

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ



МАТЕРІАЛИ
106-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького колективу
БУКОВИНСЬКОГО ДЕРЖАВНОГО МЕДИЧНОГО УНІВЕРСИТЕТУ
03, 05, 10 лютого 2025 року

Конференція внесена до Реєстру заходів безперервного професійного розвитку, які проводитимуться у 2025 році №1005249

Чернівці – 2025

УДК 61(063)

М 34

Матеріали підсумкової 106-ї науково-практичної конференції з міжнародною участю професорсько-викладацького колективу Буковинського державного медичного університету (м. Чернівці, 03, 05, 10 лютого 2025 р.) – Чернівці: Медуніверситет, 2025. – 450 с. іл.

У збірнику представлені матеріали 106-ї науково-практичної конференції з міжнародною участю професорсько-викладацького колективу Буковинського державного медичного університету (м. Чернівці, 03, 05, 10 лютого 2025 р.) зі стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

Загальна редакція: професор Геруш І.В., професорка Годованець О.І., професор Безрук В.В.

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ISBN 978-617-519-135-4

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**EFFECTS OF COMBINED ALCOHOL INTOXICATION AND LIGHT EXPOSURE ON
RENAL ANTIOXIDANT SYSTEM IN RATS**

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Introduction. Despite the widely recognized negative impacts of excessive alcohol consumption on human health, alcohol use remains common in society. The World Health Organization (WHO) reports that alcohol abuse is responsible for approximately three million deaths annually worldwide, as well as for disabilities and organ damage.

In contemporary life, ethanol consumption frequently coincides with exposure to other harmful factors, including disruptions in light cycles. Modern individuals are often exposed to light nearly constantly, with night shifts, air travel, jet lag, and active nightlife all contributing to disturbances in circadian rhythms. Under normal conditions, these biological rhythms are regulated by melatonin, a hormone primarily secreted in darkness. Even minimal light exposure can inhibit its production. Research has demonstrated that melatonin possesses a broad range of biological effects, with its primary function being a potent antioxidant action.

The aim of the study. The study aims to examine melatonin influence on activity of antioxidant enzymes, specifically catalase, glutathione peroxidase and glutathione-S-transferase in the kidneys of rats subjected to alcohol intoxication and combined alcohol and constant light exposure.

Material and methods. Subacute alcohol intoxication was induced by intragastric administration of 40% ethanol in a dose of 7 ml/kg of body weight for 7 days. Light exposure was caused by keeping animals under a fluorescent light of 1500 lux intensity for 24 hours a day.

Results. Alcohol intoxication was accompanied by a decrease of catalase activity in rats' kidneys by 21% below the control level along with a decrease of glutathione peroxidase activity by 27% and increase of glutathione-S-transferase activity by 30%. A combination of modified photoperiod with ethanol administration resulted in the decrease of catalase activity by 34% and a decrease of glutathione peroxidase activity in kidneys by 39% lower than the control level. Activity of glutathione-S-transferase was by 37 % higher than the control level. The decrease in the antioxidant enzymes activity and rise of antitoxic glutathione-S-transferase activity in case of alcohol intoxication along with the permanent light exposure was more significant than that of rats that had alcohol intake under the normal light regime, that might have resulted from a decrease in melatonin synthesis and lack of its antioxidant effect under constant light exposure. Thus, intensification of free radical generation caused depletion of antioxidant defense.

The administration of melatonin at the dose of 5 mg / kg daily at 20⁰⁰ for 7 days to animals exposed to ethanol intoxication caused normalization of catalase, glutathione peroxidase and glutathione-S-transferase activity in kidneys. Melatonin intake was revealed to be more effective in normalizing catalase activity in case of ethanol combination with constant lighting but the activity of glutathione peroxidase enzyme remained by 21% below control and glutathione-S-transferase activity was by 17% above control.

Conclusions. The administration of melatonin against the background of alcohol intoxication or its combination with constant light exposure contributed to the normalization of catalase activity in rats' kidneys but revealed less effective in normalization of glutathione peroxidase and glutathione-S-transferase activity in kidneys.

Dikal M.V.

**MITOCHONDRIAL DYSFUNCTION AS A KEY FACTOR IN THE DEVELOPMENT OF
PATHOLOGICAL PROCESSES AND DISEASES**

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Introduction. Mitochondria play a central role in cellular metabolism, performing several vital functions, including energy production in the form of adenosine triphosphate (ATP) through

oxidative phosphorylation, regulation of apoptosis, involvement in lipid, protein, and amino acid metabolism, as well as maintaining intracellular calcium homeostasis. The process of energy synthesis is accompanied by the formation of reactive oxygen species (ROS), which can affect the structure and function of cells, leading to serious health consequences and contributing to the development of various pathological processes and diseases.

The aim of the study. To investigate the causes and consequences of mitochondrial dysfunction in the pathogenesis of diseases.

Materials and methods. Databases such as Web of Science, Pubmed, and Scopus were analyzed.

Results. Mitochondrial dysfunction is a central factor in the development of many pathological processes, as it leads to disruptions in energy metabolism, insufficient energy production, and, consequently, reduced functional activity of cells. This is particularly critical for cells with high energy demands, such as those in the heart, brain, and muscles. For example, in neurodegenerative diseases like Parkinson's and Alzheimer's, energy deficiency results in metabolic disturbances in neurons, accompanied by the accumulation of reactive oxygen species, protein damage, and neuronal cell death.

Excessive formation of ROS and free radicals causes damage to the structure of DNA, proteins, and lipids, leading to genetic mutations, impaired protein functions, and destruction of cellular membranes. These processes form the basis for the development of chronic inflammatory processes and autoimmune diseases. Mitochondrial dysfunction also significantly contributes to the development of metabolic disorders such as type 2 diabetes, obesity, and other metabolic conditions. Defects in oxidative phosphorylation disrupt metabolic pathways, leading to insulin resistance and further complications.

Mitochondrial dysfunction can cause both uncontrolled activation of apoptosis and its suppression, which, in turn, contributes to the development of degenerative diseases or cancer pathologies. Disruption of the apoptosis process stimulates glycolysis reactions and the formation of ROS, creating favorable conditions for the survival and aggressive growth of cancer cells.

Conclusion. Mitochondrial dysfunction is the basis of many neurodegenerative, cardiovascular, metabolic, and oncological diseases. Studying the mechanisms of mitochondrial dysfunction and developing new therapeutic strategies can significantly improve the effectiveness of treatment and prevention of these conditions, opening new possibilities for enhancing the quality of life for patients.

Ferenchuk Ye.O.

CATALASE ACTIVITY IN THE BLOOD AND LIVER OF RATS BY EXPERIMENTAL NEPHROPATHY AND INFLUENCE OF GLUTATHIONE

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Introduction. Under the conditions of entry of toxic substances into the body and activation of redox processes, compounds of natural origin with antioxidant properties are widely used to correct metabolic disorders. Among water-soluble antioxidants, low- and high-molecular compounds containing SH-groups are popular in medicine, mono-, di- and tricarboxylic acids and other anions. Glutathione, a thiol-containing tripeptide, is the main endogenous non-enzymatic antioxidant that exerts cytoprotective and detoxifying properties. The function of the glutathione system affects the implementation of important physiological and biochemical processes: detoxification, antioxidant protection, transformation vitamins C and E, lipoic acid, ubiquinone, regulation of thiol disulfide balance, synthesis of nucleic acids, preservation of the optimal state and functions of biological membranes, participates in the exchange of eicosanoids - prostaglandins and leukotrienes. Oxidative modification or protein carbonylation, which occurs at depletion of glutathione, stimulates covalent modification of endogenous enzymes and proteins, which can lead to loss of their functions. In the kidneys, there are peculiarities of glutathione metabolism.