tion, which is, as in the quite norm, well as at the pathology.

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BLOOD ERYTHROCYTE LIPIDS IN CONDITIONS OF ADAPTATION TO ALIMENTARY STRESS-FACTORS OF RATS

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Erythrocyte phospholipids (PL) and lipid fatty acids (FA) composition was investigated at different stages of exposure to high-caloric diet in male Wistar rats. Fat diet mainly included 2% of cholesterol and 19% of tallow of the total allowance. The animals were divided into 4 groups, 10 rats in each: control group comprising intact rats kept on the vivarium standard allowance; and groups comprising animals kept on experimental diet (30 days – group 1; 90 days – group 2; and 180 days – group 3). Lipids were extracted from erythrocytes by Bligh and Dyer method (1959). Quantitative analysis of certain phospholipids classes after thin layer chromatography was made according to V.E. Vaskovsky et al method (1972, 1975). FA methyl ethers were received by Carreau and Duback method (1978), analyzed on Shimadzu GC17A gas-liquid chromatographer equipped. Results were represented in relative percentage of total FA.

Separation of erythrocyte PL mixture of rats identified six components including phosphatidylcholine (PC), phosphatidylethanolamine (PE), phosphatidylserine (PS), sphingomyelin (SM), phosphatidylinositol (PI). Rats of group 1 demonstrated PL redistribution towards PS and PE accumulation and PI and PC share reduction. Onset of the deficit PI and PC, the PL which form outer monolayer of membrane lipid frame, indicate activation of specific phospholipases and intensification of lipoperoxidation processes contributing to plasma membrane destruction. Changes in erythrocyte membrane at PS, PC and PE level in rats kept on high-caloric diet for 90 days was of the same direction as in rats of test group 1. PC deficiency in outer layer of erythrocyte membranes was compensated by SM maintenance within the range pertinent to control group animals. Such condition can be described as a compensatory response of cell to long-term exposure to stress factors of alimentary nature. Moreover, due to the high saturation with PE cholesterol is hardly built into the inner monolayer. This helps to preserve hydrophil surrounding of cell membrane integral proteins and, therefore, their function. Depletion of erythrocyte PC which forms outer shell of cell lipid matrix was evidenced at a longer exposure to high-caloric diet (180 days) as well. PC reduction in group 3 was accompanied by reliable increase in PS and SM level as compared to control group, meaning that erythrocytic cell was structurally and functionally inadequate. Due to high saturation with SM, the clusters forming phospholipid in a membrane receive large quantity of cholesterol, and this results in lower permeability of cell membrane and interference in active metabolic processes. Thus, on the 180th day of the experiment cell membrane became unable to resist the continuous flow of alimentary stress factors, and the stage of cell compensatory protection depletion occurred that had been formed by the 90th day of high-caloric diet.

Analysis of the qualitative composition of FA of erythrocyte lipids showed that rats with dyslipidemia have considerable changes in FA composition as compared to control group. 14:0, 18:0, 17:0 FA share was observed to increase in group 1. Unsaturated FA demonstrated minor increase in relative level of 18:1n9, reliable accumulation of some n6 – 20:4n6, 22:4n6, 22:5n6 family FA and drop in 18:2n6. Share of polyunsaturated fatty acids (PUFA) of n3 family slightly reduced due to the identified decrease in 20:5 and 22:6. FA saturation index in rats of group 1 was low. Growing share of 22:4n6 and 22:5n6 in erythrocyte lipids can be deemed a compensatory response to the deficiency of 22:6n3. Blood erythrocytes of group 2 showed reduction of 12:0 and 15:0 share and increase in 16:0 and 18:0. On the contrary, rats of group 3 demonstrated growth of 12:0 level. Accumulation of saturated 14:0, 16:0 and 18:0 FA in erythrocyte lipids was more evident than in rats of group 2. Modification of n6 composition in PUFA was distinguished by reducing 18:2n6 share, reliable figures being obtained from rats of group 3; and growth of relative 20:2n6,

20:3n6, 20:4n6 and 22:4n6 content, more considerable in rat of group 2. Increase in 22:5n6 share was noticed for rats of group 3 only. Rats subject to long-term high-caloric diet demonstrated deficit of n3 PUFA – 20:5n3 and 22:6n3 FA. 20:3n9 compensatory synthesis from 18:1n9 FA was a natural consequence of n3 deficit in rats.

In a summary of the experiment results it can be stated that, regardless of the time of exposure, typical features of cell membrane lipids response to nutrient stress factor included PS accumulation, increase in saturated FA content and higher n6 FA share as compared to n3 FA. At the early stage of exposure to high-caloric diet (30 days) membrane appeared to be poorly accommodated for the changing environment. In order to preserve its structural and functional integrity, cell uses prompt stress response mechanisms described by strengthening phospholipid matrix of the inner membrane layer and intensified synthesis of long-chain n6 PUFA. Long-term (90 days) exposure to alimentary stress factors forms a compensatory response, which makes it possible to maintain membrane resistance to the damaging factors and to preserve SM level. Concurrent homeostasis of 18:2n6 and 22:6n3 functioning as body n3 and n6 FA markers helps to optimize cell adaptation to and survival in unfavorable conditions. However, on the 180th day of exposure to high-caloric diet accommodation failure and compensation source depletion were observed in the rats. These processes were evidenced by loss of phospholipid matrix asymmetry in erythrocyte membrane, lower essential n3 and n6 fatty acid share. Thus, modification of erythrocyte lipids composition revealed at different stage of exposure to high-caloric diet shows specific features of membrane response to alimentary stress factors manifested as cell compensatory protection mechanisms start on the 90th day and depletion by 180th day of the exposure, being risk factor of development of "illnesses of adaptation».

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LEVOCARNITINE IN CORRECTION OF METABOLIC DISTURBANCES UNDER CONDITION OF CHRONIC CHOLECYSTITIS

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Questions of metabolic therapy of a biliary pathology are of a great importance today. One of the metabolic preparations is levocarnitine, a synthetic analogue of the natural L-stereoisomer carnitine (L-3-hydroxi-4-N-(3-methylammonium) butanoic acid). Its metabolic functions include fatty acid transport in mitochondrion, where they oxidize and emit adenosine triphosphate (ATP) energy; modulation of intercellular homeostasis of coenzyme A in mitochondrial matrix and a mediated effect on protein synthesis.

We aimed to study the influence of the levocarnitine-based pharmacological preparation on the lipid and protein metabolism at patients with non-calculous chronic cholecystitis.

Our test group consisted of patients with non-calculous chronic cholecystitis (n=47), control group included practically healthy persons (n=33). The study was conducted according to the Helsinki Declaration standards (2000), all the patients signed an informational agreement. All patients received 20%-levocarnitine solution per os during 21 days (preparation «Elcar», R№ LS-000184, 15.04.05 trademark certificate № 162966, LLC «PIK-PHARMA»). The following values of blood serum were determined: total cholesterol, high-density lipoprotein cholesterol, triglycerides, total protein and protein fractions, biochemical markers of biliary functional status. Very low- and low-density lipoprotein cholesterol was estimated by the Friedewald formula. The obtained data were evaluated by methods of the descriptive statistics using software Statistica 6.0. The difference reliability was assessed by the Student’s t test. Differences were regarded as reliable when $p<0,05$.

65,9 % of the patients with non-calculous chronic cholecystitis had dislipidemia and 100 % of them had disproteinemia. In 42,5% of the patients were observed disturbances of liver function, caused by moderate cytolytic and cholestatic syndromes. These patients had stronger metabolic disturbances, than patients with a normally functioning liver.

The study results proved, that levocarnitine has a complex effect on lipid metabolism at patients with chronic cholecystitis. It had a lipidmodulating effect on the patients with dislipidemia, who did not any signs of a disturbed functional status of liver. After the treatment course, atherogenic fraction content in their blood reduced statistically reliably. The triglyceride level reduced two-fold, low-density lipoprotein cholesterol reduced by 22,4 %, total cholesterol - by 17,8 % and did not differ from the values in control group. Tendency to a higher content of high-density lipoprotein cholesterol and weaker atherogenic properties of blood serum was observed. Hypolipidemic effect of levocarnitine could possibly result in the intensified lipid utilization by means of enzymatic degradation and activation of fatty acid transport in mitochondrion, where they enter the β-oxidation cycle. Patients with dislipidemia and disturbed functional status of liver showed no definitive dynamics of blood lipid values. A possible reason for this could be the fact, that these patients had a more intense dislipidemia, and in order to achieve a hypolipidemic effect they probably need a longer treatment course. Among the positive results,