The aim of the work was to study the activity of antioxidant enzymes ceruloplasmin and catalase in rats' blood in terms of alcohol intoxication, its combination with constant light exposure and melatonin administration.

Experiments were performed on 32 white male rats weighing 180-230 g, kept under standard conditions and vivarium diet. Subacute alcohol intoxication was induced by intragastric administration of 40% ethanol in a dose of 7 ml/kg of body weight for 7 days. The light exposure was caused by a constant fluorescent light with an intensity of 1500 lux for 24 hours a day.

We have revealed that alcoholic intoxication was accompanied by an increase in catalase activity in rats' blood by 23% above the control. Combination of ethanol poisoning with light exposure caused more significant rise of catalase activity in the blood (by 53%). The content of ceruloplasmin in blood plasma against the background of alcoholic intoxication and its combination with constant lighting was by 82% and 83% above the control level correspondingly. Such rise of catalase and ceruloplasmin activity in the blood proves activation of natural antioxidant defense in response to ethanol poisoning and lack of melatonin under constant light exposure.

The administration of "Vita-melatonin" in dose of 5 mg/kg daily at 8 p.m. for 7 days contributed to the normalization of ceruloplasmin level in the blood plasma of animals of both experimental groups and catalase activity in alcoholized rats. The rats that had received melatonin against the background of the combination of alcohol intoxication and light exposure showed permanently high catalase activity (by 83% above the control level).

Thus, melatonin administration contributed to normalization of ceruloplasmin level in the rats' blood plasma against the background of alcoholic intoxication and its combination with constant lighting whereas normalization of catalase activity in the blood of alcoholized rats was under normal photoperiod.

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EFFECT OF MELATONIN ON THE ACTIVITY OF ANTIOXIDANT PROTECTION ENZYMES IN KIDNEYS IN CONDITIONS OF ACUTE INTOXICATION

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Enzymes of antioxidant protection of the kidneys play a key role, because they neutralize reactive oxygen species (ROS) which include superoxide anion radical, hydroxyl radicals and hydrogen peroxide, which are intensively formed under conditions of acute intoxication. The accumulation of a significant amount of ROS in the body leads to the development of oxidative stress and activation of lipid peroxidation (LPO) processes with subsequent destruction of phospholipids of nephrocyte membranes.

The experiment was conducted on 36 white adult male rats weighing 0.16-0.20 kg. which were divided into three groups. The first group was a control group. In the second group of animals toxic lesions were simulated by administering a 0.1% solution of 2,4-DNF intraperitoneally at a dose of 3 mg / kg once. In the third group of animals melatonin at a dose of 10 mg / kg was administered once. Superoxide dismutase activity was determined by the Dubinina method. Catalase activity was determined by the rate of cleavage of hydrogen peroxide. Glutathione peroxidase activity was recorded by color reaction in the interaction of SH groups with Elman's reagent. The presence of protein was determined by the Lowry method.

It has been proved that acute intoxication of rats under the conditions of administration of 2,4-dinitrophenol leads to the development of oxidative stress in the body, as indicated by an increase in the content of ROS in the blood. Accompanied by increased production of free radicals and activation of lipid peroxidation processes followed by damage to cell membranes and increased activity of superoxide dismutase and catalase in the mitochondria of nephrocytes of the experimental group of animals compared with those from the control group is somewhat normalized under the influence of melatonin. In parallel, there is a decrease in the activity of glutathione peroxidase in the experimental group of animals, probably due to the accumulation of oxidized

glutathione as a consequence of reduced antioxidant protection of cells during intoxication and increased non-enzymatic oxidation of reduced glutathione by activating reoxidation processes.

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EFFECT OF GLUTATHIONE ON THE LEVELS OF OXIDATIVE MODIFICATION OF PROTEINS IN THE BLOOD BY NEPHROPATHY

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The kidney diseases cause an imbalance between free radicals' production and antioxidant capacity. Oxidative stress damages molecules and cellular structures, disorients functions of organs and systems. Lipid and protein oxidation products are metabolized by nonenzymatic and enzymatic mechanisms to eliminate oxidative stress of the organism. So, the objective of the study was to examine the effect of glutathione on the levels of oxidative modification of proteins in the blood on the experiment of kidney disease.

The experiment was conducted on 131 male albino rats with the bodyweight of 0.16-0.18 kg. Experimental nephropathy was modelled by injection of a single intraperitoneal dose of folic acid (250 mg/kg). Glutathione was introduced daily (100 mg/kg) by the intragastric way for 3 and 7 days after the injection of folic acid. All manipulations with animals were carried out according to the European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes and the law of Ukraine "On protection of animals from cruelty". In the blood plasma the levels of oxidative modification of proteins (OMP) were determined. The degree of oxidative modification of proteins was evaluated in the blood by the level of aldehyde and ketone derivatives of neutral (OMP370) and basic (OMP430) composition. The type of distribution was estimated using the Shapiro-Wilk test. Significant differences between groups were evaluated by using the Wilcoxon test and Kolmogorov–Smirnov test with $p < 0.05$ considered.

Oxidative modification of proteins is associated with the damage of both the polypeptide chain and individual amino acids with the formation of several types of radicals. The process of oxidative modification of proteins has a complex and specific nature, which is established by the amino acid composition of proteins. The level of OMP370 in rats with nephropathy was higher by 36% on the third day of the experiment compared to rats in the control group. The glutathione decreases the level of OMP370 by 24 % on the third day of the experimental period compared with the group of animals without introduction of tripeptide. According to our study results, the indices of oxidative modification of proteins of the aldehyde and ketone derivatives of the neutral character were without significant changes on the seventh day.

The level of OMP430 in the blood of rats with nephropathy was higher by 14.6% on the third day of the experiment than of those rats in the control group. On the seventh day in animals with nephropathy the activation of processes of oxidative modification of proteins was confirmed by an increase (32.6%) of indices of the aldehyde and ketone derivatives of the basic character in the serum. The increase of oxidative modification of proteins is one of the pathogenetic links in the development of pathological conditions due to oxidative stress. The glutathione decreases the level of OMP430 to the control value on the third day and by 15 % on the seventh day of the experimental period compared with the group of animals without introduction of tripeptide.

Oxidative modification of proteins can change amino acid residues, a valence and coordination of metals which leads to disruption of protein structure and facilitate proteolysis processes. Consequently, the intensity of oxidative modification of proteins can be a marker of the degree of peroxide processes and a factor that affects the state of the antioxidant system.

Our results show the potential role of glutathione in reducing complications of kidney diseases. The main function of exogenous glutathione is suppressing lipid peroxidation which occurs in the plasma membrane and damages the membrane's structure and permeability.