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TrkB.T1 in the mechanisms of genetically-determined depressive-like behavior in mice

Alsalloum M.*, Ilchibaeva T., Tsybko A., Eremin D., Naumenko V.
Institute of Cytology and Genetics, SB RAS, Novosibirsk, Russia
** mr7salloum@gmail.com*

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Depression is one of the most severe mental disorders that significantly reduces the quality of life of patients. However, since available antidepressants are still not effective for all patients, an extremely urgent problem of modern neuroscience is the study of the mechanisms of depression.

Previously, we found that the level of TrkB.T1 receptor is decreased in ASC mice (mice with genetically-determined depressive-like behavior) in comparison with C57BL/6 "normal" mice. In our research, we induced TrkB.T1 overexpression in ASC mice to study its role in the mechanisms of depressive-like behavior. By using adeno associated viruses as carriers of TrkB.T1coding gene, we induced TrkB.T1 overexpression and studied the effects of overexpression on the expression of key genes of serotonin and BDNF systems. Using behavioral tests, we assessed the effect of TrkB.T1 overexpression on behavioral level. We found that TrkB.T1 regulates aggressive-like behavior, anxiety, and depressive-like behavior in ASC mice. Considering our findings, we believe, that TrkB.T1 might serve as a drug target for new generation of anti-depressants.

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