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SCIENTIFIC PRACTICE: MODERN AND CLASSICAL RESEARCH METHODS

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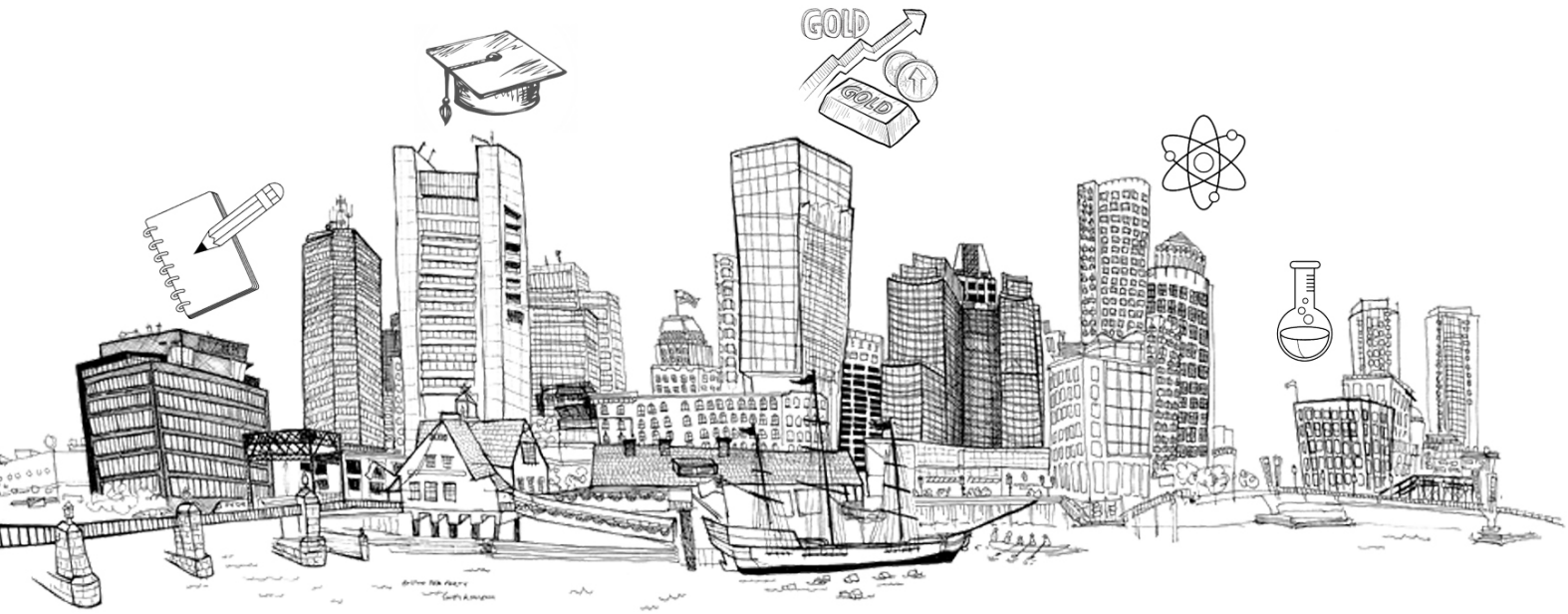

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SECTION XXIII. PHARMACY AND PHARMACOTHERAPY

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EFFECT OF COORDINATION COMPOUND OF GERMANIUM WITH NICOTINIC ACID ON THE EXPRESSION OF MARKERS OF NERVOUS TISSUE DAMAGE IN RATS UNDER CONDITIONS OF CHRONIC ETHANOL CONSUMPTION

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Introduction. Long-term alcohol consumption can lead to the development of oxidative stress, changes in the structure and functions of the brain [1], which disrupts the functioning of the body as a whole.

Neurospecific proteins such as glial fibrillary acidic protein (GFAP), a component of the glial cytoskeleton astrofibrils [2], ionized calcium-binding adaptor molecule 1 (Iba-1), which is mainly expressed in microglia and macrophages [3], and neurofilament heavy polypeptide (NF-H), a component of the intermediate filaments of the nerve cell cytoskeleton [4], were chosen as markers of nerve tissue damage.

The complex of germanium with nicotinic acid (MIGU-1) was studied in view of its potential effect on various indicators of oxidative stress under conditions of experimental pathologies with possible further use as a therapeutic agent [5,6].

The aim of the study was to evaluate the expression of neurospecific proteins GFAP, Iba-1 and NF-H in the brain of rats subjected to chronic semi-voluntary ethanol consumption and the possibility of the correction of changes detected by administration of MIGU-1.

Materials and methods. The study was carried out on Wistar rats weighing 198.0±3.0 g in autumn-winter period in accordance with the Directive 2010/63/EU [7]. Animals were divided into 3 groups. Intact animals had free access to water and food on a standard diet. Animals with chronic ethanol consumption had 20% C_2H_5OH as the only source of fluid for 3.5 months. Experimental animals had chronic ethanol

consumption and additionally received intraperitoneal administration of MIGU-1 [6] complex at a dose of 10 mg/kg/day 3 weeks before the end of the experiment. Animals were euthanised by decapitation under mild ether anaesthesia. The cerebral hemispheres were immediately removed and frozen with liquid nitrogen. The following antibodies and concentrations were used for Western blot analysis: GFAP (1:1000, sc-9065, Santa Cruz Biotechnology, USA), NF-House monoclonal NEFH Antibody RMdO-20 (1:1000, Invitrogen, USA, cat. no. 13-1300), Iba-1 (1:1000,, Invitrogen, USA, #MA5-27726) i beta Actin (1:5000, Loading Control Monoclonal Antibody (BA3R) #MA5-15739, Invitrogen,USA). The relative content of proteins was expressed in arbitrary units of optical density. Statistical processing of the results was performed using the Prism 9.5.1 software. The degree of reliability (P) of the results was assessed using the Student's t test.

Results. The study of the level of glial fibrillary acidic protein in animals subjected to chronic semi-voluntary ethanol consumption showed an increase in the expression of this protein in the brain hemispheres by 3.33 times compared to the value of the group of intact animals ($P \leq 0.05$). Such changes in GFAP indicate the activation and proliferation of astrocytes in response to the use of 20% ethanol for 3.5 months. The latter characterises the development of pathological changes in the brain of rats under chronic semi-voluntary ethanol consumption. The administration of MIGU-1 led to a 1.8-fold decrease in GFAP expression in animals of the experimental group compared to animals with chronic ethanol consumption ($P \leq 0.05$).

The expression of calcium-binding peptide Iba-1 in the studied brain regions of the experimental group rats increased 3.9-fold compared to the group of intact animals ($P \leq 0.05$). This indicates the activation of microglia in response to long-term consumption of 20% ethanol. The administration of MIGU-1 caused a 2.1-fold decrease in the level of Iba-1 in rat's cerebral hemispheres compared to animals with chronic ethanol consumption ($P \leq 0.05$).

Analysis of the expression of neurofilament heavy chains showed that in rats subjected to chronic semi-voluntary ethanol consumption, the level of NF-H in the brain hemispheres did not change significantly compared to intact animals (**$P > 0.05$**). These data indicate that there is no damage to the structure of neurons under chronic consumption of 20% ethanol. The administration of MIGU-1 in the setting of 20% ethanol resulted in an increase in the level of neurofilament heavy chains compared to animals with chronic ethanol consumption (**$P \leq 0.05$**). However, there was no significant difference between intact animals and the experimental group (**$P > 0.05$**).

Conclusions. The obtained results demonstrate the sensitivity of neuroglia to chronic consumption of 20% ethanol for 3.5 months. No changes in the level of neurofilament heavy polypeptide in rat's nervous tissue were observed. The administration of MIGU-1 reduced the level of neuroglial proteins, which opens up the prospects for its study as a therapeutic agent for the treatment of neurological and neuropsychological disorders.

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НАУКОВЕ ВИДАННЯ

ΛΌΓΟΣ

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З МАТЕРІАЛАМИ IV МІЖНАРОДНОЇ
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