PRODUCTS OF FAT OXIDATION IN THE DIET OF RATS AND THE LEVEL OF LIVER CHOLESTEROL AND PHOSPHOLIPIDS

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Researchers in recent years [2-9,11] have demonstrated that oxidative rancidification products of fats produce unfavorable effects in animals.

Certain oxidation products from fats are easily absorbed by the intestine and are found in the liver lipids. For example, peroxides are found in substantial quantities in the liver lipids of experimental animals maintained on a diet containing oil rich in fatty acid peroxides. Such a diet over the course of 2-3 months is capable of producing degenerative changes in the liver [5]. Peroxides result in the inactivation of the enzymes located in the liver mitochondria promoting β-oxidation and oxidative phosphorylation [12].

Dimers isolated from oxidized sunflower seed oil, when used in experiments on animals, have been found to produce marked fatty degeneration of the liver and granular degeneration of the kidney [3,4] in addition to a desquamative inflammation and necrosis of the gastrointestinal tract mucous membrane.

According to Reiser and co-workers [10], the amount of cholesterol in the rat liver depends upon the character of the dietary fat.

In view of the fact that oxidative products frequently accumulate in food fats, we investigated the effect produced by these products upon the liver cholesterol and phospholipid levels.

METHODS

Two series of experiments were carried out using male albino rats weighing 70-80 g (40 animals in each series). All animals were housed in individual cages and were given the following artificial laboratory diet:

Protein (casein, nutritional)	18% of the calories
Fats (in appropriate sunflower seed oil)	30% of the calories
Carbohydrate (potato starch)	52% of the calories
Bakers yeast	0.5 g per animal
Fish oil	3-4 drops per animal
Salt mixture	4% of the diet weight
Water	ad libitum

Series I consisted of two groups, each containing 20 animals; one group on the specified diet received a fat solution of the polymerized fraction (1 g/kg), specially separated from autooxidized sunflower seed oil. In that phase of the fat study, the separated fraction consisted principally of dimers. The control group in this series was given refined sunflower seed oil (acid number 0.43 mg KOH, peroxide number 0.19% iodine, iodine number 128.38%, oxirane oxygen 0.023%, oxidative products not soluble in petroleum ether 0.35%).

Series II also consisted of 2 groups of animals (20 each), one group of which, under similar conditions, was given a diet containing sunflower seed oil rich in peroxides; the control group was given a highly purified sunflower

TABLE 1. Cholesterol and Phospholipids in Livers of Rats Receiving a Diet Containing Fatty Acid Dimers

Group of animals	No. of animals	Quantity (in g% calculated on the dry weight basis)	
		Cholesterol	Phospholipids calculated as lecithin
Experimental	20	2,01 ± 0,11	1,65 ± 0,17
Control	20	1,45 ± 0,11	1,85 ± 0,15

TABLE 2. Cholesterol and Phospholipids in Livers of Rats Receiving a Diet of Sunflower Seed Oil Rich in Peroxides

Group of animals	No. of animals	Quantity (in g% calculated on the dry weight basis)	
		Cholesterol	Phospholipids calculated as lecithin
Experimental	20	1,53 ± 0,07	2,35 ± 0,18
Control	20	1,58 ± 0,10	2,29 ± 0,16

seed oil (acid number 2.33 mg KOH, peroxide number 0.08% iodine, oxirane oxygen 0.0171%, phosphatide content 0.091%).

In order to increase the peroxide content, half of the oil was treated by bubbling air through it. After this treatment, the oil had the following characteristics: acid number 2.44 mg KOH, peroxide number 0.76% iodine (an increase of 9 times over the original), oxirane oxygen 0.028%, phosphotide content 0.091%. In other words, the quantity of peroxides only had increased, the remaining indices were practically unchanged. This showed that the oxidation process had proceeded only to the first stage, i.e., to the formation of peroxides and without any secondary products of oxidation developing.

The experiments were continued 12-14 weeks, after which the total cholesterol and the lipid phosphorus were determined in the liver tissue using the method of M. N. Markova and A. A. Pokrovskii [1].

The results of the study on liver tissue from animals receiving the dimers are presented in Table 1, and those from animals receiving a peroxide-rich sunflower seed oil are presented in Table 2.

From Tables 1 and 2, it may be seen that the livers of animals,receiving the polymer fraction of autooxidized sunflower seed oil (dimers) in their diet, accumulate significantly more cholesterol than the livers of animals that were given oil with a high peroxide content. It is of interest that, while the content of cholesterol in the livers of dimer-fed animals was relatively elevated, the phospholipid level was decreased (see Table 1).

In previous histological studies, we had observed a marked fatty degeneration of the liver and granular degeneration of the kidney [3,5] in animals receiving dimers.

It is significant that peroxides, which suppress enzyme systems [12] and also produce fatty liver degeneration, do not result in an increased liver cholesterol and decreased liver phopholipids (see Table 2). This permits the conclusion that the sunflower seed oil peroxides, which appear in the liver [5], produce damage to certain enzyme systems [12] and fatty degeneration [4], but do not result in any noticeable change in the cholesterol and phospholipid metabolism in this organ during a 12-week period. Therefore, not only do the types of dietary fats have an influence on the cholesterol and phospholipid metabolism, but also certain oxidation products of the food fats may show effects in this respect.

LITERATURE CITED

1. M. N. Markova and A. A. Pokrovskii, in the book: Textbook on the Investigation of Nutrition and Public Health [in Russian], Moscow (1964), p. 159.
2. V. G. Parteshko, in the book: Physiology and Pathology of the Intestine [in Russian], Moscow (1962), p. 29.
3. V. G. Parteshko and O. M. Kucher, Vopr. pitaniya, 2, 44 (1964).
4. V. G. Parteshko and O. M. Kucher, Byull. éksp. biol., 1, 24 (1964).
5. V. G. Parteshko, Vopr. pitaniya, 5, 20 (1964).
6. V. Bens and V. A. Sedlacek, Zh. gig. épedemiol. (Prague), 3, 343 (1959).
7. H. Kaunitz, C. A. Slanetz, R. E. Johson, et al., J. Am. Oil chem. Soc., 33, 630 (1956).

8. H. Kaunitz, C. A. Slanetz, R. E. Johnson, et al., Acta chim. Acad. Sci. hung., 23, 189 (1960).
9. N. Matsuo, J. Jap. Soc. Food Nutr., 12, 118 (1959).
10. R. Reiser, M. C. Williams, et al., Arch. Biochem., 102, 276 (1963).
11. M. C. Reporter and R. S. Harris, J. Am. Oil chem. Soc., 38, 47 (1961).
12. A. L. Tappel and H. Zalkin, Arch. Biochem., 80, 326 (1959).

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