## INFLUENCE OF HIGH-ENERGY DIET ON ENZYME ACTIVITY OF MAIN COMPLEXES OF MITOCHONDRIAL RESPIRATORY CHAIN AND ACTIVITY OF H<sup>+</sup>-ATPASE OF MITOCHONDRIAL INNER MEMBRANE OF RAT HEPATOCYTES

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Previously, it has been demonstrated on rats that 20 week high-energy diet (HED) leads to non-alcoholic fatty liver disease development, which was closely correlated with visceral adiposopathy and insulin resistance. Taking into consideration a known fact that hepatocytes contain a large amount of mitochondria (each hepatocyte contains 800 mitochondria which makes approximately 18% of a hepatocyte), researchers have assumed that mitochondrial function is one of the main regulators of lipid in liver [10], and mitochondrial dysfunction contributes to hepatic steatosis development [4,8]. It is repeatedly emphasized in literature, that changes in liver which precede steatosis development as well as attend it, are still unanalyzed. For instance, findings concerning the change of enzyme activity of a respiratory chain and energy-dependent processes in hepatocytes mitochondria are inconsistent. This is the basis for formulation of the research objective [2,5,9].

The objective of this research is to study the enzyme activity of a respiratory chain of mitochondria and activity of H+ATpase of mitochondrial inner membrane of rats hepatocytes under the conditions of a long-term high-energy diet.

The research was conducted on 100 white non-pedigree rats with initial weight of 200-215 g. During a week they received standard food and afterwards were randomly divided into two groups. The rats of the first (control) group received standard food while the rats of the second group were put on a high-energy diet (HED) which consisted of standard forage (47%), sweet concentrated milk (44%), maize oil (8%), and amylum (1%) (diet No. C11024, Research Dietes, New Brunswick, NJ). In 3, 9, and 20 weeks after the beginning of the research, 10 rats were taken from each group for receiving biological material (liver homogenate). Mitochondria, namely their inner membranes, were separated out of hepatocyte fraction and using spectrometric method the activity of NA-DH-coenzyme Q oxidoreductase, succinate-coenzyme Q oxidoreductase, coenzyme Q-cytochrom C oxidoreductase, cytochrome oxidase, and H+ATpase was determined.

As a result of the research, it was ascertained, that 20 week high-energy diet did not cause significant changes in body weight index in comparison with the group of rats on standard nutrition. However, the mass of visceral lipid of rats on HED increased by 92.3% (p<0.01). The activity of main enzymes of respiratory chain of hepatocytes mitochondria was increasing during the research proportionally to the duration of HED. In 20 weeks of HED, the activity of NADH-coenzyme Q oxidoreductase, coenzyme Q-cytochrom C oxidoreductase, cytochrome oxidase increased by 26% (p<0.05), 12% (p<0.05), and 21% (p<0.05) compared to the relative results of the control group. During the experiment, the activity of succinate-coenzyme Q oxidoreductase of rats on HED did not significantly change. At the same time, a gradual suppression of the activity of H+ATpase was determined. In 20 weeks of HED the activity of H+ATpase of mitochondrial inner membrane of rats hepatocytes decreased by 43% (p<0.05) in comparison to the relative results of the control group.

Ascertained increase of the activity of NA-DH-coenzyme Q oxidoreductase, succinate-coenzyme Q oxidoreductase, coenzyme Q-cytochrom C oxidoreductase, and cytochrome oxidase at hepatocytes mitochondria may indicate the increase of electron motion at respiratory chain and increase of hydrogen gradient. The activity of succinate-coenzyme Q oxidoreductase was within normal limits, therefore, we may assume that the catabolism processes related to Krebs cycle remained unchanged.

It is known that during the destruction of mitochondrial membrane, the membrane-bound H+ATpase may change its conformational structure and lose its activity. At the same

time, the formation of lipidic hyperoxides, which appear during inflammation of mitochondria and are potential isolators, may lead to isolation of the process of coupling of oxidation and phosphorylation. One of the reasons of functional failure of H+ATpase may be oxidation of thiol groups and transformation of lipid microenvironment of enzyme.

## References

- 1. Aoun M. Dietary fatty acids modulate liver mitochondrial cardiolipin content and its fatty acid composition in rats with non-alcoholic fatty liver disease / M. Aoun, G. Fouret, F. Michel, B. Bonafos, et al. // J Bioenerg Biomembr. - 2012. - Vol. 44, № 4. - P. 439 - 452.
- 2. Aoun M. Rat liver mitochondrial membrane characteristics and mitochondrial functions are more profoundly altered by dietary lipid quantity than by dietary lipid quality: effect of different nutritional lipid patterns / M. Aoun, C. Feillet-Coudray, G. Fouret, et al. // British Journal of Nutrition. – 2012. – Vol. 107 (5). - P. 647-659.
- 3. Begriche J. Mitochondrial adaptations and dysfunctions in nonalcoholic fatty liver disease / J. Begriche, J. Massart, M. Robin et al. // Hepatology. - 2013. - Vol. 58. - P. 1497 - 1507.
- 4. Begriche K. Mitochondrial dysfunction in NASH: causes, consequence and possible means to prevent it / K. Begriche, A. Igoudjil, D. Pessayre et al. // Mitochondrion. - 2006. - Vol. 6. - P. 1 - 28.
- 5. Cardoso A.R. Diet-sensitive sources of reactive oxygen species in liver mitochondria: role of very long chain acyl-CoA dehydrogenases / A.R. Cardoso, P.A. Kakimoto, A.J. Kowaltowski // PLoS One. - 2013. -Vol. 8. - P. 77 - 88.
- 6. Day C.P. Non-alcoholic steatohepatitis (NASH): where are we now and where are we going? // Gut. -2002. - Vol. 50. - P. 585 - 588.
- 7. Garcia-Ruiz C. Mitochondrial dysfunction in non-alcoholic fatty liver desease and insulin resistance: cause or consequence? / C. Garcia-Ruiz, A. Baulies, M. Mari et al. // Free Radical Researche. - 2013. -Vol. 47. - P. 854 - 868.
- 8. Vial G. Effects of a high-fat diet on energy metabolism and ROS production in rat liver / G. Vial, H. Dubouchaud, K. Couturier K. et al. // Journal of Hepatology. – 2011. – Vol. 54(2). – P. 348 – 352. Wei Y. Nonalcoholic fatty liver disease and mitochondrial dysfunction / Y.Wei, R.S. Rector, J.P. Thyfault,
- 9. J.A. Ibdah // World Journal of Gastroenterology. – 2008. – Vol. 14, № 2. – P.193 – 199.
- 10. Younossi Z.M. et al. A novel diagnostic biomarker panel for obesity-related nonalcoholic steatohepatitis (NASH) / Z.M. Younossi, M. Jarrar, C. Nugent, M. Randhawa, M. Afendy, M. Stepanova, N. Rafiq, et al. // Obes Surg. - 2008. - Nov; 18(11): 1430-7. doi: 10.1007/s11695-008-9506-y. Epub 2008 May 24.

## EFFECT OF WHOLE-BODY VIBRATION ON BONE REMODELING

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**Key words:** vibrations, bone, mineral density, osteoporosis

Chronic mechanical vibrations combined with the physical attributes of the human body can amplify the incoming energy and present the potential for negative health effects. The aim of our study was to determine the effect of vibration oscillations of various frequencies upon the process of bone tissue remodeling.

Experimental research was conducted on 30 pubescent male rats of the weight of 180-220 g. The animals were distributed into 5 groups, 6 rats in each. Experimental animals of four study groups were exposed to heave vibration oscillations of the frequencies of 15, 25, 50 and 75 Hz correspondingly twice a day for 20 minutes, during 28 days. Then we conducted CT scanning of lumbar spine and blood sampling.

Mineral density of lumbar vertebrae of the control group was from 311,90±5,44 to