Critique on SCENIHR Preliminary Report
"The safety of dental amalgam and alternative dental restoration materials for patients and users"

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The author takes full personal responsibility for the contents of this document.

The International Academy of Oral Medicine & Toxicology – Europe (IAOMT-Europe) is dedicated to promote the health of the public at large by:

- Accumulating and Disseminating Scientific Information
- Funding Relevant Research and Education
- Provide Advisory Services.

The membership of the International Academy of Oral Medicine & Toxicology – Europe, or IAOMT, is restricted to Scientists, Medical Doctors and Dentists.

The Scientific Advisory Committee of the IAOMT issued the following Position Statement:

In spite of its long term usage, accumulated scientific evidence now clearly shows that dental amalgam (silver-mercury fillings) expose dentists, dental staff members and dental patients to substantial amounts of mercury in vapor, particulate and other forms. Chronic exposure to mercury, even in minute amounts, is known to be toxic and poses risks to human health, we must therefore conclude that dental amalgam is not a suitable material for dental restorations.

Due to mercury's inhibiting influence on the growing brain, it is incompatible with current science and experimental knowledge to endorse or condone the use mercury containing fillings - especially in children and women of childbearing age.

Physicians and dentists should, where patients are suffering from pathological states and diseases of unclear causation, consider whether exposure to mercury released from amalgam fillings may be a contributory or exacerbating factor in such adverse health conditions.

Governments of several countries have placed restrictions and/or issued advisories against the use of mercury in dental fillings - particularly in children and pregnant women. Recently a joint panel of FDA scientific advisors (1) rejected an FDA whitepaper's assurances of the safety of dental amalgam.

In light of the above mentioned facts, the International Academy of Oral Medicine & Toxicology and its Scientific Advisory Board (2) urge the dental profession to join the rest of the medical profession and abandon the use of mercury.

References:
(1) Joint Meeting of the Dental Products Panel of the Medical Devices Advisory Committee of the Center for Devices and Radiological Health, and the Peripheral and Central Nervous System Drugs Advisory Committee of the Center for Drug Evaluation and Research (September 6&7, 2006).

(2) Scientific Advisory Committee

The scientific activities of the IAOMT are overseen by an advisory committee composed of world leaders in biochemistry, toxicology and environmental medicine. They are:

Boyd Haley, PhD, FIAOMT, chairman. Professor and former Chairman of the Department of Chemistry, University of Kentucky; permanent member, NIH Biomedical Sciences, Study Section.

Thomas Burbacher, PhD, Associate Professor of Environmental and Occupational Health Sciences, Research Affiliate, Center on Human Development and Disability, Director, Infant Primate Research Laboratory, University of Washington Center for Human Development and Disability.

Louis W. Chang, PhD, Emeritus Professor of Pathology, University of Arkansas for Medical Sciences, Founding Director of the Taiwan Division of Environmental Health & Occupational Medicine.

H. Vasken Aposhian, PhD, Professor of Cellular and Molecular Biology, Professor of Pharmacology, University of Arizona, College of Medicine.

Herbert Needleman, MD, Professor of Child Psychiatry and Pediatrics, University of Pittsburgh School of Medicine.

Maths Berlin, PhD, Advisor to this Committee. Professor Emeritus of Environmental Medicine, Medical Faculty of Lund, Sweden. Dr. Berlin was the chairman of two World Health Organization conferences on mercury exposure in 1991.

IAOMT-Europe Response to SCENIHR report.

The response is deliberately kept brief so as to make it user friendly whilst still being accurate, informative and to the point.
All the issues raised by SCENIHR could have been refuted in massive scientific detail but even in this truncated form, the IAOMT Response is more than adequate in exposing the gross deficiencies of the SCENIHR report.

The SCENIHR report is grossly inadequate as a scientific document.
The report has serious omissions.
The report is contradictory.
The data that is reviewed is interpreted incorrectly.
The report confuses Toxicity and Allergy.
It makes assumptions and forms opinions to draw unwarranted conclusions even with the very limited data it reviews.
The report ignores the synergistic effect of mercury with lead, the effects of gender, diet and certain antibiotics in increasing the uptake and toxicity of mercury from dental amalgam fillings.

The conclusion drawn by the committee that dental amalgam is an adequate dental restorative material is false and will be shown to be false in this document.

This will be detailed further on in this document.

The SCENIHR report is best described as a **Fishing Expedition** rather than a scientific document; the omissions speak louder than the inclusions.

The only logical interpretation is that the committee has selected data to support a predetermined conclusion as to the safety of dental amalgam.

The SCENIHR report does nothing to enhance the reputation of its authors and experts and will only serve to bring the dental profession into disrepute in the eyes of the public.
No committee, no matter how prestigious, can turn a toxic substance, i.e. dental amalgam, into a harmless substance by decree or pseudo-scientific waffling as in this case.
They just make themselves look foolish and diminish us all by their actions.

The references at the back of the SCENIHR report are in alphabetical arrangement. The report refers to a limited number of references by authors name only.
All this makes it in most cases impossible to connect the claims made by the report to any published science.
Some of the data that is cited in the report is published only in dental trade journals after publication was refused in scientific journals on peer review as being unsatisfactory.

The Lisbon study on Portuguese orphans is a good example.
Not only was this study unethical, the data published contradicts the conclusion. Such data has no place in a supposedly scientific document as this report claims to be.

Much of the data referenced by SCENIHR is flawed and scientifically inadequate. This is illustrated by:

Mutter J et al, Amalgam Studies: Disregarding the basic principles of mercury toxicity. Int. J. Hyg. Health 207 (2004) 391-397.

A major omission in the report is the failure to acknowledge the most prestigious researchers into potential amalgam toxicity of the last 25 years.
Such names as Prof**. Boyd Haley, Prof. H.V. Aposhian, Prof. M Vimy, F. Lorscheider, A. Summers, G. Richardson and J. Pleva and M. Hansen** are entirely absent from the SCENIHR report.

This is equivalent to writing a history of space exploration but not mentioning NASA or the moon landings.

The scientists named have made the most recent, important and significant advances in the knowledge and scale of mercury toxicity emanating from amalgam fillings but their contribution have been entirely ignored by the members of the committee.
Whether this is due to gross oversight or deliberate policy as the peer reviewed published science of these scientists are diametrically opposed to the position taken by the committee is for posterity to judge.

Another omission in the report is the effect of dental amalgam on periodontal disease. This is given a cursory mention only on pages 26 and 39.
The deleterious effect of periodontal disease, the commonest disease of the human race, on general health is now well documented. To omit the causal link between dental amalgam to periodontal disease and therefore to general health effects is a gross omission rendering the SCENIHR report usefulness to a minimum.

The author is indebted to Prof. M. Vimy for the following:
“Mercury from dental amalgam restorations causes periodontitis which probably contributes to many systemic health problems such as coronary heart disease, premature and low birth weight babies, stroke and complications in diabetes to name but a few.

Mercury from dental fillings causes periodontitis:

In 1957, Zander (JADA, 55:11-15) reported "materials used in restorative dentistry may be a contributing factor in gingival disease."

In 1961, App (J Prosth Dent 11:522-532) suggested that there was greater chronic inflammation around amalgam sites than non-amalgam areas.

In 1964, Trott and Sherkat (J CDA, 30:766-770) showed that the presence of amalgam correlates with gingival disease. Such disease was not present at contralateral amalgam-free sites.

In 1969, Sanches Sotres et al (J. Periodo. l40: 543-546) confirmed Trott and Sherkat findings.

In 1972, Turgeon et al. (J CDA 37:255-256) reported the presence of very significant erythema around amalgam restorations that was not present at control non-amalgam sites.

In 1973, Trivedi and Talim (J. Prosth. Dentistry, 29:73-81) demonstrated that 62.5% of amalgam sites have inflammatory periodontal tissue reaction.

Thus, as early as 1973, a case can be made that the presence of mercury-amalgam results in chronic inflammation and bleeding in the gingival tissue adjacent to it; in other words, in situ amalgam produced chronic Gingivitis.

In 1974, Freden et al. (Odontol. Revy, 25: 207-210) showed that gingival biopsy material from sites not adjacent to amalgam had 1-10 :g mercury/gram of tissue (mean=3); whereas, gingival biopsy sites near amalgams contained 19-380 :g mercury/gram of tissue (mean=147).

In 1976, Goldschmidt et al (J. Perio. Res., 11:108-115) demonstrated that amalgam corrosion products were cytotoxic to gingival cells at concentrations of 10-6; that is, micrograms/gram of tissue.

In 1984, the year of the NIDR/ADA Workshop, Fisher et al (J Oral Rehab, 11:399-405) reported that at amalgam sites alveolar bone loss was very pronounced and statistically significant as compared to control non-amalgam sites! In other words, in situ amalgam produces chronic Periodontitis.

This suggests that placing mercury fillings leads to a dentist-induced disease, periodontal disease, which the same dentists then treat. This is iatrogenesis.

There is sound scientific evidence supporting a link between amalgam fillings and systemic diseases or chronic illness is incorrect. Periodontal disease is one of the most prevalent chronic diseases in Man, and mercury fillings contribute significantly!

Such statements by as made by the SCENIHR report suggest that the authors of the report and their advisors may be knowingly disinforming the public through

the media or they lack an understanding of the scientific research about mercury release from amalgam."

Murray J. Vimy DMD
Clinical Associate Professor
Faculty of Medicine,
University of Calgary

Additional references found by the author that support the position that mercury from dental amalgam causes periodontitis.

Rechmann, P. LAMMS and ICP-MS Detection of Dental Metallic Compounds in Not-discoloured Human Gingiva. J Dent Res., 71SI:599, A672, 1992.

Siblerud, RL. The Relationship Between Mercury From Dental Amalgam and Oral Cavity Health. Ann Dent, 49(2):6-10, 1990.

Traub, EF; Holmes, RH. Dermatitis and Stomatitis from the Mercury of Amalgam Fillings. Arch Derm Syph., 38:349-57, 1938.

Ziff, MF. Documented Clinical Side-Effects to Dental Amalgam. Adv Dent Res, 6:131-4, 1992.

DOES PERIODONTITIS CAUSE SERIOUS HEALTH PROBLEMS?

Periodontal disease affects an estimated 80% of the population over the age of 35. That both mercury fillings and poor oral health cause periodontitis is now established fact.
Mounting research studies indicate periodontitis contributes to a wide array of systemic illnesses such as heart disease, stroke, diabetes, premature and low-weight births, lung disease and arthritis. Thus not only is mercury a cause of a serious dental problems but it also many very well contribute to an array of diseases that cause so much human suffering.

The Centers for Disease Control and Prevention (CDC) reports that researchers have uncovered potential links between periodontal disease and many serious health conditions. The reason, according to many medical experts, is that the bacteria that contribute to gingivitis and periodontitis provoke inflammation or infection, which can trigger certain diseases. Periodontal disease may even aggravate or worsen existing health conditions. [1]

[1] *Life Extension Magazine, April 2006 "Preventing Disease by Improving your Oral Health" By Matthew Solan*

HEART DISEASE. Researchers have discovered that people with periodontal disease are much more likely to suffer from coronary artery disease than those without the disease. A 2004 study in the Journal of Periodontology found that 91% of 108 patients with cardiovascular disease suffered from moderate to severe periodontitis, compared to 66% of the non-cardiac patients.[2] Periodontal treatment are associated with reductions in C-reactive protein.[3] Although more studies are needed, these findings suggest that treating periodontal disease not only boosts oral hygiene, but also improves several measures of cardiovascular health. The best and most effective treatment is the removal of dental amalgam filings.

STROKE: The presence of periodontal disease also may increase risk of stroke. Previous research found that the severity of periodontal disease is proportionally related to the amount of arterial plaque located in the carotid arteries, the two major arteries on each side of the neck that supply blood to the brain. Blockage here may reduce blood flow to the brain or advance blood clots, which can lead to a stroke. A 2005 study from the University of Minnesota found a direct link between high levels of bacteria that cause gum disease and thickness of the carotid arteries. This research stands out as the first to link atherosclerosis with the type of bacteria that causes gum disease, and not with other oral bacteria.[4]

DIABETES CONTROL: Studies suggest that periodontal disease may adversely affect blood sugar control in people with diabetes. Controlling periodontal infection in diabetic individuals has been found to help improve blood sugar control, as measured by a decreased demand for insulin and decreased levels of hemoglobin A1C, a marker of long-term blood sugar control. Measures to combat complications of diabetes, especially

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[2]*Geerts SO, Legrand V, Charpentier J, Albert A, Rompen EH. Further evidence of the association between periodontal conditions and coronary artery disease. J Periodontol. 2004 Sep;75(9):1274 80.*

[3]*Elter JR, Hinderliter AL, Offenbacher S, et al. The effects of periodontal therapy on vascular endothelial function: a pilot trial. Am Heart J. 2006 Jan;151(1):47.*

[4]*Desvarieux M, Demmer RT, Rundek T, et al. Periodontal microbiota and carotid intima media thickness: the Oral Infections and Vascular Disease Epidemiology Study (INVEST). Circulation. 2005 Feb 8;111(5):576 82.*

periodontitis and gingivitis, may be important in reducing additional systemic inflammatory burden, thus potentially preventing other conditions such as cardiovascular disease.[5]

PREMATURE AND LOW-WEIGHT BIRTHS. New findings indicate that gum disease can affect the health of pregnant women and their unborn children. A University of Chile study found that women with gingivitis were at higher risk of delivering premature infants and low-weight babies than women with healthier gums. The likely reason is that periodontitis or gingivitis bacteria contribute to an inflammatory response of the placental membrane, which may induce preterm labor. Periodontal treatment reduced the risk of premature and low-weight births in women with pregnancy-related gum disease.[6]

LUNG DISEASES Some evidence suggests that periodontal disease may contribute to lung infections like pneumonia, or may worsen chronic conditions such as emphysema. Experts believe this may be due to oral bacteria that move into the airways of the throat and lungs.[7] Poor oral health may also accompany poor joint health.

ARTHRITIS People with moderate to severe periodontitis experience an increased risk of rheumatoid arthritis. [8] Gum disease is also present in many patients who suffer from juvenile idiopathic arthritis. [9]

[5] Southerland JH., Taylor GW, Offenbacher S, Diabetes and Periodontal Infection: Making the Connection, Clin. Diabetes 2005 23: 171-178.

[6] Lopez NJ et al. Periodontal therapy reduces the rate of preterm low birth weight in women with pregnancy associated gingivitis. J Periodontol. 2005 Nov;76(11 Suppl):2144 53.

[7] Scannapieco FA, Ho AW. Potential associations between chronic respiratory disease and periodontal disease: analysis of National Health and Nutrition Examination Survey III. J Periodontol. 2001 Jan;72(1):50 6.

[8] Mercado FB, Marshall RI, Bartold PM. Inter relationships between rheumatoid arthritis and periodontal disease. A review. J Clin Periodontol. 2003 Sep;30(9):761 72.

[9] *Welbury RR, Thomason JM, Fitzgerald JL, et al. Increased prevalence of dental caries and poor oral hygiene in juvenile idiopathic arthritis. Rheumatology (Oxford). 2003 Dec;42(12):1445 51.*

Comment on the abstract:
Para 2; The assertion that amalgam is an effective restoration material is certainly open to discussion.
The former director of the NIDR, Harold Loew, stated that amalgam should not be the first material to restore a tooth due to amount of unnecessary tooth destruction such a filling would require for placement.

That the use of amalgam is reducing is correct, many patients demand amalgam removal for health reasons rather than purely cosmetic ones. At the time of writing 54% of dentists in the USA no longer use amalgam and the author has not used amalgam for over 25 years.

Para 3; To say that mercury from dental amalgam causes a low amount of local effects which are easily managed, ignores the periodontal disease connection and therefore this assertion is false.

To say that amalgam does not pose a risk for neurological, psychiatric or systemic effects is false and will be dealt with in detail later on in this document. (The authors own published practice statistics reveal a different picture)

That the main exposure to mercury occurs during the placement or removal of amalgam fillings is not true.
Mercury vapour is emitted from amalgam fillings for the life of the filling and the amount of vapour emitted over a period of years exceeds the exposure at placement and removal.

To say that no studies exist to show that dental personnel suffer “classical signs of mercury intoxication” is incorrect. Studies revealing increased tremor and reduced intellectual capacity have been published and considerable data has been collected on the physiopathology exhibited by dental personnel i.e. 85% of amalgam using dentists have altered porphyrin profiles for instance indicating reduced capacity to make heme.

Para 4; The reference to allergies of different materials is interesting. Generally in the report, the authors confuse allergic response with toxic responses. Patch Testing is an unreliable method of detecting allergic response in comparison with Lymphocyte Transformation or the MELISA test.

They also make the cardinal error of not realising that it is the **RETENTION** of mercury in the individual that is the problem. Patients with high urine and blood

levels show they are excreting mercury whilst patients with low blood and urine levels exhibit the fact that they cannot excrete mercury.

Another failure of the report is the published Risk Assessment Data showing that composites are 200x safer than amalgam.

Richardson GM Assessment of exposure and Risk from Components and Degredation products of Composite Resin Dental Materials. Human Ecol Risk Assess. 3(4):683-97, 1997

No where in the report is the study of Risk assessment of Dental Amalgam

The definitive study was by Richardson in 1995
In this study, commissioned by Health Canada, Richardson showed that, based on neurological data, the maximum number of fillings allowed before effects would be evident was as follows:

Ages 3-11	0 to 1 amalgam filling
Ages 12-19	1 to 3 amalgam fillings
Ages 20+	2 to 4 amalgam fillings

Richardson, GM. Assessment of Mercury Exposure and Risks From Dental Amalgam; Final Report, Medical Devices Bureau, Environmental Health Directorate, Health Canada, 18 Aug 1995.

Richardson, GM. A Monte Carlo Assessment of Mercury Exposure and Risks from Dental Amalgam. Human Ecolog Risk Assessment, 2(4):709-61, 1996.

The conclusion of the abstract that dental amalgam is adequately ensured by the use of dental amalgam is therefore false.

Critisism of the body of the report.
Executive summary:
P8. Oral Lichen Planus is dismissed as readily managed allergic response to dental amalgam. What the authors fail to reveal is that Oral Lichen Planus is a proven precancerous condition and is caused by mercury from amalgam fillings.

Paolo D Pigatto et al. Oral Lichen Planus: Mercury and its Kin: Arch Derm. 141(11); 1472-1473; 2005

The assertions about Alzheimer's, Parkinsons etc are dealt with later on in this document.

P9. To state that amalgam restorations pose no health risk to pregnant women or the foetus is incorrect. See later in document.

Terms of Reference:
Dental Amalgam.
Is there scientific evidence that supports a link between amalgam and allergic reactions, neurological disorders or other health disorders?

IAOMT-Europe Response. Unequivocally yes.

In view of the above, is the use of dental amalgam safe for patients and users, i.e dental health professionals? Are certain populations particularly at risk, e.g. pregnant women or children?

IAOMT-Europe Response. Unequivocally yes.

The author is indebted to Prof. B. Haley for the following to illustrate scientifically the IAOMT response.

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

By Boyd E. Haley, Ph.D., Professor, Department of Chemistry, University of Kentucky, Lexington, KY

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]

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 and

European populations. 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).

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.

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.

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.
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).

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.
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]

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.

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.

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.

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)

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 pathways and the entry of low doses of mercury vapor into the nervous system.[6, 14]

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 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.

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.

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.

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.

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.
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.

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.

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 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.

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.

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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Alternative Materials:

Is there scientific evidence that supports a link between alternative materials and allergic reactions, neurological disorders or other health disorders?

In view of the above, is the use of alternative dental materials safe for patients and users, i.e dental health professionals? Are certain populations particularly at risk, e.g. pregnant women or children?

IAOMT-Europe Response:
All dental materials pose a hazard. However, as previously stated in this critique, Composite Resins are shown to be safer than dental amalgam by a factor of 200. (Richardson G)

Is Dental Health equally assured by dental amalgam and alternatives?

IAOMT Response:
The answer is no.

From a purely functional point of view, amalgam restorations are inadequate.
Over time mercury amalgam fillings corrode.
This corrosion leads to expansion of the material an inevitable consequence of which is that the tooth exhibits cracks leading to fracture of the tooth.
Below is an example of a tooth directly after amalgam filling removal. Note the cracks in the tooth produced by the expansion of amalgam due to corrosion.

This is the inevitable consequence of using amalgam as a dental restorative material.

The connection between Periodontal Disease and amalgam restorations is well established.
If other metals are in the mouth, i.e. gold alloys, palladium/platinum alloys, titanium screws or implants, nickel, cobalt, chrome etc. Faradays Law will ensure that amalgam becomes the negative electrode and corrosion rates, i.e. mercury release rates, are accelerated by up to a factor of 10.
An excellent review of this subject is*:*

M Hanson & J Pleva The dental amalgam issue. A Review; Cellular and Molecular life Sciences Vol 47,9-22; No. 1 Jan 1991

P16 Para 3 The release of mercury vapour from amalgam fillings is easily measurable in clinical practice. Such direct measurements are more reliable than estimating release from partial pressures of mercury from amalgam fillings. Mercury Vapour meters are commercially available, reliable and relatively inexpensive.
Para 4 “corrosion of restorations will occur at a very low rate”. This is unscientific. What is low or what is high?
Numbers are required in science.
Once again there is considerable data available as to the amount of mercury released from amalgam by corrosion. http://art-bin.com/art/hanson_en.html is a comprehensive article on this subject by Mats Hanson PhD.

P17 Para 4 “Exposure to mercury is difficult to measure.
Indications for exposure are obtained by blood and urine measurements”

IAOMT Response: This is nonsense.
Mercury vapour from tooth amalgam fillings is easily measured in both practice and the laboratory.
Vimy’s work estimated 10 mcg average absorbtion from amalgam fillings.
WHO paper 118 states that dental amalgam is the greatest dource of mercury exposure.

Vimy, MJ; et al. Estimation of Mercury Body Burden from Dental Amalgam: Computer Simulation of a Metabolic Compartmental Model. J Dent Res, 65(12):1415-9, 1986.

Vimy, MJ; Lorscheider, FL. Dental amalgam mercury daily dose estimated from intro-oral vapor measurements: A predictor of mercury accumulation in human tissues. J Trace Elem Exper Med, 3:111-23, 1990.

WHO. Environmental Health Criteria 118: Inorganic Mercury, pp, 28-33, 84- 113, Geneva, 1991.

90% of excreted mercury is by the fecal route.

Mackert J r et al Mercury Exposure from dental amalgam fillings. Absorbed dose and potential for adverse effects. Crit Rev Oral Biol. 8:410-436, 1997

The fact that the committee decided to use blood and urine levels shows a basic lack of understanding of mercury biochemistry and a failure to consult the literature.
Therefore to use urine levels as a marker when it represents only 10% of the mercury load with the compounding factor of retention of mercury by altered kidney function re Prof Haley’s comment, makes this assertion patently false.
The diagnostic invalidity of using Blood or Urine as a measure of mercury exposure is illustrated below.

American Dental Association: Workshop Reaffirms Dental Amalgam Safety. ADA News. Pp. 1, 5-8, 30 July 1984.

Environmental Protection Agency (USA): Health Effects Update, Health Issue Assessment, Final Report. EPA-600/8-84-019F. Office of Health and Environmental Assessment, Washington, D.C. 1984.

Goldwater, LJ; Ladd, AC; Jacobs, MB: Absorption and Excretion of Mercury in Man: VII, Significance of Mercury in Blood. Arch Environ Health, 9: 735-41,1964.

Jacobs, MB; et al. Absorption and Excretion of Mercury in Man: IV. Significance of Mercury in Urine. Arch Environ Health, 9:454-63, 1964.

Langan, DC; Fan, PL; Hoos, AA: The Use of Mercury in Dentistry: A Critical Review of the Recent Literature. JADA, 115:867-879, 1987.

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National Institute of Dental Research/American Dental Association: Workshop: Biocompatibility of Metals Used in Dentistry. JADA, 109, 469-471, 1984.

National Institute for Occupational Safety and Health (USA, NIOSH): A Recommended Standard for Occupational Exposure to Inorganic Mercury. NTIS, No. PB-222 223, 1973.

Satoh, H; et al. Selective determination of elemental mercury in blood and urine exposed to mercury vapor in vitro. J Appl Toxicol, 1(3):177-81, 1981.

World Health Organization): Environmental Health Criteria, Vol. 118: Inorganic Mercury. Pg. 61. WHO, Geneva, Switzerland, 1991.

Therefore the committees conclusions drawn on blood and urine levels of mercury are invalid.

P19 “There is no evidence that biotransformation of amalgam-derived mercury takes place intra-orally in association with bacterial activity”

IAOMT-Europe Response:
Any review of the literature will show this assertion to be false.
Mercury reacts with thiol compounds produced by anaerobic bacteria in the mouth to produce super-toxins.
These are compounds that are organically soluble. The brain and nervous system are the ones that are the most hydrophobic or the most susceptible to attack. Whenever organic thiols such as methyl thiol--CH3SH—are made, for those who know a little chemistry, that reacts with mercury and then methyl thiol mercury is formed. This drags the mercury into the central nervous system and allows it to penetrate deeper into the body and to become more capable of killing enzymes that are necessary for health.
These compounds are in the same category as the mustard gases that were used in World War I.

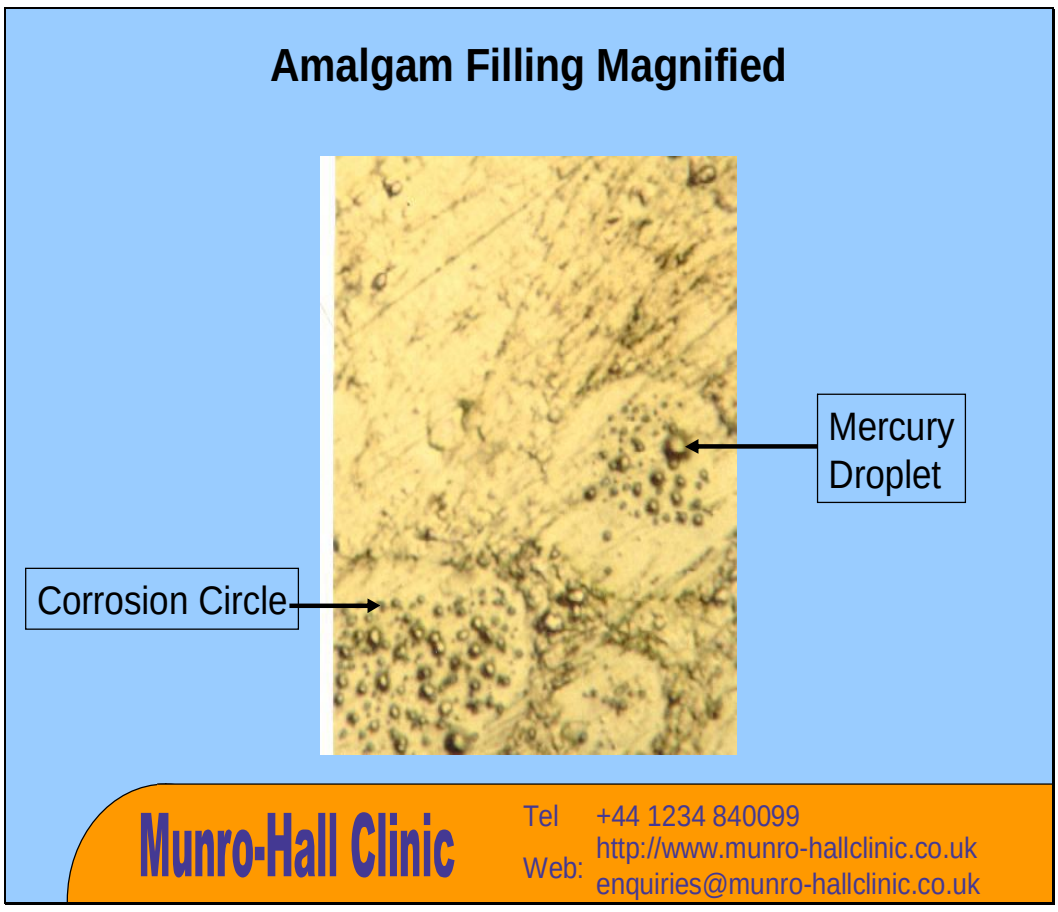
Carlson, J., Larsen, J.T., and Edlund, M.-B.: Peptostreptococcus micros has a uniquely high capacity to form hydrogen sulfide from glutathione. Oral Microbiol. Immunol., 8: 42-45, 1993

Debelian, G.J., Olsen, I., and Tronstad, L.: Systemic diseases caused by oral microorganisms. Endod. Dent. Traumatol., 10: 57-65, 1994

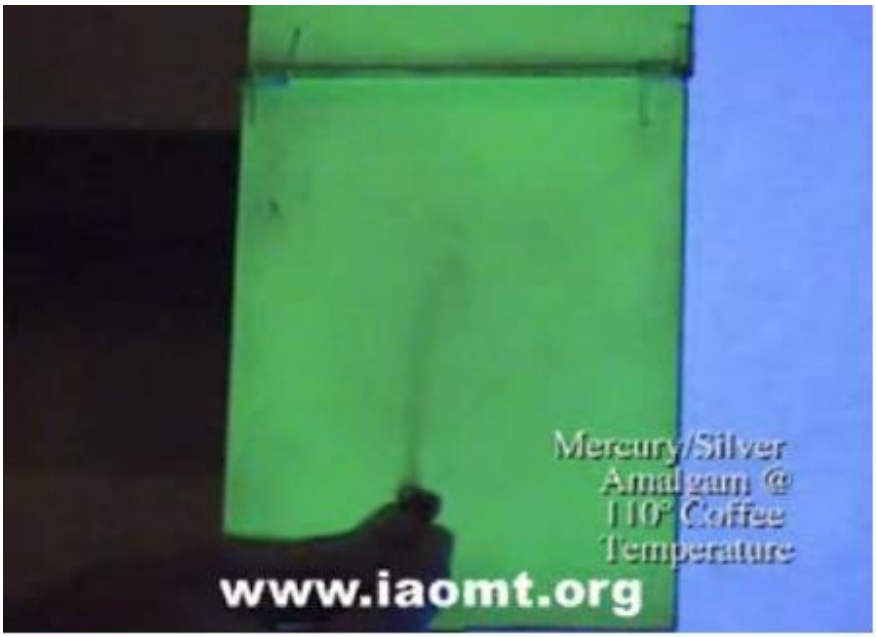
De Boever, E.H., De Uzeda, M., and Loesche, W.J.: Relationship between volatile sulfur compounds, BANA-hydrolyzing bacteria and gingival health in patients with and without complaints of moral malodour. J. Clin. Dent., 4: 114-119, 1994

Duhr, E.F., Pendergrass, J.C., Slevin, J.T., and Haley, B.E.: HgEDTA complex inhibits GTP interactions with the E-site of the brain ß-tubulin. Toxicol. Appl. Pharmacol., 122: 273-280, 1993 .

The two pictures are a graphic illustration of mercury release from amalgam fillings.



This dental amalgam filling is emitting the mercury. The amalgam filling is 22 years old.



Tooth brushing, eating and drinking will stimulate the release of mercury.

Since no studies have been done on the actual emission of mercury from amalgams in a controlled environment, the IAOMT co-operated with Prof. Haley's Lab and conducted a controlled experiment.

This is the only study of this kind ever performed.

The results are available on the IAOMT website
A single spill of amalgam was mixed by 9 dentists using 3 different manufacturers of amalgam. The samples had approx 1 square cm area.
It was found that after 3 months in distilled water, the mercury coming of these samples varied between 5 mcg/day to 20mcg/day.
This was unstimulated, any mild abrasion such as tooth brushing or raise in temperature will increase the mercury release by a factor of 10 for 90 minutes after stimulation.
At a level of 5mcg/day, it would take 182 years to release all the mercury. At a level of 20mcg/day it would take 45 years to release all the mercury.
This illustrates the chronic nature of mercury release from amalgam fillings over many years.
Thus an individual with 5 small amalgam fillings could expect to receive a minimum of 20mcg/day. Should this individual have a cast (Gold etc.) restoration as well, the rate of release would be between 4 and 10 times higher than this.

Since such a study is relatively easy and inexpensive, the question arises as to why it has not been done before?

Could it be that the results would not help the pro amalgam lobby?
The following also illustrate the quantity of mercury released from amalgam fillings.

Hanson M. & Pleva J., "The dental amalgam issue. A review." Experientia 47, 1991, 9-22

Vimy MJ et a;l Estimation of mercury body burden from dental amalgam. Computer Simulation. J. Dent. Res. 65(12);1415-19 1986

Vimy et al;Dental amalgam mercury daily dose estimated from intra-oral vapour measurements. J. Trace Elements Exper. Med. 3:111-23 1990

Where does all this mercury go?

Had the committee read the research of Lorscheider and Vimy placing radioactive mercury in sheep and primates, they would have realized that mercury is deposited in all major organs of the body, concentrating in the brain, kidney, heart and fetus.

Hahn LJ ; Kloiber R; Vimy MJ; Takahashi Y; Lorscheider F; Dental "silver" tooth fillings: a source of mercury exposure revealed by whole-body image scan and tissue analysis. FASEB J. 3:2641-2646; 1989

Vimy, MJ; Takahashi, Y; Lorscheider, FL Maternal-fetal distribution of mercury (203 Hg) released from dental amalgam fillings the American Physiology Society 0363-6119/90 R939-945

P20 mentions dental personnel having higher levels of kidney disorders than controls, but this was dismissed as of no consequence despite the wealth of data concerning the pathology of mercury on the kidney referred to on P22 & P23. This opinion has no scientific justification and is plain contradictory.
Significant adverse effects on the behaviour, mood, cognition and motor function have also been published.

D. Echeverria, H.V. Aposhian, J.S. Woods, N.J. Heyer, M.M. Aposhian, A.C. Bittner Jr., R.K. Mahurn, and M. Cianciola, "Neurobehavioral effects from exposure to dental amalgam Hg: new distinctions between recent exposure and Hg body burden," FASEB Journal 12, 971-980 (1998).

Effects of Low-Level Exposure to HgE Among Dentists. Neurotoxicol Teratol,17(2):161-8 (1995); Shapiro, I.M., et al., Neurophysiological and neuropsychological function in mercury-exposed dentists. The Lancet 1, 1147-1150 (1982);

Uzzell, B.P., et al., Chronic low-level mercury exposure and neuropsychological functioning. J of Clin and Exper Neuropsych. 8,

581-593.

P22 states that no animal studies have been done with animals with elemental mercury inhalation.

This is false.

Duhr, E; Pendergrass, C; Kasarskis, E; Slevin, J; Haley, B. Hg2+ Induces GTP-Tubulin Interactions in Rat Brain Similar to Those Observed in Alzheimer's Disease. Federation of American Societies for Experimental Biology (FESAB). 75th Annual Meeting. Atlanta, GA 21-25 April 1991. Abstract 493

In this experiment the rats breathed mercury vapour for 4 hours a day up to 28 days at a dose of between 250 and 300mcg/daily. Identical lesions were seen in the brain as in human Alzheimer's brains.

This should be enough data to ensure that, as a precautionary measure, the use of amalgam be ceased immediately.

P25 **Occupational Exposure.**
Occupational exposure is assumed to be only for 8 hours a day. To compare this to the 24 hour per day exposure to mercury from amalgam fillings and to arbitrarily assume that the mercury from amalgam is released at a rate 20 times lower is strange. Especially as the data suggests a far higher release of mercury from amalgam fillings than the report allows for.
The mercury from fish consumption is a red herring. Sorry about the pun. This is comprehensively dealt with previously by Prof. Haley.

P27 The data revealed about Lichen Planus is in contradiction to the P8 where Lichen Planus was dismissed as easily managed.

P28 States there is no link between mercury and kidney disease despite the dental personnel with a higher rate of kidney dysfunction mentioned on P20. The link between mercury and kidney dysfunction has been discussed earlier in this document.

P29. **Alzheimer's disease.** No link according to the report but any review of the literature will reveal extensive connections between mercury and Alzheimer's. The work by Prof Haley, revealed earlier in this document, certainly shows a link any layman with common sense could see between mercury and Alzheimer's. Again, this has been discussed and referenced earlier in this document.
For the sake of space only a small selection of references are shown.

Duhr, EF; et al. HgEDTA complex inhibits GTP interactions with the E site of Brain B-Tubulin. Toxicol Appl Pharmacol, 122:273-80, 1993.

Ehmann, WD; et al. Brain Trace Elements in Alzheimer's Disease. Neurotoxicol, 7(1):197-206, 1986.

Haley, B. Khatoon, S; et al. GTP binding to the b-subunit of tubulin is greatly reduced in Alzheimers Disease. ASBC 1987.

Haley, B. Duhr, E; et al. Low level HgEDTA complex specifically blocks [32P]8N3GTP interaction with human brain tubulin. ASBMB/AAI, 1990.

Haley, B. Duhr, E; et al. Hg2+ induces GTP-tubulin interactions in rat brain similar to those observed in Alzheimer's Disease. FASEB A493, 1991.

Haley, B. Bunnersen, D; et al. Detection of glutamine sythetase in the CSF of Alzheimer's diseased patients: A potential diagnostic biochemical marker. Soc Neuroscience, 1992.

Multiple Sclerosis.

Once again it is the omissions that speak the loudest. Siblerud's work for one. There is no science for a causal link but there is evidence to suggest one. The authors own experience in practice is that if amalgam is removed within 5 years of onset, the chances of recovery are good.

Ganser, AL; Kirschner, DA. The interaction of mercurials with myelin: Comparison of in vitro and in vivo effects. Neurotoxicol, 6(1):63-77, 1985.

International Labor Organization (ILO). Encyclopaedia of Occupational Health and Safety, 3rd Ed., Vol. 2. ED: Parmeggiani, L., pp. 1332-59 1983.

Siblerud, RL. A comparison of mental health of multiple sclerosis patients with silver/mercury dental fillings and those with fillings removed. Psychol Rep, 70:1139-51, 1992.

Siblerud, RL. Evidence That Mercury From Silver Dental Fillings May Be An Etiological Factor in Multiple Sclerosis. Sci Total Environment, 142:191-205, 1994.

Windebank, AJ. Specific Inhibition of Myelination by Lead in vitro; Comparison with Arsenic, Thallium, and Mercury. Exp Neurol, 94(1):203-12, 1986.

Parkinson's disease.

There is no direct causal link, but there is evidence of a connection between Parkinson's and mercury.

Finkelstein, Y; et al. The Enigma of Parkinsonism in Chronic Borderline Mercury Intoxication, Resolved by Challenge With Penicillamine.

Neurotoxicology, 17(1):291-5, 1996.

International Labor Organization (ILO). Encyclopaedia of Occupational Health and Safety, 3rd Ed., Vol. 2. ED: Parmeggiani, L., pp. 1332-59 1983
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Ngim, C-H; Devathasan, G. Epidemiologic study on the association between body burden mercury level and idiopathic Parkinson's Disease. Neuroepidemiol, 8:128-41, 1989.

Parasthesia
The work on dental personnel shows neurological effects which the report claims does not exist.

D. Echeverria, H.V. Aposhian, J.S. Woods, N.J. Heyer, M.M. Aposhian, A.C. Bittner Jr., R.K. Mahurn, and M. Cianciola, "Neurobehavioral effects from exposure to dental amalgam Hg: new distinctions between recent exposure and Hg body burden," FASEB Journal 12, 971-980 (1998).

Autism
Mercury in vaccines has long been suspected as a factor in autism.
A study by a parents group, Generation Rescue by an independent research company used by industry of over 17,000 children using CDC protocols showed that vaccinated children were 155% more at risk from autism than unvaccinated children, See http://www.generationrescue.org/survey.html.
This is contrast to the other studies financed by the vaccine industry.
One can draw one's own conclusions.
Referring back to Prof Haley's work we see that Autism is sex related and can be explained by difference in the sex hormones and the inability to excrete mercury.
Hence low levels of mercury seen in these individuals

Reduced Levels of Mercury in First Baby Haircuts of Autistic Children International Journal of Toxicology Dr. Amy S. Holmes, Mark F. Blaxill, Boyd E. Haley, Ph.D. March 14, 2003

Porphyrinuria in Childhood Autistic Disorder: Implications for Environmental Toxicity Toxicology and Applied Pharmacology, 2006. Robert Nataf, Corinne Skorupka, Lorene Amet

A Case Control Study of Mercury Burden in Children with Autism Spectrum Disorder. Journal of American Physicians and Surgeon, 2003. James Adams, PhD [Arizona State University].

A Case Series of Children with Apparent Mercury Toxic Encephalopathies Manifesting with Clinical Symptoms of Regressive Autistic Disorder Journal of Toxicology and Environmental Health, 2007 David A. Geier, Mark R. Geier

Attention-deficit hyperactivity disorder and blood mercury level: a case-control study in chinese children Neuropediatrics, August 2006 P.R. Kong [Department of Pediatrics and Adolescent Medicine, The University of Hong Kong].

Psychological conditions
According to the report, no connection between mercury from amalgam fillings and psychological conditions.
Since the measure of mercury exposure was urine levels, the result can be discounted due to the irrelevance of urine levels as a measure of body burden. This is the point to introduce statistics collected by Mats Hansen see www.iaomt.org.

FTFD SYMPTOM ANALYSIS OF 1569 PATIENTS

% of total SYMPTOM No.		Total No.	No. improved or cured	% of cure or improvement
14%	ALLERGY	221	196	89%
5%	ANXIETY	86	80	93%
5%	BAD TEMPER	81	68	89%
6%	BLOOD PRESSURE PROBLEMS	99	53	54%
5%	CHEST PAINS	79	69	87%
22%	DEPRESSION	347	315	91%
22%	DIZZINESS	343	301	88%
45%	FATIGUE	705	603	86%
15%	GASTROINTESTINAL PROBLEMS	231	192	83%
8%	GUM PROBLEMS	129	121	94%
34%	HEADACHES	531	460	87%
3%	MIGRAINE HEADACHES	45	39	87%
12%	INSOMNIA	187	146	78%

10%	IRREGULAR HEARTBEAT		159	139	87%	
8%	IRRITABILITY	132	119	90%		
17%	LACK OF CONCENTRATION		270	216	80%	
6%	LACK OF ENERGY	91	88	97%		
17%	MEMORY LOSS	265	193	73%		
17%	METALLIC TASTE	260	247	95%		
7%	MULTIPLE SCLEROSIS	113	86	76%		
8%	MUSCLE TREMOR	126	104	83%		
10%	NERVOUSNESS	158	131	83%		
8%	NUMBNESS ANYWHERE		118	97	82%	
20%	SKIN DISTURBANCES	310	251	81%		
9%	SORE THROAT	149	128	86%		
6%	TACHYCARDIA	97	68	70%		
4%	THYROID PROBLEMS	56	44	79%		
12%	ULCERS & SORES (ORAL CAVITY)			189	162	86%
7%	URINARY TRACT PROBLEMS		115	87	76%	
29%	VISION PROBLEMS	462	289	63%		

As can be seen there is a dramatic improvement on psychological disturbances, this is mirrored in the authors own practice experience.
Allergies show a marked improvement on amalgam removal
More importantly, as this symptom analysis demonstrates, the question is not whether the patient is allergic to dental amalgam but rather the direct causal relationship of mercury/amalgam dental fillings to the development of allergies to food, chemicals, and environmental factors. In the prior FTFD , this is supported by the fact that 14% of the individuals reported some type of allergy and that after replacement of their mercury/amalgam dental fillings, 89% reported their condition had improved or was totally eliminated.

Allergy and Dental Amalgam is a huge topic. Suggested reading is www. melisa.org as this covers in exhaustive detail the work of Prof V Stejskal. Suffice it here to say that the connection between allergies and dental amalgam is well established.

Women and Children

The referenced work on autism, the sheep and monkey experiments all show that mercury is preferentially absorbed by the foetus and that this can have an affect on the development of the child.

The National Academy of Sciences in the USA estimates that 60,000 newborns a year could be at risk of learning disabilities because of mercury their mothers absorbed during pregnancy. Mercury in the tissues of fetuses and infants (11-50 weeks of life) correlates significantly with the number of dental amalgam fillings of the mother.

Drasch et. al., "Mercury Burden of Human Fetal and Infant Tissues," EuropeanJournal of Pediatrics (August 1994).

This study is good starting point. There is a connection between the maternal amalgam load and the mercury level in the foetus.

Reproduction disruption, male and female is also well researched

Barnski, G; Scymczyk, J. Effects of Mercury Vapours Upon Reproductive Function On White Female Rats. Medycyna Pracy., 24(3):249-61, 1973.

Gerhard, I; Runnebaum, B. Fertility Disorders May Result From Heavy Metal and Pesticide Contamination Which Limits Effectiveness of Hormone Therapy. Zentralblatt fur Gynakologie, 14:593-602, 1992.

Gerhard, I; et al. Heavy Metals and Fertility. J Toxicol Environ Health, 54(8):593-611, Aug 1998.

Lee, IP. Effects of Environmental Metals on Male Reproduction. In: Reproduction and Developmental Toxicity of Metals; Ed: Clarkson, TW; et al.:253-78, Plenum Press, NY, 1983.

Mishanova, VN; et al. Characteristics of the Course of Pregnancy and Labor in Women Coming in Contact With Low Concentrations of Metallic Mercury Vapors in Manufacturing Work Places. Gig Tr Prof Zabol., 2:21-3, 1980.

Panova, Z; Dimitrov, G. Ovarian Function in Women Having Professional Contact With Metallic Mercury. Akusherstvoi Ginekologiya, 13(1):29-34, 1974.

Rowland, AS; et al. The Effect of Occupational Exposure to Mercury Vapour on the Fertility of Female Dental Assistants. Occupat Environ Med., 51:28-34, 1994.

Warfinge, K; et al. The Effect on Pregnancy Outcome and Fetal Brain Development of Prenatal Exposure to Mercury Vapour. Neurotoxicology, 15(4), 1994.

This is the appropriate place to show the Lisbon study was fatally flawed and that the data it did establish showed pathological effects on then children.
This inclusion of this study illustrated the fact that the SCENIHR committee had no understanding of the biochemistry of mercury.

Response to the NIDCR Funded Children's Amalgam Testing publications in the JAMA 2006.*

By Boyd Haley, Ph.D. Professor of Chemistry at the University of Kentucky
Introduction: It is of considerable importance that those interested in the health of our children consider the fact that the level of mercury in blood, urine or feces may be more a factor of the ability of the child to excrete mercury than it is of total mercury exposure.
For example, research has shown that autistic children represent a subset of the population that does not effectively excrete mercury and therefore has less mercury in the excretory materials but much more in the organs of their body. Also, autistic children have been reported to have aberrant porphyrin profiles indicating they were mercury toxic from an early exposure to mercury and that these aberrant profiles returned towards normal when the children were treated with mercury chelation procedures.
The inhibition of the porphyrin synthesis pathway inhibits the production of the final product, heme. Heme is used to bind and carry oxygen in the hemoglobin of blood.
Heme is also a necessary component of the P-450 enzymes that are critical for detoxifying the body of pesticides, herbicides and other organic toxins. Heme is also a critical factor for the ETS (electron transport system) of mitochondria where most of the energy (ATP) of the body is made. A report in the February issue of the Proceedings of the National Academy of Science established that heme is needed to flush beta-amyloid from the brain, if insufficient heme is present the beta-amyloid forms "large toxic clumps" called amyloid plaques, a major diagnostic hallmark of Alzheimer's disease.
These same amyloid plaques are regarded by many as the cause of Alzheimer's disease, but in reality the primary cause is toxins like mercury that prevent amyloid protein excretion. Therefore, mercury inhibition of the heme-producing porphyrin pathway could have major effects secondary to the primary site of mercury inhibition.
Previous publications by others have shown that adults exposed to dental amalgam mercury vapor have aberrant porphyrin profiles due to a genetic polymorphism (CPOX4), which significantly modifies the effect of mercury exposure on urinary porphyrin excretion in humans. Some were more affected

than the majority, indicating a genetic susceptibility of a subset of the population to mercury toxicity. This emphasizes the question as to why wasn't the porphyrin profile data published in these JAMA articles instead of being dismissed by the authors with only brief comments? It would be hard to explain how adults could be affected without seeing a similar effect in the children of these studies.
Below are some comments regarding these studies. Some relevant research publications regarding my comments are presented at the end of this summary.
1. In the first line of the Portugal based study entitled "Neurobehavioral Effects of Dental Amalgam in Children" Dr. Timothy A. DeRouen, et al., the author writes, "dental amalgam… emits small amounts of mercury vapor". This is neither a scientific nor quantitative statement, i.e. what is a small amount of mercury? The exposure level of a
toxin to any such study of this type is absolutely needed and this is totally ignored in these studies making any comments on safety by measuring the urine mercury levels totally invalid. The fact is these researchers are implanting into children a material that is 50% mercury and known to emit mercury vapors, but the question is how much mercury vapor are these children exposed to daily. Both the ADA and the FDA have steadfastly refused to address this question by doing the appropriate experiments and publishing them. My opinion (since I have done this) is that they know the level ofmercury vapor emission from amalgams is too high to be accepted as safe, so they stonewall this critical experiment. Now it appears as if the IRB boards of several prestigious medical schools have been convinced to do the same. It is a dereliction of duty to place a toxic material into any patient, but especially a child, and especially if the level of toxic exposure is not defined.
2. It has been published and verified that over 90% of mercury excreted by humans leaves through the biliary transport system of the liver and is excreted in the feces, not the urine. Urine mercury levels are well documented not to reflect exposure under many conditions. Therefore, a major flaw in these studies published in JAMA is that they did not measure mercury using the appropriate fecal samples and, instead, used urine, which is a minimal excretion route and vastly underestimates the total mercury exposure. Also, most mercury excreted in the urine is that bound to cysteine or other soluble, small molecule sulfur containing compounds. Therefore, the urine mercury excretion levels are as much dependent on the blood levels of cysteine or other compounds as they are on mercury exposure. Cysteine levels are dependent on diet.
The bottom line is that these studies looked for mercury in all the wrong places. One study reported that mercury in fecal materials was 13 times that in urine of the samepatients. If you don't want to find data indicating excess exposure to mercury look where it isn't, look in the urine and that's what these studies did.
3. Since the IRB's of several prestigious universities approved this research, i.e. research that exposed children to an unknown daily level of mercury vapor, the public should demand that these same universities perform experiments on the same brand of amalgams, made outside of the mouth, of known weight and surface area and determine the amount of mercury released per day by these amalgams (with and without abrasion to mimic the daily effects of chewing). They

should publish these results. *[IAOMT did it– see* article.] With this data a decent estimate of the daily exposure of the children to mercury from these amalgams can be made and an approximate determination of what fraction of the amount excreted in the urine accounts for the bulk of the mercury. Studies done in my laboratory, similar to those done by others, have demonstrated that the emission of mercury vapors were much higher than what has been "estimated" by pro-amalgam individuals. Chew et al. Clinical Preventive
Dentistry 13(3) 5-7, 1991, showed that in a study of long term dissolution of mercury from a "non-mercury releasing amalgam," it was determined that 43.5 microgram/cm ^{2} /day Hg was released and this remained constant for 2 years. What is also known is that different amalgam preparations release mercury at vastly different levels. The modern high copper amalgams were shown to release much higher levels than other older type amalgams.
4. Look at Figure 2 on page 1788 where the authors plot the urine mercury levels at each year. Years one and two show a steady increase in urinary Hg in the amalgam bearers versus the amalgam free children as expected. Yet, on years 3 to 7 the level of mercury in the urine of the amalgam bearers continuously drop until they near the levels of the amalgam free children. The paper implies that restorative treatment was used in years 6, 7 and 8, which should increase, or at least maintain the urine mercury levels. This needed explaining. In the Chew study above the amount of mercury released was steady for 2 years (the length of the study). Consider this, plus the fact that a 1gram filling would contain 500,000 micrograms of mercury, or 100,000 days of emitting a toxic
5 micrograms per day. This equates to about 275 years of mercury before it is all gone and the average life span of an amalgam before replacement is less than 10 years. Amalgams do not stop releasing mercury vapor within 7 years. So, what caused the drop after year 2?
Urine mercury levels are, in my opinion, a measure of the amount of mercury being excreted by this route. Therefore, after two years exposure the route of kidney excretion of mercury appears to be becoming less effective. This is consistent with the well known fact that increased mercury exposure inhibits it own excretion. This data is quite damning to the idea that amalgams are safe to place in children. For example, youths die of idiopathic dilated cardiomyopathy (IDCM) while under physical stress in athletic events and it has been published that the heart tissue of these individuals contains 178,400-ng/g mercury or 22,000 times more than was found in their muscle tissue, or in the heart tissue of individuals who died of other forms of cardiac disease.
Another example, a study published in J. Amer. Dental Assoc. regarding amalgams and Alzheimer's disease reported no correlations between amalgams and brain mercury levels. Yet, about 15% of the nuns in this study had brain mercury levels in the micromolar range, a very toxic level of mercury since about 1,000 fold less than this has lethal effects on neurons in culture. Again, this reflects that certain individuals appear to have less ability to excrete mercury than others, even if they live in the same location and eat the same food, etc. The point being, that mercury collects in certain tissues at levels much higher than have ever been found in blood, urine or hair and it is the primarily the

retention of mercury (or the inability to excrete mercury) that enhances its toxicity from continuous, low level exposures. The bottom line, the data in their Figure 2 gives strong indication that after two years exposure to dental amalgam mercury, the children seem to be losing their ability to excrete mercury through the urinary pathway. The real question is, have they also lost the ability to excrete mercury through the major, fecal pathway? In contrast to the recommendations made by these authors this may be a major reason to discontinue placing amalgams in children.

5. These authors say very little about the porphyrin effects in the amalgam bearers except to state that they did not indicate kidney damage. This begs the real question, what about the children's ability to make heme? Were the porphyrin profiles aberrant,as found in adults exposed to amalgams, or in autistic children? One has to questionwhy this data was not included and discussed in detail.

6. It is well described in the literature that mercury is a potent immune system suppressor and others have detailed experiments that show this. Why was this easy to test system ignored in these studies? Experiments have shown that mercury exposure dramatically effects macrophage phagocytosis of microbes at very low levels. To choose to check effects on IQ over this period of exposure and ignore the immune system, especially when the immune system is known to be affected immediately on mercury exposure, is questionable. Especially, when the object of the study was to determine if mercury from amalgams were "safe" for use in children.

7. One of the inclusion criteria for these studies was "no interfering health conditions," and Dr. Bellinger, one of the authors, stated these interfering conditions included autism and prior neurological disorders. The CDC reports that 1 in 6 American children have a neurodevelopmental disorder; I am unaware of the rate in Portugal. However, thesepapers conclude that amalgams should remain a viable clinical option in dental restorative treatment and they did not exclude use on children with neurodevelopmental disorders, the type of child they excluded from their studies. I feel that I could make a very convincing argument that the children with prior neurological disorders are children who fall into the category of children who do not effectively excrete mercury. In this waythe study has a major failing in that it excluded from the population being studied those children most susceptible to mercury toxicity.

Conclusion: These studies were poorly designed and tell us one thing of good value –that children with amalgams most likely slowly lose their ability to excrete mercury afterabout two years of amalgam exposure. This experiment should have been done on primates, not humans and presents a question of ethics in medicine.

The major problems with the studies are that they:

1. Ignored measuring the amount of mercury exposure to children by first determining the amount of mercury emitted from an average sized amalgam outside of the mouth.
2. Used urine and blood mercury levels when 90% plus of mercury is excreted in

the feces. This obviates any conclusions they make, as urine mercury levels are unreliable with regards to exposure, which is exactly what their own data shows.
3. Did not select the most sensitive clinical testing parameters for detecting mercury
toxicity but instead used testing parameters that are known to fluctuate without known cause or parameters that require long-term low level exposure to show an affect.
4. Did not state that their conclusions of amalgam safety should not include children with any prior neurodevelopmental or systemic illness.
5. Ignored the drop in mercury excretion in the urine after year 2 even though the mercury exposure from amalgams remained the same or increased. This is a sure sign of losing ability to excrete mercury with increased exposure to this toxic metal.

- *Timothy A. Rouen, et. Al., Neurobehavioral effects of dental amalgam in children, JAMA 295(15): 1784-92. 2006.*
-

David C. Bellinger, et. al., Neuropsychological and renal effects of dental amalgam in children, JAMA 295(15): 1775-83. 2006

References:

1. Escheverria, D., Woods, JS et al. Chronic low-level mercury exposure, BDNF (brain derived neurotrophic factor) polymorphism, and associations with cognitive and motor function. Neurotoxicol. Teratol, 2005 Nov-Dec; 27(6) 781-96 .

2. Escheverria, D. Woods, JS, et al. The association between a genetic polymorphism of coproporphyrinogen oxidase, dental mercury exposure and neurobehavioral response in humans. Neurotoxicol. Teratol. 2005 Dec 8.

3. Chew et al. Clinical Preventive Dentistry 13(3) 5-7, 1991. In a study of long term dissolution of mercury from an non-mercury releasing amalgam it was determined that 43.5 microgram/cm2/day Hg was released and this remained constant for 2 years.

Conclusion: These studies were poorly designed and tell us one thing of good value –that children with amalgams most likely slowly lose their ability to excrete mercury after about two years of amalgam exposure. This experiment should have been done on primates, not humans and presents a question of ethics in medicine. The major problems with the studies are that they:
1. Ignored measuring the amount of mercury exposure to children by first determining the amount of mercury emitted from an average sized amalgam outside of the mouth.
2. Used urine and blood mercury levels when 90% plus of mercury is excreted in the feces. This obviates any conclusions they make, as urine mercury levels are unreliable with regards to exposure, which is exactly what their own data shows.
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without known cause or parameters that require long-term low level exposure to show an affect.
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1. Escheverria, D., Woods, JS et al. Chronic low-level mercury exposure, BDNF (brain derived neurotrophic factor) polymorphism, and associations with cognitive and motor function. Neurotoxicol. Teratol, 2005 Nov-Dec; 27(6) 781-96.

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4. Kingman et al. J. Dental Research 77(3) 461, 1998. In a study of 1,127 military personnel by NIH the level of mercury in the urine of amalgam bearers was 4.5 times that of amalgam free controls. Some with extensive amalgams had levels 8 times or high than the amalgam free controls.

5. Hultman, P. et al. Adverse immunological effects and autoimmunity induced by dental amalgam and alloy in mice. The FASEB Journal 8 Nov 1183-1190, 1994.

6. Wataha et al. Dental Materials 10 298-303, 1994. The amalgam material with the trade name Dispersal Alloy made solutions in which it was soaked severely cytotoxic.

7. Begerow et al. International Archives of Occupational and Environmental Health66(3) 294, 1994. After removal of amalgams from 17 patients urine mercury levels dropped by a factor of 5.

8. Frustaci et al. J American College of Cardiology 33(6) 1578, 1999. Data showedthat individuals who died with IDCM (idiopathic dilated cardiomyopathy, the cause of young athletics dying during physical stress) had 22,000 times more mercury in their heart tissues than individuals who died of other forms of heart disease. Never has there been a urine or blood level reported that comes to the level of 178,400 ng/g tissue which is the same as 178.4 micrograms/g and one milliliter water weighs 1 gram. In the study under discussion they were talking about 3-5 micrograms/liter (1,000 milliliters) or so which compares t178.400 micrograms/1000g in IDCM.

Where does this mercury come from as this disease kills intercity kids as much as anyone and they are not big sea food eaters?
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Risk Assessment of Dental Amalgam.

The definitive study was by Richardson in 1995
In this study, commissioned by Health Canada, Richardson showed that, based on neurological data, the maximum number of fillings allowed before effects would be evident was as follows:

Ages 3-11	0 to 1 amalgam filling
Ages 12-19	1 to 3 amalgam fillings
Ages 20+	2 to 4 amalgam fillings

The following data is relevant.

Blais, P. Memo & Dental Amalgams and the Public Health: A View from the Health Protection Branch, Bureau of Medical Devices, Health Canada, 1976.

Clarkson, TW. Principles of Risk Assessment. Adv Dent Res, 6:22-27, 1992.

Richardson, GM. Assessment of Mercury Exposure and Risks From Dental Amalgam; Final Report, Medical Devices Bureau, Environmental Health Directorate, Health Canada, 18 Aug 1995.

Richardson, GM. A Monte Carlo Assessment of Mercury Exposure and Risks from Dental Amalgam. Human Ecolog Risk Assessment, 2(4):709-61, 1996.

USEPA. Integrated Risk Information System (IRIS). Mercury, Elemental: 1.1.98. Online. Cincinnati, Ohio: National Center for Environmental Assessment, 1998.

USPHS. Toxicological Profile for Mercury. ATSDR, TP-93/10, 1994.

Viola, PL; Cassano, GB. The Effect of Chlorine on Mercury vapor Intoxication Autoradiographic Study. Med Lavoro, 59(6-7), 1968.

Alternative Materials

Once again let us return to the science left out by this report.

The IAOMT commissioned Richardson to do a risk assessment on dental composite resins.

His results showed that composite was safer by a factor of 200 in comparison with mercury fillings

Dental Composite Biocompatibility is shown by these references.

Hamid, A; Hume, WR. Release of Estrogenic Component Bisphenol-A Not Detected From Fissure Sealants In Vitro. J Dent Res., 76(SI):321, A2459, 1997.

Olea, N; et al. Estrogenicity of Resin Based Composites and Sealants Used In Dentistry. Environ Health Perspect., 104:298-305, 1996.

Richardson, GM. Assessment of Exposure and Risks from Components and Degradation Products of Composite Resin Dental Materials. Human Ecolog Risk Assess., 3(4):683-97, 1997.

A Final Word

The FDA, Food and Drugs Administration, Advisory Panel in 2006 stated " Dental Amalgam can no longer be considered safe."

Mats Berlin Mercury in dental-filling materials— an updated risk analysis in environmental medical terms 2003
"The Safety Factor thought to exist with Dental Amalgam does not exist"

IAOMT Conclusion.

Any sensible reader would adopt at the very least the precautionary principle upon reading this document and want to cease the use of dental amalgam as a restorative material.

It has been demonstrated beyond reasonable doubt that mercury from amalgam fillings has the capacity to injure human health.

Adequate alternatives are available.

The report has been shown to be lacking in scientific rigour by the omissions of relevant data and basic misconceptions about the biochemistry of mercury in the body.

If this report is adopted it must be for reasons other than science and the health of the population.

Dental amalgam should not be allowed to be used as a restorative material within the EU unless data proving safety can be demonstrated.

The author has many years of treating patients with symptoms that have resolved upon amalgam removal under proper protocols and is more than willing to advise and share his experience with any who seek it.