The role of NMNAT2 in axon degeneration: relevance to Parkinson’s disease.

https://www.neurodegenerationresearch.eu/survey/the-role-of-nmnat2-in-axon-degeneration-relevance-to-parkinson%c2%92s-disease/

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Title of project/programme

The role of NMNAT2 in axon degeneration: relevance to Parkinson’s disease.

Source of funding information

FCT

Total sum awarded (Euro)

€ 107,640

Start date of award

01/01/15

Total duration of award in years

6.0

The project/programme is most relevant to:

Parkinson's disease & PD-related disorders

Keywords
Research Abstract

Axon pathology is a prominent feature of many neurodegenerative disorders, including Parkinson’s disease (PD). Work from our group showed, in several PD cellular models, a disrupted microtubule-dependent intracellular trafficking due to reduced acetylated tubulin levels. Nicotinamide mononucleotide adenyltransferase 2 (NMNAT2), a key cytosolic enzyme of

the NAD+ salvage pathway, has been described as an endogenous axon survival factor. NMNAT2 is essential for axon sprouting, survival and maintenance during embryonic development and to preserve axonal health in the PNS. Recently, it was also demonstrated that over-expression of NMNAT2 protected against neurodegeneration in the CNS. Interestingly, our preliminary data indicates that cytosolic NMNAT2 levels are increased in PD. NMNAT2-induced NAD+ replenishment could activate sirtuin-2, a NAD-dependent deacetylase, decreasing acetylated tubulin levels, which promotes axonal trafficking deficits in mature neurons. Our major goal is to investigate whether impaired NMNAT2 function, altered levels and/or cellular localization can contribute to the neurodegeneration found in PD.

Types:
Fellowships

Member States:
Portugal

Diseases:
Parkinson's disease & PD-related disorders

Years:
2016

Database Categories:
N/A

Database Tags:
N/A