Materials of Conferences

STATE OF RAT LIPID EXCHANGE IN CONDITION OF HIGH LIPID LOAD

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It is proved by many experimental and clinical researches that organism adaptation to unbalanced nutrition leads to significant reorganization of lipid exchange in liver. This appears in metabolic changes of fatty acids (FA) and their etherification, apoprotein synthesis and lipoprotein assembly. The influence of high fat diet on lipid and lipidprotein structure in blood serum, fatty acids of polar and neutral lipids of Wistar rat's liver was studied. The basis of hyper caloric diet was 2 % of cholesterol (CS) and 19 % of beef fat of the total diet structure. Biochemical analysis of blood serum and liver tissue was made in 30, 90, 180 days from the experiment was begun. Blood serum fats were examined at biochemical analyzer FP-901 by «Labsistems» (Finland). Dimethyl ether of liver FA phospholipids (PhL), triglycerides (TG), sterol ether (ES) were analyzed at gas-liquid chromatograph Shimadzu GC-2010 (Japan). The results were expressed in relative % from the total FA quantity. There were used estimate indicators, characterizing ferments elongase and desaturase.

Keeping rats at hyper-caloric diet promoted forming alimentary dyslipidemia. On the 30th day of the experiment it was stated the increased level of CS, TG, atherogenic fractions of lipoproteins (ChS of low-density and very little density lipoproteins – CS LPLD, CS LPVLD) in blood serum, atherogenic index (AI) and decrease of CS high-density lipoproteins (CS LPHD). In 90 days of alimentary load it was detected decrease of TG and CS LPVLD concentration, the increase of CS LPLD and AI level. On the 180th day of the experiment the content of TCS, CS LPLD was increased in blood serum and CS LPVLD was decreased. The obtained data indicates that alimentary fats show vivid hyperlipidemic effect only on the 30th day of the experiment. Prolongation of high-fat diet to 90 and 180 days does not lead to accumulation of TG and CS LPVLD in blood, does not influence on content of CS LPHD. Apparently, liver barrier function limits lipotoxic load through inhibition of apoprotein synthesis and accumulation of LPVLD in liver.

Fatty acid metabolism in rat liver on the 30th day of the experiment was characterized by intensification of biosynthesis 18:1n9, 18:2n6, 18:3n6, 18:3n3, 18:4n3, decrease of 20:4n6, 20:5n3, 22:5n3, 22:6n3. Metabolic changes of fatty acids in liver were accompanied by Δ9-, Δ6-, Δ5-desaturase and elongase activity changes. In 90 days of the experiment it was detected synthesis increase of 18:1n9, 18:2n6, 18:3n3, 20:5n3, 20:3n6. Moreover 20:5n3 was etherified mostly into sterol ether, 20:3n6 – into TG and PhL. It was also seen decrease of content 20:4n6 in PhL and its increase in TG and ES. The detected changes show switching on compensatory mechanisms on the 30-90th days of high-fat load, activating synthesis of physiologically important polyunsaturated fatty acids (PUFA). In 180 days of high-fat load action it was stated increase of Mead acid production (20:3n9) with competitive inhibition of n6 family acid synthesis (18:2n6, 18:3n6, 20:4n6) and some n3 PUFA (18:3n3, 20:5n3, 22:5n3, 22:6n3) in FA pool liver. It was seen the increase of 12:0 in PhL, TG and ES. Activity of elongase and Δ5- desaturase in liver was decreased in 180 days of the experiment.

Thus, inhibition of lipoprotein formation, synthesis activation of monoenic n9, polyunsaturated n6 and n3 FA in liver with its predominant etherification into sterol ethers and triglycerides under influence of high-fat load is one of the aspects of adaptation to alimentary stress-factors.

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THE CALBINDINE RESEARCH IN THETHYMUS MACROPHAGES

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The calbindine – the protein is being localized in the cytomembrane, in the cytoplasm, and also in the cells nucleus, it is being taken its part in the membrane folds generation, in the phagocytal processes formation at the phagocytosis early stages (e.g. Ohsawaet. al. 2001), it is being expressed by the macrophage origin cells (e.g. (Kohler C., 2007). The thymus macrophages are being related to the APUD – system, as they are