Research (What is it about?)	**Viral neuroprotective drug**
UNN authors	*Mitroshina E., Mishchenko T., Usenko A., Epifanova E., Yarkov R., Gavrish M., Babaev A., Vedunova M.*
We find (The result)	We construct the adeno-associated ***virus*** carrying the gene of brain-derived neurotrophic factor which evince a neuroprotective action in hypoxia case
Abstract	Brain-derived neurotrophic factor (***BDNF***) is one of the key signaling molecules that supports the viability of neural cells in various brain pathologies, and can be considered an effective therapeutic agent. However, several methodological difficulties, such as overcoming the blood–brain barrier and the short half-life period, challenge the potential use of BDNF in clinical practice. ***Gene therapy*** could overcome these limitations. Investigating the influence of viral vectors on the neural network level is of particular interest because viral overexpression affects different aspects of cell metabolism and interactions between neurons. Neurotrophin therapeutic delivery systems, such as viral vector constructs for long-term BDNF overexpression in nervous system cells, is a promising method for ischaemia therapy, as it may avoid the need for prolonged use of drugs and have targeted effects on a strictly defined cell type. To increase BDNF expression in brain neurons, an adeno-associated virus vector carrying the BDNF gene ***AAV-Syn-BDNF-EGFP*** was constructed. The developed AAV-Syn-BDNF-EGFP vector was tested on primary hippocampal cell cultures. Experiments *in vitro* showed that application of the developed virus vector ***increased BDNF production*** in primary hippocampal cells by ***2.7 times***. As a result of the high sensitivity of the neuronal cultures to almost all chemical treatments, including viral drugs, the developed virus was examined for possible cytotoxic effects. The experiments on the cytotoxicity of AAV-Syn-BDNF-EGFP did not reveal any changes in the viability of neural networks in primary hippocampal cultures. It opens up opportunities of using this neurotrophin for the long-term maintenance of the viability of neurons that have lost their connections with the neural network due to destruction in the post-ischaemic period.

Representative articles 2017-2018, quartiles	1. *Mitroshina E.V., Mishchenko T.A., Usenko A.V., Epifanova E.A., Yarkov R.S., Gavrish M.S., Babaev A.A., Vedunova M.V.* AAV-Syn-BDNF-EGFP virus construct exerts neuroprotective action on the hippocampal neural network during hypoxia in vitro. Int. J. Mol. Sci. **19**(8): 2295 (2018). 2. *Mitroshina E.V., Epifanova E.A., Mishchenko T.A., Yarkov R.S., Babaev A.A., Vedunova M.V.* Application of the AAV-Syn-BDNF-EGFP virus vector as a neuroprotective agent in modeling hypoxia in vitro. Sovremennye tehnologii v medicine. **10**(2), 47-56 (2018).	Q2, Q2 –
	Q-index (Qi) for the result	**1.5**
	medial yellow	

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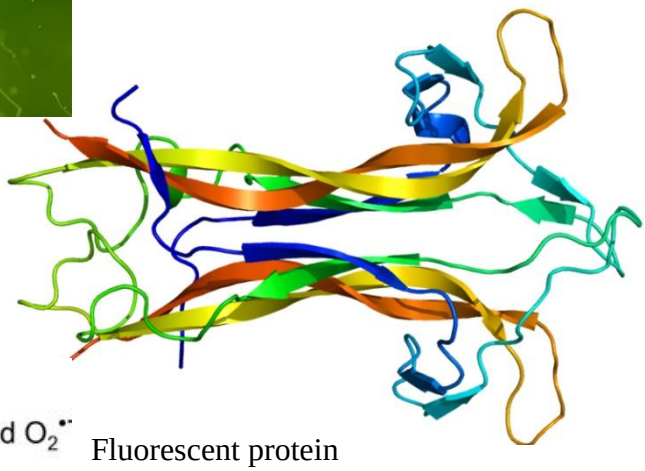
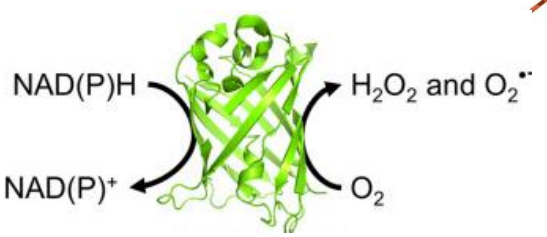
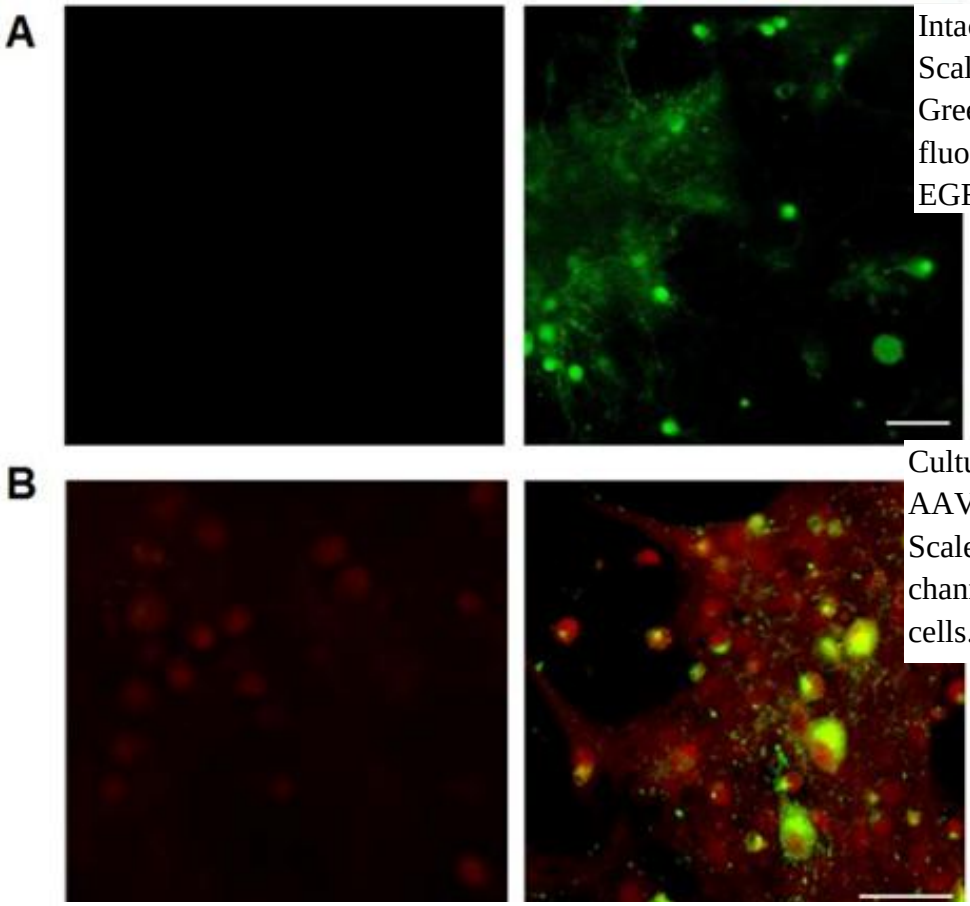
Adeno-associated virus (***AAV***)

The structure of the ***BDNF*** protein





Fluorescent protein ***EGEP***



Intact culture. Scale bar 50 μm. Green channel – fluorescence of EGFP.

Culture transduced by AAV-Syn-BDNF-EGFP. Scale bar 20 μm. Red channel – BDNF-positive cells.

Transduction by AAV-Syn-BDNF-EGFP virus vector increases BDNF expression in primary hippocampal cultures on day 7 after the virus infection.