P26. Influence on new bioactive compounds on carbohydrate metabolism markers in experimental liver disease

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Background: Currently considerable interest in the study of the various biologically active compounds, which could exert a significant influence on metabolic processes, arouses.
The research **aim** was to study the mechanisms of action of the non-platinum metal coordination compounds with halogensemicarbazides based chelation and macrolydic ligands – CMD-4, CMD-8 and CMJ-23, on metabolic processes in experimental hepatopathy (HP) and justification of their application efficiency in hepatology for the treatment efficiency monitoring.

Methods: Toxic HP was induced by the administration of ethylene glycol (EG). In the liver tissue were measured glucose metabolism markers: isoforms of lactate dehydrogenase - LDH-L (catalyzes the conversion of lactate into pyruvate) and the LDH-P (catalyses the conversion of pyruvate to lactate), as well as the activities of glucose-6-phosphate dehydrogenase (G-6-P DH) and NADP-dependent malate dehydrogenase (MDH-NADPd). The medication was carried out using new complex compounds – CMD-4, CMD-8 and CMJ-23.

Results: In ethylene glycol induced HP the activity of LDH-P, LDH-L and MDH-NADPd increased, while the functional level of G-6-PDH decreased compared with controls. CMD-4 administration to the animals with HP significantly reduced LDH-L activity by 35% ($p<0.05$) compared to animals with untreated HP and induced a tendency to restore the LDH-P and G-6-P DH levels. CMD-4 and CMJ-23 inconclusive increased the activity of MDH-NADPd by 23% -32% compared to the controls. The activation of the investigated NADP-dependent dehydrogenases - G-6-PDH and MDH-NADPd, by CMD-4 and CMJ-23, registered in our research in animals with liver disease is the expression of their anabolic effect.

CMD-8 returned the activity of NADPd MDH to the control group values, while CMJ-23 contributed to the recovery of G-6-PDH.

Conclusions: The tested non-platinum metal coordination compounds return to normal values the activity of most studied glucose metabolism enzymes in the liver tissue. Relevant changes can be appreciated as manifestations of adaptation processes in the purpose of maintaining optimal cellular homeostasis. The studied compounds exhibit selective action on the tissue enzymes, which probably depends on the degree of their engagement at different stages of the pathological process. Thus, the compounds included in the research can be used as remedies for the pathogenic correction of metabolic disorders that accompany toxic liver injury.

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