FULL TEXT LINKS



Biochem Biophys Res Commun. 2020 Jun 30;527(3):676-681. doi: 10.1016/j.bbrc.2020.05.015. Epub 2020 May 4.

Cerebral deficiency of vitamin B5 (d-pantothenic acid; pantothenate) as a potentially-reversible cause of neurodegeneration and dementia in sporadic Alzheimer's disease

Jingshu Xu¹, Stefano Patassini¹, Paul Begley², Stephanie Church², Henry J Waldvogel³, Richard L M Faull³, Richard D Unwin², Garth J S Cooper⁴

Affiliations PMID: 32416962 DOI: 10.1016/j.bbrc.2020.05.015

Abstract

Alzheimer's disease (AD) is the most common cause of age-related neurodegeneration and dementia, and there are no available treatments with proven disease-modifying actions. It is therefore appropriate to study hitherto-unknown aspects of brain structure/function in AD to seek alternative disease-related mechanisms that might be targeted by new therapeutic interventions with diseasemodifying actions. During hypothesis-generating metabolomic studies of brain, we identified apparent differences in levels of vitamin B5 between AD cases and controls. We therefore developed a method based on gas chromatography-mass spectrometry by which we quantitated vitamin B5 concentrations in seven brain regions from nine AD cases and nine controls. We found that widespread, severe cerebral deficiency of vitamin B5 occurs in AD. This deficiency was worse in those regions known to undergo severe damage, including the hippocampus, entorhinal cortex, and middle temporal gyrus. Vitamin B5 is the obligate precursor of CoA/acetyl-CoA (acetyl-coenzyme A), which plays myriad key roles in the metabolism of all organs, including the brain. In brain, acetyl-CoA is the obligate precursor of the neurotransmitter acetylcholine, and the complex fatty-acyl groups that mediate the essential insulator role of myelin, both processes being defective in AD; moreover, the large cerebral vitamin B5 concentrations co-localize almost entirely to white matter. Vitamin B5 is well tolerated when administered orally to humans and other mammals. We conclude that cerebral vitamin B5 deficiency may well cause neurodegeneration and dementia in AD, which might be preventable or even reversible in its early stages, by treatment with suitable oral doses of vitamin B5.

Keywords: Acetyl-CoA; Age-related neurodegeneration; Alzheimer's disease; Human brain; Metabolic brain disease; Pantothenate); Vitamin B5 (pantothenic acid.

Copyright © 2020 Elsevier Inc. All rights reserved.

Related information

MedGen PubChem Compound (MeSH Keyword)

LinkOut - more resources

Full Text Sources
Elsevier Science

Medical