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ABSTRACTS


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EPR study of the nitric oxide content in hippocampus and liver of rats in the simulation of cerebral ischemia followed by reperfusion

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With a decrease in the oxygen content in the inhaled air, violations of cerebral blood flow, leading to a lack of oxygen supply to the brain, cerebral ischemia occurs, which can result in an ischemic stroke, accompanied by damage to brain tissue and its functions [1]. During hypoxia and ischemia of the brain, the functioning of neurotransmitter systems, including the nitrogen monoxide system, is disrupted. Currently, there is no consensus on the role of endogenous NO in the processes occurring with damage to the nervous system [2]. In addition to vasodilating, neurotransmitter and stress-limiting properties, the involvement of NO in the reactions of oxidative stress, the glutamate-calcium cascade and inflammation has been demonstrated [3]. It has been shown that the development of cerebral ischemia and the subsequent occurrence of stroke are associated with a weakening of cerebral blood flow, as well as with violations of the regulation of blood supply to brain tissues by the NO system [4]. A violation of the oxygen supply to the brain also occurs when a vessel is thrombosed or an aneurysm ruptures, which often ends with an ischemic or hemorrhagic stroke [5]. Cerebral ischemia causes multiple and multidirectional changes in the NO content in the brain and in signal transmission. The available contradictory information suggests that there is currently no consensus on the role of endogenous NO in the processes occurring with damage to the nervous system [6]. According to the research results, the role of NO in these hypoxia-ischemia processes is contradictory: NO is able to perform both neurotoxic and neuroprotective functions [7]. The aim of our work was to study the intensity of NO production in the hippocampus of rats after ischemic brain damage by spectroscopy of electron paramagnetic resonance (EPR).

Ischemia was simulated at the Institute of Physiology of the National Academy of Sciences of Belarus, Minsk by means of a 10-minute violation of blood flow by ligation of both carotid arteries at the level of the vocal cords and taking 3 ml of blood from the common carotid artery. Measurement of NO content in brain tissue were performed by the method of electron paramagnetic resonance (EPR) [8, 9]. The method allows direct measurements, and is highly sensitive due to the use of spin traps. The components of the spin trap NO (DETC-Na, $FeSO_4$, sodium citrate) was injected 30 min before the extraction of studied tissue. The measurements of the spectra of the complex $(DETC)_2$-Fe^{2+}-NO were performed on the spectrometer EMX/plus with a temperature module ER 4112HV in the X band (9.50 GHz). The amplitude of the EPR-spectra was always normalized to the weight of the sample.

30 minutes after the introduction of the spin trap components, hippocampal and liver tissues were taken (one sample, about 100 mg). The selected areas were immediately frozen at liquid nitrogen temperature and transported from Minsk to Kazan in plastic containers with dry ice. A significant decrease in NO production in the hippocampus was shown 1 day after modeling an ischemic stroke caused by carotid artery ligation, and by 56% – during carotid artery ligation followed by taking 3 ml of blood from the common carotid artery.

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