From: [redacted] <[redacted]>
Sent: Monday, April 10, 2017 6:46 PM
To: Jeffrey Epstein
Subject: wow

This article has disappeared from the literature..having a real=y hard time locating

J Alzheimers Dis. <https://www-ncbi-nlm-nih-gov.offcampus.lib.was=ington.edu/pubmed/?term=Aluminum+and+Alzheimer%27s+disease%3A+after+a+ce=tury+of+controversy%2C+is+there+a+plausible+link%3F#> 2011;23(4):567-98. doi: 10.3233/JAD=2010-101494.

Al=minum and Alzheimer's disease:=C2[redacted]after a century of controversy</=pan>, is there a plausible link?

Tomljenovic L <https://www-ncbi-nlm-nih-gov.off=ampus.lib.washington.edu/pubmed/?term=Tomljenovic%20L%5BAuthor%5D&ca=thor=true&cauthor_uid=21157018> 1.

<https://www-ncbi-nlm-nih-gov.offcampus.lib.washi=gton.edu/pubmed/?term=Aluminum+and+Alzheimer%27s+disease%3A+after+a+cent=ry+of+controversy%2C+is+there+a+plausible+link%3F#>

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Neural Dynamics Research Group, Department o= Ophthalmology and Visual Sciences, University of British Columbia, Vancou=er, BC, Canada.[redacted]=/a> <mailto:[redacted]>

Abstract

The=brain is a highly compartmentalized organ exceptionally susceptible to acc=mulation of metabolic errors. Alzheimer's disease=/span> (AD) is the most prevalent neurodegenerative disease of the elderly and is characterized by regional specificity of ne=ral aberrations associated with higher cognitive functions. Aluminum=/span> (Al) is the most abundant neurotoxic metal on earth, widely bi=available to humans and repeatedly shown to accumulate in AD-susceptible n=uronal foci. In spite of this, the role of Al in AD has been heavily dispu=ed based on the following claims: 1) bioavailable Al cannot enter the brai= in sufficient amounts to cause damage, 2) excess Al is efficiently excret=d from the body, and 3) Al accumulation in neurons is a consequence rather=than a cause of neuronal loss. Research, however, reveals that: 1) very sm=ll amounts of Al are needed to produce neurotoxicity and this criterion is=satisfied through dietary Al intake, 2) Al sequesters different transport =echanisms to actively traverse brain barriers, 3) incremental acquisition =f small amounts of Al over a lifetime favors its selective accumulation in=brain tissues, and 4) since 1911, experimental evidence has repeatedly dem=nstrated that chronic Al intoxication reproduces neuropathological hallmar=s of AD. Misconceptions about Al bioavailability may have misled scientist= regarding the significance of Al in the pathogenesis of AD. The hypothesi= that Al significantly contributes to AD is built upon very solid experime=tal evidence and should not be dismissed. Immediate steps should be taken =o lessen human exposure to Al, which may be the single most aggravating an= avoidable factor related to AD.

P=ID:

21157018

DOI:

10.3233/JAD-2010-101494 <https://doi-or=.offcampus.lib.washington.edu/10.3233/JAD-2010-101494>