



fluid, positron emission tomography (PET) with ligands tropic to β - and fluorodeoxyglucose, which makes it possible to assess the metabolic rate of various parts of the brain; structural MRI with the ability to assess the degree of atrophy of the cerebral cortex.

Unfortunately, there are currently no drugs that stop the progression of Alzheimer's disease. To date, the FDA has approved two classes of drugs - acetylcholinesterase inhibitors (AChE) and N-methyl-D-aspartate (NMDA) antagonists - to relieve some cognitive symptoms of Alzheimer's disease, such as memory problems and other mental disorders. Donepezil, rivastigmine and galantamine are used to treat mild to moderate Alzheimer's disease (donepezil can also be used in severe Alzheimer's disease). Memantine is used to treat moderate to severe Alzheimer's disease. These drugs work by regulating neurotransmitters, chemicals that transmit messages between neurons. They can help reduce symptoms and help solve certain behavioral problems. However, these drugs do not change the underlying disease process. They are effective for some, but not all, people and can only help for a limited time. Alzheimer's therapy uses drugs and a set of measures aimed at preserving, restoring and training cognitive function (it is necessary to follow a diet, overcome the fear of death, maintain regular physical activity, communicate with friends and relatives, be socially active, etc.). As for all geriatric patients, patients with Alzheimer's disease are characterized by decreased excretory system function and the presence of a large number of comorbidities.

All this significantly limits the possibilities of pharmacotherapy and makes it important to find ways to improve the effectiveness of pharmaceutical care for these patients. Currently, there is no specific therapy for dementia, in connection with which treatment should be aimed at preventing the growth of disorders and correction of existing disorders, psychotherapy and social support of the patient.

To address the problem of Alzheimer's disease around the world, it is necessary to make research in the field of dementia a global priority, to promote international cooperation to intensify efforts to find drugs that can stop or prevent brain disorders caused by Alzheimer's.

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**THE NEPHROPROTECTIVE ACTIVITY OF LIPIN
IN GENTAMICIN-INDUCED NEPHROPATHY**

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Despite the development of diagnostics and the improvement of treatment, the number of nephrological patients in the XXI century is growing among adults and children. A significant increase in the number of kidney lesions is manifested against the background of endocrine, allergic diseases, alcohol and drug use, and under conditions of drug toxicity. Most of the overall structure of acute kidney damage is occupied by nephropathy caused by antibiotics, including aminoglycosides.

Aminoglycosides cause the development of tubular dysfunction and necrosis of tubular epithelial cells. According to the literature, the accumulation of gentamicin in the cortical layer of the kidneys causes structural and functional disorders of the proximal tubules of the kidneys, which are most associated with the formation of reactive oxygen species and weakening of antioxidant protection. Reduction of antibiotic toxicity may be achieved through the combined use of substances that can prevent or reduce the development of oxidative stress caused by gentamicin in the kidneys of animals. For this reason, a powerful antioxidant, namely lipin, has attracted our attention as a means of pathogenetic correction of gentamicin. The aim of the research is to study nephroprotective potential of lipin in conditions of gentamicin-induced nephropathy development in rats.

Research was conducted on 21 mature non-linear white rats weighting 130-180 g, randomly divided into 3 groups (n = 7): I group – intact control, II group – gentamicin- induced nephropathy (injection of 4% gentamicin sulfate solution at a dose of 80 mg/kg for 6 days),



rats of III group were daily administered lipin at a dose of 50 mg/kg. Functional state and histological changes in kidneys were estimated on the 7th day.

Administration of the investigational drug in a prophylactic-therapeutic regimen resulted in a significant reduction of the degree of damage to nephrocytes in rats with gentamicin-induced nephropathy. Lipin demonstrated high nephroprotective efficacy, as evidenced by an improvement of the excretory kidney function with an increase in GFR by 2.3 times ($p < 0,01$), diuresis – by 65.5% ($p < 0,01$), reduction of azotemia and proteinuria. The protective effect of lipin on the epitheliocytes of the proximal tubules is confirmed by an increase in reabsorption capacity and a corresponding decrease in fractional sodium excretion by 3.2 times ($p < 0,01$), as well as the normalization of tubular-channel balance. At the same time, under the influence of lipin, antioxidant protection is activated, which manifests itself in a decrease in the content of peroxidation products in both erythrocytes and kidney tissue, a decrease in catalase activity, ceruloplasmin content and an increase SH-groups compared to untreated animals. Therefore, in the gentamicin model of AKI, lipin normalizes the state of prooxidant-antioxidant balance in animals, suppressing lipid peroxidation intensity.

Lipin has a nephroprotective effect in gentamicin-induced nephropathy, and the results may serve as a basis for further study in acute renal injury of various etiologies.

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STUDY OF THE FUNCTIONAL STATE OF KIDNEYS IN CONDITIONS OF FEVER

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Kidney and urinary tract diseases, especially of microbial-inflammatory origin, occupy one of the leading places in the structure of somatic pathology. The kidneys play an important role in the maintaining homeostasis, which is characterized by a constant volume of fluid, its osmotic concentration and ionic composition. Therefore, any renal dysfunction can lead to significant changes in electrolyte and water-salt homeostasis. The extreme degree of these disorders indicates a disturbances of the basic homeostatic constants of the body.

According to the literature, about 90% of kidney diseases are accompanied by fever, which often develops in response to the effects of pyrogens of viral or bacterial nature, which in general significantly impairs the body's compensatory capacity and affects the course and consequences.

On the other hand, it is known that fever in infectious diseases is a protective response. Rise in the body temperature activates metabolic processes, functions of the nervous, endocrine, immune systems (an increase in the production of antibodies, interferon, and stimulation of the phagocytic activity of neutrophils), increase in the antitoxic function of the liver, increase in the renal circulation.

Recently, a highly active broad-spectrum immunomodulator pyrogenal has been widely used to induce fever. Pyrogenal is a protein-free exogenous highly pyrogenic lipopolysaccharide, which acts by activating the production of macrophages and polymorph nuclear leukocytes, endogenous pyrogens, which results in a shift of the set point of thermoregulation to a higher level. Pyrogenal affects the thermoregulatory centre of the hypothalamus and also has desensitizing, anti-inflammatory properties, increases the general and specific resistance of the body. Following the pyrogen administration, antigen or mitogen binds to the cellular receptor, promoting the proliferation of lymphocytes and stimulating the synthesis and secretion of Ig, potentiating the factors of nonspecific resistance and cellular immunity.

Therefore, the aim of research is to study the effect of fever on the structural and functional state of kidney and possible mechanisms of renal dysfunction; to study the function of kidney during the first and second stage of fever, the renal mechanism of autoregulation – glomerular-tubular and tubular-tubular balance in the stage of temperature decrease, biochemical and histological characteristics of the kidney cortex and medulla in the dynamics of fever development.