Nanoparticles for therapy and diagnosis of Alzheimer’s Disease (NAD)

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Title of project or programme

Nanoparticles for therapy and diagnosis of Alzheimer’s Disease (NAD)

Principal Investigators of project/programme grant

Title	Forname	Surname	Institution	Country
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Source of funding information

European Commission

Total sum awarded (Euro)

10921350

Start date of award

01-09-2008

Total duration of award in months

60

The project/programme is most relevant to

- Alzheimer’s disease and other dementias

Keywords

nanoparticles,lipids,MRI,PET,NANOPARTICLES,blood-brain-barrier,alzheimer disease,abeta,pet,antiamyloidogenic,ALZHEIMER DISEASE,ABETA,BLOOD-BRAIN-BARRIER,LIPIDS,ANTIAMYLOIDOGENIC

Research abstract in English

The search for effective therapies and early detection strategies for Alzheimer’s Disease (AD), the major cause of dementia in Europe, is imperative. It is known that beta-amyloid (Abeta) peptide plays a central role in neurodegeneration. In AD brain, Abeta is released in a soluble form that progressively becomes insoluble forming aggregates; extracellular plaques mainly composed of Abeta are a hallmark of post-mortem brains. These premises strongly suggest brain Abeta as a possible target for therapy and diagnosis of AD. In addition, it is known that brain and blood Abeta pools are in equilibrium via the blood-brain-barrier (BBB). Accordingly, it has been reported that removal of blood Abeta may withdraw the excess of brain Abeta by a sink effect. Thus, blood Abeta is another potential target. The aim of this project is to utilize nanoparticles (NPs) specifically engineered for targeting brain Abeta, for the combined diagnosis and therapy (theranostics) of AD. NPs (liposomes, solid lipid NPs, polymeric-NPs) will be multiple-functionalized with: i) a large arsenal of molecules (specific lipids, antiamyloidogenic drugs, polyphenols, heteroaromatic compounds, unnatural peptides and peptidomimetics, antibodies) interacting with A? in all aggregation forms, ii) PET or MRI contrast agents detecting such interaction, iii) molecules stimulating BBB crossing via the transcytotic route. Several artificial and cellular models will be used to fine-tune such features and to improve NPs biocompatibility, non-immunogenicity, non-toxicity and physical stability. Eventually, absorption, distribution, metabolism and excretion will be studied using animal models of AD. Different routes (i.v., oral, nasal) and protocols (two-step, NPs cocktails, aerosols) of administration will be utilized to boost NPs brain delivery. The prediction is that NPs will detect, disaggregate and remove Abeta brain deposits.

Lay summary