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,

we could observe regressing cholestatic and cytolytic syndromes and a better biliary function. This was indicated by the activated hepatic protein synthetic function, which is proved by a statistically reliable grow of total protein and albumin fraction content, which increased by 15–17%.

To sum up, levocarnitine has a lipidmodulating effect under the conditions of non-calculous chronic cholecystitis with no disturbances of the biliary functional status; it improves liver function at patients with cytolytic and cholestatic syndromes. The obtained data prove, that levocarnitine is worth using for correction of the metabolic disturbances at patients with chronic cholecystitis.

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DOSE-DEPENDENT ANTI-INFLAMMATORY EFFECTS OF SYNTHETIC CANNABINOID-RECEPTOR LIGANDS

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The research was focused on the dose-dependent effect of synthetic cannabinoid-receptor ligands WIN 55,212-2 and anandamide on proinflammatory mediator expression by blood cells *in vitro*.

Aiming to study the dose-dependent effect of WIN 55,212-2 and anandamide (0,1; 1,0; 3,0 and 10,0 mcM) *in vitro*, blood probes were taken from 12 volunteers, whose level of proinflammatory mediators: cytokines (tumor necrosis factor (TNF-α), interleukin 8 (IL-8)) and eicosanoids (leukotriene B4 (LTB4), thromboxane B2 (TXB2)) did not exceed norms. Blood cells were stimulated by lipopolysaccaride (LPS) *Escherichia coli* in dose 10 mg/ml. Basal and LPS-stimulated cytokine and eicosanoid production in whole blood was measured by means of the immunoenzymometric assay.

It was shown, that a spontaneous level of proinflammatory mediators in the blood probes, obtained from the donors, did not exceed the following values: TNF-α - 80 pg/ml, IL-8 - 115 pg/ml, LTB4 - 110 pg/ml and TXB2 - 210 pg/ml. After the LPS-stimulation of the blood cells, the expression of mediators TNF-α increased 20-fold, IL-8 eight-fold, LTB4 12-fold and TXB2 two-fold. The experiment showed, that low concentrations of WIN 55,212-2 and anandamide did not change the mediator production by LPS-stimulated blood cells. In concentration of 3,0 mcM, anandamide and WIN 55,212-2 reduced the synthesis of TNF-α, IL-8 and LTB4. The strongest inhibiting effect on blood cells was achieved at concentration of 10,0 mcM. There were revealed no influence of the studied substances in different doses on TXB2 synthesis.

To sum up, the research on the dose-dependent effect of cannabinoid substances on proinflammatory mediator expression by blood cells showed, that cannabinoid-receptor ligands WIN55,212-2 and anandamide have a unidirectional anti-inflammatory effect on TNF-α, IL-8 and LTB4 synthesis in human whole blood. The obtained data on the inhibiting effect of synthetic cannabinoids WIN55,212-2 and anandamide on proinflammatory mediator production could be used to develop new approaches to anti-inflammatory treatment.

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A MODEL OF EXAMINATION STRESS FOR THE DEVELOPMENT OF DETERMINED COLOURSTIMULATION ORIENTATED ON THE MODIFICATION OF THE FUNCTIONAL STATUS OF THE PATIENTS

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There is no other psychological term which is connected to our life as close as stress. Stress at home, in transport, at work and even during sport competitions. When stress mobilizes, for example sportsmen, it is positive factor. Otherwise, especially when it gets out of the control, it should be recognized as a destructive element of subsequent pathology formation. That includes people injured by nature-technological disasters.

Biocontrol is unique modern medical technology, which makes patient an active part of the medical-rehabilitation process.

Biocontrol is a complex of ideas, methods and computer technologies based on biofeedback, orientated on the development and perfection of physiological functions self-control mechanisms in normal condition or with a lot of pathologies. During the biocontrol procedure the information on different physiological functions will be given to the object by the external feedback arranged through a computer. It lets the examinee to learn how to control their physiological parameters and use this ability in daily life [1].

There is a problem commission on the hronobiologic and hronomedicine in Moscow (the chairman is academician F.I Komarov). The commission makes scientific researches orientated on practical use of biocontroled colourstimulation and colourtherapy (the head is F.A. Pyatakovich).

BFB-therapy for the clinical appendices is divided into: BFB-EEG- therapy, BFB-GRS- therapy