

Molecular neuroscience of Parkinson's Disease: Retromer (VPS35) dysfunction

<https://neurodegenerationresearch.eu/survey/molecular-neuroscience-of-parkinsons-disease-retromer-vps35-dysfunction/>

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Canada

Title of project or programme

Molecular neuroscience of Parkinson's Disease: Retromer (VPS35) dysfunction

Source of funding information

CIHR

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€ 500,724

Start date of award

01/04/2012

Total duration of award in years

5.0

The project/programme is most relevant to:

Parkinson's disease & PD-related disorders

Keywords

Research Abstract

Parkinson's disease (PD) is a debilitating neurodegenerative disease that affects 1 percent of the population over 65 years. PD is already a major health care burden, and will become even more so in our increasingly aging society. Currently there is no cure, and treatments only address PD symptoms. Medications do not slow disease progression and are not without serious side-effects. To develop more effective treatments for PD requires an understanding of

its molecular cause(s) (etiology) and how they influence susceptibility and progression (pathogenesis). The latest susceptibility gene (VPS35) for PD was recently discovered. VPS35 is part of the retromer, the scaffold of an ancient and elaborate cellular recycling system, selective for very specific protein cargos. Our study is to determine which cargo interactions and cellular processes are distressed by VPS35 mutations, and how these disturbances affect brain function and lead to PD. We believe that by targeting the causes of PD, rather than its consequences, and by creating and characterizing genetic models, more effective therapeutics can be developed. Our objective is to alleviate motor and non-motor symptoms, by providing neuroprotective benefit, and to lessen the burden of PD to individuals and society.

Lay Summary

Further information available at:

Types:

Investments > €500k

Member States:

Canada

Diseases:

Parkinson's disease & PD-related disorders

Years:

2016

Database Categories:

N/A

Database Tags:

N/A