Amalgam Mercury:The Effects of Retention Toxicity, Synergistic Toxicities and Genetic Susceptibility

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VISUALIZATION OF MERCURY EMITTING FROM A DENTAL AMALGAM

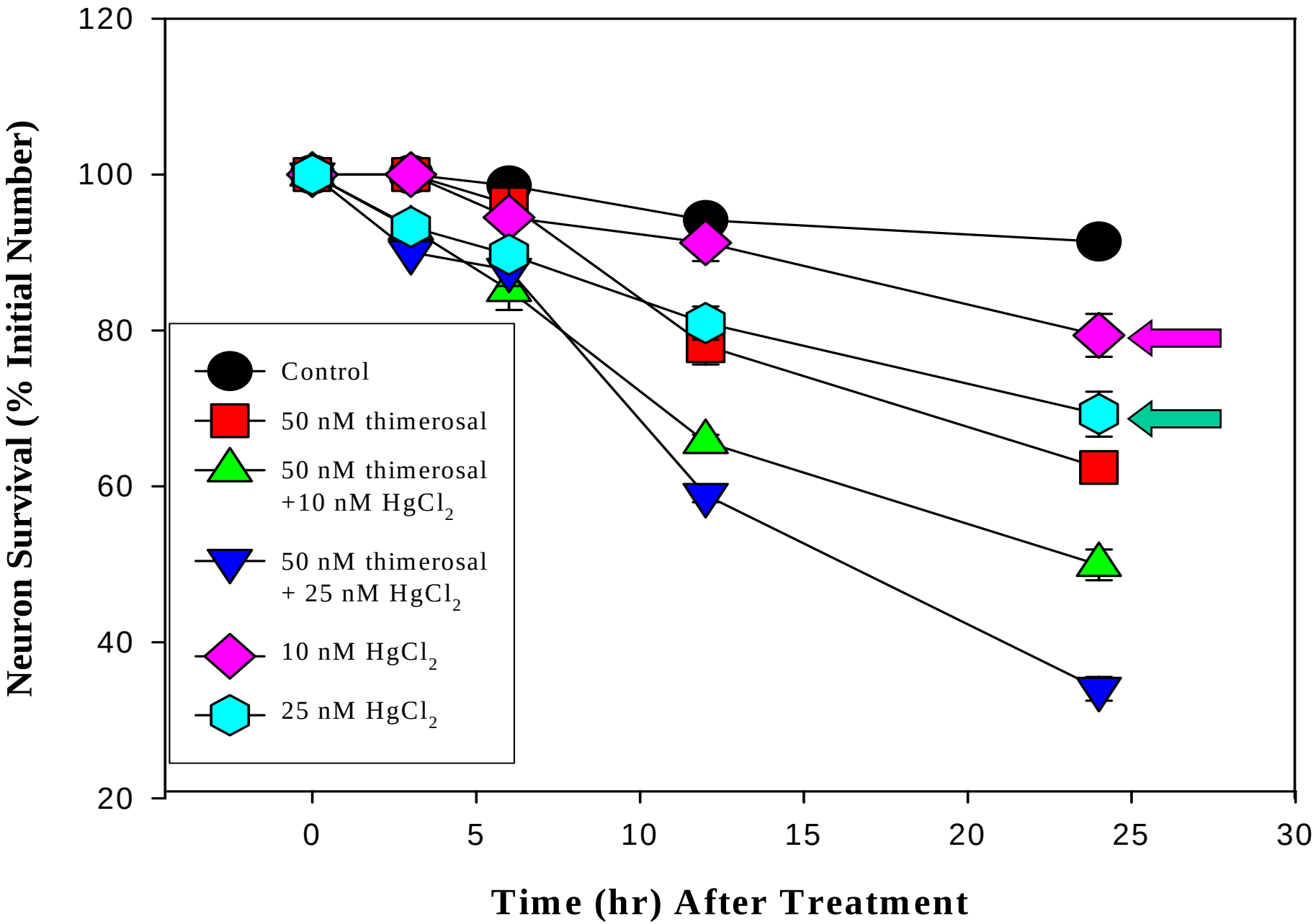
- From: www. uninformed concent.com
- David Kennedy's IAOMT tape

IN SPITE OF THE OBVIOUS EMISSION OF Hg VAPORS FROM DENTAL AMALGAM THE FDA HAS STEADFASTLY REFUSED TO TEST THEM FOR SAFETY!

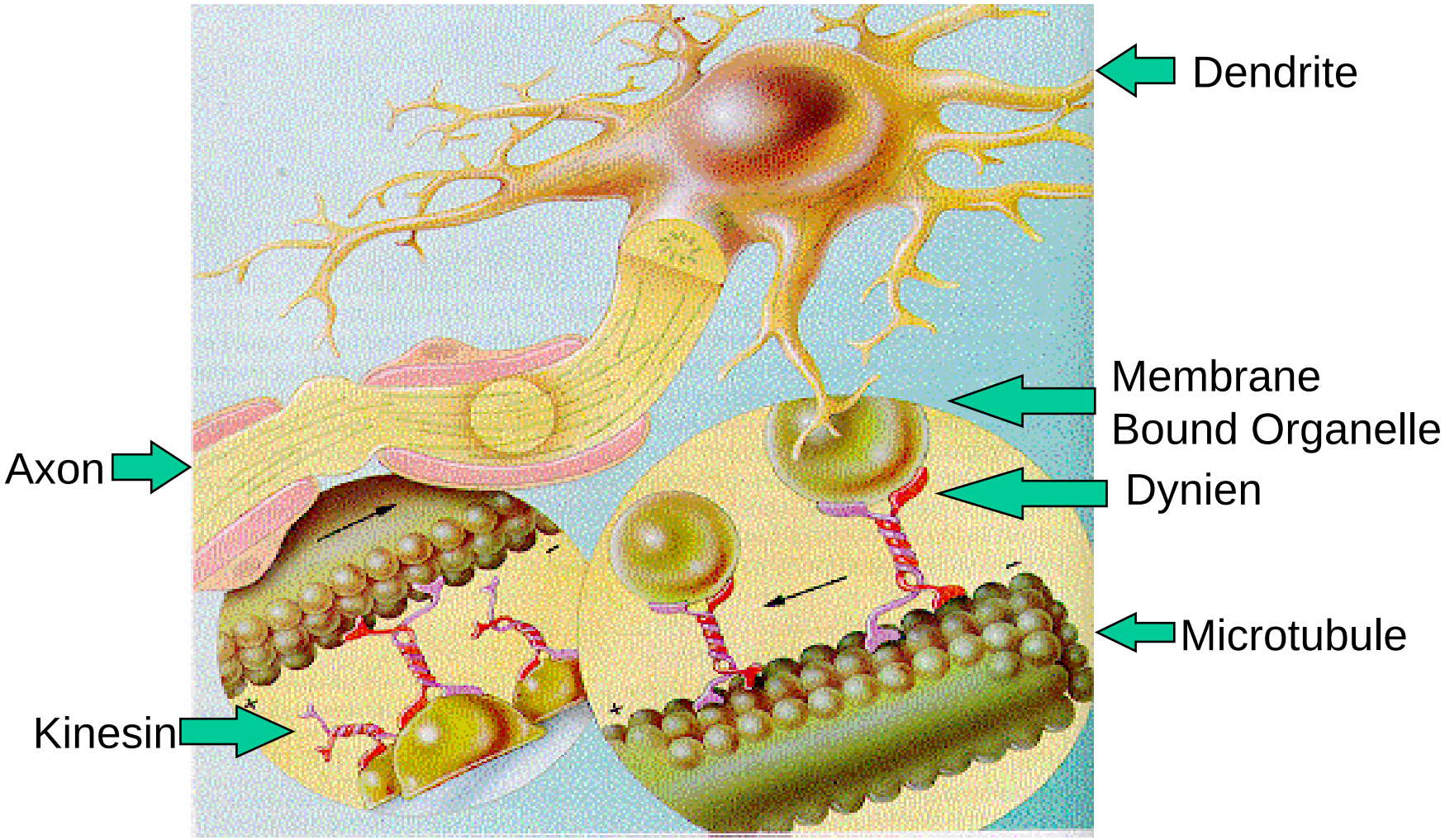
HOW MUCH Hg IS EMITTED FROM DENTAL AMALGAMS?

- It is the greatest failure of the FDA not to have required that each amalgam type be tested for mercury release in a sealed test tube before these amalgams are placed in the mouths of mothers and children. It is simple, inexpensive and easy to do, so why hasn't it been done and reported? Why isn't it required today?

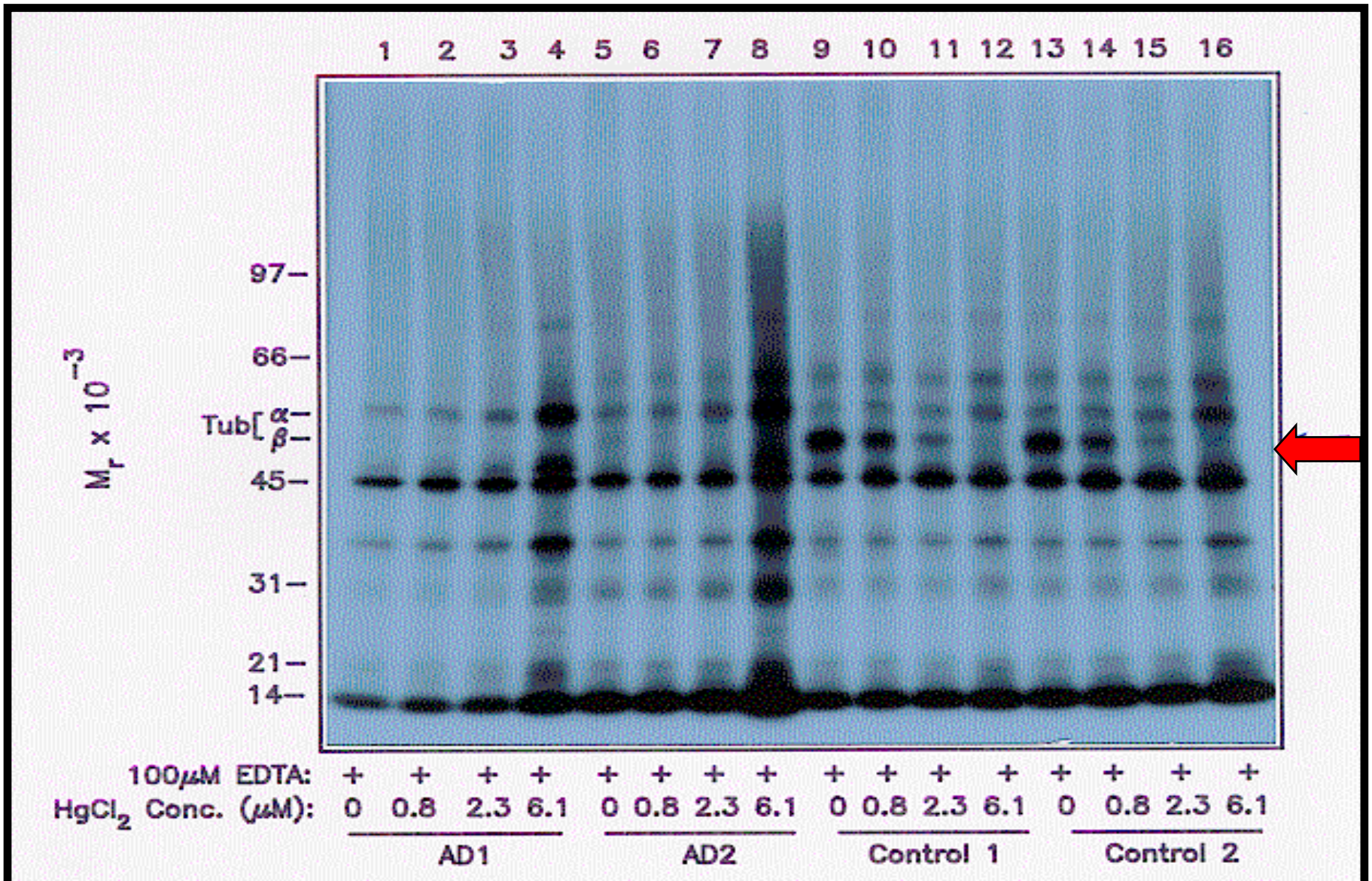
Hg & THIMEROSAL DISPLAY ADDITIVE TOXICITIES.



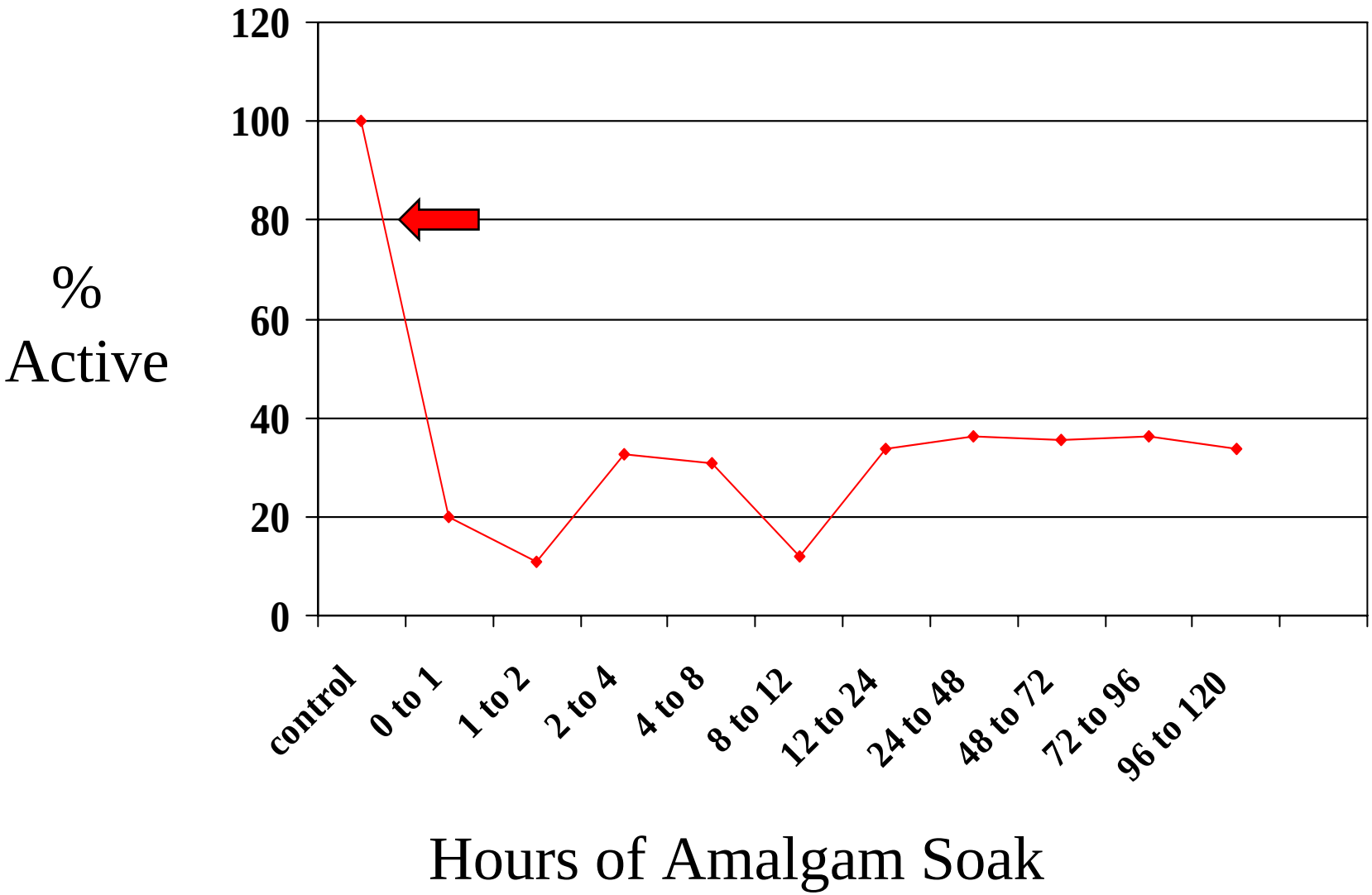
Axonal Transport - A Process Essential for the Survival of Neurons



HgEDTA Induces Aberrant [^{32}P]8N$_3$GTP-ß-Tubulin Interactions Similar to that found in AD



SEQUENTIAL AMALGAM EXTRACTION SOLUTIONS PRODUCE MERCURY-LIKE EFFECT ON HUMAN BRAIN TUBULIN



MERCURY AND ALZHEIMER'S DISEASE

Mercury has been shown to cause the following which are also observed in Alzheimer's Diseased (AD) brain.

- Inhibition of GTP-tubulin interactions and abnormal partitioning of tubulin in human brain tissue. Khatoon et al., Annals of Neurology 26, 210, 1989.
- Mercury, and only mercury, could cause the **abnormal partitioning and prevent GTP binding** as seen in AD Brain. Pendergrass & Haley, Metal Ions in Biological Systems 34, Chapter 16, 461, 1996.
- Mercury vapor exposure causes inhibition of GTP-tubulin interactions in live rats as observed in AD brain. Pendergrass et al, Neurotoxicology 18, 315,1997.
- Exposure of neurons in culture to 10^{-10} molar Hg^{2+} caused the visually observed, rapid, abnormal aggregation of tubulin producing stripped neurofibrils the precursor for **neurofibillary tangles (NFTs)**, a major diagnostic hallmark of AD. Leong et al. NeuroReports 12(4), 733, 2001
- Exposure of neuroblastoma cells to 10^{-9} molar mercury increased **Tau phosphorylation and beta-amyloid**. Both of these events occur in Alzheimer's diseased brain. Amyloid plaque formation is the "diagnostic hallmark" of Alzheimer's disease. Olivieri et al. J. Neurochemistry, 74, 231, 2000.

Therefore, Hg exposure can create elements of the diagnostic hallmarks and aberrant biochemistry of Alzheimer's Disease in appropriate testing systems!

MERCURY AND ALZHEIMER'S DISEASE

- Elevated brain glutamine synthetase in cerebrospinal fluid and serum is a diagnostic marker for Alzheimer's disease. Gunnersen & Haley PNAS, USA, 89,11949,1992. Takahashi et al. Clin. Chem. 48, 375-8, 2002.
- Levels of glutamine synthetase are elevated in astrocytes of Alzheimer's diseased brain. Tumani, et al. Arch. Neurol. 56 1241-6, 1999.
- Glutamine synthetase is inhibited by mercury and is also inhibited in Alzheimer's diseased brain. Butterfield et al. *J. Neurochem.*, 68, 2451, 1997.
- Creatine kinase is over 95% inhibited in AD brain. Brain creatine kinase is rapidly inhibited by mercury. David & Haley, Mole. Brain Research 54, 276, 1998.

MERCURY AND ALZHEIMER'S DISEASE

- Genetic susceptibility to AD by APO-E protein is consistent with loss of mercury binding capacity in the high risk APO-E4 versus the lower risk APO-E2 carriers. Pendergrass & Haley, Metal Ions in Biological Systems 34, Chapter 16, 461, 1996.
- Blood levels of mercury are 3 times higher in AD patients versus aged matched, non-demented control patients. Hock, et al., J. Neural Transm. 105,(1), 59, 1998.

Hg Levels in Human Brain

- Saxe et al, with Ehmann and Markesbery in Alzheimer's Disease, Dental Amalgam and Mercury, JADA v130, p191-199, 1999, determined Hg levels in the brains of 101 human subjects, both AD and normals.
- The histogram in this paper showed 6 of 101 subjects with brain Hg levels above 200 ng/g wet weight (C=236, 248, 319: AD=394, 622, 698). Why do these individuals alone have such high levels?
- This represents between 1.2 & 3.5 X 10^{-6} molar levels of Hg in 6% of these subjects. THESE ARE HIGHLY TOXIC LEVELS! At 100 ng/g Hg this increases to about 15% of subjects who died with highly toxic levels of brain mercury.
- ????Where does this Hg come from????

ELEVATED MERCURY IN IDIOPATHIC DILATED CARDIOMYOPATHY (IDCM).

WHERE DOES THE Hg COME FROM?

LEVELS ng/g	Hg	Sb
Controls	8.0	1.5
IDCM	178,400	19.260

Frustaci et al., J. of American College of Cardiology, 33, (6) 1578, 1999.
Controls were patients with valvular or ischemic heart disease.

ATHLETIC YOUTH DIE OF IDCM.

WHY HASN'T THE NIH OR FDA REQUESTED PROPOSALS FOR RESEARCH TO STUDY THIS??

THIS IS PROOF THAT MERCURY CAN CONCENTRATE IN SPECIFIC TISSUES OR ORGANS OF THE BODY, EVEN IF Hg BLOOD LEVELS ARE FOUND TO BE IN THE NORMAL RANGE.

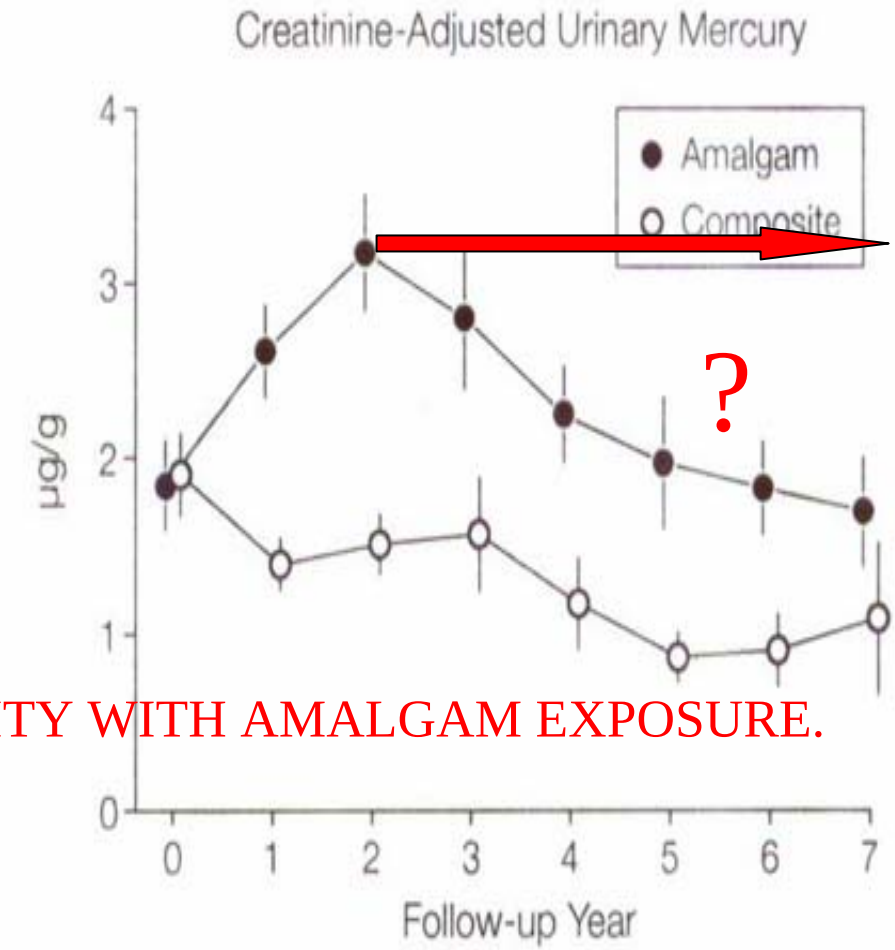
Is there other proof of high body Hg retention?

Vamnes et al. Sci. Total Envir. 308:63-71, 2003 reported the following:

1. Persons 2.5 years after amalgam removal had about the same blood Hg levels as those with existing amalgams. Those who never ever had amalgams were had blood Hg levels that were significantly lower. This indicates long term retention of Hg from amalgam exposure.
2. Chelation with DMPS caused a 24-30% decrease in blood Hg levels which rapidly returned to pre-DMPS levels within 2 hours. This indicates a high body burden of Hg exists that can rapidly replace that removed from blood by DMPS. This supports tissue retention toxicity as seen in IDCM.

Figure 2. Mean Urinary and Creatinine-Adjusted Urinary Mercury Concentrations by Treatment Group and Follow-up Year

DeRouen et al. JAMA 295, 1784-92, 2006

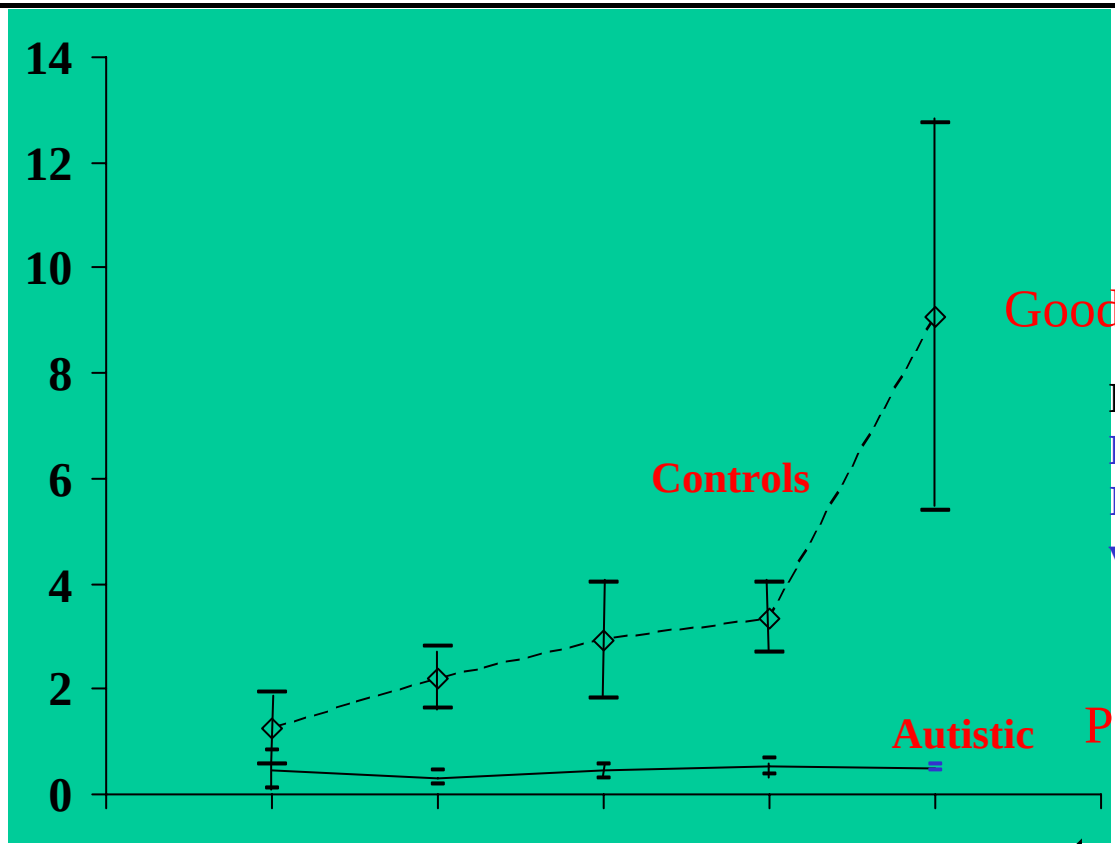


LOST URINARY EXCRETION CAPABILITY WITH AMALGAM EXPOSURE.

Error bars indicate 95% confidence intervals. Childrens Amalgam Trials 2006

- IT APPEARS THAT AFTER TWO YEARS EXPOSURE TO MERCURY FROM DENTAL AMALGAMS THE CHILDREN HAVE LOST THEIR ABILITY TO EXCRETE MERCURY INTO THE URINE.
- THIS CORRELATES WITH THE INHIBITION OF KIDNEY PORPHYRIN/HEME SYNTHESIS IN DENTISTS SHOWING PROFOUND Hg TOXIC EFFECTS ON THIS HEME SYNTHESIS PATHWAY.

MERCURY BIRTH HAIR LEVELS VS. MOTHER'S AMALGAM FILLINGS IN AUTISTIC AND CONTROL GROUPS



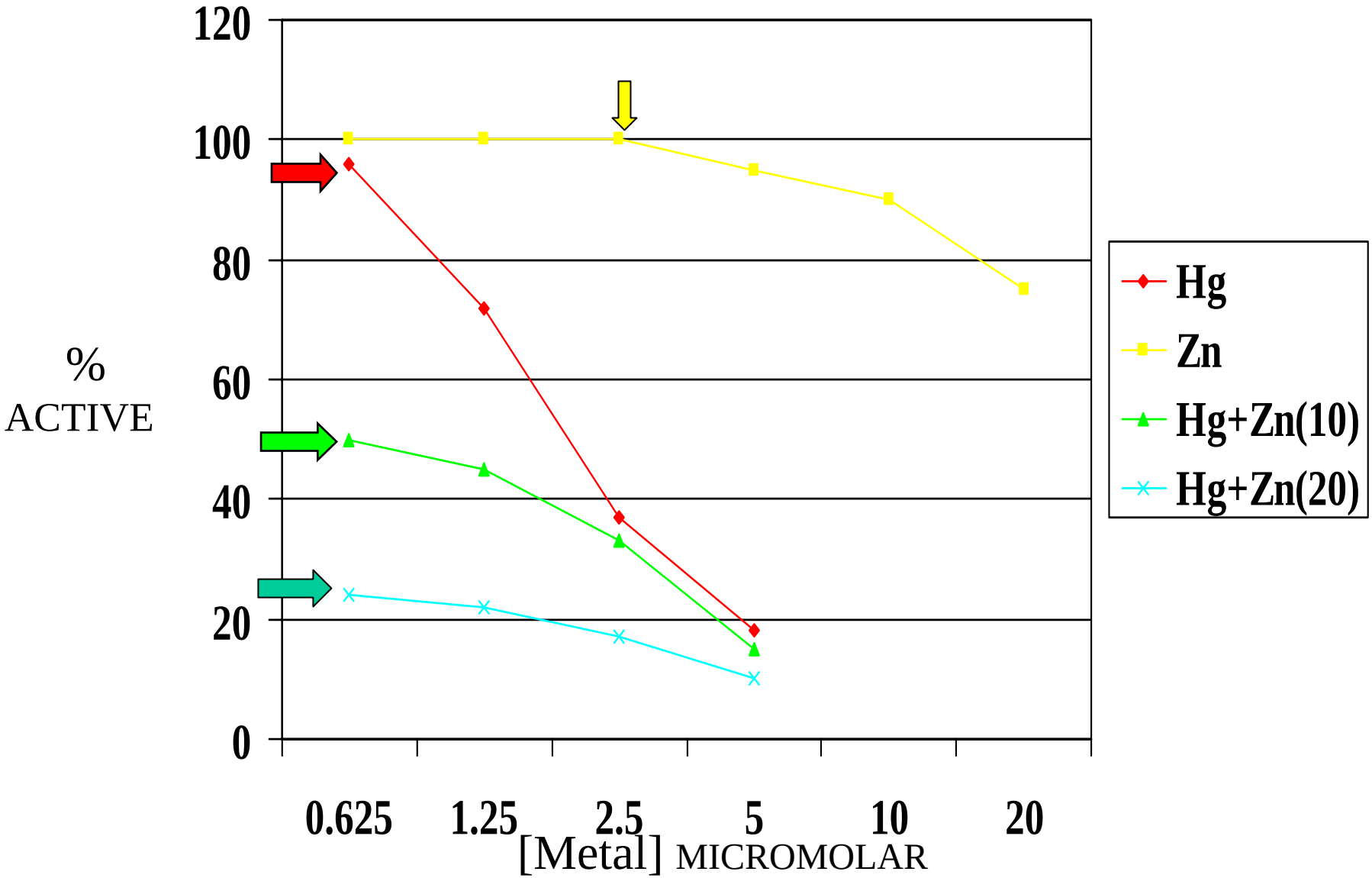
Number of amalgams:	0-3	4-5	6-7	8-9	≥10
Control: autistic ratio:	2.64	6.93	6.70	6.32	17.91
N:	15	22	29	30	43

SYNERGISTIC EFFECTS OF HEAVY METALS IS QUITE COMPLEX AND CAN GREATLY ENHANCE TOXICTY OF MERCURY

Shubert et al. Combined Effects in Toxicology--A Rapid systematic Testing Procedure:Cadmium, Mercury & Lead. J. of Toxicology & Environmental Health 4:763, 1978.

1. "the administration of an essentially no response level (LD1) of a mercury salt together with a 1/20 of the LD1 of a lead salt **killed all of the animals**."
2. "Generally, **a combination was synergistic when the most toxic member was present at or near its LD1 dose in the presence of a much less toxic member."**

SYNERGISTIC TOXICITY OF ZINC PLUS MERCURY ON HUMAN BRAIN TUBULIN VIABILITY



Antibiotics, Milk and Hg Excretion

- In a study where rats were given high doses of oral antibiotics the half-life for excretion of mercury increased from 10 days to >100 days.
- If the rats were also on a milk diet the excretion half-life increased to over 300 days.

(Rowland, *Archives of Environmental Health* 1984: 39(6); 401-408)

Mercury Effects on the Immune System

- The mitotic spindle is built on tubulin quite similar to that found in axons of neurons. Therefore, since the cells of the immune system must divide for an effective immune response Hg inhibits this and actively suppresses the immune system.
- Mercury is a **very potent inhibitor of phagocytosis** by mononuclear phagocytes, inhibiting the process at low nanomolar levels. (Rampersad et al., Transfusion 45(3):384-93,2005). This prevents removal of microbes and Hg damaged cells and proteins leading to greater susceptibility for microbe infection and widespread autoimmune problems.

GENETIC SUSCEPTIBILITY

- THE PORPHYRIN PATHWAY IS ONE OF THE MOST SENSITIVE DETECTORS OF TOXIC MERCURY EXPOSURE, GIVING A PROFILE NOT OBSERVED WITH ANY OTHER TOXIN.
- A DIRECT ASSOCIATION HAS BEEN SHOWN BETWEEN DENTAL MERCURY EXPOSURE, NEUROBEHAVIORAL RESPONSE AND A GENETIC POLYMORPHISM (CPOX4 of coprophyrinogen oxidase, work of D. Echeverria & JS Woods, U. Washington).
- PORPHYRINURIA IS ALSO HIGHLY ASSOCIATED WITH AUTISTIC DISORDER (R. Nataf, Paris, France).
- AUTISTIC CHILDREN HAVE LOWER GLUTATHIONE LEVELS THAN NORMAL CHILDREN (J. James, U. of Arkansas).

Conclusions

Several vectors make it impossible to define a safe level of mercury exposure. These are:

1. Synergistic Toxicities with other heavy metals, hormones, antibiotics, milk diets, etc.

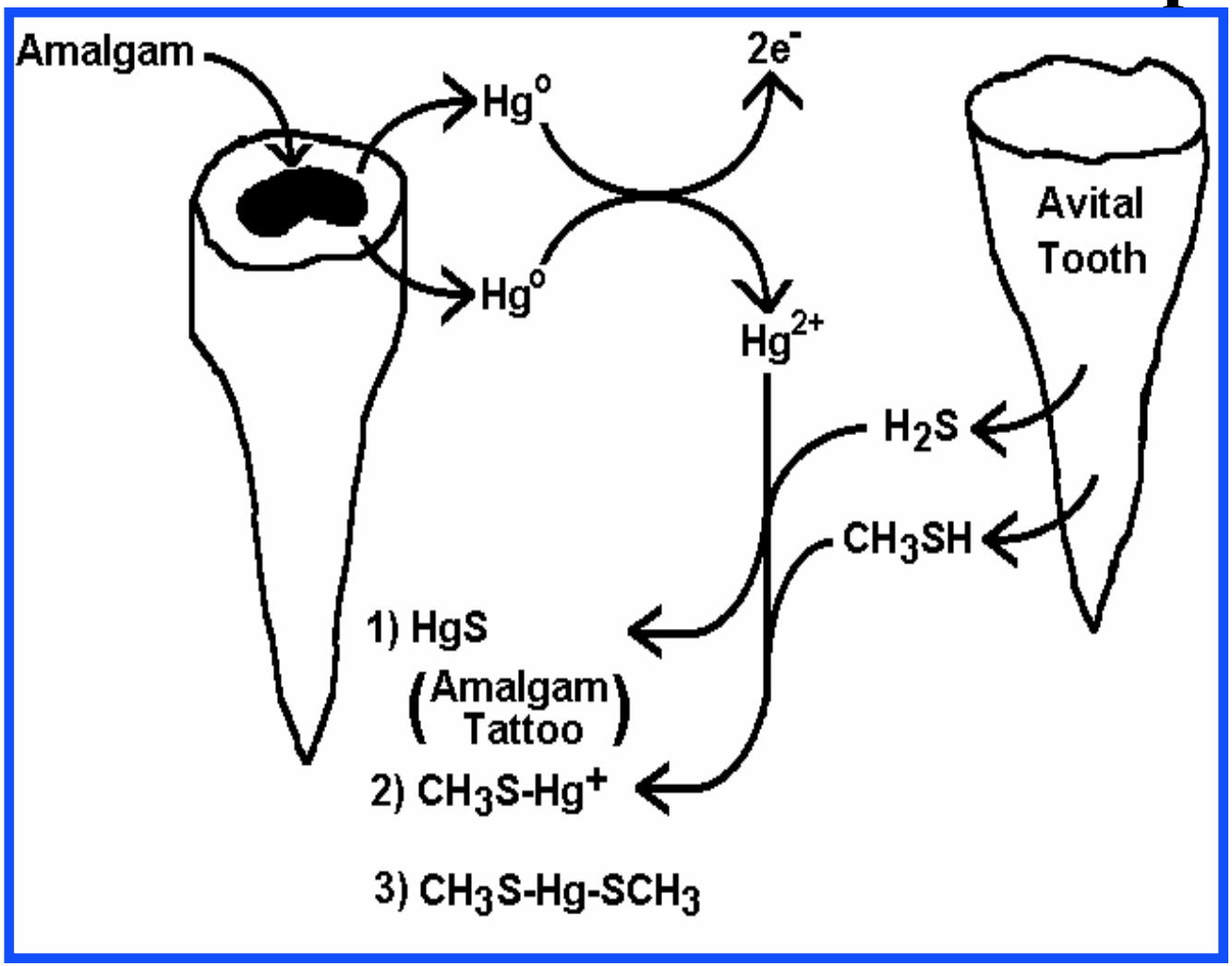
2. Genetic Susceptibility of a subset of the population.

3. High susceptibility of infants, aged and sick individuals.

4. Toxic effects that are secondary to the toxic effects of mercury, like low heme levels based on porphyrin pathway inhibition. Low heme decreases oxygen transport, P450 detox activity, and electron transport system capability representing secondary effects of mercury toxicity.

5. Removal of beta-amyloid protein in Alzheimer's disease is thought due to aberrant heme levels in the brain (Atamna & Frey, PNAS 101,#30, 11153-58). Is this another secondary mercury toxicity effect???

Amalgam Mercury Can Combine With Bacterial Toxins To Produce Even More Toxic Species



REACTION OF ORAL MERCURY WITH ANEROBIC TOXICANTS PRODUCING SUPER-TOXICANTS $CH_3S\text{-}Hg^+$ AND $CH_3\text{-}S\text{-}Hg\text{-}S\text{-}CH_3$