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Hepatoprotective and metabiotic correction and its effect on the content of free fatty acid in rats with experimental toxic liver injury

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Background. Diagnosis and treatment of liver diseases is one of the most pressing problems of modern hepatology. It is of great interest to study the peculiarities of the *in vivo* free fatty acids spectrum as important biomarkers that can serve as additional diagnostic marker for the verification of diffuse liver changes in hepatotoxic lesions. **Aim.** To evaluate morphological and biochemical changes in the liver of rats with toxic hepatitis (TH) without and after drug correction. **Materials.** Modeling of TH in rats was performed by subcutaneous injection of a solution of tetrachloromethane. Groups: control (healthy; n = 15); I — TH without medication adjustment (n = 6); II — TH + Metadoxin (Liveria IC, Ukraine); (n = 8); III — TH + Aqueous substrate of metabolic products (Hylak Forte, "Merckle GmbH"/ "Ratiopharm International GmbH", Germany) (n = 7). The parenchyma state of the liver was carried out histologically (Mallory-Slinchenko). In the liver homogenate, the quantitative content of monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) was assessed by gas chromatography. **Results.** Histological examination of the liver of the I group rats showed inflammatory cellular infiltration, tissue disorganization with steatosis of hepatocytes (cytoplasmic, macro vesicular), multiple fibrous partitions of the liver lobules. 30-days post-toxicogenic use of methadoxin (II group) and a similar duration of metabiotic use (III group) improved the morphological picture of the liver, distorted by the action of tetrachloromethane. It was established that, compared to the control, in the liver homogenate of rats of groups I–III, the level of MNSK increased by 118 times ($p < 0.001$), by 34 times ($p < 0.001$) and by 99 times ($p < 0.001$). Whereas, in comparison with groups I and III, the content of MUFA in group II tended to decrease by 3.5 times ($p = 0.430$) and 2.9 times ($p = 0.064$), respectively. The content of PUFAs increased due to rather high values of cis-9-tetradecenoic, trans- and *cis*-9-octadecenoic and *cis*-11-eicosenoic acids in group I of rats, *cis*-10-heptadecenoic, *trans*- and *cis*-9-octadecenoic and *cis*-11-eicosenoic acids in group II, *cis*-9-tetradecenoic acid in group III, *cis*-10-pentadecenoic, *cis*-10-heptadecenoic, *trans*- and *cis*-9-octadecenoic, and *cis*-11-eicosenoic acids, while in the control these fractions were either not detected or were present in small amounts. The intergroup analysis showed that the concentration of *cis*-10-pentadecenoic and *cis*-10-heptadecenoic acids in the liver homogenate of rats of group I remained significantly lower by 1.6 ($p = 0.017$) and 3.4 times ($p = 0.002$), respectively, compared to group II, and by 3.4 ($p = 0.016$) and 5.2 ($p = 0.005$) times, respectively, compared to group III. **Conclusions.** It was established that the consumption of both methadoxin and metabiotics in the post-toxic period caused a decrease in hepatocyte degeneration. TG, regardless of the correction method, was characterized by an increase in the level of PUFA. Under the influence of methadoxin, the concentration of MUFA decreased more than in the group receiving metabiotics.

K e y w o r d s: tetrachloromethane, hepatobioptates, monounsaturated free fatty acids, rats.