Interaction of systemic and central apolipoproteins in the pathogenesis and treatment of cerebral amyloid angiopathy

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Name of Fellow

Cheryl Hawkes

Institution
Funder

Alzheimer's Research UK

Contact information of fellow
Country

United Kingdom

Title of project/programme

Interaction of systemic and central apolipoproteins in the pathogenesis and treatment of cerebral amyloid angiopathy

Source of funding information

Alzheimer's Research UK

Total sum awarded (Euro)

€ 264,267

Start date of award

01/09/12

Total duration of award in years

3.3

The project/programme is most relevant to:

Alzheimer's disease & other dementias

Keywords

ApoE and Lipids | Lipid-Mediated Signaling

Research Abstract
Alzheimer’s disease (AD) affects more than 800,000 people in Britain and 35 million people worldwide, with numbers set to quadruple over the next 40 years. Accumulation of amyloid-? (A?) in the walls of cerebral blood vessels as cerebral amyloid angiopathy (CAA) damages their integrity and contributes to the observed dementia. Work from our lab suggests that CAA may develop in part due to impaired removal of A? from the brain along basement membranes in the walls of capillaries and arteries. For unknown reasons, people with high levels of plasma cholesterol and those who posses the apolipoprotein E4 (apoE) protein are at higher risk of developing AD and CAA. Apolipoproteins are necessary for cholesterol movement in the brain and periphery. In the brain, cholesterol is carried by apoE and in the blood, by apoA-I. Recently, it has been suggested that apoA-I might play a role in the development of CAA, through as-of-yet unidentified mechanisms. In the current proposal, we will test the hypothesis that i) apoA-I influences A? accumulation by altering the efficiency of the perivascular environment to remove A? from the brain and ii) that altering levels of peripheral apoA-I will have beneficial effects on the development of CAA.

Types:
Fellowships

Member States:
United Kingdom

Diseases:
Alzheimer's disease & other dementias

Years:
2016

Database Categories:
N/A

Database Tags:
N/A