Endoplasmic reticulum-mitochondria interactions and Alzheimer’s disease

https://neurodegenerationresearch.eu/survey/endoplasmic-reticulum-mitochondria-interactions-and-alzheimers-disease/

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Country

United Kingdom

Title of project or programme

Endoplasmic reticulum-mitochondria interactions and Alzheimer's disease

Source of funding information

Alzheimer's Research UK

Total sum awarded (Euro)

€ 625,932

Start date of award

01/10/2014

Total duration of award in years

3.0

The project/programme is most relevant to:

Alzheimer's disease & other dementias

Keywords
Research Abstract

A wide number of cellular functions are perturbed in Alzheimer’s disease but it is not clear how so many apparently disparate functions are all damaged together. Recently, several studies have highlighted how damage to one particular cellular feature termed the endoplasmic reticulum (ER)-mitochondria axis might precipitate many Alzheimer’s disease type changes. ER and mitochondria are two structures within brain cells that perform different functions; ER is

involved in making proteins and lipids, and mitochondria generate the energy for the cell. However, their proper functioning requires that they communicate with each other and this involves tethers that physically connect the two structures. In Alzheimer’s disease and related dementias, we and others have shown that ER-mitochondria communication is disrupted. Moreover, we have identified some of the protein tethers that connect ER with mitochondria and have shown that these are damaged in some forms of dementia. This project is to continue these studies. We aim to determine how the tethers and ER-mitochondria interactions are damaged in Alzheimer’s disease and to identify lead compounds that might correct this damage. We believe that our studies may reveal a new therapeutic target for treating Alzheimer’s disease.

Lay Summary
Further information available at:

Types:
Investments > €500k

Member States:
United Kingdom

Diseases:
Alzheimer's disease & other dementias

Years:
2016

Database Categories:
N/A

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