Addressing the contribution of JNK3 to axonal pathology in Huntingtons disease

https://neurodegenerationresearch.eu/survey/addressing-the-contribution-of-jnk3-to-axonal-pathology-in-huntingtons-disease/

Principal Investigators

MORFINI, GERARDO ANDRES

Institution

UNIVERSITY OF ILLINOIS AT CHICAGO

Contact information of lead PI
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Research Abstract

Huntington’s disease (HD) is an autosomal-dominant, progressive neurodegenerative disorder featuring devastating clinical symptoms that include motor deficits, cognitive decline, and behavioral impairments. To date, most research efforts towards the development of therapeutic strategies in HD have been largely focused on inhibition of pathways leading to the loss of neuronal somata, failing to address or even consider the progressive loss of neuritic connectivity that takes place much earlier in the disease process. Building on a solid body of published

findings and strong preliminary data, this application aims to illuminate specific therapeutic targets and mechanisms underlying axonal pathology in HD. Experiments proposed under Aim 1 will directly evaluate the contribution of JNK3, a potentially druggable protein kinase, to the axonal pathology induced by mutant huntingtin (mhtt) expression in vivo. Extending these studies, experiments under Aim 2 will identify JNK3-dependent alterations in the phosphorylation of axonal proteins induced by mhtt. Together, these studies will help illuminate a molecular basis linking JNK3 activation to mhtt-induced axonal degeneration.

Further information available at:

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