МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»



МАТЕРІАЛИ

105-ї підсумкової науково-практичної конференції з міжнародною участю професорсько-викладацького персоналу БУКОВИНСЬКОГО ДЕРЖАВНОГО МЕДИЧНОГО УНІВЕРСИТЕТУ присвяченої 80-річчю БДМУ 05, 07, 12 лютого 2024 року

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Матеріали підсумкової 105-ї науково-практичної конференції з міжнародною участю професорсько-викладацького персоналу Буковинського державного медичного університету, присвяченої 80-річчю БДМУ (м. Чернівці, 05, 07, 12 лютого 2024 р.) – Чернівці: Медуніверситет, 2024. – 477 с. іл.

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У збірнику представлені матеріали 105-ї підсумкової науково-практичної конференції з міжнародною участю професорсько-викладацького персоналу Буковинського державного медичного університету, присвяченої 80-річчю БДМУ (м. Чернівці, 05, 07, 12 лютого 2024 р.) із стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

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Dia-PTT LiQuid test results can be reported in the following units: - seconds, which means the observed clotting time; - ratio (APTT/MNPTT), which means the clotting time of the sample divided by the mean normal APTT (MNPTT)

Dia-PT test results were reported in the following units: - seconds, which means the observed clotting time; - percentage, which means the proportional part of the normal PT activity, which is calculable from the calibration curve; - INR which means the ratio raised to the power of International Sensitivity Index (ISI).

 $INR = (PT/MNPT)^{ISI}$. MNPT means normal prothrombin time.

The INR is only officially recognized dimension of the result at vit. K antagonists treated patients. The normal range expressed in INR is 0.8-1.2. Reference ranges are following on Diagon analyzers (Coag Line).

Non-parametric methods were used for statistical calculation and comparison of results between two groups: Wilkinson-Mann-Whitney and Kruskal-Wallis criteria. The influence of a factor on a sign was detected by Kruskal-Wallis criterion. The results were considered reliable with p > 0.05.

Results. The main results of the research are presented in the table.

Table.

Comparative analysis of indicators of the blood plasma coagulation system of patients with endometriosis with similar indicators of the control (healthy) group $(M \pm m)$

The research group	PT_{sec}	APTT _{sec}	INR _{ratio}	index APTT
Patients with endometriosis (n=38)	$\frac{(M \pm m)}{14.60 \pm 1.53^*}$	$(M \pm m)$ 32.50 ± 3.12	$(M \pm m)$ 1.21 ±0.08*	1.18 ± 0.07
Control group (n=23)	12.82 ± 0.95	30.42 ± 2.57	1.05 ± 0.06	1.12 ± 0.06
Note: * the groups differ reliable				

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Conclusions. When comparing indicators of the coagulation and homeostasis' system between a group of healthy women and a group of patients with endometriosis, a probable decrease in PT and INR was noted in patients with endometriosis. No significant difference in APTT was noted.

Davydova N.V.

THE STATE OF ANTIOXIDANT SYSTEM IN RATS' KIDNEYS UNDER ALCOHOLIC INTOXICATION AND ITS COMBINATION WITH LIGHT EXPOSURE

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Introduction. Although the negative effects of excessive alcohol consumption are generally known in the human population, drinking alcoholic beverages is prevalent in society. According to WHO, alcohol abuse contributes to three million deaths per year globally and millions of people's disabilities and organ damage.

In modern life, the use of ethanol is often combined with the influence of other harmful factors, such as the violation of the light regime. A modern person is exposed to light almost all the time. Night shifts, flights, jet lag, and active nightlife contribute to the disturbance of circadian rhythms. Normally, the biological rhythms are regulated by melatonin, which is known to be secreted in the dark. Even a slight lighting inhibits its synthesis. It has been shown that melatonin has a wide range of biological effects, but its main feature is a powerful antioxidant action.

The aim of the study. To investigate the effects of melatonin on antioxidant enzymes activity (catalase and glutathione peroxidase) in the kidneys of rats exposed to alcohol intoxication and its combination with 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%. 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. The decrease in the antioxidant enzymes 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 20^{00} for 7 days to animals exposed to ethanol intoxication caused normalization of catalase and glutathione peroxidase 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.

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 activity in kidneys.

Dikal M.V.

THE ROLE OF MITOCHONDRIAL REACTIVE OXYGEN SPECIES IN DEVELOPMENT OF METABOLIC DISORDERS IN CELLS

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Introduction. Oxidative stress is associated with many human diseases, including cardiovascular, neurodegenerative, hepatic diseases, and cancer. Mitochondria represent an important target for oxidative damage, which can lead to cell death because damaged mitochondria produce increasingly more reactive oxygen species (ROS). Produced ROS often activate redox-sensitive enzymes of protective signaling pathways and may directly influence cell viability. The accumulation of ROS causes damage to DNA, proteins, and lipids, and links to other pathological processes.

The aim of the study. To study the activity of the mitochondrial energy supply system under the influence of various reactive oxygen species.

Materials and methods. The databases Pubmed, Scopus, Jama were analized.

Results. Mitochondrial ROS are crucial for an organism's homeostasis. By regulating signaling pathways, they activate the adaptation and protection behaviors of an organism under stress. The accumulation of ROS causes damage to DNA, proteins, and lipids, and other pathological processes. ROS are different products from the partial reduction of oxygen, including oxygen free radicals: superoxide $[(O_2^{-}], hydroxyl [OH^{+}], alkoxyl [RO^{+}])$, and some non-radical derivatives of oxygen (singlet oxygen (¹O₂), hydrogen peroxide (H₂O₂), and hypochlorous acid (HOCl)).

Hydroxyl radicals are short-lived, highly reactive, and contribute significantly to local organelle damage through protein modification. The intensive generation of ROS can result from the action of p450 monooxygenase, mitochondrial oxidative phosphorylation, monoamine oxidase, lipoxygenase, xanthine oxidase, cyclooxygenase. Mitochondria are not only the source of energy through oxidative phosphorylation on the inner membrane, but also the process of mitochondrial oxidative phosphorylation is the main origin of free radicals. Free radicals a decrease in mitochondrial respiratory function because they impair mitochondrial structure and function by increasing mitochondrial free radical production.

Conclusions. Increased levels of reactive oxygen species and free radicals generated in damaged mitochondria cause oxidative damage and significant disruption of the metabolic processes of various tissues, impair the flow of electrons along the electron transport chain, increase mitochondrial membrane potential, respiratory control coefficients and cellular oxygen consumption. Studying the relationship between oxidative stress and mitochondrial dysfunction