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# Targeting intracellular signaling molecules in regeneration-competent cells: novel promising drug targets for treating Alzheimer's disease

Gleb N. Zyuz`kov

Goldberg research Institute of Pharmacology and Regenerative Medicine, Tomsk National Research Medical Center, Russia

**Corresponding author:** Gleb N. Zyuz`kov

Goldberg research Institute of Pharmacology and Regenerative Medicine, Tomsk National Research Medical Center, Russia

zgn@pharmso.ru

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## Abstract

Currently used drugs for the treatment of Alzheimer's Disease, based on the modulation of the functions of nervous tissue mature cells preserved in the pathology conditions, are essentially untenable. Therefore, it is relevant to develop novel approaches that can increase the efficiency of neurogenesis by synchronizing the activities of regeneration-competent cells. As part of the implementation of this direction, the search for pharmacological targets among intracellular signaling molecules is promising. The purpose of the work was to study the participation of intracellular signaling molecules in the regulation of the functions of nervous tissue progenitors and in the production of growth factors by various cells of neuroglia in modeling β-amyloid-induced neurodegeneration. Here, we shown that β-amyloid (Aβ) causes divergent changes in the functioning of neural stem cells (NSC) and neuronal-committed progenitors (NCP). Also demonstrated that different populations of neuroglia respond differently to exposure to Aβ. These phenomena indicate a significant discoordination of the activities of various RCC. Among NF-κB, IKK, PKC, PKB, PI3K, ERK ½, p38, PKA, JAKs, STAT3, JNK, p53, we identified signaling molecules that play an important role in the regulation of progenitor and glial cell functions. Inhibitors of some signaling molecules have been found to cause synchronization of pro-regenerative activity of NSC, NCP, as well as oligodendrocytes and microglial cells under conditions of Aβ-induced neurodegeneration. The results show the promise of developing a novel approach to treating Alzheimer's disease with inhibitors of intracellular signaling molecules. The study was carried out at the expense of a grant from the Russian Science Foundation No. 22-25-00069.

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## Biography

Gleb N. Zyuz`kov is a Scientist Secretary of Institute, Head of the Laboratory of Pathology and Experimental Therapy (Goldberg Research Institute of Pharmacology and Regenerative Medicine), Tomsk National Research Medical Center. Ph.D, M.D., Professor of Russian Academy of Sciences. The author of 65 patents for inventions in the field of pharmacology. International Award of Elsevier`s "SciVal/Scopus Award Russia».