UNITED STATES COURT OF APPEALS, DISTRICT OF COLUMBIA CIRCUIT

MOMS AGAINST MERCURY, CONNECTICUT COALITION for ENVIRONMENTAL JUSTICE, OREGONIANS for LIFE, CALIFORNIA CITIZENS for HEALTH FREEDOM, Kevin J. BIGGERS (Member of the Dental Board of California), Karen JOHNSON (Arizona State Senator), Linda BROCATO, R. Andrew LANDERMAN, D.D.S., Anita Vazquez TIBAU, Petitioners,	)	Docket #06-1147
vs. **AFFIDAVIT OF BOYD E. HALEY, Ph.D.**	)	
FOOD AND DRUG ADMINISTRATION ["FDA"], et al., Respondents.	)	

AN EVALUATION OF DENTAL AMALGAM AND ITS ABILITY TO INJURE HUMAN HEALTH

1) I am Professor of Chemistry/Biochemistry in the Department of Chemistry at the University of Kentucky. Throughout my career I have studied the effects of numerous compounds on the changes of the activity of enzymes, proteins and cellular function proteins and the relationship of these changes to disease states. In the past 14 years I have concentrated my research on the effects of mercury toxicity on human health. Specifically, I have researched and evaluated the contributions of dental amalgam, biologics and vaccines on the human body burden of mercury and organic-mercury compounds and the potential effects of these compounds on specific enzymes and cells. Attached is a one page biography and an abbreviated copy of my Curriculum Vitae. The full CV is approximately twenty five pages and to save space will be provided upon request.

2) Mercury exposure to humans comes from various chemical forms such as elemental vapors, inorganic salts and organic-mercurials such as thimerosal and phenylmercury acetate (PMA). All chemical forms of mercury have been proven toxic at relatively low levels. There is no doubt that mercury and mercury compounds represent the most dangerous form of metal toxicity since research on exposures show them to cause adverse effects in animals and humans at the very low levels. Mercury and mercury containing compounds are listed under the State of California's Proposition 65 as compounds that need to be evaluated for their level of toxicity to ensure the safety of the

citizens. Mercury vapor is one of the most toxic forms of mercury along with some of the organic mercury compounds. It is this vaporous form of mercury that is released from dental amalgams and is the major contributor to human mercury body burden.[22]

3) It is important to understand two concepts regarding mercury toxicity. The first is the level of exposure and the second is the contribution to human body burden. One can be exposed to mercury in the diet by eating fish, etc. This mercury is effectively excreted and does not appear to lead to a build up of mercury in the body but may cause subtle effects difficult to identify. The studies in the fish eating populations of the Faroe Islands and the Seychelles are examples of this.[36, 37] The citizens of these studies were exposed to high levels of mercury in their diets, but maintained a fairly low level of mercury body burden and urinary mercury levels not dramatically different from the USA population. In my opinion, the blood levels were higher due to excretion of the daily diet intake of bound mercury from sea food. This is most likely due to the fact that dietary mercury in fish has already reacted with protective compounds in the fish and are not as reactive or as capable of being retained on ingestion as would be other forms of mercury that have not been previously exposed to a biological system (e.g. mercury vapor).

4) In contrast to mercury from a fish diet, mercury vapor from amalgams has all of its chemical reactive potential and easily penetrates into the cells of the central nervous system where it is converted to the toxic form (Hg^{2+}), reacts with proteins in the brain, etc. and is retained for much longer periods of time and builds up in these tissues causing a significant toxic effect. Research has determined that about 80% of inhaled mercury vapor is retained by the human body and that the major contributor to human body burden is from dental amalgam. This is the position of the World Health Organization.

4) The exceptional toxicity of mercury vapor is probably due to the efficient partitioning of vaporous mercury into certain body organs (e.g CNS, kidney) and into specific cellular organelles (e.g. the mitochondria) based on mercury vapor's ability to easily penetrate membranes and the blood brain barrier. In this manner mercury vapor, Hg^0, is quite different from ionic Hg^{2+} and Hg^{1+}. For example, air and oral ingestion of mercury vapor (Hg^0) primarily affects the central nervous system whereas the kidney is the major organ affected by the cationic forms of mercury (e.g. Hg^{1+} and Hg^{2+}). Add to this problem is the fact that prolonged mercury vapor exposure can lead to inhibit the

excretion process itself. Therefore, extended exposure to mercury vapor from amalgams will, by itself, decrease the body's ability to excrete mercury. The recent data presented in the Children's Amalgam Trials, published in JAMA, shows that extended exposure to mercury from dental amalgams lead to a marked +40% decrease in the ability to excrete mercury in the urine.[27, figure 2, page 1788] from year two to year seven of the study. Even though the children (orphans in a Lisbon, Portugal orphanage) were given additional amalgams from year two to year seven the rate of mercury excretion in their urine dropped dramatically. Therefore, urine mercury levels do not represent in any way an accurate measure of the level of exposure of an individual. Another evaluation of this data, separating the urinary excretion of mercury ability of boys versus girls shows that boys, who are much more likely to have neurological illnesses as found in autism spectrum disorders, were much less capable of excreting mercury than girls[38]. In fact, the boys with amalgams placed had urinary mercury excretion rates similar to boys without amalgams indicating that within the 7 year time frame of the experiment they had lost the ability to excrete the additional mercury from their amalgam exposures.

5) The pro-amalgam group in the USA has "estimated" the amount of mercury excreted from amalgams by using urine mercury levels, which is obviously invalid, since over 90% of mercury is excreted via fecal routes, not through the urine.[34] The British Dental Association also uses this same study to infer that amalgams do not contribute significantly to human mercury exposure.[35] The pro-amalgam group are also aware of publications showing that over 90% of mercury excreted by the human body leaves through the bilary transport system of the liver and is excreted in the feces---yet they constantly refer to low urine mercury levels as their source of suggesting low exposures from dental amalgams. They make the comment that "dose make the poison"[35] yet avoid determining the actual dose but instead depend on an "estimation" based on the urine excretion rate that represents at best 10% of the total mercury being excreted.

6) It is now well known that the relative toxicity of mercury and organic mercury compounds fluctuate dramatically in humans depending on: (1) delivery route (2) the presence of other synergistic toxic metals such as lead, cadmium, aluminum, etc. (3) different diets (4) antibiotic exposure (5) genetic susceptibility[23,24] and allergic reactions (estimated as at least 1% of the human population[7] with 8.7 to 13.4% showing sensitivity

to a diagnostic patch test [5 & references therein]) (6) gender (7) state of health and (8) age of exposure[19]. Therefore, attempting to determine a generalized, lowest observable affect level (LOAEL) or no observable effect level (NOAEL) regarding mercury vapor exposure is a complicated, if not impossible, procedure as explained by the analysis of published refereed research articles (these are presented below).

7) The end point for measuring toxicity is also critical. That is, if lethality versus loss of neurological function are the end points then different values for a minimum daily acceptable limits of exposure will be arrived at. Also, when lethality is compared to loss of neurological function, or suppression of the immune system, as the end points a different minimum acceptable daily exposure would be expected. In today's medicine the health of the individuals metabolism and neurological is of prime concern and this has lowered the level of mercury exposure that is considered a NOEL.

8) It is obvious that lethality requires a higher level of exposure to mercury vapor than does neurological, immunological or developmental damage. For example, adverse immunological effects and autoimmunity induced by dental amalgam and alloy in mice has been demonstrated.[25] This has been further supported by observations that the phagocytosis by macrophages, the first step in the innate and acquired immune systems, is inhibited by low nanomolar levels of mercury.[30] Neurotoxicity combined with a suppressed immune system in an aged patient would be considered a danger for an amalgam exposed person with a neurological disease, such as a motor neuron diseased. Low nanomolar levels of mercury are reached in the blood and urine of individuals with amalgam fillings. For example, in a urine or blood with a low 3 micrograms/liter of mercury the concentration would be about 15 nanomolar or 15×10^{-9} molar (3×10^{-6} grams divided by 201 grams/mole for Hg). One to five nanomolar levels of mercury can have dramatic effects on certain enzymes or neurons or immune system cells in culture. Porphyrin profiles (see below), leading to the synthesis of heme, in dentists show mercury induced aberrancies at urine levels in the 3 microgram/liter range[23,24]

9) Many individuals may appear normal and have apparently non-toxic levels of blood and urine mercury and still suffer from extreme mercury toxicity. For example, young athletes and others who died from Idiopathic Dilated Cardiomyopathy (IDCM) have been found to have 22,000 times the mercury in their heart tissue when compared to

their muscular levels or the mercury in the hearts of individuals who died of other forms of heart disease[18]. This level, 178,400ng/g, would have definitely have been lethal to the kidney and CNS cells and this level has never, to my knowledge, been observed in a blood, urine or hair sample of a human. In my opinion, the unexplained, abnormal partitioning of huge levels of mercury into specific organs in certain individuals essentially renders it impossible to identify a hair, blood or urine level of mercury that is safe for all, a NOEL. It certainly indicates that a person with an existing motor neuron disease would be at elevated risk if constantly exposed to low level mercury vapors. It is important to note that mercury toxicity is a retention toxicity, where mercury is extracted from the blood and retained in certain tissues, leading to elevated levels that can cause illnesses.

10) For an accurate determination of a LOEL or NOEL for injury causing mercury exposure it is clear that using data from one strain of a genetically inbred rat or mouse strain could result in a very inaccurate answer, going either way.[4] However, this has been done. Humans are not genetically inbred and their diets differ dramatically. Some are on antibiotic medications that would enhance the toxicity of all mercury compounds. Further more, it has been established in the literature that different strains of mice and rats give different sensitivities to mercury and that there can be dramatic differences in sensitivity to specific toxicants between species such as rats and humans. Therefore, basing safety on animal data is often very misleading.

11) Recent studies on dentists and dental technicians (selected as they are exposed to mercury vapor) has shown that a specific polymorphism in the CPOX gene leads to enhanced disruption of the porphyrin pathway which leads to the synthesis of heme. About 85% of all dentists had abnormal porphyrin profiles that indicated their ability to make heme was being impeded, and 15% of this 85% displayed a marked inhibition that correlated with their mercury exposure. [23,24] Similar data has been reported for autistic children, where 53% have shown abnormal porphyrin profiles indicative of mercury toxicity.[26] Treating a subset of these autistic children with a mercury chelator effected a porphyrin profile change back towards the normal range indicating that the cause of the abnormality was toxicity, not genetics.[26] This implies that very low levels of mercury exposure as determined by urinary mercury levels can have an

effect on 85% of the population and a dramatic affect on certain susceptible individuals who represent 15% of the population.

12) It is very important to note the negative contributions secondary to the mercury inhibition of heme synthesis. Heme is required for oxygen carrying capacity of blood, it is also necessary for a critical step in the electron transport system of the mitochondria. Both of these steps, if impeded, will decrease the ability of the body to make energy for physiological functions that are necessary for good health. Also, heme is a needed cofactor for the P450 enzymes that have a primary role in detoxing the body of many organic toxins such as pesticides, PCBs, herbicides, etc. Without adequate heme a human will have an impeded ability to detox many different toxins that they may be exposed to.(ref. Any good biochemistry textbook)

12) Additionally, recent research has shown that the removal of beta-amyloid protein from the brain in a normal fashion requires a specific heme, and that a lack of this heme prevents beta-amyloid excretion and leads to the formation of amyloid plaques (senile plaques) in the brain.[32] The amyloid plaque build up is a major pathological, diagnostic hallmark of Alzheimer's disease.[27] Therefore, the mercury inhibition of heme synthesis could lead to a secondary systemic abnormality that contributes to severe neurological illnesses, including the neuronal disease classified as Alzheimer's disease. The observation of increased amyloid build up due to inadequate forms of the proper heme molecule is also supported by the observed formation of neurofibillary tangles (NFTs) from neurons in culture by the exposure to sub-nanomolare levels of mercury, much lower (by about 1,000 fold) than is found in many human brains.[31] NFTs are also a major pathological, diagnostic hallmark of Alzheimer's disease. This data is consistent with the observations published earlier where mercury, and again, only mercury could cause a major biological abnormality in a major brain protein when added to normal human brain tissues or in rat brain on exposure to mercury vapor.[12, 13] Therefore, mercury, and only mercury at very low levels, can generate the two major pathological hallmarks of a major neurological disease as well as mimic the protein level aberrancies. The exposure to mercury and its known effects on neurons may explain the uptake of inorganic mercury by olfactory patways and the entry of low doses of mercury vapor into the nervous system.[6, 14]

13) Synergistic toxicity of two or more toxic metals has been known for some time. It has been shown that the relative toxicity of mercury containing compounds appears to be dramatically affected by the presence of other compounds and heavy metals that synergistically enhance the toxicity of mercury. For example, mixing of an LD1 dose of mercury with a 1/20 dilution of an LD1 of lead produces a mixture with an LD100, not an LD2 or less that would be expected with additive toxicities[1]. Since there is considerable concern about the lead levels in the drinking water in our nation's capital and other major cities it seems the citizens there would be under more toxic stress from dental amalgams than those in locations with little or no lead exposure.

14) Consider also that mercury from different exposures are at the least additive in their toxicity effects and they may come from different types of iatrogenic exposures.[15, 16, 17] A report from the National Center for Health Statistics, Center for Disease Control and Health in 2003 stated that approximately 8% to 10% of women of child-bearing age had concentrations of mercury higher than the US EPA's recommended reference dose, below which exposures are considered to be without adverse effects[3]. One would expect similar mercury levels, or higher, in the male population and in the population of individuals with motor neuron disease or other neurological illnesses.. This blood level in women caused more recent concern with data showing that cord blood was 1.7 times the level of maternal blood indicating that more than 8% of children being born are being exposed to toxic levels of mercury from their mother's blood. All of these individuals would definitely be more at risk during transient mercury exposures than would the general population and are certainly not comparable to animals in a pristine environment being exposed to only one mercury toxicant and fed a chow that is designed to be free of other toxic metals. Therefore, a 10-fold reduction for urinary mercury levels, as is common in converting a LOEL into a NOEL, most likely does not provide the protection factor predicted as it would not account for exposures to materials that synergistically enhance mercury toxicity nor does it account for the reduction of urinary mercury excretion caused by prolonged mercury vapor exposures.

15) It is well known that diet plays a major role in the ability of mammals to excrete mercury[2]. Studies have shown that three different diets fed to adult female mice (high protein synthetic diet; standard rat chow diet; milk diet) dramatically changed the

rate of fecal excretion of mercury. Mercury was introduced orally as methyl-mercury (MeHg) and diet caused differential rates of whole body mercury elimination. The results showed that mice fed a synthetic, high protein diet had the lowest tissues levels of mercury whereas those fed the milk diet retained the highest mercury levels. This was confirmed by the total percentage of mercury excreted in the feces after 6 days of 43%, 29% and 11% in the high protein, rat chow and milk diets, respectively. Therefore, diet plays a major role in the fecal excretion rates of mercury from an organic mercury compound. As expected, diet also affected the excretion rate of mercury from body tissues. The obvious importance of this data is that the retention of mercury in the body of someone on a milk diet would be much higher. Twenty year old studies report that suckling animals absorb about 50% of Hg^{2+} versus 5% in non-suckling animals[11]. Since the level of toxicity would likely increase with retention time, especially if the exposure rate to mercury were consistent over any significant period of time, then the diet can have a major affect on a calculated NOELs and minimum acceptable daily levels.

16) Gender effects of mercury toxicity appear to be based on both the protective effects of the female hormone[28] and the enhancement of mercury and ethylmercury toxicity by testosterone, the male hormone[29]. Research in our laboratory showed that testosterone dramatically enhanced the toxicity of mercury and ethylmercury whereas estrodiol showed a potent protective effect. A significant quote from another lab states "The estrogenic effects were associated with a reduction of mercury content of the anterior pituitary gland and medial hypothalmus, suggesting a protective estrogenic effect."[28] Further, a study has found that amniotic fluid testosterone levels appear higher in mother who give birth to children with autism spectrum disorders. The conclusions of one paper stated "These finding implicate foetal testosterone in both social development and attentional focus. They may also have implications for understanding the sex ratio in autism."[33] What is of importance here is the fact that gender plays a major role in susceptibility to mercury toxicity with the male gender appearing to be more susceptible.

17) Toxicity is also known to vary with the chemical species of mercury that exists in the body's tissues. Diets can change this as it was observed that foods ingested played a major role in the mercury chemical species that existed in the mice given oral doses of MeHg. Hg^{2+} was the species found at the highest level in test animals on a

synthetic protein diet (35.3%) and was the lowest in test animals on a milk diet (6.6%). It is reasonable to predict that diet changes the conversion of MeHg to Hg^{2+} and would likely do so for other organic mercury compounds, such as ethyl-mercury (Et-Hg), which is released from thimerosal. The toxicity of organic mercury compounds (e.g. MeHg versus EtHg), which partition into the body organs similar to mercury vapor, has been suggested to be greater than Hg^{2+} (inorganic mercury). It is also reasonable to expect the toxicity to be partially determined by the rate that the organic mercury compounds are converted to Hg^{2+} after the chemical nature of the mercury source has allowed effective partitioning across the blood brain barrier.

18) Other studies confirm that the renal uptake and toxicity of circulating mercury is significantly enhanced in rats by the co-ingestion of the essential amino acid L-cysteine[8] and disease marker homocysteine[9]. Elevated blood homocysteine level is also a major risk factor for cardiovascular disease. Therefore, humans with risk for cardiovascular disease would be more at risk by low level mercury exposure than others due to the more effective mercury uptake stimulated by elevated homocycteine levels.

19) Medical status is of concern when considering mercury compound toxicity, especially when bacterial infections are being treated. Treatment of adult female mice with widely used antibiotics 7 days prior to MeHg exposure dramatically influenced mercury retention of tissues from mice receiving similar organic mercury exposures[2]. The calculated whole body mercury elimination half-times from day 1 to day 6 varied from 34, 10 and 5 days for mice fed a milk diet, mice chow or high protein diet. A remarkable 6.8 fold increase in retention half-life existed between a milk diet and high protein diet that was caused by antibiotic treatment that also changed the gut microflora. Antibiotic treatment dropped the fecal mercury excretion to near zero in the high protein and milk diets and to less than 8% with the mouse chow diet.[2] Therefore, it can be concluded that the relative toxicity of mercury and organic-mercury compounds would be dramatically increased if the test subjects were on certain antibiotics.

20) The toxicity of mercury vapor is dependent on retention and excretion and these vectors are dramatically affected by diet and antibiotic treatment as well as other factors. This makes it nearly impossible to define a safe level of exposure for any individual, but especially individuals with other types of neurological illnesses like motor

neuron diseases or impending dementias. Being exposed minute by minute to mercury vapor for years has never been established as safe, but it has been effectively avoided by the dental organizations with the exception of giving their opinions regarding perceived safety. It is incredible that the responsible US government agencies and the organizations and companies using dental amalgam have not felt the need to produce such research. Especially with the obvious severe toxic nature mercury vapor and the ease at which the level of mercury vapor that would escape from a dental amalgam could be measured. The quality data is just not available in the literature to evaluate and determine the level at which mercury vapor is emitted from the various types of dental amalgam. However, it is my opinion that the reason is not because it would be difficult to do, but to do so would place the manufacturers and users of dental amalgam at risk for major lawsuits and they would lose their businesses.

21) One has to ask the simple question "Why are producers of amalgam products not required to produce data in the packages that describe the amount of mercury vapor that escapes daily from their amalgam of known weight and surface area under conditions that mimic the mouth with regards to temperature, pH and brushing?" In my opinion, the reason they don't is well known since to do so would quickly establish their amalgam products as dangerous to human health.

22) The process of placing or removing dental amalgam's in a pregnant mother has to increase the exposure of the *in utero* infant to elevated mercury vapors as it would dramatically increase the mother's blood mercury levels. It is well known that mercury vapor can cross the placenta, and is even concentrated in the cord blood versus the mother's blood. Other studies have shown that mercury increases in the birth hair of normal children in response to increasing dental amalgams in the birth mother[20]. Other similar studies point to aberrant mercury hair levels in children with neurological problems[20,21]. There can be little doubt that the exposure of a pregnant mother to mercury vapor by aggressive dental amalgam treatment could cause harm to her infant *in utero*. It also points out that the most effective protection of the body cannot keep mercury from spreading throughout the most susceptible of our population, the very young, the very old and the very ill.

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Subscribed and sworn to by Boyd E. Haley, Ph.D. this May 30, 2006.

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Robert E. Reeves, Notary Public
State At Large
Commonwealth of Kentucky
My commission expires Oct 6,2006

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