

Generation of effective carbohydrate-based inhibitors of a glycosidase implicated in Alzheimer disease and their molecular mechanism of action.

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Principal Investigators

Vocadlo, David

Institution

Simon Fraser University (Burnaby, B.C.)

Contact information of lead PI

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Generation of effective carbohydrate-based inhibitors of a glycosidase implicated in Alzheimer disease and their molecular mechanism of action.

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Research Abstract

Alzheimer disease (AD) manifests both by the development of extracellular amyloid deposits (plaques) and intracellular aggregates known as neurofibrillary tangles (NFTs). The plaques result from accumulation of an insoluble peptide known as amyloid beta (Abeta). The NFTs

arise from a form of the protein tau that assembles into aggregates. A feature of AD is that patients show impaired brain glucose metabolism and this correlates with conversion of patients from mild cognitive impairment to AD. Recently, a sugar modification of proteins has been implicated in AD. It is believed that because this sugar modification is derived from glucose its levels decrease as glucose impairment occurs in early stage AD. Data suggests that increasing levels of this sugar modification could be beneficial to slow the progression of AD. We aim to generate useful inhibitors that can efficiently increase levels of this sugar modification and to determine how this sugar modification acts to prevent the tau-driven death of neurons.

Further information available at:

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Investments < €500k

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Canada

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