CHANGES IN EXTRACELLULAR LIPID METABOLISM PARAMETERS UNDER EXPERIMENTAL METABOLIC SYNDROME

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Introduction. Metabolic syndrome and type 2 diabetes make up the main, the most global problems of health, and their level is increasing at an alarming rate. Hepatic lipase (HL) - lipolytic enzyme, which is involved in the regulation of plasma triglycerides level. High triglyceride levels can increase the risk of coronary heart disease. HL facilitates the TG output from the pool of the very low density lipoproteins (VLDL), and this function is controlled by the composition and structure of the particles of high-density lipoprotein (HDL). The composition of HDL regulates the "liberation" of the HL from the liver, and the structure of HDL controls the HL transportation and activation of it in the bloodstream. Changes in the HDL composition can disrupt the function of the HL by disruption the output and the activation of the enzyme. The structure of HDL could therefore affect the plasma levels of TG and risk of coronary heart disease.

Aim. The aim of present work was to investigate some correlations between activity of HL and blood lipoproteins content and composition.

Materials and methods. In our work we measured HL activity and some parameters of lipid metabolism under experimental metabolic syndrome caused by fructose-enriched high calorie diet in hamsters.

Results and discussion. It has been shown that levels of apolipoprotein B-containing lipoproteins, LDL, and chylomicrons were increased and it was connected with the atherosclerosis development. Podendotelial delay and modification of ApoB - containing lipoproteins are milestones in the initiation of atherosclerosis. In podendotelia implantation of modified lipoproteins by macrophages leads to the formation of fat cells that store excessive amounts of cholesterol ethers and subsequently to apoptosis. Deactivating the hepatic lipase hamsters that are fed according to a diet high in cholesterol, caused dyslipidemia including hypercholesterolemia, hypertriglyceridemia and increased levels nonesterefecized fatty acids. These changes were accompanied by intolerance to glucose, and hepatic inflammation of the pancreas. Moreover, that promotes deposition of monocytes and macrophages on the subendotelial layer of the artery cells. Deposition of lipids in monocytes and macrophages leads to the further development of atherosclerosis.

Conclusions. Our results indicate that dyslipidaemia caused by deficiency HL in combination with a high calorie causes atherogenic changes. Thorough understanding of these mechanisms will help to develop new therapeutic strategies.