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ROLE OF PHAGOCYTES IN OXIDATIVE DAMAGE OF LIVER PROTEINS DURING SHORT-TERM ALCOHOLIZATION

Mykytenko A. O.
Ph.D in medicine
Ukrainian medical stomatological academy
Poltava, Ukraine

Abstract: Article reveals the role of liver tissue phagocytes in development of oxidative damage to proteins during short-term alcoholization. The experiment was carried out on 18 sexually mature male rats weighing 180-220 g. Given the fact that superoxide anion radical production does not increase on the 3rd day of experiment, when oxidatively modified proteins are increased, we reached a conclusion that Liver phagocytes are not a source of reactive oxygen species that damage liver proteins in the early stages of modeling alcoholic hepatitis.

Keywords: alcohol, rat, liver, OMP, superoxide, $NADPH_2$-oxidase

Alcohol abuse is a complex social problem of our time. Alcoholism not only destroys a person's personality, but also leads to various pathological changes in the internal organs. The liver, providing the neutralization of toxic substances, is one of the first targets of the damaging effect of alcohol. Long-term alcohol consumption can lead to the development of alcoholic hepatitis and liver cirrhosis.

Molecular mechanisms of the development of alcoholic hepatitis are associated with a change in the polarization of macrophages in the liver [1, p. 651]. Polarized macrophages can increase the production of reactive oxygen and nitrogen species. Nitric oxide is an important regulatory molecule and changes in its production can alter the course of the inflammatory process [2, p. 59]. Excessive production of reactive oxygen species can increase tissue protein damage during inflammation. [3, p. 122].

At the moment, the effect of short-term alcoholism on the production of reactive oxygen species and oxidative damage to proteins in liver tissues has not been sufficiently studied.

The aim of this work was to determine the production of superoxide radical anion from $NADPH_2$-oxidase of phagocytes and the content of oxidatively modified proteins in rat liver on the 1st and 3rd day of modeling chronic alcoholic hepatitis.

Materials and methods. The experiment was carried out on 18 sexually mature male rats weighing 180-220 g. During the experiments, the recommendations of the "European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes" (Strasbourg, 1986) were followed. Animals were divided into 2 groups: 1 - control group (n = 6), 2 - group of animals (n = 12), which were simulated alcoholic hepatitis by the method of Stepanov Yu.M. [4, p. 196]. The control animals underwent the same manipulations, however, alcohol was replaced with saline. Animals were removed from the experiment under thiopental anesthesia by bloodletting. The object of the study was the liver. In 10% liver tissue homogenate, the production of superoxide radical anion from $NADPH_2$-oxidase of phagocytes was investigated according to the method proposed by Tsebrzhinsky O.I. [5, p. 96]. The content of oxidatively modified proteins (OMP) was determined by the method of Dubinina E.E. [6, p. 24].

Результаты. The production of superoxide anion radical from $NADPH_2$-oxidase of phagocytes in the liver of rats on the 1st day of the experiment statistically significantly increased by 2.85 times when compared with the parameters of control animals. On the 3rd day of the experiment, the production of superoxide anion-radical from NADPH2-oxidase of phagocytes is statistically significantly reduced by 4.64 times in comparison with the 1st day of the experiment and decreases by 38.66% when compared with the control group. The OMP content on the first day of the experiment does not statistically significantly change when compared with the control group. The concentration of OMB in the liver of rats on the 3rd day of the experiment statistically significantly increased 6.66 times when compared with the 1st day of the experiment and 10.0 times when compared with the control group.

Conclusions. Modeling of alcoholic hepatitis causes oxidative damage to liver proteins on the 3rd day of the experiment. Liver phagocytes are not a source of reactive oxygen species that damage liver proteins in the early stages of modeling alcoholic hepatitis.

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