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THE STATE OF MITOCHONDRIA IN HIPPOCAMPAL NEURONS OF RATS WITH METABOLIC SYNDROME AND AFTER CARBACETAM ADMINISTRATION

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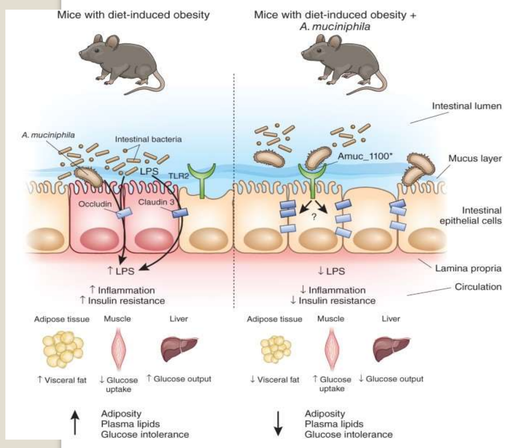

Urbanization and the associated sedentary lifestyle and overeating are the main causes of the modern health epidemic – metabolic syndrome. This complex and multifactorial disorder, which affects a significant portion of the world's population and is characterized by a group of interrelated conditions that increase the risk of cardiovascular disease, stroke and type 2 diabetes. Considering the fact that mitochondria plays an important role in energy metabolism and oxidative stress, which is one of the molecular mechanisms damaging different organs and tissues with metabolic syndrome, their functional changes are of certain interest.

In this respect, learning the alterations of the parameters characterizing the functional state of the hippocampal mitochondria under effect of carbacetam as a (GABA)-ergic system is rather interesting.

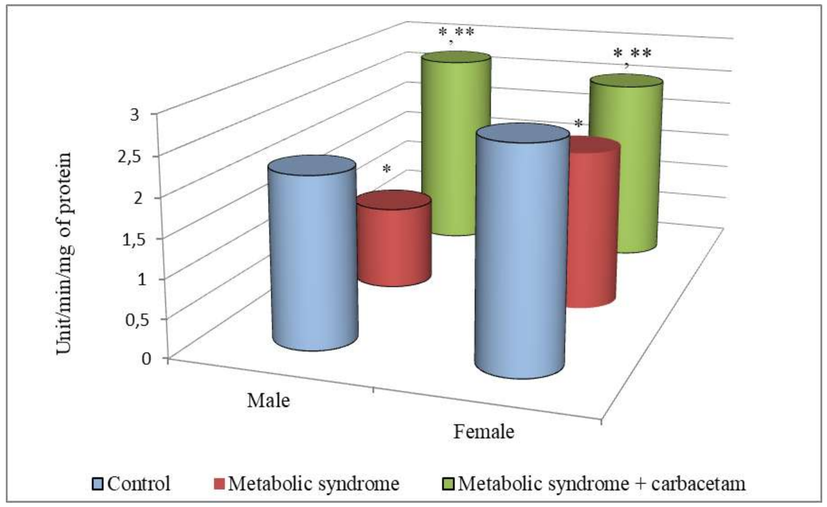

Fig. Relative rate of mitochondrial swelling in the hippocampus of rats with metabolic syndrome after carbacetam administration during 14 days in the dose of 5 mg/kg (M±м, n=7)

Notes: – reliability of differences compared with the control group of rats, ** – reliability of differences compared with the group of rats with metabolic syndrome.*

Results: Simulated metabolic syndrome was found to manifest by a decreased light scattering and an increased relative rate of mitochondrial swelling in the hippocampal mitochondrial fraction; increased free radical lipid and protein oxidation with more marked changes in males. When rats with metabolic syndrome receive carbacetam during 14 days, in their mitochondrial fraction light scattering and relative rate of mitochondrial swelling decrease, both in males and females.

Conclusions: Thus, a decreased intensity of mitochondrial swelling and improved condition of the antioxidant system of the hippocampal mitochondria of rats with metabolic syndrome irrespective of their sex is indicative of the effective correction of GABA receptors by means of carbacetam under conditions of the experiment.