

# Alzheimer's Disease & Mercury - Definitive Research

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SPECIAL NOTE: The University of Calgary has placed  
a copy of the video on their site at: <http://movies.commonscalgary.ca/mercury/>

Research conducted at the University of Calgary Faculty of Medicine has demonstrated that trace amounts of mercury can cause the type of damage to nerves that is characteristic of the damage found in Alzheimer's Disease. The level of mercury exposure is consistent with those levels found in humans with mercury/silver amalgam dental fillings. The exposure to mercury caused the formation of "neurofibrillar tangles," which are one of the two diagnostic markers for Alzheimer's Disease. The scientists found that other metals, including aluminum, did not cause the damage. Previous research has shown that mercury can cause the formation of the other Alzheimer's Disease diagnostic marker, "amyloid plaques." The research, published in a peer-reviewed medical journal, is accompanied by a video visual presentation of the effect. Utilizing digital time-lapse photography, this video shows rapid damage to the nerve cells after introduction of minute amounts of mercury. Funding for this video was provided by the International Academy of Oral Medicine and Toxicology (IAOMT).

"Retrograde Degeneration of Neurite Membrane Structural Integrity of Nerve Growth Cones Following In Vitro Exposure To mercury."

Leong, CW; Syed, NI; Lorscheider, FL. NeuroReport, 12(4):733-737, 2001.

**BIOPROBE COMMENT:** This study should remove all doubt regarding the role that dental mercury from amalgam fillings plays in the development of Alzheimer's Disease (AD). Although the American Dental Association would like to have you believe otherwise, science has clearly demonstrated that there is a positive correlation between brain mercury levels and the number and surfaces of "mercury/silver" amalgam dental fillings. The mercury levels that caused the devastating damage to nerve cells in the above referenced study were 100 to 1000 times below those found in the brains of people with "mercury/silver" amalgam dental fillings.

In 1997, researchers at the University of Calgary Medical School and the College of Pharmacy at the University of Kentucky clearly demonstrated that exposing rats to the same levels of mercury vapor that can be released from "mercury/silver" amalgam dental fillings caused the mercury to interact with brain tubulin and disassemble microtubules that maintain neurite structure. The identical neurochemical lesion of similar or greater magnitude is evident in Alzheimer brain homogenates from approximately 80% of patients, when compared to human age-matched neurological controls. (Neurotoxicology 1997;18(2):315-324)

In 2000, researchers at the Neurobiology Laboratory, Psychiatric University Hospital in Basel, Switzerland using neuroblastoma cells exposed to mercury demonstrated an increase in production of amyloid protein that makes up the amyloid plaques as well as significantly increasing the phosphorylation of Tau protein. (J Neurochem 2000 Jan;74(1):231- 236)

Studies demonstrating a correlation between amalgam dental fillings and brain mercury levels:

Lakartidningen 1986 Feb 12;83(7):519-522 Swedish Dental Journal  
1987;11(5):179-187, Sci Total Environ 1987 Oct;66:263-268, J Prosthet Dent

1987 Dec;58(6):704-707, FASEB J 1989 Dec;3(14):2651-2646, Sci Total Environ 1990 Dec 1;99(1-2):1-22, Sci Total Environ 1993 Sep 30;138(1-3):101-115, J Trace Elem Med Biol 1995 Jul;9(2):82-87, Zentralbl Hyg U:mweltmed 1996 Feb;198(3):275-291 FASEB J 1998 Aug;12(11):971-980, Biometals 1999 Sep;12(3):227-231

### **[Alzheimer's Disease & Mercury (cont)]**

#### **Following is the editorial which accompanied the publication of this study;**

Mercury induced growth cone collapse: Owen Hamill, Physiology and Biophysics, UTMB, Galveston, TX, USA

Cases where exposure to heavy metals in the domestic and work environment have contributed to human disease extend back to antiquity with the use of lead in water pipes and wine storage vessels. It has been proposed that pandemic lead poisoning, resulting in mental incompetence and declining birth rate, especially amongst the ruling class, contributed to the fall of Rome [1] (see [2] for another view). More recent lead poisoning in the general population has arisen from lead-based paints and lead-additives in petrol. A well-documented case of occupational poisoning arose in workers of the 19th century felt hat industry due to the use of mercury as a stiffener of rabbit fur. Increased irritability, mood swings, tremulousness, ataxia and impairment in intellectual capacity characterize Mad Hatter's disease [3]. Currently there is ongoing public health debate on whether low level chronic exposure to mercury due to dental repair work results in subclinical behavioral changes associated with CNS damage (see [4] for review). For example, in the USA the most common material used in dental fillings is a mercury/silver mixture (amalgam) in which an estimated 70 000 kg is used in 100 million fillings/year. Furthermore, evidence indicates that mercury vapor is continuously released from tooth fillings where it is breathed in by the lungs and converted into mercuric ions. Although there is no debate on the toxic effects of high concentrations of mercury (i.e. associated with urinary concentrations .50 µg/l), a challenge exists to demonstrate more subtle, preclinical effects associated with chronic low level mercury exposure in the general population with fillings. At least consistent with this notion is the study published in this issue [5] showing that exposure to mercury concentrations of 0.1 µM results in rapid (i.e. within 10 min) retraction of growth cones in snail neurons and is correlated with disruption of microtubules. Interestingly, the authors point out that similar disruption of microtubules is associated with Alzheimer's disease. These recent findings give added impetus for the development and implementation of alternative materials for fillings and may provide parents with added ammunition in teaching their children to floss.

#### **REFERENCES**

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Scarborough J. J Hist Med Allied Sci 39, 469±475 (1984).  
O'Carroll RE, Masterton G, Dougall N et al. Br J Psychiatry 167, 95±98  
1995  
Lorscheider FL, Vimy MJ and Summers AO. FASEB J, 9, 1499±1500 (1995).  
Leong CCW, Syed NI and Lorscheider FL. Neuroreport, 12, 733±737.

