 613.

4

The effect of chlorpromazine on phosphate exchange in the phospholipids of rat brain *in vivo* and *in vitro*

by

E.F. Marshall and G.B. Ansell

It has been considered that the incorporation of ^{32}P orthophosphate into brain phospholipids is depressed *in vivo* by large doses of chlorpromazine (*Wase, Christensen & Polley*, 1956; *Ansell & Dohmen*, 1956). At concentrations of 10^{-4} *M in vitro* the uptake of phosphate into phosphatidylcholine and phosphatidylethanolamine was also depressed but the uptake into a phosphoinositide and phosphatidic acid was significantly enhanced. Higher concentrations (10^{-3} *M*) depressed incorporation into all phospolipids (*Magee, Berry & Rossiter*, 1956). Further investigations have now been carried out *in vivo* and *in vitro*.

Litter mates were subjected to chronic treatment with chlorpromazine (10 mg/kg body weight/day) for a fortnight and maintained in a warm atmosphere with litter mate controls. The capacity of the brain phospholipids to incorporate [^{32}P]-orthophosphate over a short exchange period was measured. There was a significant depression for phosphatidylcholine and phosphatidylinositol but other alkali-labile phospholipids were unaffected. No effect was obtained on phosphorylethanolamine or phosphorylcholine. Those results will be compared with those obtained after a single injection of chlorpromazine.

In subsequent experiments brain cortex slices were incubated in a Krebs-Henseleit medium containing glucose. With a concentration of 10^{-3} *M* chlor-

promazine there was complete inhibition of the uptake of [^{32}P]-orthophosphate into the choline-, ethanolamin-, and serine-containing phospholipids, a 75% inhibition of uptake into phosphatidylinositol and no action on phosphatidic acid. Lower concentrations (10^{-4} *M*), however, stimulated phosphate exchange in phosphatidic acid but had no effect on other phospholipids in the medium used (*Magee, Berry & Rossiter*, 1956). That the point of action of chlorpromazine on the biosyntheis of phosphatidylcholine is subsequent to the formation of phosphorylcholine has been corroborated by experiments with ^{32}-P-labelled phosphorylcholine.

Addition of adenosine triphosphate (10^{-3} *M*-ATP) to the ^{32}P-containing medium simultaneously with chlorpromazine had no effect on the action of the drug. If, however, the ATP, ^{32}P and slices were equilibrated for 20 minutes before the addition of chlorpromazine, the uptake into phosphatidic acid and phosphatidylinositol was partially restored to control levels but the uptake into phosphotidylcholine and -ethanolamine was still completely inhibited. These restlts will be discussed in relation to the several independent investigations of the action of chlorpromazine on brain lipid metabolism.

Ansell, G.B. & Dohmen, H. (1956). J. Neurochem., **1**, 150.

Magee, W.L., Berry, J.F. & Rossiter, R.J. (1956). Biochim. Biophys. Acta, **21**, 408,

Wase, A.W., Christensen, J. & Polley, E. (1955). Arch. Neurol. Psychiat. **73**, 54.

5

The effect of psychotropic agents on brain lipid biosynthesis and metabolism

by

P. Paoletti, E. Grossi and R. Fumagalli

In previous experiments a psychotropic ageut, chlorpromazine (CPZ), has been shown to exert a remarkable effect on brain phospholipid synthesis. CPZ, at low doses, increases the incorporation of 1-^{14}C acetate into the brain phospholipids of growing rats both *in vivo* and *in vitro* (*Grossi, Paoletti & Paoletti*, 1960). As shown by other investigators, CPZ, in higher doses, decreases ^{32}P incorporation and turnover rate in brain phospholipids, particularly into phosphoinositide, phosphatidylcholine and -ethanolamine fractions (*Ansell & Dohmen*, 1956). Other psychotropic agents, Azacyclonol (*Rossiter*, 1957) and Pipradol (*Magge et al.*, 1957) stimulate the incorporation of ^{32}P into brain phospholipids. 2-Dimethylaminoethanol has been reported to raise the incorporation of ^{32}P

into brain phosphatidyl choline and phosphatidic acid and to increase phosphorylcholine levels both in liver and in brain (*Ansell & Spanner*, 1961).

We have recently investigated the effects of imipramine (Tofranil) and of desmethylimipramine (DMI) on lipid metabolism. DMI is the metabolite of imipramine recently shown to be responsible for the antidepressant action of the drug(*Gillette et al.*, 1961). Both drugs at levels as low as 10^{-5} *M*, stimulate the incorporation of [1-^{14}C] acetate into the fatty acids of brain phospholipids *in vitro*. No effect is evident on the biosynthesis of cholesterol or the fatty acids of neutral fat. This action is confined to nervous tissue. DMI, when administered *in vivo* in doses as low as 3.4 mg/Kg, stimulates brain phospholipid fatty acid synthesis, while Tofranil shows a less marked and delayed effect.

In another series of experiments, the fatty acid turnover of brain phospholipids in animals treated with DMI was investigated. Newborn rats were injected with [1-^{14}C]-acetate and the single fatty acids isolated from the brain phospholipids and assayed for specific radioactivity at different times (1 to 30 days after [1-^{14}C]-acetate administration). The effect of DMI treatment in these experiments will be evaluated.

The implications of the biochemical effects or brain lipids in relation to the pharmacological mode of action of these psychotropic agents are discussed.

Ansell, G.B. & Dohmen, H. (1956). J. Neurochem., **1**, 150.

Ansell, G.B. & Spanner, S. (1961). In *Neuropsychopharmacology*, **2** (ed. Rothlin, E.), p. 458, Elsevier: Amsterdam.

Gillette, J.R., Dingell, J.V., Sulsen, F., Kuntzman, R. & Brodie, B.B. (1961). Experientia, **17**, 417.

Grossi, E., Paoletti, P. & Paoletti, R. (1960). J. Neurochem. **6**, 73.

Magee, W.L., Pritchard, E.T. & Strickland, K.P. (1957). Federation Proc. **16**, 215.

Rossiter, R.J. (1957). In *Metabolism of the Nervous System* (ed. Richter, D.) p. 355, Pergamon: London.

6

The total enzymic synthesis of cerebrosides

by

R. O. Brady

The elucidation of the complete sequence of reactions for the biosynthesis of the cerebroside molecule has now been accomplished using an enzyme preparation obtained from young rat brain tissue. Earlier studies on the formation of sphingosine and psychosine suggested that the acylation of the secondary

amino group of O^1-galactosyl-sphingosine (psychosine) might be the terminal step in this process. Recent experiments with stearyl-1-[^{14}C]-coenzyme A confirm this assumption and indicate that the formation of cerebrosides occurs according to the following scheme:

(1) Palmityl coenzyme A + TPNH + H^+ → palmitic aldehyde + TPN^+

(2) Palmitic aldehyde + serine-pyridoxal phsophate-Mn^{++} → dihydrosphingosine + CO_2 + pyridoxal phosphate + Mn^{++}

(3) Dihydrosphingosine + flavoprotein → sphingosine + flavoprotein•2H

(4) Sphingosine + uridine diphosphate galactose → psychosine + uridine diphosphate

(5) Psychosine + stearyl CoA → N-stearyl-O^1-galactosyl-sphingosine + coenzyme A.

7

Die Wirkung verschiedener Lipoide auf die Lysolezithin-Zytolyse

von

J. Domonkos

Das Vorhandensein von Lysolezithin ist in mehreren Organen, in erster Linie im Lipoidextrakt des Gehirns nachgewiesen worden. Nach *Thompson* und Mitarbeitern enthält das Gehirngewebe Phospholipase-A, auf deren Wirkung Lysolezithin entstehen kann.

Bei der Untersuchung der Bildungsmöglichkeiten für die Lysophosphatide beobachtete Verfasser den Abbau Verschiedener Phosphatide im Gehirngewebe. Nach den bishergen Untersuchungen ist im Laufe der vierstündigen Inkubation von Gehirnhomogenisaten ein Abbau der Plasmalogene bzw. die Bildung freien Fettaldehyds festzustellen. Von den Gehirn-Phosphatiden haben sich die Plasmalogene als die empfindlichsten, am leichtesten zerfallende Verbindungen erwiesen. Welche Rolle dem Abbau der Plasmalogene in der Bildung der Lysophosphatide zukommt, werden weitere Untersuchungen zu entscheiden haben.

Das auch unter physiologischen Bedingungen vorkommende Lysolezithin schädigt die Zellen nicht. Um der Ursache hierfür näher zu kommen, hat Verfasser die Wirkung der verschiedenen Hirnlipoide und Fettsäuren auf die Lysolezithin-Zytolyse untersucht. Es zeigte sich, dass nicht nur das Cholesterin, sondern auch die Phosphatide und andere Lipoide die Wirkung des Lysolezithins hemmen. Cholesterin setzt in der Konzentration von 1×10^{-5} *M* die hämolytische Aktivität des Lysolezithins (1.5×10^{-5} *M*) um 90–100%, Kolaminkephalin, Lezithin und Sphingomyelin um 75–80% und Cerebroside um 60–65% herab. Die lankettigen Fettsäuren (Palmitinsäure, Stearinsäure, Ölsäure, Linol-

säure und Linolensäure) bewirken in Konzentrationen von 3.8×10^{-6} *M* eine Steigerung des durch 1×10^{-5} *M* Lysolezithin entfalteten hämolytischen Effekts um 60-90%.

Die Methylester der Fettsäuren dagegen haben—in der gleichen Konzentration wie die Fettsäuren zur Anwendung gebracht—eine 40-60% ige Hemmung der Lysolezithin-Hämolyse zur Folge.

8

The enzymic synthesis of hexosamine
in nervous tissue
by
N. Canal and L. Frattola

In the last ten years the authors have been interested in the hexosamine-containing components of nervous tissue. Amino-sugars (mainly glucosamine and galactosamine) are essential constituents of cerebral lipids (gangliosides), and are present in the mucopolysacchaccharides of the ground substance of grey matter; this probably plays a role in the functioning of the blood-brain barrier. In some pathological conditions (familial leucodystrophy, amaurotic idiocy) a marked rise of hexosamine-containing compounds in nervous tissue has been demonstrated.

While the topographical distribution of hexosamine and its changing levels with increasing age have been extensively investigated, data on the mechanism of its biosynthesis in nevous tissue are still lacking. The present paper deals with the enzymic step by which glucosamine is formed from glucose-6-phosphate and glutamine. This step was first demonstrated by Leloir and Cardini in *Neurospora crassa* and subsequently found in many animal tissues.

In rabbit brain the distribution of the glucosamine-synthesizing activity is, in decreasing order: cortical grey matter, cerebellum, brsin stem, white matter and basal ganglia. In the whole brain of developing rats the highest activity is found some days before birth; it falls progressively in brains examined at the age of one day, one week, fifteen days and thirty days. The hexosamine-synthesizing activity has been studied also in human normal and neoplastic nervous tissue taken during intracranial operations.

The results will be discussed.

Section IV: Localisation and distribution of enzymic activity

1. A comparative study on the distribution of certain enzymes and their metabolic significance in the cerebellum of the rat, *Tewari H.B. & Bourne G.H.*

2. The fine localization of certain hydrolytic oxidative and hydrogen-transfer enzymes within living nerve-cells of vertebrates, *David G.B., De Almeida D.F., Castro G. De O. & Brown A.W.*

3. Über die histochemische Lokalisierung der unspezifischen Cholinesterase im Zentralnervensystem der Ratte, *Teocharov B.*

4. Regional, comparative and ontogenetic studies on lactate dehydrogenase of nervous tissue, *Bonavita V.*

5. Brain guanase : Purification, properties and regional distribution in monkey brain, *Mansoor, M., Kalyankar G.D. & Talwar G.P.*

6. Changes durin maturation in the enzmic activity and composition of subcellular fractions obtained from rat brain, *Davison A.N. & Gregson N.A.*

7. Observations on the differential distribution of acid and neutral proteinases in brain subfractions, *Marks N. & Lajtha A.*

8. A method for the histochemical demonstration of noradrenaline in nervous tissue, *Falck B. & Torp A.*

9. The distribution of monoamine oxidase in the brain and pituitary gland of the albino rat, *Smith B.*

10. Aktivitätsanstieg von Enzymen der Oxydoreduktion in Axonen des zentralen und peripheren Nervensystems nach Durchschneidung, *Kreutzberg G.*

11. Localization of phosphatases and oxidative enzymes in neural tissue. Effect of post-mortem autolysis and comparison with cytological localization in Tay-Sachs' disease, *Lazarus S.S., Wallace B.J. & Volk B.W.*

12. Histochemical research on some aspects of acetylcholinesterase and its inhibitors in the CNS, *De Giacomo P.*

Section V : Enzymic activity related to neural function

1. Effects of environmental complexity and training on brain cholinesterase and brain anatomy in the rat, *Bennett, E.L., Krech D. & Rosenzweig M.R.*

2. The enzymic activation of the electroencephalogram, *Serra C.*

3. Solitäre hyperaktive Nervenzellen, *Thomas E. & Pearse A.G.E.*

4. Bonnd acetylcholine and entrapped cytoplasm in subcellular frabtions of guinea-pig brain homogenates, *Johnson M.K. & Whittaker V.P.*

5. Electrophoretic analysis of human brain esterases, *Barron K.D., Bernsohn J. & Hess A.R.*
6. Electrophoretic separation of rat brain esterases in starch gel medium, *Lagnado J.R.*
7. Electrophoretic separation of enzymes in the central nervous system, *Van der Helm H.J.*
8. Multiple forms of dehydrogenase activity revealed with tetrazolium salts. Cytoplasmic enzymes of the central and peripheral nevous system, *Van Sande M., Lowenthal A. & Karcher D.*
9. The succinic dehydrogenase activity in the reactive glia, *Mossakowski M.J.*
10. The presence of cholinesterases in long-term cultures and subcultures of adult mammalian brain cells, *Geiger R.S. & Stone W.G.*
11. The effect of central nervous system drugs on the enzyme activity of rat brain, *Albaum H.G.*

Lection VI: Enzymic changes in disease

1. Microchemical enzyme studies of the rat nervous system in experimental thiamine deficiency, *Dreyfus P.M.*
2. The toxicity of copper in the brain of the pigeon and its attempted reversal, *Peters R.A. & Walshe J.M.*
3. Inhibition of enzymic activity by intravenous drug therapy, *Dunlop E.*
4. Morphological and enzymic alterations in reacting glia, *Koenig H. & Barron K.D.*
5. Die Enzymaktivitat in Liquor bei Kindern mit Meningitis, *Szász G.*
6. Enzymic changes in the blood plasma in organic cerebral disease of children, *Naka, S., Mori H. & Nakaosa T.*
7. The generation of energy-rich phosphate in the erythrocyte in multiple sclerosis at different temperatures, *Huszák I., Szechenyi F. & Heiner L.*
8. Serum aldolase activity in degenerative neurological diseases, *Ionásescu V. & Luca N.*
9. Effects of the internal radiation (^{32}P) on the ATP content of the encephalon, *Egaña E. & Lanas J.*

10. Studies of traumatic lesions by means of determination of malic dehydrogenase activity, *Kaluza J.*

11. Changes in the activity of some enzymic systems determined in the cortical scar and at the level of secondary degenerative lesions, *Mison-Crighel N., Constantinescu E., Costa-Foru D. & Crighel E.*

12. Comportement enzymatique de la cicatrice cérébrale, *Hornet Th., Petresco Ar. & Anghelesco N.*

13. Brain enzyme changes in drum shock in the rat, *Nimomiya H., Greisman S.E., Buxton R.W. & Michaelis M.*

第5回　神経化学懇話会：1962年11月，大阪

11月17日（土），18日（日）の両日，大阪市東区森之宮西之町，大阪府厚生会館で開催された第5回神経化学懇話会の演題を記録としてとどめておく．この懇話会の記録も，昨年と同様“神経研究の進歩”に掲載される．

第1日——11月17日（土）——

シンポジウム：内因性精神病及び精神薄弱の代謝異常……〈座長　大阪市大・精神　中　　脩三）

S1.（15分）精神病者血清の Phenothiazine 脱水素酵素に及ぼす影響
……°木住野幸二（慈雲堂病院），上野陽三・植松宗悟・鶴岡孝人（日大・神経）

S2.（15分）分裂病患者の体液と解糖抑制との関連についての検討
……°伊藤　斉・森　文彦・開沢茂雄・三浦貞則（慶大・神経）

S3.（15分）精神分裂病患者の尿中有毒因子に関する研究……藤田繁雄（札幌医大・神経）

S4.（15分）精神分裂病患者の尿中アミンと精神薄弱患者の尿中アミノ酸
……°武貞昌志・金沢　彰・柿本泰男・中嶋照夫・佐野　勇（阪大・精神）

休　憩

S5.（15分）小児の器質的脳疾患における血液の酵素学的変化について
……°中脩三・森　一・川北幸男・鶴丸泰男・中筬豊久（大阪市大・精神，国立河内長野病院）

S6.（15分）実験的フェニールケトン尿症の神経化学的研究
……塚田裕三・°平野修助・松谷天星丸（東邦大・第2生理）

S7.（15分）兄妹に見られたフェニール焦性ブドウ酸精神薄弱症についての第1報（妹の場合）
……°東　漸・東　徹・市山卓爾（東病院），高坂睦年・秋山順子・寺尾　章（岡大・精神），森昭胤（岡大・陣内外科）

昼　食（委　員　会）

一　般　演　題

1.（15分）分離神経細胞に関する研究Ⅰ．分離法……浜中　昭彦（大阪市大・精神）

2.（15分）可逆性グリア反応の限界と意義（その1）——特に膜様原形質について——
……°中沢恒幸・富永　淳・原　洋二・笠原俊彦（慶大・神経）

3.（15分）無細胞系における燐タンパクの研究……°織壁永次・松井英男・石川晋次・島薗順雄（東大・生化）

4.（15分）発生過程における実験的代謝異常……°宮本侃治・平山　晧・成瀬　浩・秋元波留夫（東大・神経），黒川正則（東大・脳研）

5.（15分）頭部外傷の生化学第2報——Endogenous uncoupling factor（Mitochondria）——
……荒木千里・石井昌三（京大・第1外科），早石　修・久野　滋・°小沢和恵・辻　宏（京大・医化）

6.（15分）脳損傷による髄液並びに血清中酵素活性の変動——基礎的研究——
……°高倉公明・早川　勲・三宅浩之（東大・脳外科）

休　憩

7. (15分) 覚せい剤中毒の生化学的研究第6報——脳ミトコンドリアの解糖について——
…… 江副　勉・°加藤伸勝・二階堂　亨 (都立松沢病院)

8. (15分) 中枢神経刺激剤の脳組織呼吸及び蛋白質代謝に及ぼす影響
…… °中沢省三・古沢善文・原　隆夫 (新潟大・脳外科)

9. (15分) 向神経薬物と脳蛋白の相互反応 …… °清田一民・上野貞造・藤田英介・寺岡　奏 (熊大・神経)

10. (15分) 脳内アンモニヤ処理における肝機能の関与 …… °融　道男・小林健一・高橋　良・仮屋哲彦・小林暉佳・長尾佳子 (東医歯大・神経)

11. (15分) 脳代謝の動的観察 …… 相沢豊三・後藤文男・村松文雄・浜口勝彦・奈良昌治・°富田　稔・垣見亮次 (慶大・相沢内科)

特別講演：“Biological-Active Polypeptides” ……E. Werle (Klinisch-Chemisches Institut, Chirurgische Klinik der Universität München)

第2日——11月18日 (日)——

一般演題

12. (15分) L-DOPA (Dihydroxyphenylalanine) の脳切片への active transport について……吉田　博・°蟹池健一 (阪大・薬理)

13. (15分) β-フェニールエチルアミンの哺乳動物体内における存在ならびにその中枢作用
…… °中嶋照夫・柿本泰男・清水宏俊・佐野　勇 (阪大・精神)

14. (15分) Catechol の痙攣作用……松本淳治・西　博通 (阪大・高次神経研)

15. (15分) Picrotoxin 痙攣に対する脳 Catecholamine, 5-Hydroxyptamine および GABAの役割
…… °斎藤章二・徳永友喜子・多田　穣・望月安雄 (慶大・薬理)

休　憩

16. (15分) GABA と γ-メルカプト酪酸について
…… 西沢義人・児玉貞介・三宅正朝・菅原重道・°建石保子 (阪大・小児)

17. (15分) Homocarnosine (γ-Aminobutyryl-L-Histidine) に関する研究 (第1報)
…… °森　昭胤・杉生了亮・増川禎彦 (岡大・陣内外科), 高坂睦年 (岡山・精神)

18. (15分) 炭酸脱水酵素阻害剤の抗てんかん作用について……°谷向　弘・西村　健・乾　正・播口之朗 (阪大・神経)

19. (15分) 肝臓におけるトリプトファン代謝と視床下部自律中枢の役割……嶋津　孝 (阪大・第3解剖)

昼　食 (常任委員会)

総　会

シンポジウム：脳細胞成分 (Subcellular units) の生化学　(座長　東邦大・第2生理　塚田裕三)

S8. (30分) 電子顕微鏡で見た神経細胞の Subcellular units について ……藤原　忠 (大阪市大・電顕研)

S9. (20分) ミトコンドリアにおけるエネルギー転換の基本単位 (基本粒子) について
……小田琢三 (岡大・病理)

S10. (15分) 腦細胞内成分の分離に関する形態学的検討……
…… 井上章 °品川嘉也・榊村純正・入交昭彦・伊達慶宗（京大・生理）

S11. (15分) 腦のリボソームの調製と検定……山上 栄（大阪市大・精神）

休 憩 （座長 東大・腦研 黒川正則）

S12. (15分) 腦 Subcellular units の物質分布について……
…… °植村慶一・塚田裕三（東邦大・第2生理），飯田慈子（東大・理・生物化学）

S13. (10分) 腦 Subcellular units における ATPase について……
…… °荏原光夫・明石勝禎（東邦大・第2外科），植村慶一（東邦大・第2生理）

S14. (15分) γ-アミノ酪酸（GABA）と腦顆粒……佐野 馨（阪大・第2内科）

S15. (15分) 兎腦に於ける化学伝達物質（Cholinergic 及び Non-cholinergic）の細胞内分布
…… 井上 章・°片岡喜由・品川純子・今泉正臣・光村民子・矢原庄二（京大・生理）

S16. (15分) 腦アセチルコリンの構成 …… 黒川正則（東大・腦研），°町山幸輝・加藤誠・秋元波留夫（東大・神経）

神経化学文献

凡　例

1) * 印はこの号に，その雑誌から拾い出した神経化学文献表題がのつていることを示します．

2) 右側の巻（号）は，その雑誌のその巻（号）について目を通してしらべたことを示します．従つて右側に巻（号）が記載されていながら * 印のないものは，神経化学文献が見当らなかつたことを示すわけです．

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神経化学懇話会

会　　則

第1条　本会は神経化学懇話会(The Japanese Neurochemical Society)という．神經化学の発展を促し，会員相互の連絡，内外の関連機関との連絡を図ることを目的とする．

第2条　本会の会員は次のとおりとする．

1．正会員　本会の目的に賛同し，所定の会費を納める者．会費は1，000円とする．

2．賛助会員　本会の事業を援助し，1口以上の賛助会員費を納める者または団体．1口は年額1万円とする．

第3条　会員は本会の行う事業に参加し，会誌（神経化学）および印刷物の配布をうける．

第4条　会員になろうとする者は，会費を添えて所定様式の入会申込書を事務局に提出する．退会しようとする者は，退会届を提出する．

第5条

1．本会には役員として委員若干名をおく．委員は総会で選任する．委員の中から会長1名，常任委員若干名を定める．

2．会長は，毎年選出され，研究発表会および総会を主催する．

3．委員会は，必要に応じて会長が招集し，本会の運営につき必要な事項を審議する．

4．常任委員の中より常任委員長を選出する．常任委員会は，必要に応じて常任委員長が招集し，研究発表会の開催，会誌の編集に関する事項，その他会の目的達成に必要な事項を処理する．

第6条　委員の任期は2年として，再任を妨げない．

第7条　本会は年1回全国的研究発表会を行い，その際総会を開く．

第8条　総会において，会計報告および常任委員会で必要と認めた事項について承認をうる．

第9条　総会は会員の10分の1以上，委員会は委員の4分の1以上，常任委員の2分の1以上の出席を必要とする．但し委任状をみとめる．

第10条　本会の会計は，常任委員会の議決を務て，常任委員長が管理する．

第11条　この会則は，委員会および総会において，3分の2以上の議決を経て変更することが出来る．

第12条　会則施行についての細則は，常任委員会の議決を経て別に定める．

付　則

1）　本会の事務局は，当分の間，東邦大学医学部第2生理学教室（東京都大田区大森4の77）におく．

委　　員　（ＡＢＣ順・○印は常任委員）

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昭和37年11月18日第5回神経化学懇話会総会（於大阪）に於いて会則・委員は上記の如く決定した．

編集委員（ABC 順）

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