Effects of a fatty acid deficiency on lipids of whole brain, microsomes, and myelin in the rat

Grace Y. Sun

Laboratory of Neurochemistry, Cleveland Psychiatric Institute, Cleveland, Ohio 44109

Abstract The lipid compositions of whole brain homogenates and microsomal and myelin fractions isolated from the brains of 6-month-old rats raised on a lab chow diet, a fatty acid-deficient diet, and a deficient diet supplemented with 5% (w/w) corn oil were determined. Brain and body weights were significantly lower in the fatty acid-deficient group. The compositions of alk-1-enyl groups and phospholipids of whole brain homogenates of rats maintained on the three diets were not different. However, marked alterations were found in the acyl group compositions of the major phosphoglycerides from whole brain homogenates and from the myelin and microsomal fractions of rats maintained on the fatty acid-deficient diet. With the deficient diet, 20:3(n − 9) was found in the major phosphoglycerides as well as in the myelin and microsomal fractions. In addition, the levels of 20:4(n − 6) and 22:4(n − 6) were decreased. The levels of 20:4(n − 6), 22:4(n − 6), and 22:5(n − 6) were higher in the brain phosphoglycerides of rats maintained on the corn oil-supplemented diet than on the lab chow control diet, and the elevation in these acyl groups was more evident in the microsomal fraction than in the myelin fraction.

Supplementary key words phosphoglycerides · acyl groups

ALTHOUGH the chemical composition of the brain is known to be relatively constant after maturity and is more resistant to the influences of external factors than are other organs, the brain may be vulnerable to protein undernutrition and other dietary deficiencies imposed during early periods of growth and development (1–3). Recent experimental findings showed that the fatty acid composition of the developing brain can be altered by variation of the dietary fatty acid levels, especially the essential fatty acids (3–8). Any alteration of brain fatty acids may also affect brain metabolism, since fatty acids are important molecular components of the subcellular membranes. Examples of a diet-related alteration of mitochondrial functions have been demonstrated in the liver (9–12).

Recently, Clausen and Møller (13) showed that rats reared on a polyunsaturated fatty acid-deficient diet were more susceptible to experimental allergic encephalomyelitis (EAE) than control rats reared on a normal diet. This fact as well as the other results cited may be taken as evidence of a diet-induced change in the chemical structure of cerebral membranes. Since diet-induced changes in the composition of rat brain membranes may not be as evident in studies of whole brain homogenate as in subcellular fractions, it seemed appropriate to study acyl group composition of isolated myelin and microsomal fractions in addition to whole brain homogenates of rats maintained on various deficient diets. The chief aim of the present study was to compare the lipid and acyl group compositions of whole brain homogenate and microsomal and myelin fractions isolated from the brains of rats fed a lab chow diet, a fatty acid-deficient diet, and a fatty acid-supplemented diet.

MATERIALS AND METHODS

Animals and diets

Pregnant female rats of the Wistar strain supplied by A & D Farms (Belleville, N.J.) were caged individually in wire-bottomed galvanized cages. They were given

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Abbreviations: CPG, choline phosphoglycerides; EPG, ethanolamine phosphoglycerides; GPE, *sn*-glycero-3-phosphorylethanolamine; SPG, serine phosphoglycerides; IPG, inositol phosphoglycerides; TLC, thin-layer chromatography; GLC, gas–liquid chromatography. Fatty acid designations, number of carbon atoms:number of double bonds.

tap water ad lib. and were started on the following diets approximately 2 wk before delivery: (*a*) a fatty acid-deficient diet; (*b*) a fatty acid-deficient diet supplemented with corn oil; and (*c*) Purina laboratory chow. Chromatographic analysis of the fatty acid compositions showed that the corn oil-supplemented diet contained 16:0, 13%; 18:0, 2%; 18:1, 25%; and 18:2, 60%. The Purina laboratory chow diet contained 16:0, 19%; 18:0, 7%; 18:1, 31%; 18:2, 35%; and 18:3, 3%.

The fatty acid-deficient diet was obtained from General Biochemicals (Chagrin Falls, Ohio) and contained the following ingredients (g/kg): casein, 240; sucrose, 710; salt mix no. 2, U.S.P. XIII (catalog no. 170870), 39; vitamin supplement (GBI Technical Bulletin V-17), 10; and nonnutritive fiber, 9. The fatty acid-supplemented diet had the same composition as the deficient diet except that corn oil (5%, w/w) was added. The group receiving Purina laboratory chow was included as an additional control.

Litters were weaned at 30 days of age and placed on the same diet as their mothers. All of the animals were individually caged and received water and diet ad lib. They were weighed and inspected weekly for manifestations of fatty acid deficiency (14).

Extraction of lipids from total brain homogenate

Rats were maintained on the experimental diets until they were 6 months old. For extraction of lipids from total brain homogenate, four rats (two male and two female) from each dietary group were killed. The brains were removed, weighed, combined according to sex of the rat, and homogenized in 20 vol of chloroform–methanol 2:1 (v/v). The procedure for lipid extraction has been described previously (15).

Isolation of rat brain microsomes and myelin

Four female rats (6 months of age) from each dietary group were killed, and the brains were weighed and homogenized individually in 20 vol of 0.32 M sucrose. The procedure for subcellular fractionation has been described previously (16). The crude myelin fractions were purified by osmotic shock, reflotation, and centrifugation of the floating layer at 40,000 *g* for 15 min. The final microsomal and myelin pellets were resuspended in 3 ml of distilled water. For extraction of the lipids from the subcellular fractions, 20 vol of chloroform–methanol 2:1 (v/v) was added to each fraction. The nonlipid residue was removed by adding 0.2 vol of 0.58% NaCl to the lipid extract (16). The extracted brain lipids were finally dissolved in 10 ml of chloroform and stored at 4°C.

Analysis of phospholipids by thin-layer chromatography

Portions of the lipid extracts from total brain homogenate were analyzed for phospholipid composition. Samples were applied to thin-layer plates containing a 0.5-mm layer of silica gel G that had been slurried in 0.01 M Na_2CO_3. The brain phospholipids were separated in the first dimension with chloroform–methanol–15 N NH_4OH 65:25:4 (v/v/v). After exposure to HCl fumes, the thin-layer plates were developed in chloroform–methanol–15 N NH_4OH 100:50:12 (v/v/v) (17). After separation, the lipid spots were visualized by exposing the thin-layer plates to iodine vapor. Individual lipid spots were scraped into test tubes and analyzed for phosphorus content (18).

Analysis of the nonpolar side chains by gas–liquid chromatography

Lipid extracts from total brain homogenate and the subcellular fractions were separated by two-dimensional TLC, but without exposure to HCl. Lipid spots were visualized under an ultraviolet lamp after spraying the plates with 2′,7′-dichlorofluorescein. The acyl groups from EPG, CPG, and SPG were converted to their methyl ester derivatives by reacting the phosphoglycerides with 0.5 M NaOH–methanol (19). The fatty acid methyl esters were purified on thin-layer plates developed in toluene. For the analysis of alk-1-enyl group composition, portions of the lipid extract from total brain homogenate were reacted with 1,3-propanediol and *p*-toluenesulfonic acid to produce the cyclic acetal derivatives which were analyzed by GLC. The conditions for the analysis of cyclic acetals and methyl esters by GLC have been described (20).

RESULTS

Brain weights, body weights, and fatty acid deficiency

Differences in the weight and growth rates of the deficient and the control groups appeared after 6 wk of age. After 8 wk, disorientation of the fur, hair loss, and the development of a scaly appearance became evident in the deficient group. Hair loss, water consumption, and scaling of the skin increased as the deficiency continued. None of the animals on laboratory chow exhibited deficiency signs during the period of the experiment.

The mean body weights of the lab chow control, fatty acid-supplemented, and fatty acid-deficient groups evaluated at 4 months of age were (g): 378 ± 11.7, 338 ± 9.3, and 220 ± 14.1, respectively, for the male rats, and 213 ± 8.1, 215 ± 10.4, and 176 ± 10.4, respectively, for the female rats. The body weights of

both males and females of the deficient groups were significantly lower than those of rats fed the control and supplemented diets ($P < 0.001$). The mean brain weights at 6 months of age for the three dietary groups were (g): 1.99 ± 0.04 (lab chow diet), 1.92 ± 0.04 (fatty acid-supplemented diet), and 1.83 ± 0.06 (fatty acid-deficient diet). The weights were significantly lower in the fatty acid-deficient animals ($P < 0.01$).

Phospholipid and alk-1-enyl group compositions of whole brain homogenates

The phospholipid composition of whole brain homogenates prepared from rats in the three nutritional groups is shown in Table 1. Very little difference appears in the major classes of phospholipids. The levels of the various alk-1-enyl phospholipids (Table 2) were similar for the control and fatty acid-deficient groups, but the fatty acid-supplemented group had a slightly higher level of 18:0 and a lower level of 18:1 compared with the other two dietary groups.

TABLE 1. Phospholipid composition of whole brain homogenates in rats maintained on control, fatty acid-supplemented, and fatty acid-deficient diets

Phospholipids	Nutritional Group		
	Control	Fatty Acid-supplemented	Fatty Acid-deficient
	%		
SPG and IPG	19.6 ± 0.32	19.3 ± 0.47	19.6 ± 0.47
Sphingomyelin	5.4 ± 0.26	4.8 ± 0.17	4.8 ± 0.34
CPG	33.4 ± 0.57	32.8 ± 0.33	32.6 ± 0.70
Alk-1-enyl, acyl GPE	21.9 ± 0.39	22.3 ± 0.36	23.1 ± 0.36
Diacyl GPE	17.4 ± 0.23	18.3 ± 0.24	17.5 ± 0.67
Unknown	2.3 ± 0.24	2.5 ± 0.16	2.0 ± 0.22

The values represent percentages ± SEM of total lipid phosphorus measured after a two-dimensional TLC separation of the total lipid extract of whole brain homogenates. The values are means of a total of six separate determinations of two pooled male and two pooled female rat brains per dietary group.

TABLE 2. Alkenyl group compositions of whole brain homogenates in rats maintained on control, fatty acid-supplemented, and fatty acid-deficient diets

Alkenyl Groups	Nutritional Group		
	Control	Fatty Acid-supplemented	Fatty Acid-deficient
	%		
16:0	22.5 ± 0.27	21.8 ± 0.16	22.3 ± 0.15
16:1	1.0 ± 0.20	1.2 ± 0.12	1.0 ± 0.20
18:0	39.6 ± 0.48	41.9 ± 0.31	38.7 ± 0.32
18:1	37.3 ± 0.36	35.7 ± 0.21	38.1 ± 0.24

Values represent percentage weights ± SEM of 10 GLC determinations of two pooled male and two pooled female rat brains per dietary group.

Acyl group composition of whole brain phosphoglycerides

The acyl group composition of the major phosphoglycerides isolated from whole brain homogenates of rats on lab chow, fatty acid-supplemented, and fatty acid-deficient diets is given in Table 3. Each of the major phosphoglycerides contained acyl groups in distinctly different patterns. Diet-related differences in acyl group composition were also apparent. Notably, 20:3(n − 9) was present exclusively in all major phosphoglycerides of the fatty acid-deficient animals. In addition, a possible increase in 22:3 and decreases in 20:4(n − 6) and 22:4(n − 6) were observed in the same deficient group. Differences in acyl group composition between the lab chow diet and the fatty acid-supplemented groups were less obvious. However, small increases in

TABLE 3. Acyl group composition of whole brain phosphoglycerides in control (C), fatty acid-supplemented (+FA), and fatty acid-deficient (−FA) rats

Acyl Groups	Total Phosphoglycerides			SPG			CPG			EPG		
	C	+FA	−FA	C	+FA	−FA	C	+FA	−FA	C	+FA	−FA
	% of total fatty acids											
16:0	19.7	19.4	21.0	4.4	4.9	5.2	40.1	39.2	38.6	6.7	6.4	7.2
16:1			0.8	1.7	1.3	3.4	1.6	2.0	2.2	1.0	1.0	1.2
18:0	21.0	20.9	21.1	37.5	40.2	39.5	14.6	16.1	15.2	18.0	17.8	19.9
18:1	27.6	26.5	28.9	20.2	21.6	21.0	33.1	35.0	37.1	25.1	24.2	28.3
20:1	4.1	3.6	3.3	3.1	2.5	2.0	3.1	2.0	1.9	6.2	5.9	5.5
20:3(n − 9)			5.5			6.4			2.6			7.5
20:4(n − 6)	11.2	12.0	6.8	10.7	13.0	7.3	6.4	5.5	2.8	13.7	16.2	9.6
22:3[a]	1.6		1.7	1.3		2.0						2.6
22:4(n − 6)	2.4	3.2	1.9	3.5	1.8	1.6				6.5	7.5	3.2
22:5(n − 6)		2.5	2.5	0.9	1.8	3.5				1.3	2.0	3.2
22:6(n − 3)	13.1	12.3	10.4	15.7	12.6	13.4				19.9	19.2	15.5

The values for the acyl group composition are the means of at least four GLC determinations on two pooled male and two pooled female rat brains from each dietary group. Sex differences were not evident and the values were combined. Values of duplicate samples were within a variation of ±4%.

[a] Tentative identification.

20:4(n − 6), 22:4(n − 6), and 22:5(n − 6) were observed in the acyl groups of EPG and total phosphoglycerides of the fatty acid-supplemented group.

Acyl group composition of myelin and microsome phosphoglycerides

The isolated myelin fraction contained higher levels of monoenoic acyl groups (18:1 and 20:1) and lower levels of 22:6(n − 3) in the phosphoglycerides compared with the acyl groups in total brain homogenate and in the microsomal fractions (Table 4). The rats which received the fatty acid-deficient diet showed higher levels of 20:3(n − 9) and 22:3 acyl groups and lower levels of 20:4(n − 6) and 22:4(n − 6) than the animals on lab chow or fatty acid-supplemented diets. In fact, no deposition of 20:3(n − 9) was detected in myelin of lab chow control or fatty acid-supplemented rats. The percentage by weight of 20:3(n − 9) in myelin of the fatty acid-deficient rats was 8.7% in SPG, 3.2% in CPG, and 6.8% in EPG. The acyl group compositions of total phosphoglycerides of myelin isolated from the lab chow and fatty acid-supplemented groups were similar, except that higher levels of 20:4(n − 6) and 22:4 (n − 6) were observed in the acyl groups of the fatty acid-supplemented group. Such differences were especially evident in the myelin EPG. In addition, a reduction of monoenoic and (n − 3) acyl groups was shown in the myelin phosphoglycerides and EPG of the fatty acid-supplemented group. 22:3 was only tentatively identified, but its presence in the myelin fraction was more evident than in the total brain homogenate. A small increase in the level of 22:3 in the myelin phosphoglycerides of the fatty acid-deficient rats was also observed.

Table 5 shows that the microsomes of fatty acid-deficient animals had higher levels of 20:3(n − 9), 22:3, and 22:5(n − 6) and lower levels of 20:4 and 22:4(n − 6) than microsomes from the fatty acid-supplemented and lab chow control rats. Also, microsomes of the supplemented rats contained relatively higher levels of 20:4 (n − 6), 22:4(n − 6), and 22:5(n − 6) and lower levels of 18:1 and 20:1 than the microsomes from control rats. It is also evident that the acyl group composition of microsomes differed markedly from that of myelin. Isolated microsomal membranes have considerably more 20:4(n − 6) and 22:6(n − 3) than does myelin.

DISCUSSION

Results of the present experiment are generally in good agreement with reports from previous studies associating changes in the composition of acyl groups in brain homogenates with variations in dietary fatty acids (3–8). The effects of essential fatty acid deficiency on the lipid composition of different body organs have been extensively studied (1, 6, 7, 21–24). In the liver and heart, changes in the acyl group composition related to a fatty acid-deficient diet may be evident in a short period after feeding the deficient diet. Moreover, changes in acyl group composition can be alleviated after supplementing the diet with essential fatty acids (1, 24). The most obvious change in acyl groups during essential fatty acid deficiency is the rapid synthesis of 20:3(n − 9), which is not present under normal circumstances. The 20:3(n − 9) acyl group is presumably synthesized from oleic acid to be used as a replacement for 20:4(n − 6), which is normally derived from linoleic acid (22). Although the matured brain is comparatively more resistant to alterations due to deficient dietary conditions, changes in acyl group composition similar to those in the liver and heart are evident when deficiency is imposed during the early developmental period (3, 5, 8). Present results further showed that these diet-related changes were also found among the acyl groups of the myelin and microsomal fractions.

Previous analyses of acyl group compositions of subcellular membranes from the mouse brain indicated that the myelin membranes were rich in monoenoic acyl groups and 22:4(n − 6), but the microsomal membranes contained high levels of 22:6(n − 3) (15). Although the presence of 20:3(n − 9), and possibly 22:3, was balanced by decreasing of 20:4(n − 6) and 22:4(n − 6) in the phosphoglycerides of total brain homogenate, the level of 22:6(n − 3) was not appreciably altered. The relative resistances of 22:6(n − 3) in brain towards essential fatty acid deficiency was also reported by Walker (1). It may be significant that myelin membranes, which are considered to be relatively inert metabolically, also reveal differences in acyl group composition in brain phosphoglycerides as a consequence of fatty acid deficiency in the diet.

Although changes in acyl groups can also be detected in total brain homogenate, these diet-related alterations were not as evident as in the individual subcellular fractions. Separate analyses of microsomal and myelin fractions isolated from the fatty acid-deficient rats revealed some membrane-related changes in acyl group composition. A major difference is the rise in 22:5(n − 6) of the microsomal phosphoglycerides from the deficient and supplemented rats. This acyl group was not present in either myelin or microsomal lipids of the lab chow control rats. The increase in 22:5(n − 6) shown in the microsomal phosphoglycerides of the fatty acid-supplemented group is probably due to the increase in levels of the linoleate family. However, the increase in 22:5 (n − 6) in the phosphoglycerides of the total brain homogenate of deficient rats is surprising, although Galli, White, and Paoletti had reported similar results pre-

TABLE 4. Acyl group composition of myelin phosphoglycerides in control (C), fatty acid-supplemented (+FA), and fatty acid-deficient (−FA) rats

Acyl Groups	Total Phosphoglycerides			SPG			CPG			EPG		
	C	+FA	−FA	C	+FA	−FA	C	+FA	−FA	C	+FA	−FA
	% of total fatty acids											
16:0	10.1	12.4	10.9	3.5	4.0	3.0	25.4	24.8	23.0	4.4	4.3	3.4
16:1	1.4	1.6	1.9	1.9	1.4				1.4	1.5	1.6	1.0
18:0	17.9	19.8	17.4	33.8	33.5	35.7	17.6	16.2	15.6	9.0	9.3	8.2
18:1	38.5	37.7	38.1	31.6	30.9	28.4	41.8	39.7	41.6	43.8	40.1	41.9
18:2	1.1	1.2	0.6		1.4		1.4	1.7		1.1	0.8	
20:0	1.1	2.1	1.4	1.7	2.3	0.6	1.7	1.9	2.3	1.7	1.4	1.9
20:1	13.5	11.8	10.7	9.1	9.7	8.4	7.4	7.2	6.3	18.9	17.4	18.9
20:3(n − 9)			5.0			8.7			3.2			6.8
20:3(n − 6)	0.7			1.6	2.4		1.0	1.7			1.0	
20:4(n − 6)	6.5	6.6	3.5	8.5	7.6	4.0	3.2	3.3	2.6	7.7	9.7	3.7
22:3[a]	3.5	3.0	5.5	2.1	4.0	4.5		2.7		4.1	1.5	5.5
22:4 (n − 6)	3.2	3.4	1.9	2.7	2.0	1.4	1.2	1.7	2.1	4.8	7.8	2.9
22:5(n − 6)											1.7	1.2
22:6(n − 3)	3.5	1.7	1.8	3.6	1.3	2.5				3.7	4.3	2.5

The values for the acyl group composition are the means of at least two GLC determinations on two to four female animals per dietary group. Values of duplicate samples were within a variation of ±4%.

[a] Tentative identification.

viously (8). Our results further indicated that this change was found mainly in the microsomal fraction. It is possible that unidentified acyl groups of (n − 9) series having chromatographic properties similar to 22:5(n − 6) are included in this fraction. Another difference is the increase in 22:3 level associated with the fatty acid-deficient rats. This acyl group is more concentrated in the myelin fraction than in the microsomal fraction. The elucidation of double bond position of 22:3 has not been completed. However, Klenk and Montag (25) reported 22:3 in phosphoglycerides of human brain and suggested that it could be derived from the oleic acid family.

Both male and female rats maintained on the fatty acid-deficient diet in this study not only had significantly lower body weights, but also lower brain weights. Although the growth rates for body and brain were similar for the lab chow control rats and the rats on the corn oil-supplemented diet, differences in brain acyl group composition were observed between these two groups. The elevated levels of 20:4(n − 6), 22:4(n − 6), and 22:5(n − 6) shown in the brain phosphoglycerides of the fatty acid-supplemented rats are most probably due to the high content of linoleic acid present in the corn oil.

Although changes in acyl group composition were detected in brain phosphoglycerides, our results were in good agreement with those of Galli et al. (8) in that comparable alterations were not observed in the alkenyl groups and the phospholipids. However, we have not

TABLE 5. Acyl group composition of microsome phosphoglycerides in control (C), fatty acid-supplemented (+FA), and fatty acid-deficient (−FA) rats

Acyl Groups	Total Phosphoglycerides			CPG			EPG		
	C	+FA	−FA	C	+FA	−FA	C	+FA	−FA
	% of total fatty acids								
16:0	20.0	22.9	20.0	37.9	41.3	40.5	6.1	8.7	5.5
16:1	1.2		0.8			1.2	1.8	1.2	0.8
18:0	21.8	20.7	22.2	14.5	15.1	13.7	21.7	23.7	20.8
18:1	23.6	18.7	21.7	29.8	27.5	29.4	17.9	12.3	14.5
18:2	1.3			1.0	0.8			0.6	
20:1	3.6	3.2	3.1	3.5	3.1	3.1	5.9	3.9	4.0
20:3(n − 9)			6.0			3.6			8.8
20:4(n − 6)	9.4	10.7	6.7	5.6	6.3	3.2	10.5	12.5	9.0
22:3[a]	1.6	1.2	4.4	2.0	1.7	1.8	3.4	2.9	3.8
22:4(n − 6)	2.9	3.5	1.8	1.1	1.4		5.7	7.0	3.7
22:5(n − 6)		2.8	4.2		0.9	1.4		4.1	5.8
22:6(n − 3)	15.5	15.4	15.9	4.8	4.1	3.7	24.9	21.7	21.0

The values for the acyl group composition are the means of at least two GLC determinations on two to four female animals per dietary group. Values of duplicate samples were within a variation of ±4%.

[a] Tentative identification.

observed sex-related differences in the acyl group composition of the control rats as were reported by the same authors (8). The alkenyl groups are more resistant to dietary variations because they are not polyunsaturated. Since there was no alteration in the levels of individual phospholipids, it may be assumed that the synthesis of 20:3(n − 9) by animals maintained on the deficient diet was to replace the deficient acyl groups.

Since EPG is the major phosphoglyceride of the myelin and microsomal fractions, the proportions of saturated and unsaturated acyl groups in myelin and microsomal EPG of control, fatty acid-supplemented, and fatty acid-deficient rats were summarized (Table 6). The proportion of 22:3 was not included in the calculation because the positions of double bonds in this fatty acid were not elucidated. Compared with the normal controls, the acyl groups from the fatty acid-deficient and the fatty acid-supplemented groups showed increases in the polyenoic acyl groups, with corresponding decreases in monoenoic acyl groups. Except for the microsomal EPG of the fatty acid-supplemented group, there was no apparent change in levels of the saturated acyl groups. Our results were in good agreement with those of Galli et al. (8), who suggested that the brain phosphoglycerides could maintain their level of unsaturation during fatty acid deficiency. The increase in polyenoic acyl groups in the fatty acid-supplemented rats was mainly due to an increase in (n−6) acyl groups. No apparent diet-related alteration was observed in the (n − 3) acyl group in the myelin, but the level of (n − 3) acyl groups in the microsomal fractions of the fatty acid-deficient and supplemented rats was slightly lower.

Results from Clausen and Møller (13) showed that rats reared on a fatty acid-deficient diet were more susceptible to EAE. Preliminary results from our laboratory supported their findings. This may provide some support for the etiology of multiple sclerosis as proposed by Bernsohn and Stephanides (26), who suggested that the disease may be the result of a diet deficient in polyunsaturated fatty acids. It is conceivable that demyelination may ultimately occur as a consequence of deleterious alteration in membrane structure. Moreover, as in the liver, the replacement of the linoleate acyl groups by 20:3(n − 9) in brain membranes, as well as the presence of excess amounts of 20:3(n − 9), may cause changes in enzymic activity, membrane permeability, and possibly other functions at the cellular and subcellular levels which may have an important consequence in the etiology of various neurologic disorders.

TABLE 6. Summary of acyl group composition of microsome and myelin EPG in control (C), fatty acid-supplemented (+FA), and fatty acid-deficient (−FA) rats

Acyl Groups	Microsome EPG			Myelin EPG		
	C	+FA	−FA	C	+FA	−FA
	% of total fatty acids					
Saturated	27.8	32.4	26.3	15.1	15.0	13.5
Monoenes	25.6	17.4	19.3	64.2	59.1	61.8
Polyenes						
(n − 9)			8.8			6.8
(n − 6)	16.2	23.6	18.5	12.5	20.2	7.8
(n − 3)	24.9	21.7	21.0	3.7	4.3	2.5
Total polyenes	41.1	45.3	48.3	16.2	24.5	17.1

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