Design and Optimisation of New Chemical Entities that Prevent the Neurotoxic Oligomerization and Misfolding of both beta-amyloid and tau Proteins: A Disease Modifying Therapeutics Approach for Alzheimer's Dementia

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Contact information of lead PI Country

United Kingdom

Title of project or programme

Design and Optimisation of New Chemical Entities that Prevent the Neurotoxic Oligomerization and Misfolding of both beta-amyloid and tau Proteins: A Disease Modifying Therapeutics Approach for Alzheimer's Dementia

Source of funding information

The Wellcome Trust

Total sum awarded (Euro)

€ 3,601,588

Start date of award

19/07/2013

Total duration of award in years

The project/programme is most relevant to:

Alzheimer's disease & other dementias

Keywords

Research Abstract

Numerous studies support a causative role for b-amyloid (A) and tau in the aetiopathogenesis of Alzheimer's disease (AD).1,2 These proteins tend to abnormally clump ,3,4 and such protein misfolding processes give rise to neurotoxic aggregates of b-amyloid (plaques) and tau (tangles) the pathological hallmarks of AD.5 In vitro studies have verified that Ab is significantly neurotoxic when in small aggregates (dimers, trimers and other oligomers).6,7,8 Since diseasemodifying (in preference to merely symptomatic) drugs represent the most desirable therapeutic approach to AD,9 protein misfolding of A and tau represents a leading target in the rational design of a disease-modifying drug.1

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

Database Tags:

N/A