МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»

М А Т Е Р І А Л И

105-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького персоналу
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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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assessments.Statistical processing of the obtained data was performed using the SPSS Statistics 17.0 software. All data are represented as a mean ± standard error of the mean (M±m). Estimation of the differences between the samples was conducted using a parametric Student's t-test and a nonparametric Mann-Whitney U test. P.

Results. Cellular toxicity of acetaminophen is associated with translocation and dysfunction of Na+-K+-ATPase, which ensures effective sodium reabsorption. In rats with acetaminophen-induced AKI a decrease in sodium reabsorption and, accordingly, an increase in fractional sodium excretion was found. An increase in the sodium concentration in the tubular fluid led to the activation of tubuloglomerular feedback with a 2-fold decrease in glomerular filtration rate (GFR), reduced urine output, and development of retention azotemia. Significant proteinuria compared to the control confirms the severe toxic damage to renal tubular cells. In animals that received melatonin treatment (group III) renal dysfunction was less pronounced. Melatonin counteracted the nephrotoxic effect of acetaminophen, as evidenced by the prevention of significant sodium loss due to maintenance of the reabsorption capacity of tubular cells, restoration of urine output due to maintenance of GFR, and prevention of retention azotemia and significant proteinuria. Acetaminophen overdose induced the oxidative stress from the intensification of ROS production, lipid and protein peroxidation processes and the simultaneous decline of the enzymatic antioxidant capacity. Melatonin showed a significant antioxidant effect manifested in attenuation of both lipid and protein peroxidation in the kidney tissue, along with an increase in the GPx and CAT activity compared to untreated animals.

Conclusions. In conditions of acetaminophen-induced AKI nephroprotective effect of melatonin manifests by the preservation of the kidney function and restoration of the local prooxidant-antioxidant balance. Results of research complement to existing data on the nephroprotective activity of melatonin and substantiate the high therapeutic potential and prospects of melatonin use as adjunctive therapy of drug-induced nephropathy.

Lisnyanska N.V.

THE ROLE OF OXIDATIVE PROCESSES IN THE DEVELOPMENT OF THE PATHOLOGICAL COURSE OF CHRONIC ENTEROCOLITIS IN EXPERIMENTAL ANIMALS WITH STREPTOZOTOCIN DIABETE

Department of Pathological Physiology
Bukovinian State Medical University

Introduction. Diabetes is the 3rd most common disease after cardiovascular diseases and cancer. According to WHO, diabetes increases mortality by 2-3 times and shortens life expectancy. Almost 3% of the primary disability of the adult population of Ukraine occurs precisely because of diabetes. Annually, the number of patients increases by 5-7%. According to the Diabetes Atlas of the International Diabetes Federation (IDF), as of 2021, the number of people with diabetes (20-79 years old) in Ukraine is 2 million 325 thousand.

Diabetes mellitus has been found to be one of the three most common comorbidities in patients with chronic enterocolitis. This creates prerequisites for a deeper study under experimental conditions of biochemical processes occurring in the intestinal wall and blood in diabetes, enterocolitis and their combination.

The aim of the study. To reveal the peculiarities of oxidative processes and establish their role in the development of the pathological process in experimental animals with chronic enterocolitis on the background of streptozotocin diabetes.

Materials and methods. The study was performed on white outbred sexually mature male rats, which were divided into 4 groups of 12 animals. Modeling of diabetes mellitus was carried out by a single intraperitoneal injection of streptozotocin (Sigma Aldrich, USA, at a dose of 60 mg/kg of body weight) to 2-month-old animals. The control group of animals was injected with the appropriate amount of citrate buffer. Chronic enterocolitis was reproduced by free access of 2.5-month-old animals to 1.0% carrageenan solution in drinking water for 1 month. The development of diabetes in experimental animals was confirmed by determining the fasting blood glucose level,

chronic enterocolitis was confirmed morphologically (for qualitative and quantitative analysis of the degree of structural damage to the small intestine).

Results. Certain aspects of the course of experimental chronic enterocolitis against the background of streptozotocin diabetes have been clarified, which indicate a violation of biochemical processes in the small intestine in the pathology under study. It has been established that in conditions of chronic enterocolitis on the background of diabetes, oxidative processes in the tissues of the small intestine are activated: the level of neutral and basic aliphatic aldehyde- and ketone-dinitrophenylhydrazones increases with the predominance of protein fragmentation phenomena ($p<0.05$), the content of diene conjugates increases (by 104.84%) and TBC-active products (by 115.02%) ($p<0.01$) and the activity of superoxide dismutase (by 131.09%) and catalase (by 21.65%) decreases, which is also reflected in biochemical indicators of the blood of the studied animals.

In rats with streptozotocin diabetes during the development of chronic enterocolitis, the development of endogenous intoxication was recorded for the first time, as indicated by an increase in the total pool of substances of low and medium molecular weight in the plasma (by 2.4 times, $p<0.001$). The metabolic result of endotoxicosis in chronic enterocolitis against the background of streptozotocin diabetes in rats is an increase in cell destruction, which is confirmed by an increase in the level of nucleic acids in the blood of rats by 76.2%, compared to the control ($p<0.001$).

Conclusions. In the case of chronic enterocolitis against the background of streptozotocin diabetes, the intensification of oxidative destruction processes is accompanied by changes in the enzymatic activity of energy supply processes, which leads to destructive damage to the cells of the small intestine.

Semenenko S.B.

FEATURES OF THE INFLUENCE OF NUTRITION ON THE CIRCADIAN RHYTHMS OF THE HUMAN BODY

Ya.D. Kirshenblat Department of Physiology
Bukovinian State Medical University

Introduction. Based on scientific literary sources the influence of nutrition, energy exchange and biorhythms on body weight control and prevention of metabolic diseases is described. Interference with the sleep-wake cycle, as well as disorders of diet, lead to unfavorable metabolic processes. Maintaining the integrity of the system of biorhythms is an antidote to the development of metabolic disorders.

The aim of this study. To investigate the peculiarities of the influence of nutrition on the circadian rhythms of the human body.

Material and methods. Literary sources of foreign and domestic authors were used in the work, and their systematic analysis was carried out.

Results. Nutrition is one of the constant, most effective external factors affecting the human body in many ways. Scientists have accumulated a lot of information about the interaction of nutrition and the system of biological rhythms. Biorhythms are considered as one of the main mechanisms of human adaptation to environmental conditions — changes in the length of the light period, temperature regime, geomagnetic influences, as well as to the organization of work and nutrition. A powerful motivation for the study of the connection between nutrition and biorhythms, in particular circadian rhythms, was the study of energy metabolism disorders. Scientific studies indicate that eating fatty food leads not only to obesity, but also to disruption of the body's daily biological rhythm. In addition to the influence of light on chronobiorhythms, there is a cause-and-effect relationship between the peculiarities of nutrition and the imbalance of a biological clock. Scientists have investigated that a diet with a high fat content affects, like the biological clock, the process of releasing adiponectin - a protein secreted by adipose tissue cells and associated with the processes of glucose assimilation and lipid metabolism, which leads to inhibition of chronobiorhythms. It has been studied that each meal causes fluctuations in the activity of digestive enzymes, fluctuations in the concentration of hormones in the blood, which go beyond the time of