

# Validated, unified theory of Alzheimer's Disease (AD) illuminates two essential research questions

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## BACKGROUND:

1992

2001\*

2015

2017 +

**EVIDENCE OF THIAMINE DEFICIENCY (TD) IN AD HAS BEEN MOUNTING OVER THE YEARS.**

Thiamine (Vitamin B1) is an organic micronutrient required in the human diet.(1)

**1992:** A University of Manitoba nutrition study suggested the possibility of a secondary TD in AD correlating with scores of cognitive function. This deficiency was detected in blood and occurred in spite of normally adequate dietary intakes of thiamine. (1, p .iii)

**\*2001:** Thesis examination committee at the University of Manitoba approved "The Potential Role of Thiamine in the Pathogenesis and Treatment of AD" a M.Sc. Dissertation that **PROPOSED A NEW THEORY THAT AD IS A TD DISEASE** (1-p.85).

In AD and TD models there are similar patterns of:  
a. clinical signs and symptoms (1-p.37)  
b. biochemical abnormalities, (1-p.13, 1-p.29) and  
c. brain pathology abnormalities (1-p.41)

In addition, evidence was documented that thiamine supplementation can potentially correct key targets of AD research: the AD cholinergic deficit (1-p.64-70) and abnormalities in B-Amyloid (1-p.77-81)

**2015:** The measurement of blood thiamine metabolites by HPLC is an ideal diagnostic test for AD (2). AD subjects have significantly reduced blood thiamine diphosphate levels (TDP) as compared to control subjects in both exploration and validation phases of this landmark study (2). TDP comprises 80% of the tissue stores of thiamine (1-p.24). **THIS STUDY VALIDATES THE 2001 THEORY** ( 1-P.85).

**2017:** "Scientific evidence supporting a unified theory of AD" published in *Alzheimer's and Dementia Journal* (3)

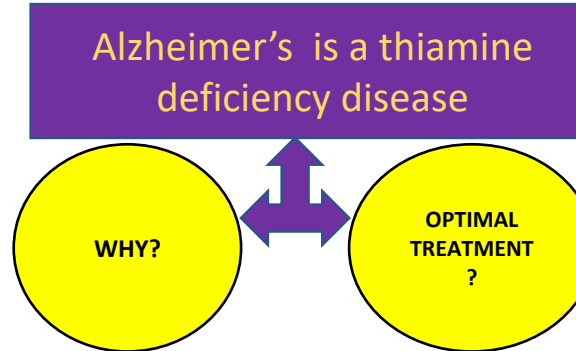
## UPDATE?

**METHOD:** Scientific literature review on topic of thiamine and/or AD PUBMED, e library. wiley.com., mspace.lib.umanitoba.ca

## RESULT:

1. Thiamine supplementation can potentially correct the AD cholinergic deficit (1-p.64,4), abnormalities in B-Amyloid (1-p.77,3), tau (5), neuro inflammation (4,5,6,7) and memory/cognition (1-p59,3), key targets of AD research.
2. Limited oral thiamine supplementation trials in AD have had mixed results These have been reviewed elsewhere (1-p.49-54,6).

## CONCLUSION:



1. What causes TD in AD? Altered thiamine requirements, excretion, absorption, transport, metabolism or diet and other factors?
2. What is the optimal treatment of TD in AD? Control for medications\*, medical conditions\* and dietary components affecting thiamine intake and utilization (\*1-p.52, p.53, 9). Consider advantages of intramuscular injections vs. oral treatments (8), need for other nutrients (1-p.52), proven thiamine treatments in TD diseases.

**ACKNOWLEDGEMENTS:** Deep gratitude goes to my graduate school advisory committee at the University of Manitoba and the countless scientists in the fields of nutrition and AD research who conducted the scientific studies leading to this poster presentation.. This poster represents the culmination of thirty years of independent study. May this work inspire an exciting, newly illuminated frontier in AD and nutrition research.

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