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Changes in the Fatty Acid Pattern of Plasma Fractions of Rats Fed Coconut, Olive or Sunflower Oil

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The fatty acid composition of plasma phospholipids, triacylglycerols and cholesteryl esters from rats fed diets supplemented (10 % w/v) with coconut, olive or sunflower oil during six days has been studied. Rats fed the olive oil diet showed an increased amount of oleic acid whereas the animals fed the sunflower oil diet showed a higher content of linoleic acid than that of the other two groups. n-6 polyunsaturated fatty acids were mainly carried in phospholipid and cholesteryl-ester fractions. There were no differences in the amount of n-6 and n-3 polyunsaturated fatty acids. The effect of diet supplementation was shown after only six days of treatment and the results were similar to those reported in longer treatments.

Key Words: Fatty acids, Plasma, Rat, Coconut oil, Olive oil, Sunflower oil.

Plasma lipids are good indicators of the rapid modification produced by dietary fats, because of its turnover and directional flux of various lipoproteins (16). In plasma, the principal dietary effects were found in phospholipids and cholesteryl ester fractions (19) and although there is no change in the polyunsaturated/saturated fatty acid ratio, there were some in the individual fatty acids (13).

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Previous studies carried out in our laboratory (8) had shown that long-term supplementation of the diet with olive or sunflower oil caused changes in fatty acid plasma profile. This study was designed with the object of assessing whether dietary short-term supplementation affects plasma fatty acid composition.

Materials and Methods

Male weanling Wistar rats weighing 100-110 g were randomly divided into

three groups of seven animals each. Groups were fed the same basal diet with a different 10 % (w/v) fat supplement: coconut oil, olive oil and sunflower oil. The composition of the basal diet was starch 65.6 %, vitamin-free casein 10 %, cellulose 8 %, mineral mix 4.5 %, vitamin mix 1 %, DL-methionine 0.5 % and choline bitartrate 0.33 % (11). Dietary fatty acid composition was assessed by gas chromatography of fatty acid methyl esters of each diet total lipids (7, 15). Fatty acid composition of the diets is shown in table I. Food and water were provided *ad libitum*. Food intake was similar in all groups, and all animals had similar weights at the end of the experiment (150-160 g).

After feeding the experimental diets for six days, 12 h fasted rats were killed by decapitation and the blood was collected in heparinized plastic tubes. A group of seven weanling rats (control) was also sacrificed under the same conditions.

Table I. *Fatty acid composition of diets.* Fatty acid composition was determined by GLC as described in Material and Methods. Results are expressed as the percentages of total fatty acid methyl esters.

Fatty acid	Coconut oil	Olive oil	Sunflower oil
10:0	4.8		
12:0	55.6		
14:0	22.3		
16:0	11.0	11.1	7.5
16:1 n-7	0.8	0.1	
18:0	6.9	3.2	3.8
18:1 n-9		78.8	21.5
18:2 n-6		4.1	65.9
18:3 n-3		1.3	0.3
20:2 n-6		0.6	0.2
20:3 n-6			0.6

Plasma lipids were extracted with chloroform/methanol (2:1, v/v) containing the antioxidant 2,6-di tert-butyl-p-cresol (BHT) (50 mg/l) (7). Phospholipids (PL), triacylglycerols (TG) and cholesteryl-esters (CE) were separated by one-dimensional thin layer chromatography (TLC) on Silica Gel 60G using as a solvent a mixture of hexane:ethyl ether:acetic acid (80:20:1, by vol.) (18). The spots were scraped off and analyzed for fatty acid composition. Fatty acids were methylated with a 14% boron trifluoride in methanol (15) and analyzed by gas chromatography (Hewlett Packard, model 5880A) equipped with a flame ionization detector and a 30 m x 0.15 mm (i.d.) capillary column. The temperature program for the GLC run consisted of 5 min at 150 °C, followed by a 2 °C/min increase to 186 °C and a 3 °C/min increase to 226 °C.

The results are reported as area percentages and shown as means ± S.D. The data were analyzed by a one-way analysis of variance followed by a Tukey test (5).

Results and Discussion

The purpose of this work was to study the effects of dietary oils with marked differences in the fatty acid composition (saturated, coconut oil; monounsaturated, olive oil; polyunsaturated, sunflower oil) on plasma fatty acid pattern in short-term period.

The fatty acid profile of plasma phospholipids, triacylglycerols and cholesteryl-esters from rats fed a basal diet supplemented with coconut, olive or sunflower oil during six days are shown in tables II-IV. The effect of dietary supplementation appeared just after two days of supplementation. Thereafter, no changes in the amount of the different fatty acids were found.

Table II. *Fatty acid composition of plasma phospholipids in control and coconut, olive or sunflower oil-fed rats for six days.*
The results are expressed as means ± S.D. (n = 7). a and b: significance between the coconut and olive and sunflower groups, respectively. c: significance between olive and sunflower groups. d: significance with respect to the weanling rats. 1: $p<0.05$ and 2: $p<0.01$.

Fatty acid	Control	Coconut	Olive	Sunflower
16:0	31.7 ± 0.9	28.4 ± 0.9	24.9 ± 0.9 d_2	29.4 ± 2.2
18:0	17.2 ± 0.7	17.7 ± 1.0	18.4 ± 0.8	20.1 ± 1.7
16:1 n7	1.9 ± 0.3	2.0 ± 0.2	1.3 ± 0.1 a_1	1.6 ± 0.2
18:1 n9	11.1 ± 0.5	11.2 ± 0.8	20.8 ± 0.8 a_2d_2	7.2 ± 0.6 c_2
18:2 n6	17.0 ± 0.7	15.3 ± 1.6	10.7 ± 0.5 d_2	22.5 ± 1.8 c_2
20:2 n6	1.1 ± 0.4	0.9 ± 0.0	2.7 ± 0.4 a_2	0.6 ± 0.1 c_1
20:3 n6	1.4 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	0.5 ± 0.1 $b_2c_2d_2$
20:4 n6	14.5 ± 0.5	18.4 ± 1.3	16.7 ± 0.6	14.6 ± 1.9
22:5 n6	1.1 ± 0.2	1.6 ± 0.1	1.2 ± 0.1	1.5 ± 0.3
22:6 n3	2.6 ± 0.3	2.9 ± 0.2	2.3 ± 0.2	1.7 ± 0.3

Table III. *Fatty acid composition of plasma triacylglycerols in weanling and coconut, olive or sunflower oil-fed rats for six days.*
Legend as in table II.

Fatty acid	Control	Coconut	Olive	Sunflower
16:0	37.4 ± 1.5	34.8 ± 1.1	24.7 ± 4.0 d_1	22.5 ± 2.6 b_2d_2
18:0	5.6 ± 0.4	5.8 ± 0.9	4.0 ± 0.8	10.3 ± 2.0
16:1 n7	9.3 ± 0.9	9.2 ± 1.0	3.1 ± 0.4 a_2d_2	3.1 ± 0.3 b_2d_2
18:1 n9	35.2 ± 0.5	34.2 ± 2.5	56.1 ± 4.5 a_1d_2	26.6 ± 1.1 b_2
18:2 n6	10.2 ± 0.6	9.2 ± 0.8	5.6 ± 0.2 d_2	29.6 ± 4.2 $b_2c_2d_2$
20:2 n6	0.9 ± 0.2	1.4 ± 0.3	1.6 ± 0.4	1.5 ± 0.2
20:4 n6	1.0 ± 0.2	2.2 ± 1.6	1.7 ± 1.2	2.6 ± 0.4
22:5 n6	0.0 ± 0.0	1.8 ± 0.2 d_2	2.5 ± 0.8 d_1	0.0 ± 0.0 b_2c_1
22:6 n3	2.0 ± 0.2	1.2 ± 0.7	2.2 ± 1.3	2.8 ± 1.5

Table IV. *Fatty acid composition of plasma cholesteryl esters in weanling and coconut, olive or sunflower oil-fed rats for six days.*
Legend as in table II.

Fatty acid	Control	Coconut	Olive	Sunflower Rats
16:0	17.5 ± 1.0	15.4 ± 2.0	10.5 ± 0.7 d_2	19.3 ± 1.8 b_2
18:0	9.5 ± 1.3	5.0 ± 0.8	3.1 ± 0.4 d_2	6.9 ± 2.2
16:1 n7	4.2 ± 0.2	12.0 ± 0.8 d_2	3.6 ± 0.2 a_2	4.9 ± 0.6 b_2
18:1 n9	13.9 ± 1.1	17.1 ± 1.1	28.1 ± 2.0 a_2d_2	19.1 ± 2.5 c_2
18:2 n6	13.5 ± 0.7	13.3 ± 1.2	10.0 ± 0.3	23.9 ± 2.5 $a_2c_2d_2$
18:3 n6	1.8 ± 0.0	1.6 ± 0.2	0.0 ± 0.0 a_2d_2	1.2 ± 0.3 c_1
20:3 n6	1.9 ± 0.3	1.0 ± 0.1	3.4 ± 0.9	4.5 ± 1.3
20:4 n6	32.4 ± 2.6	34.9 ± 3.6	34.7 ± 3.0	43.0 ± 5.6
22:6 n3	0.0 ± 0.0	0.9 ± 0.1	2.4 ± 0.3	0.5 ± 0.0

As expected, palmitic acid (16:0) was the main saturated fatty acid in plasma TG, since TG fatty acids are directly related to dietary intake (6, 12, 19) and palmitic acid is the main saturated fatty acid in the three diets. In PL, stearic acid (18:0) percentage was similar in the three experimental groups and in control weanling rats (approximately 20 %). However, as stearic acid has a low level in the three experimental diets and in rat milk (4), it may be obtained by endogenous synthesis (19). It is interesting to note that there were no differences in plasma saturated fatty acid level in the three groups despite the fact that coconut oil is the more saturated.

Palmitoleic acid (16:1 n-7) was always higher in the coconut oil group. As the coconut oil lacks this fatty acid, it should come from an increased delta-9-desaturase activity of the palmitic acid provided by the diet (GIRÓN *et al.*, unpublished results).

Oleic acid (18:1 n-9) was higher under an olive oil feeding than in weanling rats and coconut and sunflower oil fed rats in all lipid fractions analyzed and was mainly carried in plasma CE and TG (8). n-6 polyunsaturated fatty acids were mostly transported in the PL and CE fractions; in the CE fraction, arachidonic acid (20:4 n-6) was in a higher amount than linoleic acid (18:2 n-6). This has been described in rats (17) and it is opposite to what happens in humans (9) and dogs (8).

The only n-3 fatty acid detected in plasma fractions has been docosahexaenoic acid (22:6 n-3) which was in general less abundant than the n-6 metabolites. The ocurrence of n-6 and n-3 fatty acids in the coconut oil group is important despite the lack of potential precursors in the coconut oil. This fact could be explained by its liberation from the reservoirs formed during the weanling period (2).

In the PL fraction, the 22:6 n-3 content was similar in the three groups. The PL is the main fraction which carries fatty acids to the brain (3); it is therefore convenient to maintain a constant percentage of the plasma n-3 fatty acids because their deficiency alters dramatically the fatty acid composition of various organs, including brain (3, 14). Moreover, unlike other organs the speed of recuperation from these abnormalities is very slow, and in rats many weeks are required for the brain to recover (1).

In summary, the modification of plasma fatty acid composition in response to the diet can be achieved in a short period of time. These results were similar to those found for longer periods of time (6, 8, 10, 19).

M. D. GIRÓN, M. D. CRIADO, A. LARA y M. D. SUÁREZ. *Cambios en el perfil de ácidos grasos en las fracciones plasmáticas de ratas alimentadas con aceite de coco, de oliva o de girasol.* Rev. esp. Fisiol. (J. Physol. Biochem.), 51 (2), 65-70, 1995.

Se estudia la composición de ácidos grasos en las fracciones de fosfolípidos, triglicéridos y ésteres del colesterol del plasma de ratas alimentadas durante seis días con una dieta base suplementada al 10 % (g/v) con aceite de coco, oliva o girasol. Las ratas alimentadas con aceite de oliva presentan mayor cantidad de ácido oleico, mientras que las de la dieta de girasol tienen mayor proporción de ácido linoleico. En todos los grupos, los ácidos grasos poliinsaturados de la serie n-6 se transportan principalmente en las fracciones de fosfolípidos y ésteres del colesterol. No se observan diferencias en el contenido de ácidos grasos poliinsaturados de las series n-6 y n-3, entre grupos. El efecto de la suplementación de la dieta se muestra en sólo seis días, con resultados similares a los publicados en tratamientos más largos.

Palabras clave: Acidos grasos, Plasma, Rata, Aceite de coco, Aceite de oliva, Aceite de girasol

References

1. Bourre, J. M., Durand, G., Pascal, G. and Youyou, A. (1989): *J. Nutr.*, 119, 15-22.
2. Bourre, J. M., Piciotti, M. and Dumont, O. (1990): *Lipids*, 25, 354-356.
3. Bourre, J. M., Dumont, O., Pascal, G. and Durand, G. (1993): *J. Nutr.*, 123, 1313-1319.
4. Dhami, M. S. I., De la Iglesia, F. A. and Fewer, G. (1981): *Res. Commun. Chem. Pathol. Pharmacol.*, 33, 319-329.
5. Dixon, W. J., Brown, M. B., Engelman, L. and Jenrich, R. I. (1990): BMDP statistical software manual. University of California Press. Berkeley.
6. Dougherty, R. H., Galli, C., Ferro-Luzzi, A. and Iacono, J. M. (1987): *Am. J. Clin. Nutr.*, 45, 443-455.
7. Folch, J., Lees, M. and Stanley, G. H. S. (1957): *J. Biol. Chem.*, 226, 497-503.
8. Girón, M. D., Mataix, F. J. and Suárez, M. D. (1992): *Arch. Int. Phys. Biochim. Biophys.*, 100, 321-326.
9. Holman, R. T., Smythe, L. and Johnson, S. (1979): *Am. J. Clin. Nutr.*, 32, 2390-2399.
10. Huang, Y. S. and Horrobin, D. F. (1989): *Ann. Nutr. Metab.*, 31, 18- 28.
11. ILAR's Committe on Laboratory Animal's Diets (1979): *Nutr. Abs. Rev.*, 49, 413-419.
12. Iritani, N. and Fujikawa, S. (1982): *J. Nutr. Sci. Vitaminol.*, 28, 621-629.
13. Leth-Espensen, P., Stender, S., Ravn, H. and Kjeldsen, K. (1988): *Arteriosclerosis*, 8, 281-287.
14. Mohrhauer, H. and Holman R. T. (1989): *J. Neurochem.*, 10, 523-530.
15. Morrison, W. R. and Smith, L. M. (1964): *J. Lipid Res.*, 5, 600-608.
16. Phillip, G. B. and Dodge, J. T. (1967): *J. Lipid Res.*, 8, 676-681.
17. Rayssiguier, Y., Gueux, E., Cardot, D. V. M., Thomas, G. and Robert, A. (1986): *Nutr. Res.*, 6, 233-240.
18. Shipsky, V. P. and Barclay, M. (1969): *Meth. Enzymol.*, 14, 548-550.
19. Vericel, E., Lagarde, M., Mendy, F., Courpron, P. and Dechavanne, M. (1987): *Nutr. Res.*, 7, 569-580.