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TABLE OF CONTENTS

BUSINESS ECONOMICS

REGIONAL ECONOMY

FINANCE AND CREDIT

ACCOUNTING AND AUDITING

Proceedings of the 6th International Scientific and Practical Conference «Science in the Environment of Rapid Changes»

(June 16-18, 2025). Brussels, Belgium

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MEDICINE AND PHARMACY

The state of the prooxidant system in the cerebral cortex of rats with scopolamine-induced neurodegeneration and under the influence of enalapril

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Neurodegenerative diseases are characterized by progressive damage to nerve cells and loss of neurons, leading to impaired motor and cognitive functions. The pathophysiological processes that accompany this pathology, in particular amyotrophic lateral sclerosis, Parkinson's disease, Alzheimer's disease, are far from being fully explained. The complex pathogenesis of neurodegenerative diseases remains largely unknown; however, growing evidence suggests that reactive oxygen species may play a critical role, since high levels of them are commonly observed in the brains of patients with neurodegenerative conditions [1]. Therefore, oxidative stress has been proposed as one of the factors that plays a potential role in the pathogenesis of neurodegeneration.

Neurons are known to be highly sensitive to oxidative stress, as their functions depend on oxidative phosphorylation, which is used as an energy source compared to other cells. However, as neurodegenerative conditions progress, the ability of cells to maintain redox balance decreases, leading to free radical accumulation, mitochondrial dysfunction, and neuronal damage [2]. This will result in complex changes in cellular metabolism and damage to cell membranes, oxidative modification of proteins and lipids, which reduces or completely eliminates their

MEDICINE AND PHARMACY

functional activity.

For many years, clinicians have used modulators of the renin-angiotensin system (RAS) to combat hypertension. However, it has recently been demonstrated that they have other pleiotropic properties independent of their hypotensive effects, such as enhancing cognition [3]. Interestingly, crosstalk between the RAS and other systems, such as the cholinergic, dopaminergic, and adrenergic systems, has been demonstrated. The process of cognitive progression may either be caused or exacerbated by RAS dysfunction through an imbalance of angiotensin receptors to affect cognition. In this regard, the effect of enalapril on protein and lipid peroxidation processes has attracted our attention.

Therefore, the aim of our work was to study the effect of enalapril on peroxidation of proteins and lipids in the cerebral cortex of rats with a scopolamine-induced model of neurodegeneration.

Materials and methods. The experiments were conducted on non-linear white male rats weighing 0.18-0.20 kg, which were kept under standard vivarium conditions. The studies were carried out in compliance with the Council of Europe Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes. To create a model of neurodegeneration, scopolamine hydrochloride (Sigma, USA) was administered intraperitoneally at a dose of 1 mg/kg. On the 28th day, the rats with the neurodegeneration model were blindly divided into two groups: I- with intraperitoneal administration of enalapril at a dose of 1 mg/kg; II - with the administration of only 1 ml of saline solution (14 days). The control rats were administered 1 ml of saline solution from the 28th day. The content of oxidative modification of proteins in homogenates was determined by the number of products of their oxidative modifications by spectrophotometry at wavelengths of 370 and 430 nm. The intensity of lipid peroxidation was estimated by the content of products reacting with 2-thiobarbituric acid (TBCAP). Statistical analysis of the results of the study was performed using the Student's t-test.

Results. It was found that compared to the control group, in rats with a neurodegeneration model, the content of neutral proteins increased by 1.3 times, and basic proteins by 1.2 times. At the same time, neutral proteins were more susceptible to peroxide oxidation. An increase in the degree of oxidative modification of proteins in the studied

MEDICINE AND PHARMACY

homogenates is evidence of damage to brain proteins under neurodegeneration conditions. After the use of enalapril, peroxidation of proteins in the cerebral cortex, which was registered at λ=370 nm, decreased by 1.1 times compared to the data of the model pathology. Registration of oxidative modification of proteins at λ=430 nm revealed a decrease in the content of neutral and basic proteins by an average of 1.1 times under the influence of enalapril. It is worth noting that in rats with scopolamine-induced neurodegeneration after 14 days of enalapril administration, the degree of protein damage decreased.

When analyzing the results, rats with scopolamine neurodegeneration showed an increase in the amount of TBCAP by 1.3 times compared to the control group. In rats that were administered enalapril, the content of TBCAP decreased by 1.1 times compared to rats with model pathology. However, this indicator remained higher than in the control group.

Conclusion. Thus, the results of the study of peroxidation of proteins and lipids of the cerebral cortex under the conditions of suppression of central cholinergic effects by scopolamine indicate the development of cerebral degeneration. After the administration of enalapril, the degree of oxidative modification of proteins decreases and the content of TBCAP significantly decreases. The results obtained indicate the ability of enalapril to inhibit the development of neurodegeneration in scopolamine-induced brain damage.

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