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Changes in the Number of CD38 and Cx43-Immunopositive Cells in the Neurovascular Unit of the Brain in Experimental Alzheimer's Disease

E. D. Khilazheva[a, *], A. I. Mosiagina[a], A. V. Morgun[a], N. A. Malinovskaya[a], Ya. V. Gorina[a], E. V. Kharitonova[a], O. L. Lopatina[a], and A. B. Salmina[a, b]

[a] *Research Institute of Molecular Medicine and Pathobiochemistry, Voyno-Yasenetsky Krasnoyarsk State Medical University, Krasnoyarsk, 660022 Russia*

[b] *Brain Research Department, Research Center of Neurology, Moscow, 125367 Russia*

**e-mail: elena.hilazheva@mail.ru*

It is known that mitochondrial dysfunction can be a trigger or a concomitant mechanism in the development of Alzheimer's disease. It is also known that the restoration of intracellular mitochondrial activity of neurons and cerebral endotheliocytes is possible due to the transfer of intact mitochondria from other brain cells, in particular, astrocytes, and this transfer of mitochondria is mediated by the CD38 protein and functionally associated with it Cx43, which makes these proteins promising for study both in relation to the study of the mechanisms of development of neurodegeneration, and in relation to the possible modulation of their activity for the correction of neurological deficits. The aim of this work was to study changes in the number of CD38 and Cx43-immunopositive cells in the neurovascular unit and the blood-brain barrier of the brain in experimental Alzheimer's disease. We have shown that the toxic effect of beta-amyloid leads to a significant increase in the number of CD38- and Cx43-positive cells both in the hippocampus of animals in an in vivo experiment and as part of an in vitro blood-brain barrier model. We also showed that the cultivation of isolated astrocytes in the presence of beta-amyloid leads to an increase in the content of Cx43 in cells, and the permeability of these half-channels significantly increases.

Keywords: astrocytes, neurons, endotheliocytes, Alzheimer's disease, mitochondria, CD38, Cx43