MYOSIN V A LINK BETWEEN ISCHEMIC INJURY AND NERVOUS SYSTEM REGENERATION.

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Many forms of acute or chronic neurodegenerative diseases have been related to neuronal death mediated by glutamate release. A variety of neuronal types undergo excitotoxic death when exposed to high concentrations of this neurotransmitter. During excitotoxicity associated with ischemia, glutamate produces a massive neuronal Ca^{2+} influx leading to the activation of calpain. Myosin-Va (MVa) is a molecular motor protein involved in vesicle transport that is proteolysed by calpain. Previous reports in our lab showed that MVa is connected with nerve regeneration and that the protein is locally synthesized after injury, having a fivefold increase in expression. In addition, we introduced the possibility that MVa is involved in the process of cell death of CGN elicited by excitotoxic conditions and the protein is a target for calpain-I during an excitotoxic injury addressing the participation of molecular motors in neurotoxicity. Since thyroid hormones (TH) are essential for the development of the brain and are part of the response to metabolic stress, we addressed TH effect in MVa synthesis. Our data show that MVa synthesis is severely affected by TH levels. These data together let us to propose that MVa is a target protein to excitotoxicity-induced damage and neuronal cells try to upregulate its synthesis after injury.

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