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Alteration of fatty acid blood composition in the development dynamics of experimental hypercholesterolemia

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Overview
Studies of serum fatty acids in rabbits during 2 months of cholesterol show a 13.71% increase in all fatty acids compared to intact animals. The increase in saturated fatty acids was 65.75% compared to intact animals. In animals with experimental atherosclerosis, the sum of monounsaturated and polyunsaturated acids is reduced by 1.58 and 1.67 times, respectively, in comparison with intact animals.

Currently, the role of fatty acids in the development of atherosclerosis, pathologies of the cardiovascular system has been proven. Therefore, it is necessary to reduce the amount of palmitic fatty acid (LC), increase oleic LC, polyunsaturated LCD, while increasing the share of medium chain LCD, among which lauric LCD is especially prominent. In recent years, medium chain fats (triglycerides) of coconut oil, dominated by lauric saturated fatty acid, have been officially recommended for use as vegetable oil for salads and cooking. Unlike long chain fatty acids, which dominate most vegetable oils, medium chain fats do not deposit (1).

Fatty acids can participate in human metabolism, can undergo elongation, desaturation and oxidation. But among them it is very important to highlight the irreplaceable - those that the body can get only with food. The most important fatty acids are considered polyunsaturated LCD. Numerous studies have shown that their increased consumption reduces the risk of developing malignant tumors, reduces insulin resistance, reduces the severity of cardiovascular pathology (2. 3). The most studied are eicosapentaenoic (EPC, timnonone) and docosahexaenoic (DGC, cervonic) acids contained in marine fish fat, as well as vegetable alpha-linolenic (ALC). It is omega-3 acids, which means finding the first unsaturated (double) bond between alkyl radicals at 3 from the methyl end of the molecule.

The aim of the research to study the fatty acid content of the blood serum dynamics of development of experimental hypercholesterolemia.

Research methods: Experiments were conducted on 30 male rabbits of the Shinchell line. The first group was composed of intact animals. The second group is experimental hypercholesterolemia, reproduced by daily intragastric cholesterol administration of 0.2 g per kg of body weight for 2 months (4).

The fatty acid composition of the serum was determined on a triple quadrupole chromato-mass spectrometer with gas chromatograph (GH-MS/MS) TRACE 1310 TSQ 8000 and robotic autosampler CTC TriPlus RSH by ThermoFiScific(USA).

Statistical Analysis: The standard application package "Jamovi" was used for statistical processing. The normality of the distribution of quantitative indicators was assessed using the Chapero-Wilk criterion and/or the analysis of excesses and asymmetries. In cases of distributional normality, quantitative data were presented as mean and standard deviation (M 50), 95% confidence intervals. If the distribution of the data differs from the normal one, they were presented as median (Me) and

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percentile (25%-75%). The level of statistical significance of the indicators was defined as $p<0.05$.

Research findings and discussion

Free fatty acids promote apoptosis/necroptosis of endothelial cells (ECs) (5-7) and mediate many of the harmful effects on endothelial cell precursors - (PECs) (8).

The results of the study of fatty acid content in rabbit serum with experimental hypercholesterolemia are shown in the table. As the table shows, when feeding rabbits for 2 months with cholesterol, there is a 13.71% increase in the amount of all fatty acids compared to intact animals. At the same time, the increase in saturated fatty acids was 65.75% compared to intact animals. Such an increase in saturated content

Table

Fatty acid content of blood serum in experimental atherosclerosis rabbits

ty acids,%	Animals group	
	Intact	Monitoring
urina (C12:0)	0,48[0.33;0.55]	0,72[0,6;0,87]
ristina (C14:0)	2,3[2.07;2.51]	4,11[342;4,6]
mitina (C16:0)	24,1[23,1;25]	37,7[35,8;41,3]
rgarine (C17:0)	1,77[1.63-1.84]	2,8[2,45;3,04]
aric (18:0)	9.14[8,76-9,38]	16,8[15,1;19,2]
oundnut (C20:0)	0,67[0,5;0,79]	0,94[0,8;1,1]
stylene (C14:1)	0,77[0,99;1,3]	1,07[0,94;0,35]
mitoleic (C16:1)	1,83[1,69;2,04]	0,84[0,52;1,07]
ic (C18:1)	3,07[2,57;3,75]	2,88[2,48;3,26]
oleic (18:2)	27,6[26,8;28,5]	13,3[11,3;14,4]
omo-γ-linolenic (C20:3)	2,11[0,75;1,47]	2,23[1,78;2,41]
oundnut (C20:4)	1,59[0,75;1,71]	1,83[1,1;2,3]
cozahexaene (C22:6)	1,34[1,14;1,5]	2,08[2,89;3,15]
eryone	76,77	87,3
urated	38,5	63,07
saturated	38,31	24,23
lyunsaturated	32,64	19,44
urated/unsaturated	0.99	2,60

Fatty acids are produced by increasing lauric, palmitic, margarine, myristic and stearic acids. These acids have increased by 1.5, 1.56, 1.58, 1.78 and 1.83 times, respectively, compared to intact animals. This data points to the main role of palmitic acid in the development of hypercholesterolemia, an increase in its content may be a predictor of atherosclerosis (9). With an increased content of palmitic acid in food, a large amount of palmitic LDL is formed in the body, which disrupts the synthesis of LDL. Fatty acid binding (HIPC) proteins are insufficient to transfer the fatty acids to the cell, or the LDL proteins are not activated.

Thus, a large amount of ligandless palmitic LDP is formed, which in consequence turn into blood into «biological debris», which the body cannot assimilate and by any means tries to destroy. The active components of the complement system «recognize» marked by neutrophils m-LDP and contribute to the formation of the ligand with the help of which the «biomusor» enters the intimate artery (10).

Sedentary macrophages (a species of phagocytes) begin to act in the intrime of arteries on m-LDP. Macrophages perceive m-LDP as macromolecules of the protein. In macrophage lysosomes, proteolysis (decay) of the apoV protein occurs. However, cholesterol-treated essential fatty acids (cholesterol esters) cannot be hydrolyzed, which are found in large amounts in LDL macrophages. Non-hydrolyzed cholesterol esters first accumulate in macrophage lysosomes, later they occupy all of the cytoplasm and form foaming cells (11), triggering a pathophysiological process called "accumulation disease". Lysosomes breakdown leads to autolysis, macrophage death (12). When the number of dead macrophages produced exceeds the capacity limit of the artery intima, it is inflated and detached. An atherosclerotic plaque is formed.

In animals with experimental hypercholesterolemia, the amount of unsaturated and polyunsaturated acids drops by a factor of 1.58 and 1.67, respectively, compared to intact animals. The ratio of saturated/unsaturated fatty acids in animals by experimental hypercholesterolemia increased by 2.62 times compared to intact animals. The content of polyunsaturated fatty acids in intact rabbits was statistically higher than the control group, The presence of polyunsaturated fatty acids increases cholesterol absorption in the intestine and its conversion to cholic acid followed by elimination. It should also be noted that when the blood increases palmitic LDL at the same time for the cells decreases the intake of essential HPLC (linoleic and linolenic)which are the structural elements of cell membranes and ensure the normal development and adaptation of the human body to adverse environmental factors. Essential acids in the LDL, together with palmitic acid, also cannot penetrate the cell and this may be the main link in the beginning of the pathological process cascade.

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