Synthesis *in vitro* of 1-alkyl-2-acyl- and 1,2-diacyl-*sn*-glycerophosphorylcholines and Ethanolamines by Neuronal, Glial and Synaptosomal Fractions from Adult Rabbit Brain

by

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Summary. The *in vitro* incorporation of radioactivity from cytidine-5′-diphosphate-[^{14}C]choline and cytidine-5′-diphosphate-[^{14}C]ethanolamine into choline and ethanolamine phosphoglycerides was increased markedly by 1-alkyl-2-acyl-*sn*-glycerols and 1,2-diacyl-*sn*-glycerols. The neuronal-glial (predominately astrologial) ratio of the specific activities of the choline phosphotransferase (E.C. 2.7.8.2.) ranged from 3.4–6.1 depending on the lipid substrate. The corresponding range of ratios for the ethanolamine phosphotransferase (E.C. 2.7.8.1.) was 4.5–4.9. The specific activities of the phosphotransferases in synaptosomal fractions were lower than in glial cells and less than 10% of the specific activity in microsomal fractions. Thus, the *de novo* synthesis of choline and ethanolamine phosphoglycerides is much more active in neuronal perikarya than in glial or synaptosomal fractions. In the presence of 1-alkyl-2-acyl-*sn*-glycerols, the formation of both alkylacyl and alk-l-enylacyl phosphoglycerides was increased. Choline plasmalogens (1-alk-1′-enyl-2-acyl-*sn*-glycerol-3-phosphorylcholines) were synthetized at a rate of 6.0 nmol/h/mg neuronal protein. This relatively high synthetic rate is consistent with the rapid rate of metabolic turnover found by other workers.

In the present study with neuronal perikarya, glial cells and synaptosomes, we have determined the degree of increase of choline and ethanolamine phosphoglyceride synthesis by additions of 1-alkyl-2-acyl-*sn*-glycerols and 1,2-diacyl-*sn*-glycerols and we have determined the synthetic activity for each of the different types of CPG and EPG in the presence and absence of 1-alkyl-2-acyl-*sn*-glycerols.

Materials and methods

Preparation of fractions enriched in neuronal cell bodies and in glial cells. The procedure used for preparing the cell fraction was that of Blomstrand & Hamberger [6, 7]. Five rabbits, weighing 2–3 kg, were anaesthetised with sodium pentobarbitone and perfused through the heart with Ringer's solution. The cerebral cortices were removed and sliced at 0.4 mm thickness and incubated in a shaking water bath at 37°C for 30 min. Incubation took place in a 500 ml Erlenmeyer flask, gassed with 100% oxygen, in 60 ml of the incubation medium containing (in final concentrations): 35 mM

Abbreviations used: -GPC—*sn*-glycero-3-phosphorylcholine, -GPE—*sn*-glycero-3-phosphorylethanolamine

tris-HCl buffer pH 7.4, 120 mM NaCl, 5 mM KCl, 5 mM Na phosphate buffer pH 7.4, 2.5 nM $MgCl_2$, 20 mM glucose and 2% Ficoll.

After incubation the slices were disrupted in the cold (0-4°C) by passage through increasingly fine nylon mesh down to 50 μm pore size. The cell suspension was diluted with sucrose-salt isolation medium (0.32 M sucrose; 120 mM NaCl, 10 mM tris-HCl buffer pH 7.4 and 0.5 mM EDTA) and centrifuged for 5 min at 150 *g*.

The resulting pellet was resuspended in 20% Ficoll solution. A discontinous Ficoll gradient was prepared in tubes for the MSE 3×65 ml swing-out rotor in the following manner: first 9 ml of 20% Ficoll+25% sucrose was added, followed by successive additions of 9 ml 32% Ficoll, 27 ml of cell suspension in 20% Ficoll, 9 ml of 16% Ficoll and finally 9 ml of 12.8% Ficoll. The tubes were centrifuged immediately for 110 min at 55,000 *g*. After centrifugation the neuronal fraction was obtained at the interface below the 32% Ficoll layer and the glial fraction at the interface below 12.8% Ficoll. Cell fractions were aspirated from the gradients, diluted with 500 ml of the sucrose-salt solution and collected as pellets by centrifugation for 15 min at 2,000 *g*. After morphological examination using light microscopy, the fractions were resuspended in 0.1 M tris-HCl buffer, pH 7.4 and used for experiments. By light microscopy, the neuronal fraction contained about 90% neuronal cells, with capillaries and free nuclei as the major contaminants and glial cells as a minor contaminant. Glial preparations appeared to be 80-90% pure with equal numbers of neurons and capillary endothelial cells as the contaminants. The glial cells were predominately astrocytes.

Isolation of synaptosomes. Synaptosomes were isolated by the method of Whittaker & Baker [26]. Brain cortices from anaesthetised and perfused rabbits were chopped and dispersed in nine volumes of 0.32 M sucrose, pH 7.4. The dispersion was centrifuged for 10 min at 1,000 *g*. The supernatant fraction was then centrifuged for 10 min at 18,000 *g* to sediment the crude mitochondrial fraction, which was resuspended in 0.32 M sucrose and placed over layers of 1.2 M, 1.0 M and 0.8 M sucrose in tubes for the MSE 3×65 swing-out rotor. The inclusion of the 1.0 M sucrose layer was a modification of the cited method.

After centrifugation at 55,000 *g* for 2 h, the band between 1.2 M and 1.0 M sucrose was collected, diluted with 0.32 M sucrose, and pelleted by centrifugation for 10 min at 18,000 *g*. The synaptosomal pellet was resuspended in the 0.1 tris-HCl buffer, pH 7.4 for use in the experiments.

Materials. ATP, CTP and pancreatic lipase were obtained from Sigma Chemical Company, St. Louis, Mo., U.S.A. CDP-[14C]choline and [1,2-14C]ethanolamine were from the Radiochemical Centre, Amersham, Bucks, England. Diacylcyclohexylcarbodiimide was obtained from Ralph Emanuel Ltd, Wembley, England. Dowex 1×8 200-400 mesh (formate form) was from Serva, Feinbiochemica, Heidelberg, FRG. *Chimera monstrosa* (ratfish) liver oil was from Western Chemical Industries Ltd, Vancouver, Canada. Bile salt were obtained from Polfa Laboratories, Warsaw, Poland. Egg lecithin and Silica Gel G were from E. Merck A. G. Darmstadt, FRG. Methyl borate was from Koch-Light Company Ltd, Colnbrook, England. Tween 20 was from Atlas-Goldschmidt GMBH, Essen, FRG. Phospholipase C, *Clostridium welchii*, was obtained from Calbiochem, Los Angeles, California.

1-Alkyl-2-acyl-*sn*-glycerols were prepared from *Chimera monstrosa* liver oil which was subjected to lipolysis as previously described [22]. The product was purified by TLC on a 0.5 mm layer of Silica Gel G by development with 90:10:8 (v/v/v) toluene-acetone-methyl borate. A 40 mM emulsion of alkylacylglycerols was prepared in 0.1 M tris-HCl, pH 7.4 containing 0.1% Tween 20 by sonication (MSE Ultrasonic Disintegrator, London) for 20 min. 1,2-Diacyl-*sn*-glycerols were prepared from egg lecithin by treatment with phospholipase C according to Renkonen [24]. A 40 mM emulsion of diacylglycerols was prepared as described above for alkylacylglycerols.

The preparation of [14C]phosphorylethanolamine was described by Ansell & Chojnacki [2]. CDP-[14C]ethanolamine was prepared by the method of Chojnacki & Metcalfe [9] with the use of CTP. The purification of the product was performed in all cases by chromatography on Dowex 1×8 200-400 mesh. The amounts of CDP-[14C]choline and CDP-[14C]ethanolamine were assayed spectrophotometrically at 260 and 280 nm.

Chemical determinations. Protein was determined according to Lowry *et al.* [21]. Phospholipid P was determined in lipid extracts according to Bartlett [3].

Incubation conditions. The incubation mixture contained in a final volume of 0.2 ml: 75 mM tris-HCl buffer, pH 7.6; 30 mM $MgCl_2$; 0.1 mM dithiothreitol; 1.26 mM CDP-[^{14}C]choline, 1.03×10^5 dpm, or 1.75 mM CDP-[^{14}C]ethanolamine, 9.6×10^5 dpm; 0.01% Tween 20; 4 mM alkylacylglycerols or 4 mM diacylglycerols; and either 150 µg neuronal protein, or 270 µg glial protein, or 305 µg synaptosomal protein.

Reactions were stopped after 30 and 60 min of incubation at 37°C by the addition of 3.0 ml of chloroform-methanol, 2:1 (v/v).

Extraction and analysis of phospholipids. Lipids were extracted according to Folch *et al.* [12]. In some experiments, portions of the total extract were taken for the assay of the radioactivity.

In other experiments, carrier lipids from rat brain (for CDP-ethanolamine experiments) or from rabbit heart (for CDP-choline experiments) were added before separation of diacyl, alkylacyl and alkenylacyl types of ethanolamine or choline phosphoglycerides by the thin-layer chromatography method of Horrocks & Sun [20].

Lipid areas were scraped from the TLC plates into counting vials, scintillation fluid [8] was added, and the radioactivity was measured in a Nuclear-Chicago Isocap/300 Liquid Scintillation System.

Results

Increased CPG and EPG synthesis by diradylglycerols.

Experiments were carried out to determine the capacity of neuronal perikarya, glial cells and synaptosomes for the synthesis of choline and ethanolamine phosphoglycerides, with and without alkylacylglycerols and diacylglycerols. Without exogenous diradylglycerols, the rate of ethanolamine phosphoglyceride synthesis was 4.4 times higher in neurons than in glial cells and 4.8 times higher in neurons than in synaptosomes (Table I). Similar ratios were found with exogenous diradylglycerols. In the presence of 4 mM alkylacylglycerols, ethanolamine phosphoglyceride synthesis was increased by 110, 90 and 90% and with 4 mM diacylglycerols the increase was 40, 40 and 50% for neurones, glial cells and synaptosomes, respectively.

TABLE I

Stimulation by added diradylglycerols of the incorporation of radioactivity into choline and ethanolamine phosphoglycerides of neuronal and glial cells and synaptosomes from adult rabbit brain

Addition	Incorporation into ethanolamine phosphoglycerides			Incorporation into choline phosphoglycerides		
	Neurons	Glia	Synaptosomes	Neurons	Glia	Synaptosomes
None	10.7	2.4	2.2	10.2	3.3	2.4
4 mM alkylacylglycerols	22.2	4.5	4.2	63.2	10.3	4.8
4 mM diacylglycerols	15.3	3.4	3.2	57.4	10.8	5.9

Incubation mixture: 75 mM tris-HCl (pH 7.6), 30 mM $MgCl_2$, 0.01% Tween 20, 1.75 mM CDP-[^{14}C]ethanolamine (9.6×10^5 dpm) or 1.26 mM CDP-[^{14}C]choline (1.03×10^5 dpm), 0.1 mM dithiothreitol and either 150 µg neuronal protein, por 270 µg glial protein, or 305 µg synaptosomal protein. The final volume was 0.2 ml and incubations were carried out at 37°C for 30 min and 60 min. Values (nmol/mg protein/h) are the means from experiments with two preparations each of neurons, glia and synaptosomes.

The rate of choline phosphoglyceride synthesis without exogenous diradylglycerols was 3.1 times higher in neurons than in glial cells and 4.2 times higher in neurons than in synaptosomes. Higher ratios were found with exogenous diradylglycerols. With added 4 mM alkylacylglycerols, the increase of choline phosphoglyceride synthesis was 6.2 fold in neuronal cells, 3.1 fold in glial cells and 2.0 fold in synaptosomes. The increase was 5.6, 3.3 and 2.4 fold for neuronal cells, glial cells and synaptosomes respectively with 4 mM diacylglycerols.

TABLE II

Effect of added alkylacylglycerols on the distribution of radioactivity in choline and ethanolamine phosphoglycerides of neuronal and glial cells from rabbit brain

Addition	Distribution in ethanolamine phosphoglycerides			Distribution in choline phosphoglycerides		
	diacyl -GPE	alkenylacyl -GPE	alkylacyl -GPE	diacyl -GPC	alkenylacyl -GPC	alkylacyl -GPC
	Neuronal cells					
None	6.9	2.3	1.2	9.4	2.9	1.4
4 mM alkylacyl- glycerols	2.7	3.0	9.0	10.5	6.0	46.2
	Glial cells					
None	1.8	0.2	0.04	2.7	0.8	1.2
4 mM alkylacyl- glycerols	2.8	0.6	2.7	2.5	1.5	10.0

Assays were performed as described in Table I. Values (nmol/mg protein/h) are the means from two preparations of cells

Distribution of radioactivity in the phosphoglycerides. With only the endogenous diglycerides, the neuronal and glial cells incorporated a substantial amount of radioactivity into diacyl GPE, a small amount into alkenylacyl-GPE and very little into alkylacyl-GPE (Table II). For calculation of the degree of increase of ether lipid synthesis the rates for alkylacyl-GPE and alkenylacyl-GPE were combined because the latter is formed from the former (see [17]). Without exogenous alkylacyl-glycerols, we believe that most of the newly synthesized alkylacyl-GPE is oxidized to alkenylacyl-GPE. Newly synthesized alkylacyl-GPE accumulates only when the rate of synthesis exceeds the rate of oxidation, namely when the formation of alkylacyl-GPE is stimulated by the addition of alkylacylglycerols. In the presence of 4 mM alkylacylglycerols with the neuronal cells, a 3.4-fold increase was found for the formation of the ether lipids in comparison with the values found without an exogenous lipid substrate. With the glial cells, the addition of exogenous alkylacylglycerols increased the incorporation of radioactivity into ether lipids 14-fold.

As noted for the ethanolamine phosphoglycerides, without exogenous diglycerides the neuronal and glial cells incorporated a substantial amount of radioactivity into diacyl-GPC. Small amounts of radioactivity were found in the ether lipids. In the presence of 4 mM alkylacylglycerols with the neuronal perikarya, a 12-fold increase

was found for the formation of the ether lipids as compared to the values found without exogenous diglycerides. The corresponding value with glial cells was a 6-fold increase.

Discussion

The synthesis of phospholipids in neuronal and glial cell fractions was examined for the first time by Freysz Bieth & Mandel [13] who studied the *in vivo* turnover of phospholipids using labelled ortophosphate. The *in vitro* synthesis of diacyl-GPC and diacyl-GPE in dispersions of isolated neuronal and glial cells from adult rabbit brain was investigated by Binaglia *et al.* [4] with labelled nucleotides and added 1,2-diacyl-*sn*-glycerols. They concluded that the cytidine-dependent enzymic system for the synthesis of these diacyl phosphoglycerides is concentrated mostly in the neuronal cells as compared to glial cells. Similar results for the synthesis of diacyl-GPC in chicken brain neuronal and glial cells were reported by Freysz & Mandel (14). A similar cytidine-dependent enzymic system is involved in the synthesis of alkylacyl phosphoglycerides [18,19, 22, 23], which are the precursors of the alkenylacyl phosphoglycerides [19].

Ethanolamine and choline phosphotransferases (E.C. 2.7.8.1. and 2.7.8.2. respectively), have a considerably higher specific activity in neuronal perikarya than in glial cells (predominately astrocytes) or in synaptosomes. The neuronal-glial ratio of the specific activities of the ethanolamine phosphotransferase with diacylglycerols was 4.5 (present study), 4.8 (Binaglia *et al* [4]) and 2-6 depending on the source of the diacylglycerols (Binaglia *et al.* [5]). A greater disparity has been found for the choline phoshotransferases with diacylglycerols. We obtained a neuronal-glial ratio of 5.2, Binaglia *et al.* [4] reported a ratio of 8.8, and for chicken cells Freysz & Mandel [14] reported a ratio of 2,7.

For the ethanolamine phosphotransferase with alkylacylglycerols, we obtained a neuronal-glial ratio of 4.9, whereas Roberti *et al.* [25] found a ratio of 1.9. Another difference is in the extent of desaturation of the alkylacyl-GPE. Of the total radio-activity in the alkenylacyl-GPE and alkylacyl-GPE, from 13—18% was in alkenyl-acyl-GPE in our studies but Roberti *et al.* [25] found 40%. Our procedure for the isolation of neuronal perikarya and glial cells was very similar to that employed by Binaglia *et al.* [4,5] and Roberti *et al.* [25]. The major difference in incubation conditions is that we used 30 mM $MgCl_2$ and Roberti *et al.* [25] used 10 mM $MnCl_2$. The effect of this difference on desaturating activity is now being investigated.

Choline phosphotransferases have not been assayed previously in brain cells with alkylacylglycerols. The synthesis of ether containing choline phosphoglycerides was quite active in the presence of alkylacylglycerols (Table II) as found previously for microsomes from rat brain (Radomińska-Pyrek *et al.*, [23]). The neuronal-glial ratio of the specific activities of the cholinephosphotransferase with alkylacylglycerols was 5.2. Thus, in the present study, the range of neuronal-glial ratios with or without added diradylglycerols was 4.5—4.9 for the ethanolamine phosphotransferase and 3.4—6.1 for the choline phosphotransferase. The higher specific activity of

these phosphotransferases in neuronal perikarya as compared with glial cells was expected because Freysz *et al.* [13], Woelk *et al.* [27] and Goracci *et al.* [15] reported that neuronal ethanolamine and choline phosphoglycerides were more active metabolically in neuronal perikarya than in glia. Freysz *et al.* [13] also reported that the choline plasmalogens in rat brain cortex had the highest metabolic activity of all the types of choline and ethanolamine phosphoglycerides in both the neuronal perikarya and glial cells. In the present study, the rate of formation ot choline plasmalogens with alkylacylglycerols was twice as high as the corresponding rate of formation of ethanolamine plasmalogens in both neuronal and glial cells. Such a high synthetic capacity for a relatively small amount of lipid is consistent with a high rate of metabolic turnover.

Cohen & Bernsohn [10, 11] have studied the incorporation of fatty acids into the phospholipids of various cell types from rat and calf brains. In contrast to results with phosphotransferases, for fatty acid incorporation they report that astrocytes are as active or more active than neuronal perikarya. Oligodendroglia from calf brain were 9 to 170 times as active as neuronal perikarya in incorporating stearic, linoleic and linolenic acids into phospholipids.

We found low rates of phosphotransferase activity for synaptosomes. Neuronal-synaptosomal ratios of the specific activities of the ethanolaminephosphotransferase ranged from 4.8–5.3. For the cholinephosphotransferase, the neuronal--synaptosomal ratios were 4–13 depending on the nature of the diradylglycerol substrate. The glia were always more active than the synaptosomes. The diacylglycerol-stimulated cholinephosphotransferase specific activity is 7 times greater for microsomes than for synaptosomes according to Abdel-Latif *et al.* [1], In our hands, much higher activities for this enzyme have been obtained, namely 64 nmoles/ /mg protein/h for microsomes (Radomińska-Pyrek *et al.* [23] and 5.9 nmoles/mg protein/h for synaptosomes. since the activity of the synaptosomal fraction is only 9 % of the activity of the microsomal fraction, all of the activity found in the synaptosomal fraction may be due to contamination by microsomes [16].

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REFERENCES

[1] A. A. Abdel-Latif, M. B. Roberts, W. B. Karp, J. P. Smith, J. Neurochem., **20** (1973), 189.

[2] G. B. Ansell, T. Chojnacki, Biochem. J., **98** (1966), 303.

[3] G. R. Bartlett, J. Biol. Chem., **234** (1959), 466.

[4] L. Binaglia, G. Goracci, G. Porcellati, R. Roberti, H. Woelk, J. Neurochem., 21 (1973), 1067.

[5] L. Binaglia, R. Roberti, G. Goracci, E. Francescangelli, G. Porcellati, Lipids, **9** (1974), 738.

[6] C. Blomstrand, A. Hamberger, J. Neurochem., **16** (1969), 1401.

[7]   —   ,   —   , ibid., **17** (1970), 1187.

[8] G. A. Bray, Anal. Biochem., **1** (160), 279.

[9] T. Chojnacki, R. F. Metcalfe, Nature, **210** (1966), 947.

[10] S. R. Cohen, J. Bernsohn, Brain Res., **60** (1973a), 521.

[11]   —   ,   —   , Trans. Amer. Soc. Neurochem., **4** (1973b) 68.

[12] J. Folch, M. Lees, G. H. Sloane-Stanley, J. Biol. Chem., **226** (1957), 497.

[13] L. Freysz, R. Bieth, P. Mandel, J. Neurochem., **16** (1969), 1417.

[14] L. Freysz, P. Mandel, FEBS Letters, **40** (1974), 110.

[15] G. Goracci, E. Francescangelli, G. L. Piccini, L. Binaglia, H. Woelk, G. Porcellati, J. Neurochem., **24** (1975), 1181.

[16] J. W. Gurd, L. R. Jones, H. R. Mahler, W. J. Moore, J. Neurochem., 22 (1974), 281.

[17] L. A. Horrocks, in: *Ether lipids: Chemistry and biology*, 1972, ed. by F. Snyder, Acad. Press, New York, pp. 177–272.

[18] L. A. Horrocks, *Abstracts, 9th International Congress of Biochemistry, Stockholm*, 1973 p. 416.

[19] L. A. Horrocks, A. Radomińska-Pyrek, FEBS Letters, **22** (1972), 190.

[20] L. A. Horrocks, G. Y. Sun, in: *Research methods in neurochemistry*, ed. by R. Rodnight, N. Marks, Plenum Press, New York, 1972, pp. 223.

[21] O. H. Lowry, N. J. Rosebrough, A. L. Farr, R. J. Randall, J. Biol. Chem., **193** (1951), 265.

[22] A. Radomińska-Pyrek, L. A. Horrocks, J. Lipid Res., **13** (1972), 580.

[23] A. Radomińska-Pyrek, J. Strosznajder, Z. Dąbrowiecki, T. Chojnacki, L. A. Horrocks, *Abstracts, 9th FEBS Meeting, Budapest, 1974*, p. 361.

[24] O. Renkonen, Biochim. Biophys. Acta, **125** (1966), 288.

[25] R. Roberti, L. Binaglia, E. Francescangelli, G. Goracci, G. Porcellati, Lipids, **10** (1975), 121.

[26] V. P. Whittaker, L. A. Baker, in: *Methods in neurochemistry*, vol. 2, ed. by R. Fried, Marcel Dekker Inc., New York, 1972, p. 1.

[27] H. Woelk, K. Kanig, K. Peiler-Ichikawa, J. Neurochem., **23** (1974), 1057.

И. Строшнайдер, А. Радоминьска-Пырек, Е. Лазаревич, Л. А. Хоррокс, **Синтез** *in vitro* 1-аклил-2-ацил- и 1,2-диацил-sn-глицеро-3 фосфорилхолинов и этаноламинов фракции нейронов, глии и синаптосом выделенных из мозга взрослых кроликов.

Содержание. 1-алкил-2-ацил-Sn-глицеролы и 1,2-диацил-sn- глицеролы заметно увеличивали включение *in vitro* радиоактивной метки из цитидин-5-дифосфоро(^{14}C) холина и цитидин-5-дифосфоро-(^{14}C)-этаноламина в холин и этаноламинфосфоглицериды. Отношения специфических активностей холинофосфотрансферазы (Е.С. 2.7.8.2.) для нейрон-глия (прежде всего астроглия) в пределах 3,4–6,1 является зависимым от липидных субстратов. Соответствующий предел отношения для этаноламинософотрансферазы (Е.С. 2.7.8.1) составлял

4,5–4,9. Специфическая активность фосфотрансфераз фракции синаптосом ниже, чем в глияльных клетках и составляет менее 10% специфической активности микросомальной фракции. Таким образом, синтез холина и этаноламин фосфоглицеридов значительно активнее в нейрональном перикарионе, чем в глияльном или во фракции синаптосом. В прсутствии 1-алкил-2-ацил-sn-глицеролов образование как алкилацил- как и алк-1-энилацил фосфоглицеридов было повышено. Холиновые плазмалогены (1-алк-1-энил-2-ацил-Sn-глицеров-3-фосфорилхолины синтезировались со скоростью 6,0 нмоль/лас/мг нейронального белка. Эта сравнительно высокаю скорость синтеза согласуется с высокой скоростью метаболического обмена найденного другими авторами.