

Reversal of neuronal and cerebrovascular pathology in Alzheimer's disease

<https://neurodegenerationresearch.eu/survey/reversal-of-neuronal-and-cerebrovascular-pathology-in-alzheimers-disease/>

Principal Investigators

Hamel, Edith

Institution

McGill University

Contact information of lead PI Country

Canada

Title of project or programme

Reversal of neuronal and cerebrovascular pathology in Alzheimer's disease

Source of funding information

CIHR

Total sum awarded (Euro)

€ 493,802

Start date of award

01/04/2013

Total duration of award in years

5

Keywords

Research Abstract

Despite very active research on Alzheimer's disease (AD) over the past years, there is still very little to offer to patients as a choice of new therapy. It has been argued that this disappointing outcome is due to our poor understanding of the initial culprit of the disease. Several studies in patients indicate that a chronic reduction in blood supply to the brain occurs before the cognitive decline, and actually contributes to the neuronal failure. Dysfunctional blood vessels would impact on basic needs of the brain for fuel, and lead to neuronal injury, accumulation or faulty clearance of deleterious molecules and, ultimately, to neuronal degeneration. Studying the relationships between cerebrovascular pathology and memory deficit in AD using a classic

mouse model of the disease, we found that it is possible to totally rescue both memory and brain perfusion. We also developed a new mouse model that recapitulates the full spectrum of neuronal, cognitive and cerebrovascular landmarks of the human disease. We will use these two models to better identify the relationships between cerebrovascular dysfunction and memory loss, and understand how drugs currently used for the treatment of cardiovascular diseases can rescue both deficits. We hypothesize that preserving a healthy cerebral circulation together with intact neuronal function will offer more effective treatments for AD. Further, we suggest that it is possible to rescue memory independently from reducing the levels of amyloid-beta, a highly toxic protein that accumulates in the brain of AD patients. Our goals are to identify the altered molecular pathways in brain and blood vessels that can be targeted therapeutically to concurrently rescue memory and brain perfusion.

Further information available at:

Types:

Investments < €500k

Member States:

Canada

Diseases:

N/A

Years:

2016

Database Categories:

N/A

Database Tags:

N/A