

The role of intracellular trafficking in Alzheimer's disease

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Funder

FCT

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Country

Portugal

Title of project/programme

The role of intracellular trafficking in Alzheimer's disease

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FCT

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The project/programme is most relevant to:

Alzheimer's disease & other dementias

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Research Abstract

In late-onset Alzheimer's disease (LoAD), the causes of the associated cerebral beta-amyloid (A β) accumulation with aging are unknown. A β accumulation results from overproduction

and/or reduced degradation. Growing evidence implicates intracellular trafficking as a disease mechanism. Two trafficking regulators, bin1 and CD2AP, were recently identified as putative AD-risk factors but their role in Abeta metabolism is unknown. Protein trafficking regulators are also found altered in the aging brain, potentially acting as aging-risk factors for loAD. My project aims to determine how intracellular trafficking dysfunction in neurons is mechanistically involved in Abeta accumulation. I will investigate how bin1 and CD2AP altered function and aging-risk factors deregulate intracellular trafficking thus shifting Abeta metabolism towards accumulation. I will employ state of the art cell biology methodology using primary cultures of neurons to demonstrate that deregulation of trafficking contributes to Abeta accumulation. Understanding the cellular basis of loAD is essential to identify new therapeutic targets.

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