Short communication

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Neurology & Brain Injury 2019 - A low molecular weight GAPDH binder reduces secondary damage after traumatic brain injury on rats

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Abstract

Extensive death of neurons can be caused by neurodegenerative pathologies or massive cell damage due to ischemia or traumatic brain injury (TBI). Such pathological conditions lead to the emergence of various proteins in the intercellular space. These proteins can regulate the development of secondary damage due to a clearly expressed toxic effect on the surrounding cells. In this work, we demonstrated the possibility of inhibiting the cytotoxic effect of such aggregates by treatment with specific drug that bind the GAPDH protein, a well-known participant of aggregation, in the intercellular space. Selected chemicalhydrocortisone derivative RX624 prevented the interaction of exogenous GAPDH with cell membrane and reduced the death of the acceptor cells. We also demonstrated efficiency of RX624 treatment on rat model of TBI. This chemical blocked formation of GAPDH aggregates, inhibited cytotoxic effects of cerebrospinal fluid and rescue motor function of injured rats. Importantly, that RX624 treatment of rats had the similar effect with specific anti-GAPDH antibodies intracranial injection.

Biography :

Vladimir F Lazarev has completed his PhD from the Institute of Cytology (RAS) and Post-doctoral studies were continued at the same place. Now, he is the Senior Researcher in the Laboratory of Cell Protection Mechanisms. He has published over 20 papers in reputed journals. He is a Supervisor of several students and has been serving as an Editorial Board Member of repute.

Note: This work is partly presented at Joint Event on Neurology & Brain Injury (March 14-15, 2019 | Paris, France)

