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## Study Of The Influence Of A-Linolenic And Linoleic Acid On The Development Of Atherosclerosis In Experimental Mice.

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### ABSTRACT

The article presents the results of the effect of modified beef on the progression of atherosclerosis. The type and amount of PUFAs that will be needed to overcome the potentially proatherosclerotic effect of saturated fatty acids in beef and the effectiveness of the action of n-3 PUFAs compared with n-6 PUFAs for normalizing lipid metabolism.

**Keywords:** polyunsaturated fatty acids, beef, atherosclerosis prevention.

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## INTRODUCTION

In previous studies, a number of differences in the fatty acid composition of beef were noted, including a marked change in the relative ratio of PUFAs n-3: n-6. This is largely due to the lower content of linoleic acid and a higher content of  $\alpha$ -linolenic acid in grass-fed beef. The second experiment was designed to find out whether the relative difference in the proportions of these fatty acids is responsible for differences in plasma lipids and whether large changes in the amount of these fatty acids can influence the development of atherosclerosis.

## MATERIAL AND METHODS

To study the effect of enriched beef on the development of atherosclerosis in the experiment, we used live models of mice with artificially induced atherosclerosis, which allowed us to form a lipoprotein profile similar to that in humans. Such mice easily develop dialysis hyperlipidemia and atherosclerosis and are very sensitive to changes in the content of fatty acids in the diet.

In order to obtain freeze-dried beef, we fattened 30 animals of Kalmyk breed (22 weeks old) kept on one of two rations:

- 1) TO (grass / white clover during grazing, and grass silage in winter),
- 2) KO (barley straw and feed (40:60, per 100 kg of dry matter).

For the preparation of freeze-dried beef from each animal of each fattening diet, the internal oblique muscle of the abdomen was selected. Samples were ground, freeze dried, and ground to a powder. Samples from each animal of each fattening ration were thoroughly mixed before freeze-drying to ensure uniformity of the total sample.

The fatty acid composition of the rations for each group of mice was determined using gas chromatography FAME.

Mice were randomly divided into 3 groups of food (n = 10) with an average age of 12 weeks. Three food groups received an "atherogenic diet" (regular food, supplemented with 15% cocoa butter, COB diet) or a similar diet, in which a third of cocoa butter was replaced with either linseed oil ( $\alpha$ -linolenic acid rich, FLO diet) or sunflower oil (rich linoleic acid, diet SO). All diets were supplemented with 0.25% cholesterol. Duration of feeding was 12 weeks, the animals were given fresh food every morning, and the remains were weighed, so daily food intake could be observed throughout the study.

## RESULTS AND DISCUSSION

Table 1 shows that the substitution of the share of nutrition for COB, FLO or SO reduced the content of saturated fatty acids (16: 0 and 18: 0). As expected, the FLO diet was relatively rich in  $\alpha$ -linolenic acid, and the SO-diet was linoleic acid. This difference was reflected in the fatty acid liver of mice that consumed these types of food, the liver of mice fed the type of food FLO, an increase in the proportion of  $\alpha$ -linolenic acid in those that received an SO increase in the proportion of linoleic acid.

FLO-diet significantly reduced plasma cholesterol levels compared to COB and SO-diets (Table 2). Plasma triacylglycerol was also lower in animals fed FLO, but this was only a statistical difference compared to the COB group. No significant differences in plasma HDL were found.

**Table 1: Fatty acid composition of the diets and liver lipids of mice treated with various sources of PUFA**

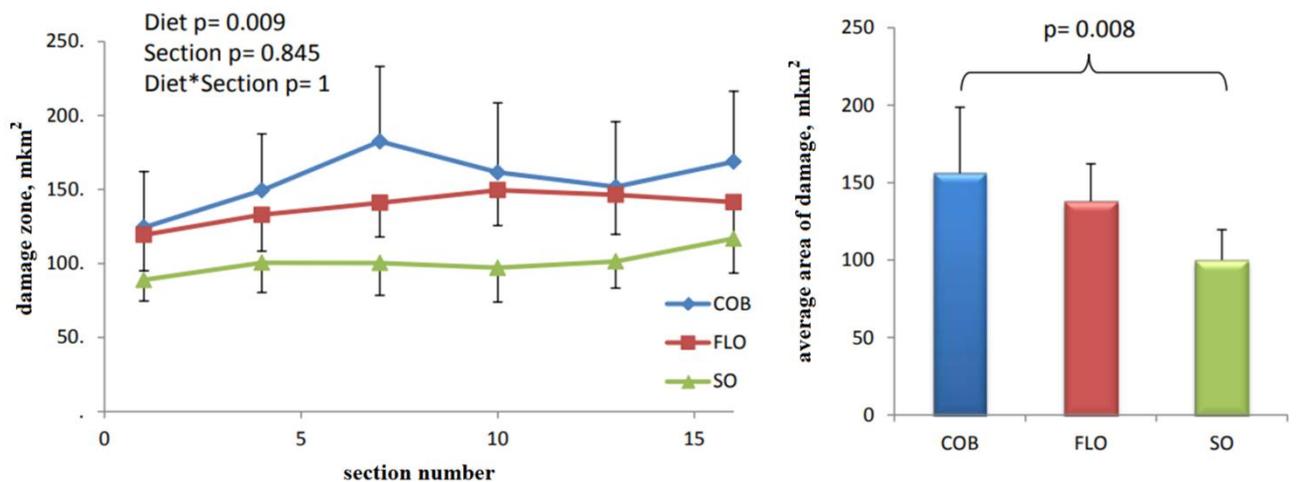
Total content of fatty acids,%	Fatty acid composition of diets			Fatty acid composition of mouse liver lipids		
	COB	FLO	SO	COB	FLO	SO
C16: 0 palmitic	24.36	18.58	18.67	16.82±0.05	14.35±0.29	14.14±0.56
C18: 0 stearic	30.34	21.41	21.21	8.15±0.4	6.83±0.37	6.47±0.49
C18: 1n-9 oleic	31.4	27.40	28.96	51.06±1.29	44.08±0.89	44.2±0.98
C18: 2n-6 linoleic	10.97	15.04	28.17	9.75±0.20	13.85±0.33	21.48±0.41
C18: 3n-3-linolenic	1.10	15.93	1.15	0.87±0.14	6.74±0.17	0.78±0.11
C20: 4n-6 arachidonic	CD	CD	H CD	5.78±0.35	2.16±0.14	5.55±0.55
C20: 5n-3 eicosapentaenoic	CD	CD	CD	CD	2.01±0.10	CD
C22: 5n-3 docosapentaenoic	CD	CD	CD	CD	0.98±0.04	0.03±0.03
C22: 6n-3 docohexaenoic	CD	CD	CD	2.56±0.18	4.10±0.26	1.61±0.15
The amount of saturated fatty acids	55.96	41.08	41.13	25.76±0.91	21.65±0.55	21.19±0.10
Amount n-3	1.1	15.93	1.15	3.43±0.11	13.84±0.32	2.42±0.10
Amount n-6	10.97	15.04	28.17	16.5±0.54	16.73±0.49	28.43±0.92

CD - cannot be determined.

**Table 2: Plasma lipids and liver lipoproteins in mice treated with various sources of PUFA**

mMol/l	COB	FLO	SO	P
Total cholesterol	9.6 ± 0.31	8.79 ± 0.19	9.76 ± 0.33	0.046
Cholesterol HDL (high density lipoprotein)	2.00 ± 0.11	1.97 ± 0.11	1.90 ± 0.13	0.847
LDL cholesterol (low density lipoprotein)	7.8 ± 0.24	6.83 ± 0.17	8.07 ± 0.31	0.003
LDL / HDL ratio	3.95 ± 0.22	3.6 ± 0.28	4.37 ± 0.39	0.230
Total triacylglycerol	1.7 <sup>a</sup> ± 0.1	1.31 ± 0.07	1.6 ± 0.15	0.041

As noted in Figure 1, a significant effect of diet was observed on the affected area, but there was no influence of the section number and the interaction between them. PostHoc analysis indicates a significantly lower average lesion area in the SO group compared with the COB group.



**Figure 1: Analysis of atherosclerosis in mice treated with different sources of PUFA.**

## CONCLUSION

The effect of  $\alpha$ -linolenic acid FLO on plasma lipids (lowering both cholesterol and triacylglycerol) suggests that such a diet may be more prophylactic of atherosclerosis than a diet rich in linoleic acid SO. However, the determination of the degree of atherosclerosis is not confirmed by the direct influence of the types of diets SO or FLO on a significant decrease in the area of damage. This suggests that these oils affect other factors than plasma lipids, which can affect the development of atherosclerosis.

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