Benfotiamine in Alzheimers Disease: A pilot study

https://neurodegenerationresearch.eu/survey/benfotiamine-in-alzheimers-disease-a-pilot-study/

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Country

USA

Title of project or programme

Benfotiamine in Alzheimers Disease: A pilot study

Source of funding information

NIH (NIA)

Total sum awarded (Euro)

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01/09/2014

Total duration of award in years

3

The project/programme is most relevant to:

Alzheimer's disease & other dementias

Keywords

Acquired Cognitive Impairment... Aging... Alzheimer's Disease... Alzheimer's Disease including Alzheimer's Disease Related Dementias (AD/ADRD)... Behavioral and Social Science... Brain Disorders... Clinical Research... Clinical Research - Extramural... Clinical Trials and Supportive Activities... Dementia... Neurodegenerative... Neurosciences... Nutrition... Prevention... Translational Research

Research Abstract

DESCRIPTION (provided by applicant): Despite major advances in early diagnosis, neuroimaging, and biomarker research, no disease-modifying therapies are available for Alzheimer’s Disease (AD). Reduced brain glucose metabolism always accompanies AD and is an outstanding biomarker of disease progression. While the sentiment in AD research is that the changes in glucose metabolism are secondary to diminished neuronal function and synaptic loss, reduced glucose metabolism can lead to diminished synaptic function, reduced brain function including cognition, and the development of AD like pathology. Thus, increasing brain glucose utilization is an attractive therapeutic target. Although the cause of the decline in glucose utilization is unknown, plausible mechanisms link it to a decline in thiamine (vitamin B1) dependent processes in the brain. Thiamine dependent enzymes are critical to normal brain glucose utilization, and all are diminished in AD in parallel with a decline in clinical dementia rating scores. In humans and/or animals, thiamine deficiency diminishes brain metabolism and cognition, while promoting AD like pathology including plaques and tangles. On the other hand, elevating brain thiamine increases brain metabolism and cognition in humans and animals. In animal models of AD, thiamine diminishes AD-like pathology. These findings suggest that increasing brain thiamine should be beneficial in AD. The most effective way to increase blood and brain thiamine is with the thiamine derivative benfotiamine. Multicenter trials in humans show that benfotiamine prevents diabetic retinopathy and peripheral neuropathy and that it is safe. Thus, we propose a proof of concept pilot study in patients with Amnestic Mild Cognitive Impairment (AMCI) and mild AD dementia to test the hypothesis that increasing brain thiamine availability by administration of benfotiamine will delay the reduction in glucose utilization as shown by FDG-PET and slow further decline in cognition. The proposed randomized, double blind, placebo controlled clinical pilot trial will treat 76 patients with benfotiamine for one yea. The proposed pilot clinical trial has the potential to identify a new, safe intervention aimed at modifying biological and clinical processes of AD progression that could inform a larger clinical trial.

Lay Summary

PUBLIC HEALTH RELEVANCE: Despite major advances in early diagnosis, neuroimaging, and biomarker research, no disease- modifying therapies are available for Alzheimer’s Disease (AD). Reduced glucose metabolism is an invariant feature of AD and an outstanding biomarker of disease progression. This pilot study will test whether a vitamin B1 derivative that increases glucose utilization and diminishes AD symptoms in mouse models will be beneficial in patients with AD.

Further information available at:

Types:
Investments > €500k

Member States:
United States of America

Diseases:
Alzheimer's disease & other dementias

Years:
2016

Database Categories:

N/A

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