ИЦДГ печень (мкмоль НАДФ×мин/мг белка)	71,69 [70,65; 78,64]	63,34 [59,25; 66,28] ***
ИЦДГ миокард (мкмоль НАДФ×мин/мг белка)	514,58 [473,15; 539,04]	425,31 [354,75; 456,51] **

Таким образом, снижение активности ферментов ЦТК могут свидетельствовать о нарушении энергообразующей функции митохондрий в печени и миокарде. При этом выявленные изменения активности ферментов ЭТЦ митохондрий миокарда и печени при ОАИ могут свидетельствовать об их адаптивной реакции для защиты сердца и печени от повреждения этанолом.

Выводы. Острая алкогольная интоксикация сопровождается снижением энергопродукции в печени и сердце, при этом, выявленные изменения активности комплексов ЭТЦ митохондрий могут свидетельствовать об адаптивной реакции при ОАИ. Понимание сложной регуляторной системы митохондриальной биоэнергетической дисфункции позволит разработать более специфические терапевтические стратегии для лечения заболеваний сердца и печени опосредованных острой алкогольной интоксикацией.

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CHANGES IN THE CONTENT OF REDUCED GLUTATHIONE AND MALONDIALDEHYDE IN BLOOD OF RATS WITH EXPERIMENTAL PERITONITIS UNDER NO SYNTHASE MODULATION

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Introduction. The mortality rate in peritonitis has not considerably dropped despite extensive research to determine the mechanisms underlying the illness's development. Specifically, little is known about the role that NO produced by constitutive and inducible isoforms of NO synthase plays in the development of oxidative stress, a critical pathogenetic feature of peritonitis [1, 2]. Further research in this area is necessary because NO has both pro- and antioxidant effects. This is essential for controlling blood flow, vascular permeability, immune system modulation, and mucosal defense.

Aim. The aim of the study was to investigate changes in the content of reduced glutathione and malondialdehyde in blood of rats with experimental peritonitis and NO synthase modulation.

Materials and methods. The experimental animals received five equal series of intraperitoneal injections (IP injections) at a rate of 0.6 ml per 100 grams of body weight: series 2-5 – 15% fecal suspension [3]; additionally Series 1 (control) and Series 2 (experimental peritonitis) were injected intramuscularly with of 0.85% sodium chloride solution, when Series 3 – with Aminoguanidine, 15 mg/kg, an inhibitor of the inducible isoform of NOS; Series 4 – with L-arginine, 300 mg/kg, a substrate of NO synthase (NOS); and Series 5 – with L-arginine +Aminoguanidine. After the samples were incubated at room temperature for ten minutes, the concentration of reduced glutathione and malondialdehyde in blood was determined using a SOLAR spectrophotometer. The statistical data was processed using nonparametric methods with Kruskal-Wallis test.

Results and discussion. When compared to the values of these indicators during EP without drug administration or with their isolated use, the level of reduced Glutathione, an indicator of antioxidant defense, increased more significantly in the blood plasma of rats with peritonitis and the combined administration of the investigated NO synthase modulators. Rats with EP and the combined administration of L-Arginine and Aminoguanidine demonstrated an increase in the reduced glutathione level in the blood plasma after half a day by 2.6 times ($p<0.01$), after one day by 3.8 times ($p<0.01$), and after three days by 2.5 times ($p<0.01$) when compared to the results in animals with peritonitis without their administration. The blood plasma of rats with peritonitis and combination treatment of the studied NO synthase modulators showed the biggest decrease in Malondialdehyde concentrations when compared to the values seen in rats with peritonitis either with or without their usage alone. Specifically, the Malondialdehyde concentration in the blood plasma decreased to 1.1 (0.8; 1.3) μmol/l, or three times ($p<0.01$), and 42 (35; 50)%, $p<0.01$, after half a day; 54 (51; 56)%, $p<0.01$, and 34 (33; 35)%, $p<0.01$ after 1 day, and 61 (56; 67)%, $p<01$, and 43 (35; 47)%, $p<01$ after 3 days.

Rats administered with the combined dose of the investigated NO synthase modulators showed an increase in the reduced glutathione levels in their blood plasma of 1.2 times ($p>0.05$) after half a day, 1.4 times ($p<0.05$) after one day, and 1.2 times ($p > 0.01$) after three days. However, after half a day, one day, and three days the amount of reduced glutathione in the blood plasma increased by 69 (61; 76)% ($p<0.01$), 91 (88; 115)% ($p<0.01$) and 67 (61; 75)% ($p<0.01$), respectively, in comparison to the results with the administration of either L-arginine or aminoguanidine. Rats with experimental peritonitis showed altered plasma levels of reduced glutathione and malondialdehyde, a consequence of lipid peroxidation, when they were administered both L-arginine, a substrate of NO synthase, and aminoguanidine, an inhibitor of the enzyme's inducible isoform.

Conclusion. It was discovered that the usage of L-arginine and aminoguanidine and elevated levels of reduced Glutathione in the blood plasma of rats with experimental peritonitis indicated an increase in antioxidant qualities. This may be explained by the amino acid L-Arginine's function as a precursor in the synthesis

of the tripeptide glutathione and the suppression of NO and ONOO-induced cytotoxicity. In its turn, a decrease in the amount of the lipid peroxidation product Malondialdehyde in the blood plasma of rats with experimental peritonitis and the administration of a combination of L-arginine and aminoguanidine indicates a decrease in oxidative stress activity. This could be because the activity of inducible NO synthase is suppressed, which in turn prevents NO from having a cytotoxic effect on cell membranes and the resulting peroxynitrite.

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