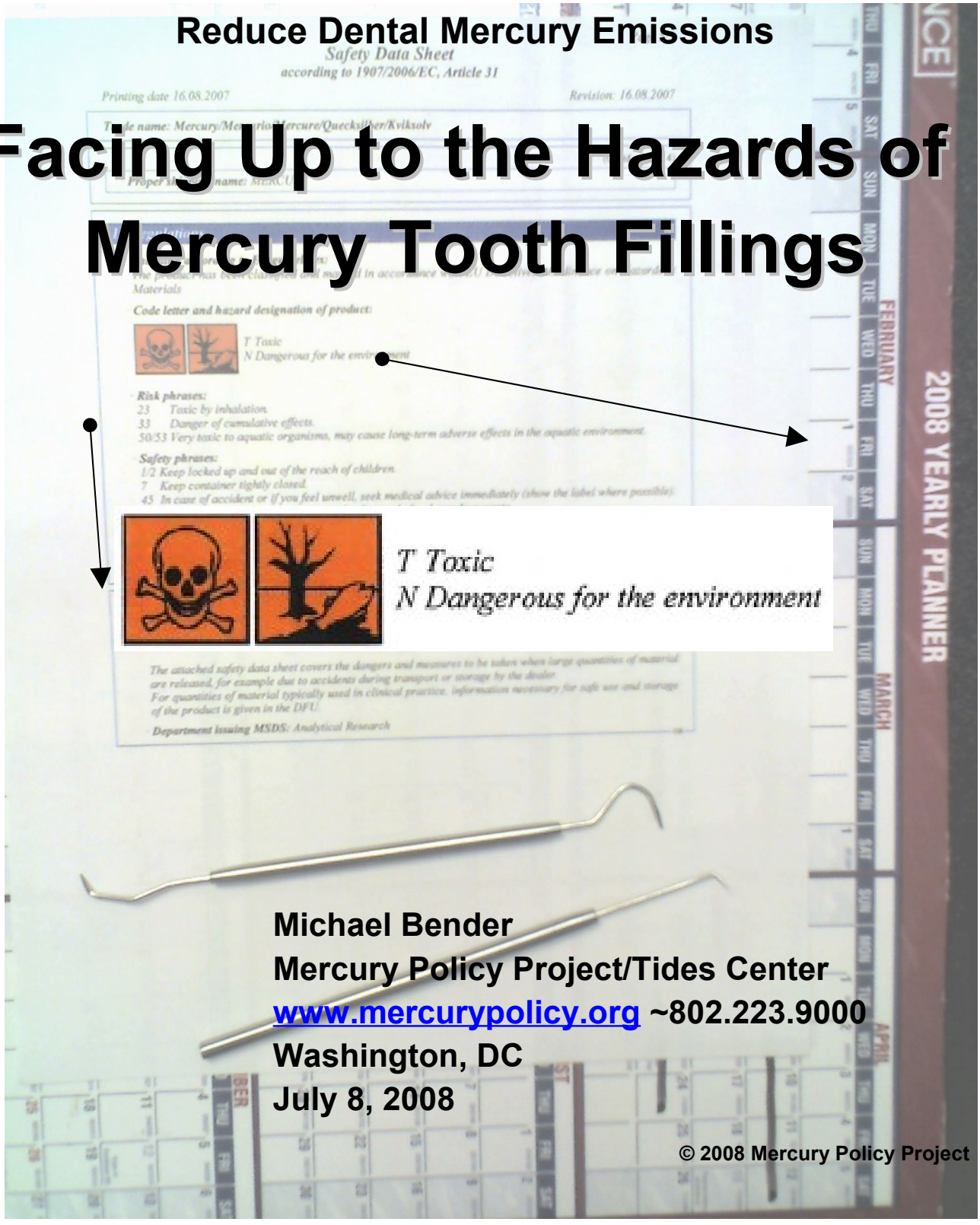
A Report to US House of Representatives
Government Oversight Subcommittee on Domestic Policy

Assessing State and Local Regulations to
Reduce Dental Mercury Emissions

Facing Up to the Hazards of Mercury Tooth Fillings



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The Mercury Policy Project (MPP) is a project of the Tides Center and works to promote policies to eliminate mercury uses, reduce the export and trafficking of mercury, and significantly reduce mercury exposures at the local, national, and international levels. We strive to work harmoniously with other groups and individuals who have similar goals and interests.

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Introduction

It's becoming increasing clear that the recent improvements in technology for the non-mercury filling—most commonly the "composite"—have rendered the mercury tooth filling—aka "amalgam"—obsolete. One only has to look at the recent bans on new amalgam placement in Norwegian or Swedish dental patients or elimination of insurance coverage for amalgam restorations in Danish patients to document mercury-free tooth restoratives as a viable substitute.

Practically speaking, the age of amalgam is over.

So why do over 60 million mercury tooth fillings still get placed into Americans' mouths every year?

Is it because it is simply cheaper and quicker for your dentists to place an amalgam and they make more money doing so?

Is it because, as the expression goes, "you can't teach an old dog new tricks," and in some cases dentists are reluctant to change or take the time to master the new techniques for placement of composites?

Or is it because the US dental sector, led by the American Dental Association and its state associations, remains in denial that mercury is a neurotoxin — a hazardous material before it is placed in the mouth, and a hazard that releases toxic vapors after it is in the mouth? And could concerns about potential legal liability reinforce this denial?

Or finally, is it because dentists are not aware or held accountable to the fact—undisputed by the US EPA since it was presented to the US House subcommittee last fall-- that the continued use of amalgam is resulting in the release of upwards of 10 tons—and growing—of mercury into the air and water each year in the U.S. And that at least some of that mercury gets taken up in the fish Americans eat and, in particular, poses the most acute risk to pregnant women and their developing fetus and young children?

The answer certainly includes some or all of the above points, depending upon the expert you may be talking with.

While the calculations here are necessarily based on a certain number of assumptions, estimates and projections, the basic fact remains that up until now significant added costs of using amalgam—the so-called "externalities"—have not been factored into the fee charged by your dentist. This report demonstrates when factoring in these external costs, even under multiple scenarios, the cost of placing an amalgam filling virtually meets or surpasses the cost of placing a non-mercury composite filling.

Assuming that it is not yet politically viable for decision-makers in the US to ban amalgam outright, this report – for the first time ever-- lays out the rationale for placing a user fee on the continued use of dental mercury as a means to cover the costs of preventing dental mercury pollution from environmental release.

This report also clearly shows the cost-effectiveness of amalgam separators at preventing mercury from getting into the environment. It also clearly demonstrates that voluntary programs are not effective in convincing dentists to install and properly maintain separators.

1 Dental mercury, wastes and emissions

1.1 Mercury in the environment

Mercury is a naturally occurring metal and a persistent, bio-accumulative neurotoxin, especially affecting the brain and nervous system. It enters the environment via natural events, such as volcanic eruptions, but more-so through human activities. Methylmercury is more mobile and even more toxic than elemental mercury, and it easily finds its way into the food chain, contaminating fish. Methylmercury is synthesized by microbial action on mercury-polluted sediments and soils. The consumption of fish from waters contaminated with mercury is the source of greatest risk of exposure to this pollutant (NACWA 2002).

While mercury releases to wastewater should clearly be avoided, most methylmercury is generated from the by-products of the combustion of mercury-containing materials. The release of mercury by combustion occurs in a variety of settings, including coal-fired power plants, municipal incinerators, sludge incinerators, hazardous waste incinerators, industrial boilers, cremation chambers and other industrial processes including metal refining and cement production.

The widely documented effects of mercury exposure on human health and wildlife have driven a great range of efforts, in the US and overseas, to significantly reduce the level of this toxic, persistent, and bio-accumulative metal in the environment. The rest of this paper will address one key source of mercury releases to the environment, which is the use of mercury in dentistry.

1.2 Dental mercury wastes

The primary sources of mercury waste that originate in the dental clinic include amalgam waste generated producing amalgam for use in the procedure; the excess material carved from new amalgam fillings; the removal of old amalgam fillings; the removal of teeth containing amalgam; other mercury going to solid waste or wastewater; mercury emissions directly to the air; the traps, filters and other devices in dental clinics to remove mercury from the wastewater – and the “downstream” flows of mercury from there.

Most dental mercury waste results from the removal of previous fillings from patients’ teeth. Together with waste generated during the replacing of fillings, removed teeth, etc., these dental wastes typically follow these main paths. They may be

- Captured for subsequent recycling or disposal,
- Washed down drains that lead to the general municipal wastewater system,
- Placed in special containers as medical waste, or
- Discarded as municipal waste.

It is commonly accepted that most municipal wastewater systems encounter significant levels of mercury, and it has been determined that typically close to 50% of that mercury originates from dental practices (AMSA 2002a). Some observations are summarized in the following table.

City	Mercury load from dental offices
Duluth, Minnesota	36%
Seattle, Washington	40-60%
Palo Alto, California	83%
Greater Boston Area, MA	13-76%

1.3 Dental mercury emissions

Dental amalgam is a large source of mercury waste in the environment. According to EPA, "Mercury discharges [in wastewater] from dental offices far exceeded all other commercial and residential sources." (EPA 2006) EPA cited an estimate that 36 percent of mercury reaching municipal sewage treatment plants is released by dental offices. Other investigations have put the figure closer to 50 percent (NEG-ECP 2007). The costs of largely eliminating discharges of dental mercury to wastewater are assessed in Section 3 of this report.

Despite regulations regarding the characterization and disposal of mercury bearing wastes, many solid dental wastes still follow the low-cost route of disposal as municipal solid waste and are subsequently disposed of in landfills or by municipal incineration. Depending on the characteristics of the landfill, dental amalgam may decompose over time and the mercury may enter the leachate (which may itself be disposed of in a manner that permits the mercury to be released), groundwater, soils, or volatilize into the atmosphere. Studies have documented methylmercury in gases emitted from landfills (Lindberg *et al.* 2001).

Mercury from dental amalgams is also a significant source of airborne emissions. EPA has estimated airborne mercury attributable to wastewater sludge incineration to be 0.6 ton per year, but the discussion in Section 4 below provides evidence that the EPA estimate is seriously underestimated. Among other failings, EPA emissions estimates do not include total mercury emitted during the cremation of human remains. However, cremation has been shown to be a significant source - over 3 tons of emissions - due to the large amount of mercury in existing dental fillings. In comparison, the largest source of airborne mercury is coal-burning power plants, which emit an estimated 48 tons of mercury per year.

The 2002 EPA National Emissions Inventory (version 3) put atmospheric emissions related to dental mercury at 1.5 tonnes, as in the first column of the table below. The EPA numbers are compared with the more rigorous estimates submitted in testimony last fall, summarized in the second and third columns, which suggest air emissions at least 5 times higher than the EPA estimates. (Bender 2007) The EPA has not contested these revised estimates.

Atmospheric emissions of dental mercury (tons)			
Pathway	**EPA National Emissions Inventory 2002**	**This report 2005 (low estimate)**	**This report 2005 (high estimate)**
Human cremation	0.3	3.0	3.5
Dental clinics	0.6	0.9	1.3
Dental mercury sewage sludge incineration	0.6	1.5	2.0
Dental mercury sludge spread on land and landfilled	n.a.	0.8	1.2
Dental mercury MSW incineration and landfill	n.a.	0.2	0.5
Dental mercury infectious and hazardous waste	n.a.	0.5	0.7
Human respiration	n.a.	0.2	0.2
Total	**1.5**	**7.1**	**9.4**

1.4 Quantities of dental mercury consumed

Contrary to what the US dental sector maintains, there has been very little evidence of reduction in the amounts of mercury used in dental restorations in recent years.

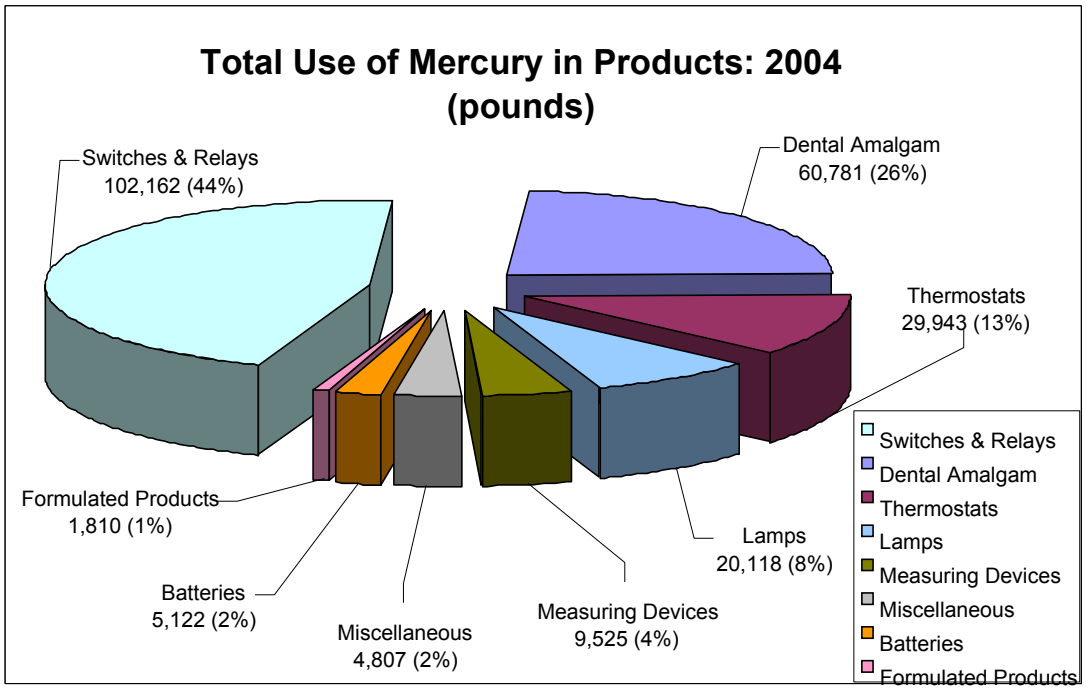
The Interstate Mercury Education and Reduction Clearinghouse (IMERC), a program of the Northeast Waste Management Officials' Association (NEWMOA), published a report online showing that mercury use in products sold in the U.S. declined from 131 tons in 2001 to 117 tons in 2004, an 11 percent reduction. The IMERC study, *Trends in Mercury Use in Products: Summary of the Interstate Mercury Education and Reduction Clearinghouse (IMERC) Mercury-added Products Database* (IMERC 2008), summarizes mercury use in products sold in the United States in 2001 and 2004 from information submitted by hundreds of manufacturers.

From IMERC's latest report, we see little change from 2001-2004 in the amount of amalgam provided to dental facilities from these five major manufacturers. For both years analyzed, 2001 and 2004, about 30 tons (61,537 in 2001 and 60,781 pounds in 2004) of mercury was used for the placement of almost 60 million amalgam fillings. This is detailed in the following table provided by IMERC.

Total Amount of Mercury Sold in Fabricated & Formulated Products U.S. For Calendar Years 2001 & 2004				
Products/Components	**Total Mercury (pounds)**		**Number of Total Manufacturers Reporting**	
	2001	**2004**	**2001**	**2004**
Switches & Relays	119,660	102,162	53	46 + 3 nr*
Dental Amalgam	61,537	60,781	5	5
Thermostats	30,971	29,943	9	8 + 1 nr
Lamps	21,438	20,118	177	185 + 8 nr
Miscellaneous	8,505	4,807	12	10 + 2 nr
Batteries	5,914	5,122	40	41
Measuring Devices:				
Sphygmomanometers	4,305	2,219	2	2
Thermometers	5,347	4,524	13	8 + 4 nr
Manometers	1,936	2,545	4	4
Barometers	353	234	1	1
Psychrometers/Other Measuring Equipment	4	3	3	3
Chemicals & Solutions	2,060	1,810	20	20 + 1 nr
Total	**262,030** (131 tons)	**234,268** (117 tons)	**339**	**352**

With regard to nationwide consumption of mercury, as shown in the NEWMOA figure below, dental offices are the second largest user of mercury, after switches and relays.

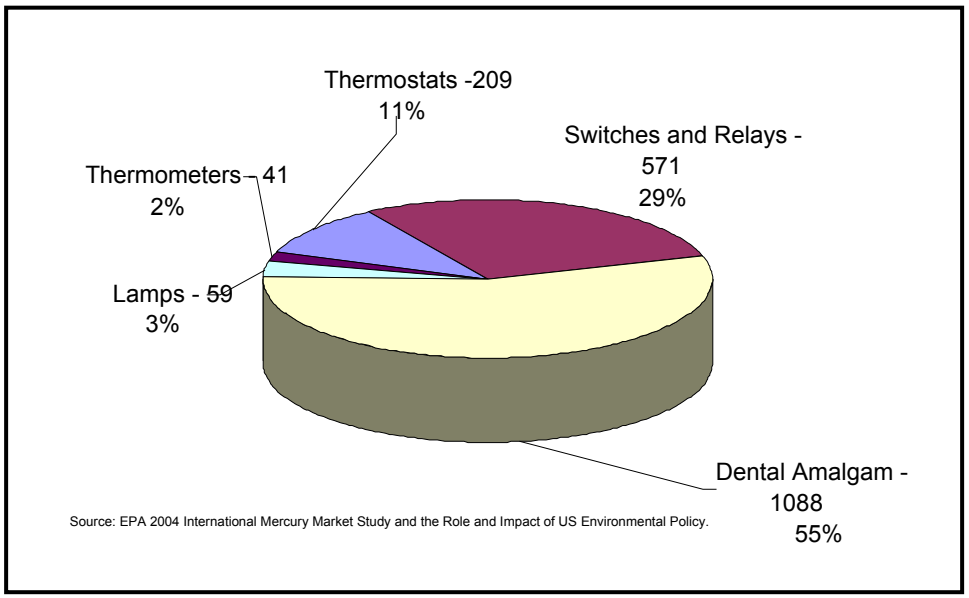
Figure 1 – Mercury consumption in the USA in 2004



Finally, as seen in the following EPA figure, mercury contained in the existing dental fillings of Americans comprises over half of all mercury "circulating in the economy" today,

amounting to over 1000 tons. (EPA 2004) All of this mercury will eventually have to be dealt with in order to keep it out of the environment.

Figure 2 - Mercury circulating in the U.S. economy



1.5 Quantities of mercury in dental wastes

Following the methodology used by EPA (Cain 2007), of the 30 tons of "new" mercury consumed in a typical year by dental clinics, some amalgam is carved away or otherwise lost during a typical clinical procedure – averaging some 20-25% of the total amalgam used. However, most of the mercury lost is not due to "carving" and fitting a new filling, but due rather to the amount of old amalgam that is removed to make room for the new filling. Considering that about 70% of fillings are replacements, that not all new fillings are amalgams, etc., some 31 tons of mercury have been calculated to go to emissions and waste (Bender 2007).

The quantities of mercury consumed and mercury wastes generated by the dental profession are directly related to the average life of a filling. In a US Geological Survey report published in 2000, it was noted that the average life of a mercury amalgam filling is reported to be from 5 to 8 years, while a 1995 article in a Swiss dental medical journal reported the average life to be 10 years. Other estimates have ranged as high as 10-20 years (Reindl 2007).

2 Status of efforts to minimize the risks of amalgam

2.1 Norway, Sweden Ban Amalgam

Starting in January 2008, Norway banned amalgam. In announcing the ban, Norwegian Minister of Environment Erik Solheim said:

> "Mercury is among the most dangerous environmental toxins. Satisfactory alternatives to mercury in products are available, and it is therefore fitting to introduce a ban. When the environmental toxin mercury is released to the environment it is very harmful, and *inter alia* the development of children may be damaged as a result."

According to the Norwegian Ministry, mercury is among the most dangerous environmental toxins. Satisfactory alternatives to mercury in products are available, and it is therefore fitting to introduce a ban. Minister Solheim further stated that the Norwegian ban shows that Norway is taking responsibility at home. It is an important signal, to the EU and other countries scrutinizing various uses of mercury, that there are satisfactory alternatives to mercury, the minister concluded.

Sweden announced a similar ban on amalgam, and Denmark announced that it will not provide public insurance to cover mercury in fillings after April 1, 2008. Such measures would be politically impossible if entirely satisfactory mercury-free alternatives were not available, or if these governments were not absolutely convinced that amalgam carries a higher risk than mercury-free alternatives.

2.2 FDA Settles Lawsuit, Agrees to Classify Amalgam as a Medical Device, Revamps Website

After 32 years of delay, the Food and Drug Administration has finally agreed to comply with Federal law and set a date to classify mercury amalgam as a substance that poses a health risk, especially to pregnant women and unborn babies, and to children. This about-face resulted from settling the lawsuit, *Moms Against Mercury et al. v. Von Eschenbach, Commissioner, et al.*, in which the judge cited FDA for an "*unreasonable delay*" and "*a reasonable case of failure to act*." As reflected in the May 16, 2008, court transcripts, Judge Ellen Huvelle stated that the "*probability of harm is enormous*," and asked the FDA: "*How could you drag your feet for 32 years? Do what you are supposed to do*." Judge Huvelle also stated that she couldn't "*order a ban, but can compel [FDA] to act*," observing that this was "*government at its worst*" and that she wanted this "*public safety issue to be resolved*." The FDA must now finish classification within one year of the close of the public comment period on its amalgam policy, that is, by July 28, 2009.

As part of the settlement, the FDA agreed to, and with uncharacteristic speed has already, change its website— dramatically. The updated June 3, 2008 FDA website now states, for example:

> "*Dental amalgams contain mercury, which may have neurotoxic effects on the nervous systems of developing children and fetus*." … "*Pregnant women and persons who may have a health condition that makes them more sensitive to mercury exposure, including*

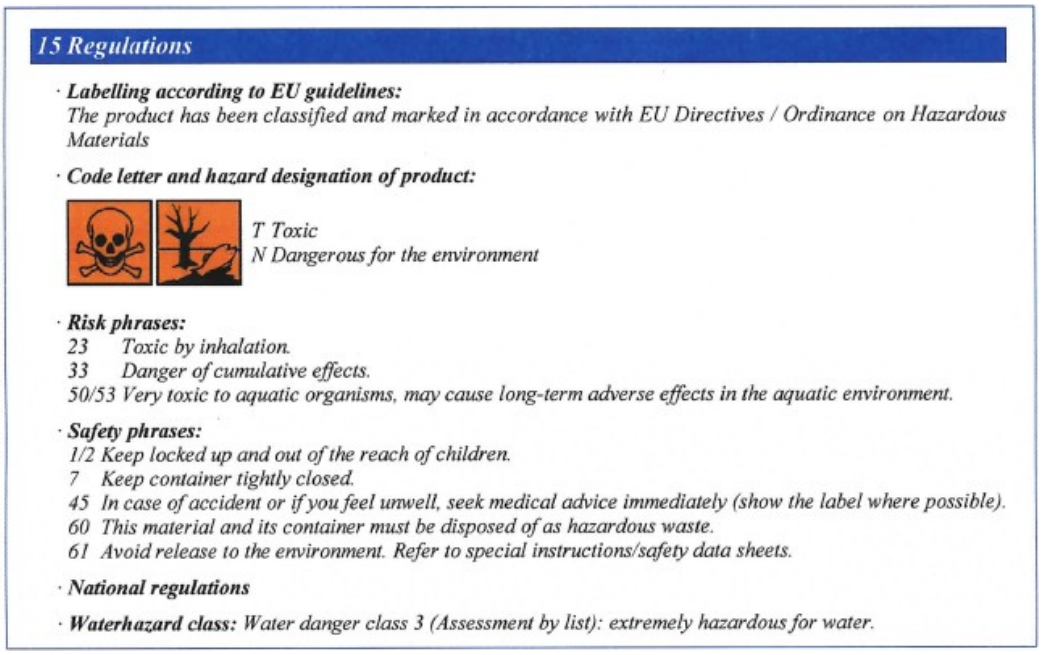
> *individuals with existing high levels of mercury bioburden, should not avoid seeking dental care, but should discuss options with their health practitioner*."

The FDA website (FDA 2007) also states, "Some other countries follow a 'precautionary principle' and avoid the use of dental amalgam in pregnant women," and provides links to advice about amalgams from regulatory agencies in other countries, including Canada, France and Sweden. For example, the FDA website link to Health Canada advises dentists to take the following measures:

- Non-mercury filling materials should be considered for restoring the primary teeth of children where the mechanical properties of the material are suitable.
- Whenever possible, amalgam fillings should not be placed in, or removed from, the teeth of pregnant women.
- Amalgam should not be placed in patients with impaired kidney function.

These warnings are similar to those sent by amalgam manufacturers. Encapsulated dental amalgam is shipped from manufacturers to a dentist's office with a skull-and-crossed-bones affixed next to the words: "*POISON, CONTAINS METALLIC MERCURY*." (MSDS 2007) Amalgam manufacturers – Kerr, Vivadent and Dentsply, among others – advise dentists against placing amalgam in the teeth of pregnant women, nursing mothers, children under six, and anyone with kidney disease. Dentsply, for example, warns:

> "Contraindication [N.B.: "Contraindication" is a directive to forbid, not just a "warning"]: "*In children 6 and under*" and "*In expectant mothers*."



15 Regulations

· ***Labelling according to EU guidelines:***
The product has been classified and marked in accordance with EU Directives / Ordinance on Hazardous Materials

· ***Code letter and hazard designation of product:***

T Toxic
N Dangerous for the environment

· ***Risk phrases:***
23 Toxic by inhalation.
33 Danger of cumulative effects.
50/53 Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

· ***Safety phrases:***
1/2 Keep locked up and out of the reach of children.
7 Keep container tightly closed.
45 In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible).
60 This material and its container must be disposed of as hazardous waste.
61 Avoid release to the environment. Refer to special instructions/safety data sheets.

· ***National regulations***

· ***Waterhazard class:*** *Water danger class 3 (Assessment by list): extremely hazardous for water.*

However, these warnings are apparently not being passed along to the public, based on the results of a national poll conducted for the Mercury Policy Project by Zogby International whereby:

- Most Americans (76 percent) don't know mercury is the primary component of amalgam fillings;
- 92 percent of Americans overwhelmingly want to be informed of their options with respect to mercury and non-mercury dental filling materials prior to treatment; and
- 77 percent of Americans would choose higher cost fillings that do not contain mercury if given the choice.

2.3 ADA & State Dental Associations Blocking Amalgam Separator Installations

The American Dental Association (ADA) now recommends that amalgam separators be installed in all dental offices as part of their "best management practices (BMPs)," but they maintain that adequate levels of compliance with their recommendation can be achieved through a voluntary program. (ADA 2007) Meanwhile, they have successfully blocked amalgam separator initiatives across the country. For example, it's clear that the ADA is actively helping State Associations find ways to avoid installing separators, or block any kind of requirements to do so, at least in the following states and local jurisdictions.

California The CA Dental Association (CDA) was the sole opponent of Assembly Bill 966 in 2005, authored by Assemblymember Lori Saldaña, and stopped the bill in the Assembly. The bill would have mandated separators. In 2003, CDA was sole opponent of AB 611, authored by Assemblymember Gloria Negrete-McLeod, which also would have required separators. They actually hijacked the bill and got the author to substitute a mere codification of BMPs. The bill then died in Appropriations Committee.

Michigan In Michigan, a colleague had a very brief conversation with a MI Dental Association director who informed him that the ADA lawyer who was "helping" with the separator issue told him that they would not have to deal with the issue until 2011.

Montana According to the *ADA News,* "Immediately after the drafting of HB 665, members and staff of the Montana Dental Association, including two dentists in the Montana legislature, promptly met with the bill's sponsor, Rep. Teresa Henry. At what MDA executive director Mary McCue described as a congenial, professional meeting with a very reasonable lawmaker, the MDA explained its nearly two-year efforts, statewide, to educate dentists and promote voluntary adoption of the ADA's Best Management Practices for handling amalgam waste. The one-two punch was successful; MDA was able to convince Rep. Henry to amend her bill, who shortly removed all language Feb. 18 requiring dentists to install separators. The issue is no longer on the table. "Thanks to the assistance of the ADA, we got out ahead of the issue and it certainly helped us," said Ms. McCue.

Oregon After many delays, an amalgam separator bill was passed with an extraordinarily long compliance date (2011) due to the efforts of the lobbyist for the Oregon Dental Association. Yet the Oregon Dental Association was a bit too clever in how it arranged for such a long lead time. The provision that the ODA inserted into SB 704 deferred the effective date if the dentist is "certified by a special district that manages wastewater

treatment to be following 'best management practices.'" There are a few such districts in the state, but none of them were the least bit interested in becoming a certifying agency for 11,000 Oregon dentists. So, in Oregon's first-ever even-year legislative assembly, the ODA dropped a bill seeking a fix to SB 704, expanding the kind of entities that could certify a dentist's BMPs. Instead, a shorter time frame was adopted for the separator requirement to become law (2010).

Philadelphia Last year, the PA Dental Association blocked a proposed ordinance by the Philadelphia City Council would have required most dentists residing in Philadelphia to install amalgam separators. According to their newsletter, the PA Dental Association worked in conjunction with the ADA, its lobbyists and public relations team and other dental organizations in what they termed a "strong lobbying effort to amend the ordinance." The ADA and PDA were explaining the financial hardships that would be encountered by the Dentists and the city's poorer population because composites were more expensive and the "poor", who could not afford the more expensive fillings, would not take their children to the dentist, causing untold hardships and disease to the less fortunate.

While multiple and complex factors may influence the success, or lack thereof, of a voluntary program, there is a growing body of evidence that a mandatory approach, while administratively more demanding, is necessary to achieve a faster and more comprehensive result. Even more importantly, this creates a level playing field that does not discriminate against the vast majority of dentists who wish to comply with the ADA recommendation to install separators.

The use of amalgam separators is highly cost effective in preventing releases of mercury to the environment, particularly when compared to the cost to remove mercury at a wastewater treatment plant of approximately $21 million per pound, or $46,000 per gram (AMSA 2002b).

Recent data from the Boston area Metropolitan Water Resources Authority (MWRA) (see figure below) showed a 48% reduction in mercury concentration in sludge as amalgam separator use increased from less than 20% to over 80%. Additional data is being collected and assessed to evaluate whether these reductions are typical across the region, and to estimate the overall regional reduction in mercury releases attributable to these programs (NEG-ECP 2007).

King County in Seattle may be taken as an example. King County employed three distinct strategies to limit or control the amount of mercury discharged from dental offices over the 13-year time frame of this case study. The initial resistance of the ADA and dental community to installing separators contributed to the length of time and the changing strategies that had to be employed by the county. The King County Program 1995-2000 focused on an intensive outreach program for dentists, including an annual poster, monthly ads in a local journal, a Voucher Incentive Program, EnviroStars, information dissemination, and trade shows/mercury roundups.

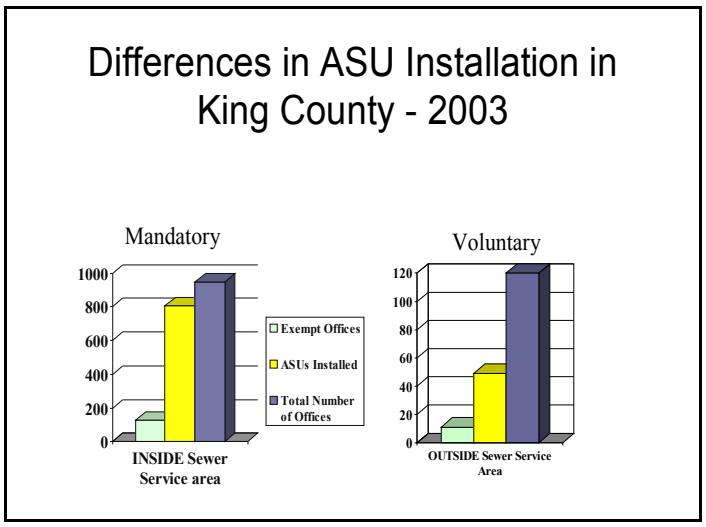
Even after these efforts, a 2000 study in King County found that more than three-quarters of dental offices did not recycle or sequester mercury-bearing waste captured in chairside traps and vacuum pump filters. Rather, they put it in the waste bin, included it with medical waste, stored it onsite for eventual disposal or flushed it down the drain (Savina 2003).

As a result, the following practices were made mandatory by July 1, 2003:

- Use best management practices (BMPs) for amalgam waste;

- Demonstrate compliance with King County local limits (0.2 mg/l) for mercury discharge to sewer (0.1 mg/l for > 5000 gpd, and 0.2 mg/l for < 5000 gpd). These limits are readily achievable for dental offices with adequate amalgam separators.

The following figure demonstrates the difference in compliance by 2003 in King County between an area with mandatory requirements and an area with voluntary requirements, despite the fact that the county's outreach program was targeted at the entire county. By 2005 there was a 97% compliance rate in the King County sewer service area – where separators are mandatory.



For these reasons, a growing number of states (9 states thus far) have opted for a mandatory requirement for amalgam separators in dental offices, either through law or regulation.

3 Costs of Controlling Amalgam Releases to Wastewater

The purpose of this section is to calculate the cost of removing Hg from the wastewater effluent of dental clinics. A formula to calculate this cost was developed and is explained below. (It should be noted that in order to account for uncertain developments in the future with regard to inflation, and also to facilitate cost comparisons, "constant dollars" of 2005 have been used in the calculations.)

$$C_t = N\,(E/10 + I/10 + O)$$

C_t = total cost for all US dental offices
N = number of dental offices requiring an installation
E = average equipment cost per separator (amortized over 10 years)
I = installation costs per separator
O = operating and maintenance costs per year

In order to derive the total cost (C_t) for installing dental amalgam separators nationally, the total number of dental offices (N) was obtained from ADA records. This information included the number of dentists in general practice as well as those operating as dental specialists. These specialists include oral surgeons, orthodontists, and cosmetic dental

specialists. It could be assumed that about half of these might require amalgam separators since patients would have dental work done that would affect restorative materials and allow this material to get into the wastewater discharge from that office. We chose to use only the number of GP dental facilities for our baseline and made the worst case scenario all GP and specialist facilities having to install the separators. ADA's records indicate the number of general practice dental facilities in the USA operating at 183,480. The additional registered dental specialist facilities number 44,635, for a total of 228,115 dental facilities in operation throughout the USA.

The average costs for equipment (E), installation cost (I), and operating and maintenance (O) were derived from an industry publication on the efficacy of amalgam separators. This document made comparisons between the costs and efficacies of amalgam separators and the American Dental Association's Best Management Practice (vacuum pump filters) for diverting amalgam materials from being transferred outside the facility in wastewaters.

Three manufacturers' amalgam separators were chosen for the comparison. Equipment cost ranged from a low of $595.00 to as high as $1195.00 and averaged $846.67. This cost was then amortized over 10 years as the expected life of the system, rather than the traditional five years which is the usual IRS timeline for fully depreciating equipment. We assumed that the lifetime of the operation of the unit was a more reasonable timeframe rather than the depreciation of costs since the units were designed to operate over a longer period of time.

Estimated installation costs by the manufacturer for all options were considered to be identical. To plumb a separator into the existing systems was defined as costing $250.00 for labor and miscellaneous materials not included with the separator. This cost was also amortized over a 10-year timeframe to reflect cost over the lifetime of the unit.

Operating and maintenance costs varied with the separators. These costs ranged from $474.00 to $570.00, and averaged $528.00 per year. Included in these costs are the removal and replacement with a new separator or replacement of the filter material under a maintenance contract depending on the manufacturer's recommended O&M guidance.

Final calculation of the total annual cost (C_f) only for GP dental facilities to install, operate and maintain dental amalgam separators was then calculated at $117 million, with the worst case scenario for installation at all dental facilities of about $145 million.

Based on IMERC data showing that at least 30 metric tons of Hg were used in the US in 2004 for amalgam fillings, it is evident that at least 60 million amalgam fillings were placed in 2004, and probably 2005 as well, since this quantity has been relatively stable since 2001.

Therefore, the "best-estimate" cost of adequately controlling the mercury releases from one amalgam filling in the United States through the use of typical separator equipment would run $1.95 per filling in 2005 dollars, or about $2.42 per filling if all specialist dental facilities are included in the calculation as well. Based on a further sensitivity analysis, i.e., varying some of the basic assumptions, this estimate could vary by perhaps plus-or-minus 20%.

Dental facility amalgam separator cost per amalgam filling		

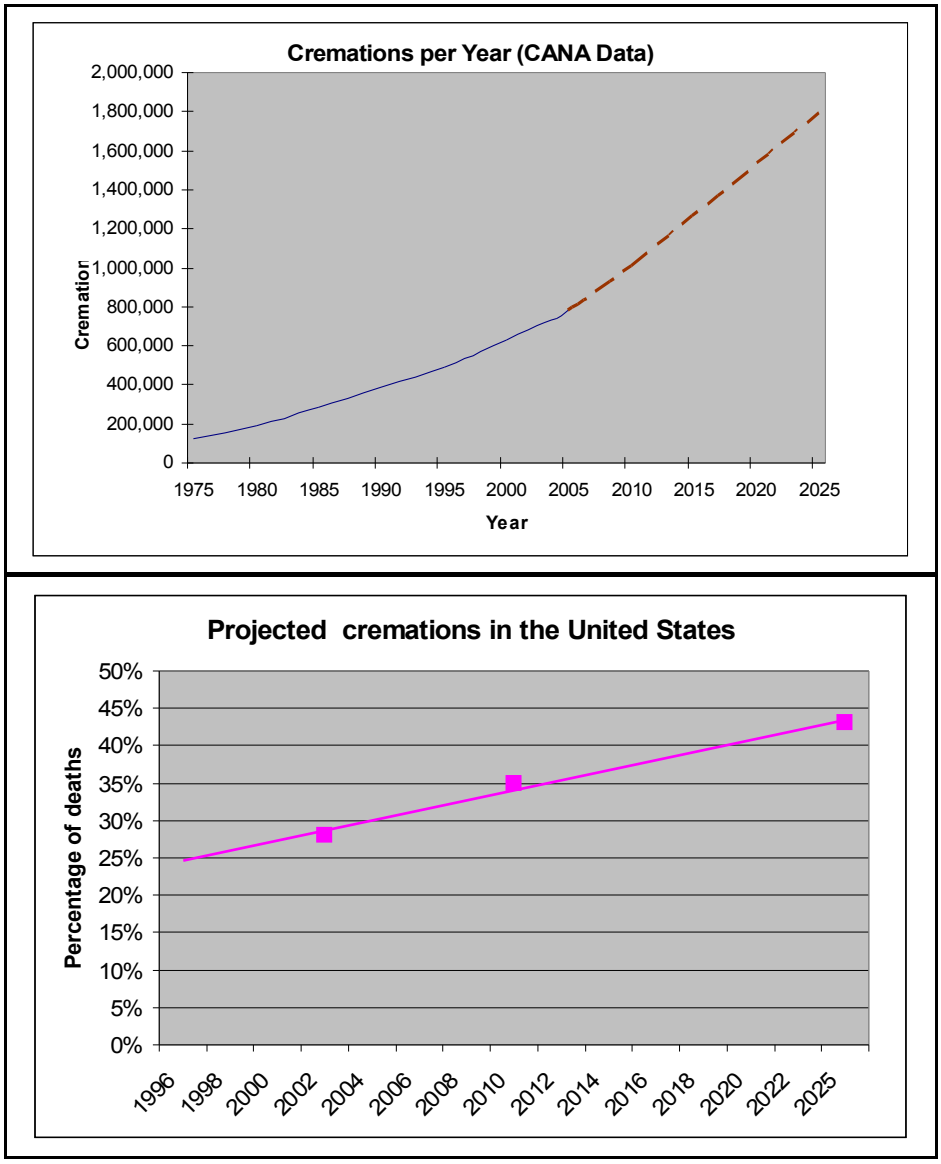
[All costs given in "2005 dollars"]			
	low	high	average
Separator equipment cost	$595	$1,195	$846.67
Equipment installation cost			$250.00
Combined equipment & installation cost			$1,096.67
Lifetime of separator equipment (yrs.)			10
Amortized equipment & installation cost per year			$109.67
Operating, maintenance, recycling cost per year	$474	$570	$528
Total equipment and operating cost per year per facility			$638
General practice (GP) dental facilities			183480
Registered dental specialist (RDS) facilities			44635
Total GP and specialist facilities			228115
Total cost for all GP facilities per year			$116,999,141
Total cost for all GP & RDS facilities per year			$145,461,408
Total mercury used in dental amalgam (metric tons/yr.)			30
Approx. mercury per amalgam filling (gram)			0.5
Number of amalgam fillings placed per year			60000000
Separator cost per filling for all GP facilities			**$1.95**
Separator cost per filling for all GP & RDS facilities			**$2.42**

4 Costs of Controlling Mercury Releases During Cremation

4.1 Cremation trends

Cremation is an increasingly common practice in the US, as the cost of burials rises. Cremation is typically carried out at a high temperature that vaporizes virtually all of the mercury in any dental amalgams, although it has proven quite difficult to balance the amount of mercury present in dental amalgams with measurements of mercury emissions in the crematorium flue gases. Often crematoria are located within cities and close to residential areas, and stacks tend to be relatively low (UNEP 2003). According to the Cremation Association of America, there are about 1,900 crematoria in the US. Nationally, over 30% of Americans are now cremated, a figure that is anticipated to rise to 43% by 2025. Figure 3 provides an indication of US cremation trends and projections to 2025.

Figure 3 – Projected cremations in the USA (1996-2025)



Source: Derived from CSGB 2004; Reindl 2007.

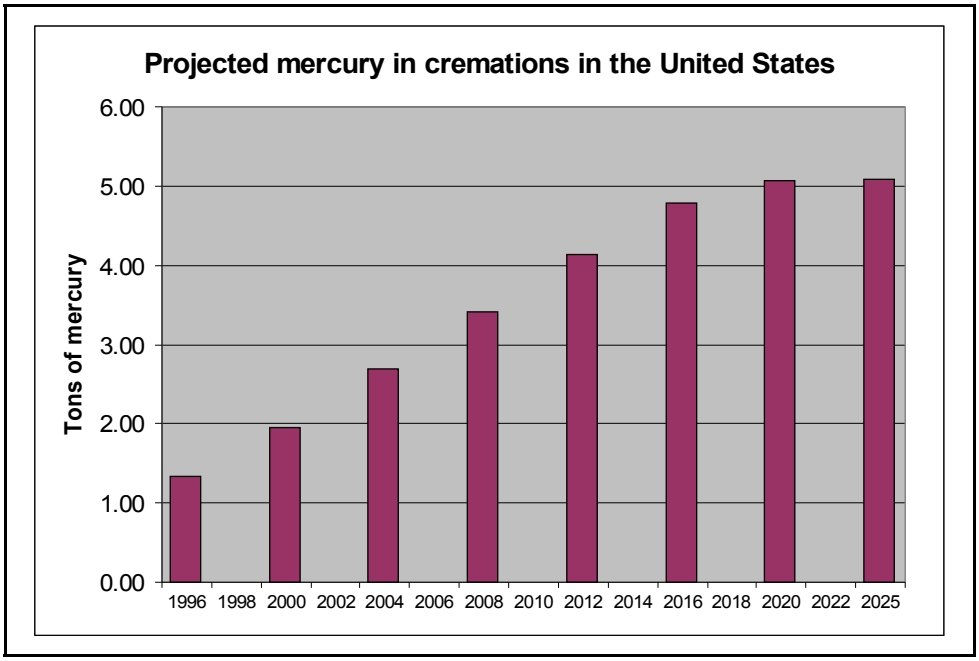
The 1998 Northeastern States Mercury Study estimated that each person cremated had an average of 2.9 grams of mercury in fillings.

Cain et al. (2007) have estimated that about 3.3 tons of mercury were emitted by crematoria in 2005. In the model used, 25% of these emissions were assumed attached to particulates, which would settle to the ground locally and be classified as land deposition, and 75% assumed to be elemental mercury emissions to the atmosphere. Based on a literature review including ground deposition studies in New Zealand and Norway (Reindl 2007), it appears justifiable to allocate up to 90% of the mercury entering crematoria as emissions to the atmosphere, with some of the balance retained, at least temporarily, in combustion equipment and the stack.

In the next 15 years, emissions from crematoria are expected to rise considerably. There are two simultaneous trends contributing to this: a rise in the average number of fillings per person cremated and a rise in the number of cremations. Figure 4 demonstrates how the

increasing number of cremations combines with the increased retention of teeth per person cremated to magnify the quantities of mercury potentially released during cremations.

Figure 4 – Rapidly increasing quantities of dental mercury to be dealt with by crematoria



Source: P. Maxson projections based on data in Reindl (2007)

4.2 Cremation mercury control costs

The purpose of this section is to calculate the cost of removing Hg from US crematoria flue gases. A formula to calculate this cost was developed and is explained below. (It should be noted that in order to account for uncertain developments in the future with regard to inflation, and also to facilitate cost comparisons, "constant dollars" of 2005 have been used in the calculations.)

$$C_f = (E + L*M + L*N_c*O)/(L*N_c*N_f)$$

C_f = total cost for a crematorium to treat mercury air emissions from one amalgam filling
E = the total cost for equipment installation and operation
L = lifetime of pollution control equipment
M = the additional annual maintenance cost for monitoring emissions
N_c = number of cremations per year
O = increased environmental service costs per cremation
N_f = number of fillings per cremation

We were unable to find any detailed examples of flue gas control devices installed at crematoria in North America to date. On the other hand, we were able to find a report from the UK by the Department of Environment, Food and Rural Affairs used in a consultation from 2003 and 2004 on *Mercury Emissions from Crematoria*. In that consultation, costs for installation of pollution control devices and their operation were given for the crematoria in operation in the UK.

For the flue gas control equipment installation and operation, the document specifies costs consisting of equipment, building and commissioning costs, the running costs including energy, maintenance and supervision, and the purchase and disposal of sorbent used for the removal of mercury.

For (*E*) the actual flue gas control equipment purchase and installation, based on real costs at facilities in operation in the UK, DEFRA estimated this cost at about $525,000 (£265,000). The cost of this pollution control equipment is assumed here to be amortized over 15 years (*L*). This was felt to be a reasonable lifespan for these pollution control systems, although we have also looked at the implications if we were to assume a lifespan of 20 years.

In order to determine the number of cremations carried out by the typical crematorium, we took the most recent Cremation Association of North America's report from 2006. In this report it is identified that in 2005, there were 1971 registered crematoria in the United States. Next, we took the 740,698 cremations in 2005 and divided that by the number of crematoria to get a throughput of the average facility of 376 cremations. Assuming some consolidation of crematoria in the future, especially as the total number of cremations (and the number of crematoria) are expected to increase significantly in the coming years, we roughly estimated 400 cremations per year (N_c) at the typical crematorium during the period 2005-2020.

Estimates for the increase in operation (*O*) costs due to the presence of the pollution control were based on real cost data and placed at $17.43 (£8.80) per person cremated. These are defined as the cost for environmental services, and include the costs of additional labor, sorbent purchase and disposal, and any increase in costs for operation.

Additional maintenance costs (*M*) were included by DEFRA to reflect the need for monitoring the emission source for compliance assurance. This was estimated to run about £500-1000 per crematorium per year. For simplification, we used a conservative annual cost of $2000.

As the typical mercury releases during one cremation are estimated at 3 grams, it may be assumed that the average person cremated has about 6 amalgam fillings (N_f).

The final numbers we arrived at were on the order of $660,000 total costs (in 2005 dollars) for one crematorium to deal with 6,000 cremations comprising some 36,000 amalgam fillings over the period 2005-2020.

Based on these figures, the "best-estimate" cost (C_f) of adequately controlling the mercury releases from one amalgam filling at a crematorium in the United States would run $18.32 in 2005 dollars. Based on a further sensitivity analysis, i.e., varying some of the basic assumptions, this estimate could vary by perhaps plus-or-minus 30%.

Crematorium Hg treatment cost per filling	

[All costs given in "2005 dollars"]	
Best estimate assumptions:	
Take a single facility as an example	
Install pollution control equipment in "base year"…	2005
Investment for pollution control equipment	$525,000
Lifetime of pollution control equip. (yrs.)	15
Actual US cremations in base year	740698
Number of US crematoria in base year	1971
Actual US cremations per crematorium per year	376
Assume average yearly cremations 2005-2020	400
Total cremations handled by this equipment 2005-2020	6000
Average Hg per cremation (grams)	3
Average Hg per amalgam filling (grams)	0.5
Average number of amalgam fillings per cremation	6
Total amalgam fillings handled by this equipment	36000
Additional environmental services cost per cremation	$17.43
Total additional environmental services cost	$104,580
Annual emissions monitoring cost	$2,000
Total emissions monitoring cost for this equipment	$30,000
Total costs for this pollution control equipment	$659,580
Total fillings cremated and sequestered	36000
Effective crematorium Hg treatment cost per filling	**$18.32**

It should also be noted that the basic flue gas controls for mercury will also control dioxins/furans, so a co-benefit of the mercury controls would also be achieved.

5 Conclusion: Costs of Composites Similar to Amalgam When Pollution Control Costs Are Factored In

Dentists typically charge more for composite fillings than for amalgams. Dental outlets and insurance companies say these cost differences are largely due to increased time required to place composite fillings, especially in rear teeth. Consolidating dental fees in urban areas across the US, as in the table below, confirms the estimate of dental colleagues that the cost of an average composite filling is 20-25% higher than an average amalgam filling.

Silver Amalgam Fillings, Permanent teeth	
Ave. 1, 2 and 3 surface	$108
Composite Resin Filling - Front & Rear teeth	
Ave. 1, 2 and 3 surface	$139

Reference: Dental fees (2004)

In order to understand the true cost of amalgam use, however, one needs to factor in “external” costs associated with preventing mercury pollution due to amalgam. This pollution comes primarily from wastewater releases during placement and removal of amalgam, and the growing culturally acceptable practice of cremation. Ultimately, society pays for the uncontrolled mercury pollution from dental amalgam through additional pollution control costs, the loss of common resources, and the heath effects associated with mercury contamination.

Even with chair-side traps in place for biologic material control and vacuum pump filters to remove materials suctioned from a patient's mouth, dental offices can release amalgam waste as very fine material that eventually ends up at sewage treatment plants. Here, they add to the other dental mercury that we inhale or ingest that passes through our systems and into sewerage. While our mercury dose comes mostly from food (fish), one must add the mercury continually released from amalgam in our mouths. Specifically because of dental mercury, many publicly owned treatment works are out of compliance with water quality standards for their effluent. Where separators have been required, effluent levels have returned to compliance with Clean Water Act standards.

Controls that remove more than 95% of amalgam from dental office wastewater have been used for years in many practices where dentists have voluntarily installed them as a choice of conscience. Amalgam separator technology is well-refined and has been in use in numerous U.S. Armed Forces dental clinics, including a very large facility operated at the Great Lakes Naval Training Center in North Chicago, IL.

Amalgam reaches the end of its useful life when we do. As demand for cremation as a culturally-acceptable practice grows, and more people retain their teeth throughout their lives, the release of mercury into the air from uncontrolled cremation flue gases increases the amount of mercury that amalgam is responsible for releasing to the environment. As with other combustion processes used to destroy materials – such as medical waste incinerators – cost-effective pollution controls for mercury exist that can be applied to crematoria.

The following table shows that when only two of these external costs are included, the real cost of using amalgam is already quite close to that of mercury-free fillings.

	Amalgam	Composite
Filling cost at the dental clinic	$108	$139
"External" costs:		
- separator mercury removal	$2	--
- crematorium mercury removal	$18	--
- municipal solid waste mercury removal, etc.	??	--
"Full cost" of an amalgam filling	**$128+**	**$139**

Drawing obvious conclusions from this simple cost comparison, combined with the clear risks of using amalgam, as finally admitted by the FDA, Congress should follow in the path already blazed by some progressive European countries that have decided to adopt strong measures to either discourage or ban amalgam use.

Measures that Congress should consider include:

- Require dental clinics that replace amalgam to install and properly operate amalgam separators, and to report annually on quantities of mercury collected.
- Assess a modest user fee of $30.00 for the production of each additional mercury tooth filling, payable by the manufacturer at time of sale. Funds collected should be placed into a designated account to cover the costs of controlling mercury pollution.
- Phase-out the use of mercury tooth fillings within the next 3-5 years.

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AFFIDAVIT: AN EVALUATION OF DENTAL AMALGAM AND IT'S ABILITY TO INJURE HUMAN HEALTH

2 February 2010

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 20 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. It is my opinion that the most critical mistake of modern medicine is the lack of understanding of the synergistic toxic effects associated with mercury and organic mercury toxicity. Synergistic effects drive the toxic level of mercury exposure to levels much lower than expected and can change the toxicity profiles substantially.

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 shows them to cause adverse effects in animals and humans at very low levels and that a "retention toxicity" where seemingly non-toxic levels, when constantly present as in dental amalgam vapors, can slowly build up in tissues causing severe illnesses. 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. Much of the mercury in seafood is bound to selenium and render much less toxic to mammals. 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. Recent studies show that released Hg vapor from dental amalgams setting quietly in sealed test tubes is in the range of 4 to 21 μg/cm2/day.[55] This surface area is approximately the size of a small, single spill amalgam filling. It has been shown that fecal mercury levels average about 65 μg per day in amalgam bearers. These are exact measurements and agree well with each other. However, many publications "estimate" the amount of mercury released by amalgams based on the blood or urine levels. In one study it was stated that "The integrated daily Hg dose absorbed from amalgam was estimated up to 3 microg for an average number of fillings and at 7.4 for a high amalgam load." [50] The "estimated levels" defy explanation as the numbers would not allow for more than 2 amalgam fillings and would never reach the 65 μg average mercury in fecal material plus the urinary mercury excretion. We also know that abrasion by a toothbrush elevates the daily mercury excretion in sealed amalgams by over 10-fold. This points out the major problem of most reported experiments on dental amalgam, the amount of

patient exposure is mostly "estimated" and almost always estimate very low compared to the level measured outside the mouth under rigorously maintained conditions. However, even after amalgam removal, inorganic Hg dropped rapidly in plasma and red cells, stabilizing at 27% of pre-removal levels after 60 days. Concentrations of organic Hg in plasma remained unchanged, indicating no change in dietary uptake of organic Hg.[50]

The 73% decrease in blood/plasma mercury levels supports the concept that dental amalgams account for the vast majority of inorganic mercury found in the human body of amalgam bearers.[50] However, the "estimated" levels of mercury released from amalgams in this study (3μg on average) is refuted by other studies which found oral emission of mercury ranged up to 125 μg Hg/24 h, and urinary excretions ranged from 0.4 to 19 μg Hg/24 h.[51] Also, fecal excretions ranged from 1 to 190 μg Hg/24 h, which was 100 times the mean intake of total Hg from a normal Swedish diet. These data, done on the same patients, also point out explicitly that urinary excretions do not reflect amalgam release or exposure of mercury and that the concept of low 3 to 8 micrograms release of mercury per day as an estimate of amalgam contribution to human exposure is not at all accurate, in fact it is absurdly low.[50] In an earlier paper from this same group they had stated that "In saliva, there was an exponential decline in the Hg concentration during the first 2 weeks after amalgam removal (t 1/2 = 1.8 days)" and concluded that amalgam fillings were a significant source of mercury in saliva and feces."[52] However, they later stated "The Hg concentrations in saliva remained elevated for at least 1 wk, suggesting that dissolved Hg vapor is not the major source of mercury in mixed saliva."[50] They also reported that fecal levels in amalgam bearers were 11.7 times higher than found in amalgam free subjects (2.7 vs 0.23 mumol Hg/kg dry weight, $p < 0.001$) and increased 2 days after amalgam removal to a median 280 mumol Hg/kg dry weight, a fecal increase of over 100 fold.[52] This is one of the negative effects of placing amalgams, they may have to be removed and repaired and doing so can lead to a bolus exposure to mercury.

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 inhibition of the mercury 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 at year 7 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.

Since this data in the Children's amalgam trial only evaluated urine mercury it must be considered with caution as this measure does not accurately reflect what may be happening with regards to total exposure, excretion or retention. For example, research has shown that the oral emission or mercury in amalgam bearers ranged up to 125 micrograms Hg/24 h, and urinary excretions ranged from 0.4 to 19 micrograms Hg/24 h.[42] In 10 subjects, urinary and fecal excretions of mercury and silver were also measured. Fecal mercury excretions ranged from 1 to 190 micrograms Hg/24 h. The worst-case individual showed a fecal mercury excretion amounting to 100 times the mean intake of total Hg from a normal Swedish diet.[42] These studies also imply that urinary measures are not indicative of the total mercury intake at all and the mercury levels reported are orders of magnitude higher than that speculated by the ADA from "estimations" by dental researchers.[34,50]

5) The pro-amalgam group in the USA has "estimated" the amount of mercury excreted from amalgams by using urine mercury levels[34], which is obviously invalid, since over 90% of mercury is excreted via fecal routes, not through the urine.[41] 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 "the 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 and even this is not accurate in individuals who are low in glutathione and unable to effectively excrete mercury.

In a recent study the level of mercury in feces and saliva were measured in amalgam free controls and amalgam bearers before and after removal of the amalgams.[41] Before removal, the median Hg concentration in feces of amalgam bearers was more than 10 times higher than in samples from an amalgam free reference group consisting of 10 individuals (2.7 vs 0.23 mumol Hg/kg dry weight, $p < 0.001$). A considerable increase of the Hg concentration in feces 2 days after amalgam removal (median 280 mumol Hg/kg dry weight) was followed by a significant decrease. Sixty days after removal the median Hg concentration was still slightly higher than in samples from the reference group.[41]

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. For example, mercury is a potent immunomodulator and a well known relationship exists between impaired B-cell receptor (BCR) signal strength and autoimmune disease. A group that had previously shown that in mouse B cells, non-cytotoxic concentrations of inorganic mercury interfered with BCR-mediated growth control, suggesting that BCR signal strength was impaired by Hg^{+2}, later showed that the kinetics and magnitude of BCR-mediated activation of ERK-MAPK are markedly attenuated in these same cells and in spleenic B cells that have been exposed to low and nontoxic burdens of Hg^{+2}. [53] It therefore appears plausible that suppression of the immune system can occur at levels of mercury that are not considered toxic by many.

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 disease. 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]

Hg has been shown to induce autoimmune disease in susceptible animals with effects including overproduction of specific autoantibodies and pathophysiologic signs of lupus-like disease. However, these effects are only observed at high doses of Hg that are above the levels to which humans would be exposed. A study was done to test the hypothesis that Hg does not cause autoimmune disease directly, but that mercury interacts with triggering events, such as genetic predisposition, exposure to antigens, or infection, to exacerbate autoimmune disease.[46] They found that treatment of mice not susceptible to Hg-induced autoimmune disease with very low doses and short term exposures of inorganic Hg (20-200 μg/kg) exacerbates disease and accelerates mortality in the graft versus host disease model of chronic lupus. Also, low dose Hg exposure increased the severity and prevalence of experimental autoimmune myocarditis. In a human study involving Amazonian populations exposed to Hg through small-scale gold mining, with and without current or past malaria infection they reported a significantly increased prevalence of antinuclear and antinucleolar antibodies and a positive interaction between Hg and malaria. They proposed that their findings supported a new model for Hg immunotoxicity. Namely, mercury can serve as a co-factor in autoimmune disease, increasing the risks and severity of clinical disease in the presence of other triggering events, either genetic or acquired.[46]

It is well known that the initiation and severity of systemic autoimmune diseases is influenced by genetic and environmental factors, including bacterial infections. To explore the involvement of innate immunity in mercury-induced autoimmunity in mice a recent study employed bacterial lipopolysaccharide (LPS), which non-specifically activates the immune system.[47] Resistant mice were rendered susceptible to mercury-induced autoimmunity by co-administration of LPS. These findings indicate that activation of the innate immune system by bacterial infection plays a key role in both the induction and severity of mercury induced autoimmunity. [47]

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.

Another study showed the irreversible effects of occupational exposure to color blindness. About 3 years after exposure the mercury level had dropped to 1.4 ± 1.4 μg/g creatinine for exposed patients, a level considered non-toxic, compared with 0.5 ± 0.5 μg/g creatinine for controls. However, the findings indicate that following a long-term occupational exposure to Hg vapor, even several years away from the source of intoxication, color vision impairment remains irreversible.[43] Such studies point out that mercury damage cannot be evaluated by the current level of mercury in the urine. Another study evaluated the automated visual field perimetry in 35 ex-workers (30 males; 44.20±5.92 years) occupationally exposed to mercury vapor and 34 controls (21 males; 43.29±8.33 years). Compared to controls, visual field sensitivities of the Hg exposed group measured were lower for the fovea as well as for all five eccentricity rings of vision ($p<0.05$).[49]

Another study compared neutrophil function in non-exposed and exposed populations (with a mean +/- s.d. urinary mercury concentration of 24.0 +/- 20.1 μg/1 creatinine) in which 44 of the workers urinary mercury levels were below the accepted threshold level (TLV) of 50 μg/1 creatinine.[45] The neutrophil functions were significantly reduced in the mercury-exposed workers compared with the controls. In 28 of these workers, neutrophil chemotaxis was re-evaluated 6 months later after the daily exposures were decreased significantly and urinary mercury concentrations showed a significant reduction. However, neutrophil migration *did not return* to within the normal range in these subjects. These results suggest that a current 'safe' level of mercury exposure may lead to impairment of neutrophil function.[45]

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] A more recent study states that mercury was elevated in the plasma of Alzheimer's disease patients when compared to age-matched controls.[39]

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. A recent report states "Levels of Hg in the cord blood were significantly associated with the number of maternal amalgam fillings ($rho=0.46$, $P<0.001$) and with the number of years since the last filling ($rho=-0.37$, $P<0.001$); these associations remained significant after adjustment for maternal age and education. Dental amalgam fillings in girls and women of reproductive age should be used with caution, to avoid increased prenatal Hg exposure.[40] 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. Another study examined the effects of inorganic mercury (mercuric chloride) exposure exclusively through maternal milk on the biochemistry related to oxidative stress in the cerebellum of weanling mice.[54] Their results showed that with pups, the lactational exposure to mercury increased cerebellar glutathione reductase activity as well as cerebellar lipoperoxidation. However, these changers were not observed in dams. The authors concluded that their results imply that inorganic mercury exposure through maternal milk is capable of inducing motor deficits as well as biochemical changes in the cerebellum of weanling mice.[54]

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. A study confirming this was done on 7 male plus 7 female rats that were exposed to the same level of thimerosal. At doses of 38.4–76.8 mg/kg using 10% DMSO as diluent, seven of seven male mice compared to zero of seven female mice tested succumbed indicating a definite gender effect.[44] Since boys are dramatically more susceptible to neurological illnesses, such as autism, than are girls it seems reasonable to consider environmental insults from mercury and organic mercury as the most likely cause.

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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56. **Transport of thiol-conjugates of inorganic mercury in human retinal pigment epithelial cells. Christy C. Bridges[a], Jamie R. Battle[a] and Rudolfs K. Zalups[a]** data suggest that Cys-S-Hg-S-Cys and Hcy-S-Hg-S-Hcy are taken up into ARPE-19 cells by Na-dependent amino acid transporters, possibly systems B0, + and ASC. These amino acid transporters may play a role in the retinal toxicity observed following exposure to mercury.
57. **Amalgam dental fillings and hearing loss.**
Rothwell JA, **Boyd PJ**. Int J Audiol. 2008 Dec;47(12):770-6. In this study we investigated the effects of amalgam dental fillings on auditory thresholds. The results suggest an association between more amalgam fillings and poorer thresholds at higher frequencies, which could contribute to presbyacusis in developed countries.
58. **Enhanced toxicity for mice of pertussis vaccines when preserved with Merthiolate.**
Nelson, E.A. Gottshall, R.Y. Appl Microbiol. 1967 May;15(3):590-3. Pertussis vaccines preserved with 0.01% Merthiolate are more toxic for mice than unpreserved vaccines prepared from the same parent concentrate and containing the same number of organisms. An increase in mortality was observed when Merthiolate was injected separately, before or after an unpreserved saline suspension of pertussis vaccine.
59. **Fish from 291 streams test positive for mercury in USGS survey**"Data on Mercury in Water, Bed Sediment, and Fish from Streams Across the United States, 1998–2005," the United States Geological Survey (USGS), a division of the Department of the Interior (DOI) More than two-thirds of the fish exceeded the U.S. EPA level of concern for fish-eating mammals.
60. HEPATITIS B VACCINATION OF MALE NEONATES AND AUTISM
CM Gallagher, MS Goodman, Graduate Program in Public Health, Stony Brook University Medical Center, Stony Brook, NY Annals of Epidemiology Vol. 19, No. 9 ABSTRACTS (ACE) September 2009: 651–680 p. 659 P24*Boys who received the hepatitis B vaccine during the first month of life had 2.94 greater odds for ASD (Nz31 of 7,486; OR Z 2.94; p Z 0.03; 95% CI Z 1.10, 7.90) compared to later- or unvaccinated boys. Non-Hispanic white boys were 61% less likely to have ASD (ORZ0.39; pZ0.04; 95% CIZ0.16, 0.94) relative to non-white boys.CONCLUSION: Findings suggest that U.S. male neonates vaccinated with hepatitis B vaccine had a 3-fold greater risk of ASD; risk was greatest for non-white*
61. **Gender-selective toxicity of thimerosal**

Donald R. Branch Departments of Medicine and Laboratory Medicine and Pathobiology, University of Toronto, 67 College St., Toronto, Ontario, Canada M5G 2M1 Experimental and Toxicologic Pathology (accepted 22 July 2008) **At doses of 38.4–76.8 mg/kg using 10% DMSO as diluent, seven of seven male mice compared to zero of seven female mice tested succumbed to thimerosal. it was completely unexpected to observe a difference of the MTD between male and female mice. Thus, our studies, although not directly addressing the controversy surrounding thimerosal and autism, and still preliminary due to small numbers of mice examined, provide, nevertheless, the first report of gender-selective toxicity of thimerosal and indicate that any future studies of thimerosal toxicity should take into consideration gender-specific differences.**

62. **Mercury in saliva and feces after removal of amalgam fillings.**
Björkman L, et al. Toxicol Appl Pharmacol. 1997 May;144(1):156-62. Department of Basic Oral Sciences, Karolinska Institutet, Stockholm, Sweden. The purpose of this study was to obtain data on Hg concentrations in saliva and feces before and after removal of dental amalgam fillings. **Before removal, the median Hg concentration in feces was more than 10 times higher than in samples from an amalgam free reference group consisting of 10 individuals (2.7 vs 0.23**

mumol Hg/kg dry weight, p < 0.001). Sixty days after removal the median Hg concentration was still slightly higher than in samples from the reference group. In saliva, there was an exponential decline in the Hg concentration during the first 2 weeks after amalgam removal (t 1/2 = 1.8 days). It was concluded that amalgam fillings are a significant source of Hg in saliva and feces.

63. **Human exposure to mercury and silver released from dental amalgam restorations.**

Skare, I and Engqvist, A. Arch Environ Health. 1994 Sep-Oct;49(5):384-94. National Institute of Occupational Health Stockholm, Sweden. In 35 healthy individuals, the number of amalgam surfaces was related to the emission rate of mercury into the oral cavity and to the excretion rate of mercury by urine. Oral emission ranged up to 125 micrograms Hg/24 h, and urinary excretions ranged from 0.4 to 19 micrograms Hg/24 h. In 10 cases, urinary and fecal excretions of mercury and silver were also measured. Fecal excretions ranged from 1 to 190 micrograms Hg/24 h and from 4 to 97 micrograms Ag/24 h. Except for urinary silver excretion, a high interplay between the variables was exhibited. The worst-case individual showed a fecal mercury excretion amounting to 100 times the mean intake of total Hg from a normal Swedish diet. With regard to a Swedish middle-age individual, the systemic uptake of mercury from amalgam was, on average, predicted to be 12 micrograms Hg/24 h.

64. **Maternal amalgam dental fillings as the source of mercury exposure in developing fetus and newborn.** *Journal of Exposure Science and Environmental Epidemiology* (2008) **18,** 326–331. Lubica Palkovicova[a], Monika Ursinyova[a], Vlasta Masanova[a], Zhiwei Yu[b] and Irva Hertz-Picciotto[b]". **The main aim of this analysis was to assess the relationship between maternal dental amalgam fillings and exposure of the developing fetus to HgThe median values of Hg concentrations were 0.63 mcg/l (range 0.14–2.9 g/l) and 0.80 mcg/l (range 0.15–2.54 mcg/l) for maternal and cord blood, respectively. Levels of Hg in the cord blood were significantly associated with the number of maternal amalgam fillings (=0.46, *P*<0.001) and with the number of years since the last filling (=- 0.37, *P*<0.001); these associations remained significant after adjustment for maternal age and education. Dental amalgam fillings in girls and women of reproductive age should be used with caution, to avoid increased prenatal Hg exposure. None of the cord blood Hg concentrations reached the level considered to be hazardous for neurodevelopmental effects in children exposed to Hg *in utero* (EPA reference dose for Hg of 5.8 mcg/l in cord blood).**

65. **Irreversible color vision losses in patients with chronic mercury vapor intoxication**
66. **CLÁUDIA FEITOSA-SANTANA[a1a2] [c1], MIRELLA T.S. BARBONI[a1a2], NESTOR N. OIWA[a1a2], GALINA V. PARAMEI[a3], ANA LUISA A.C. SIMÕES[a2], MARCELO F. DA COSTA[a1a2], LUIZ CARLOS L. SILVEIRA[a4a5] and DORA F. VENTURA[a1a2] Visual Neuroscience (2008), 25:487-491. This longitudinal study addresses the reversibility of color vision losses in subjects who had been occupationally exposed to mercury vapor. These findings indicate that following a long-term occupational exposure to Hg vapor, even several years away from the source of intoxication, color vision impairment remains irreversible.**
67. **Alzheimer's Metal Concentrations in Plasma and Cerebrospinal Fluid in Patients with Disease. Dement Geriatr Cogn Disord. 2008 May 5;25(6):508-515. Gerhardsson L, Lundh T, Minthon L, Londos E. <u>The plasma concentrations of manganese and total mercury were significantly higher in subjects with AD (p < 0.001) and AD + vasc (p </= 0.013) than in controls.</u> <u>Besides the raised plasma mercury concentrations, no consistent metal pattern in plasma or CSF was observed in patients with AD.</u>**
68. **Blood mercury levels rising among U.S. women. Dr. Dan Laks, UCLA August 09. Using data from the U.S. Centers for Disease Control and Prevention's National Health and Nutrition Examination Survey (NHANES), a researcher from the University of California, Los Angeles, found that while inorganic mercury was detected in the blood of 2 percent of women aged 18 to 49 in the 1999-2000 NHANES survey, that level rose to 30 percent of women by 2005-2006. "My study found compelling evidence that inorganic mercury deposition within the human body is a cumulative process, increasing with age and overall in the population over time," study author and neuroscience researcher Dan R. Laks said in an UCLA news release. "My findings also suggest a rise in risks for disease associated with mercury over time.“ Laks also found a connection between levels of the pituitary hormone lutropin and chronic mercury exposure, which he said might help explain mercury's link to**

neurodegenerative disease. “these results suggest that chronic mercury exposure has reached a critical level where inorganic mercury deposition within the human body is accumulating over time," Laks said. "It is logical to assume that the risks of associated neurodevelopmental and neurodegenerative diseases will rise as well."

The Scientific Case Against Amalgam

Dental amalgam has been controversial ever since it was introduced, early in the nineteenth century, because of its mercury content. People of the Napoleonic era knew full well that mercury was poisonous, and the best that anyone has ever claimed about amalgam is that the mercury exposure may be too small to hurt anyone. Over time, though, a great body of evidence has accumulated showing that mercury is release from amalgam in significant quantities, that it spreads around the body, including from mother to fetus, and that the exposure causes physiological harm. A growing number of dentists, physicians, researchers, citizen activists, politicians, and regulators have come to the conclusion that the time has come to consign amalgam to the "dustbin of history." This article will sketch out the main points of the scientific case against amalgam.

The history of amalgam is, of course, familiar. The alchemists of China and Europe were fascinated with mercury, the only metal that is liquid at room temperature, and which would evaporate with mild heat. They knew that liquid mercury could dissolve powders of other metals, such as tin, copper or silver. European methods for using a paste of silver shavings dissolved in mercury as dental restorations were introduced to America by the Crowcour brothers about 1830. Problems with excessive expansion in early amalgam were solved in time by adding the other, now customary metals – tin, zinc, and copper. The formula and technique for using amalgam has remained virtually unchanged for the past one hundred years.

The "first amalgam war" started almost immediately. The toxic effects of mercury, including dementia and loss of motor control, were common knowledge in the post–Napoleonic era, and many dentists objected to the obvious disadvantage of using such a dangerous material in people's mouths. In 1845, the American Society of Dental Surgeons asked its members to sign a pledge never to use it. The economics were compelling, though,

as they remain today. At a time when the only other feasible restorative material was gold, amalgam looked to be the restorative material for the masses. Then, as today, patients did not show signs of acute poisoning as they left the dentist's office, so there did not appear to be a problem. As the use of amalgam grew, the American Society of Dental Surgeons fell apart, and in 1859, the pro–amalgam faction formed the American Dental Association, the same organization that leads the dental profession in the USA to this day, and remains steadfast in its defense of amalgam.

The "second amalgam war" was provoked in the 1920's by Professor Alfred E. Stock, a leading chemist at the Kaiser Wilhelm Institute in Germany. Adverse effects on his own health from mercury in the lab led him to question the supposed safety of mercury from dental amalgam. His research concluding that there were adverse health effects was published in leading scholarly journals of the day. It touched off a debate that raged through the 1930's without a clear resolution, only to fade away in the storm of World War II.

We are currently in the advanced stages of the third amalgam war. The argument was reopened in the late 1970's, as modern methods of detecting the presence of trace amounts of mercury were introduced, including mass spectrophotometry and the Jerome mercury vapor detector. We have accumulated a formidable body of evidence establishing the chain of toxic events: 1) amalgam releases significant amounts of mercury; 2) the mercury distributes to tissues around the body, and is the biggest source of mercury body burden; 3) the mercury from amalgam crosses the placenta and into breast milk, resulting in significant pre- and post-partum exposures for infants; and 4) adverse physiological changes occur from that exposure on the immune, renal, reproductive and central nervous systems, as well as the oral and intestinal flora.

A succinct but comprehensive review of this topic is: Lorscheider, FL, Vimy, MJ, Summers, AO. *Mercury exposure from "silver" tooth fillings: emerging evidence questions a traditional dental paradigm.* FASEB J. 9: 504-508 (1995). FASEB is the Federation of American Societies for Experimental Biology, and their journal is one of the world's highest rated scientific sources. They have published a number of important papers on this issue.

Organized dentistry could examine the emerging evidence and decide that it is time to change their minds about the traditional dental paradigm, although it appears more likely that they'll soldier on in denial. The four percent of dentists who think of biocompatibility first have long since abandoned amalgam, and the greater number who have joined the "esthetic dentistry" movement have, by and large, moved away from it as well. About 27% of US dentists are reported in 2001 to be practicing mercury free.[1] Will our profession accept a future of scientific progress and handle the legacy of amalgam in an enlightened way, or will we go down like DDT and asbestos, like big tobacco and nuclear waste?

This brief review will touch on the high points, the blockbusters in the case against amalgam. There is a vast literature on the subject, which can be further accessed in other articles available on this website, the *Bibliography of Mercury Topics*, the *Swedish Government 2003 Report on Dental Amalgam*, and *Status Report on Dentistry in the Environment*, and on other websites provided in the Links section.

Amalgam releases significant quantities of mercury.

What kind of metal is amalgam? All the technical information we learn in dental school about an intermetallic matrix of gamma and mu phases only serves to obscure the fact that the mercury is not all reacted. Figure 1 is a photomicrograph of a polished metallurgic sample of amalgam which has been pressed on by a micro-probe.[2] Where the probe touched the surface, droplets of free liquid mercury are squeezed out into view. This process does not require heating the sample, as some have objected; it was repeated down to the temperature of liquid nitrogen.[3]

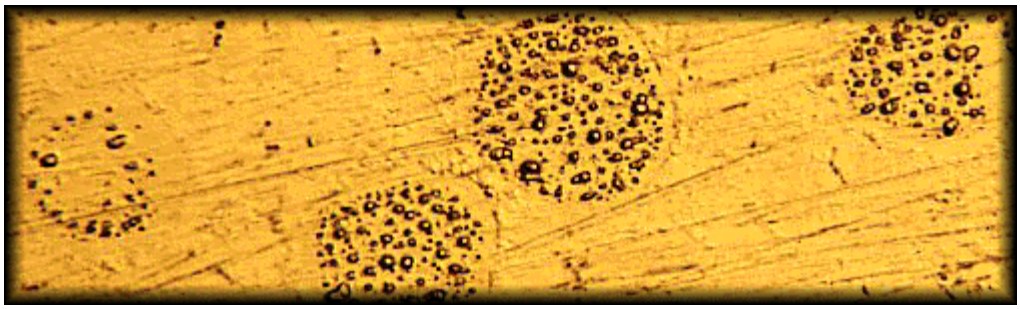

Figure 1 – Microscopic beads of liquid mercury expressed from the surface of amalgam metallurgical sample, following pressure from a microprobe. (from Masi, 1994)

The clearest, most gut wrenching way to comprehend that amalgam contains free mercury was discovered by IAOMT member Roger Eichmann, DDS. An extracted tooth containing an old amalgam filling is held in the light of a miner's blacklight, which is nothing but a fluorescent tube without phosphors – a pure mercury vapor discharge lamp. By the principles of atomic absorption spectrophotometry, the only cold vapor that could absorb the wavelength of mercury emission light and cast a shadow would be that of mercury itself. The filling in the photo in figure 2 has been dipped in 110^0 F water, to simulate the type of mild heating one would expect from chewing, grinding the teeth, or drinking hot liquids. The smoke visibly emerging is the shadow of mercury vapor. A video version of this alarming demonstration entitled, "The Smoking Tooth," is available for download on the home page of this website. Click on the link, and watch the steady emission of mercury vapor, like smoke from a smoldering fire, from a filling that had been in someone's mouth for years. A pdf version with still photos is available for those without broadband internet.

Figure 2 – The smoking tooth.

This graphically dramatic process was hinted at by the fact that old amalgams contain significantly less mercury than new ones.[4 5] It was quantified in the human mouth by Svare, et. al., Gay et. al., Vimy and Lorscheider, and others.[6 7 8 9 10] By using a Jerome Mercury Vapor Detector and other methods, these groups were able to measure the mercury content of the air in the mouths of people with or without amalgams, before and after chewing. The baseline mouth air of people with amalgams contains more mercury than that of people without amalgams. After ten minutes of chewing gum, the mercury concentration in mouth

air does not change in subjects without amalgams, while for those with amalgam fillings it increases 8 – 10 fold, and remains elevated for at least 90 minutes.

Vimy and Lorscheider derived an average absorbed mercury dose of 10 μg per day from amalgam fillings from their measurements of mouth air.[9] Other groups have reported varying estimates. On the low end, Mackert[11] and Berglund et. al.[12], by applying assumptions and inferences concerning how much mouth air is actually inhaled, arrived at average daily doses for subjects with twelve or more amalgam surfaces, of 1.83 and 1.7 μg, respectively (not zero). The question of inhaling mouth air should be moot, though, because elemental mercury vapor is lipophilic, and is absorbed easily through cell membranes and mucosal barriers. On the high end, Patterson et. al.[13] reported absorbed doses of as much as 27 μg per day. Skare and Engqvist,[14] by metabolic methods, arrived at a figure of 12 μg per day for a group of subjects with an average of 47 amalgam surfaces.

The current best accepted reference on absorbed dose of mercury from amalgam fillings comes from the World Health Organization proceedings of 1991 [15], which was the report of a meeting of toxicologists and environmental health specialists (few dentists and no industry lobbyists, the opposite of the 1997 WHO meeting!). The conclusion of that group was that the average person in the industrial world with an average number of amalgam fillings, and no occupational exposure to mercury would absorb between 3 – 17 μg per day, with an average of 10 μg, from the fillings; 2.3 μg from all dietary sources; and 0.3 μg from all other environmental sources.

Richardson[16] presented a chart (figure 3) summarizing seventeen separate estimates of mercury exposure due to amalgam in adults. The range of the estimates intersects with limits recommended for non-occupational exposure by several agencies, including the Agency for Toxic Substances and Disease Registry of the US Public Health Service, Health Canada, and the US Environmental Protection Agency, as shown by the vertical red lines.

Mercury distributes to tissues around the body.

One of KO Frykholm's experiments in his landmark 1957 study [17] of mercury in amalgam involved giving eight volunteers four new fillings each, labeled with radioactive ^{203}Hg. He was able to detect excretion of the radioactive mercury in urine for seven days, and in feces for thirteen days. From this he concluded that the release of mercury from the fillings, while not zero, was self limiting, and should therefore be no problem for the exposed people. The "no problem" conclusion was not supported by toxicology, and there was no discussion of the possible retention in the body of some of that radioactive mercury. Nevertheless, this study has been relied upon by supporters of amalgam ever since, as proof that there is "no problem."

In the late 1980's, Murray Vimy, Fritz Lorscheider and their group undertook to use radioactive mercury to examine the question of tissue retention of mercury from amalgams fillings, in a series of experiments supported by the IAOMT. Vimy, a founding member of the IAOMT, is a general dentist in Calgary, Alberta, and Lorscheider, now retired, was a professor of physiology at the University of Calgary Medical School. They enlisted the help

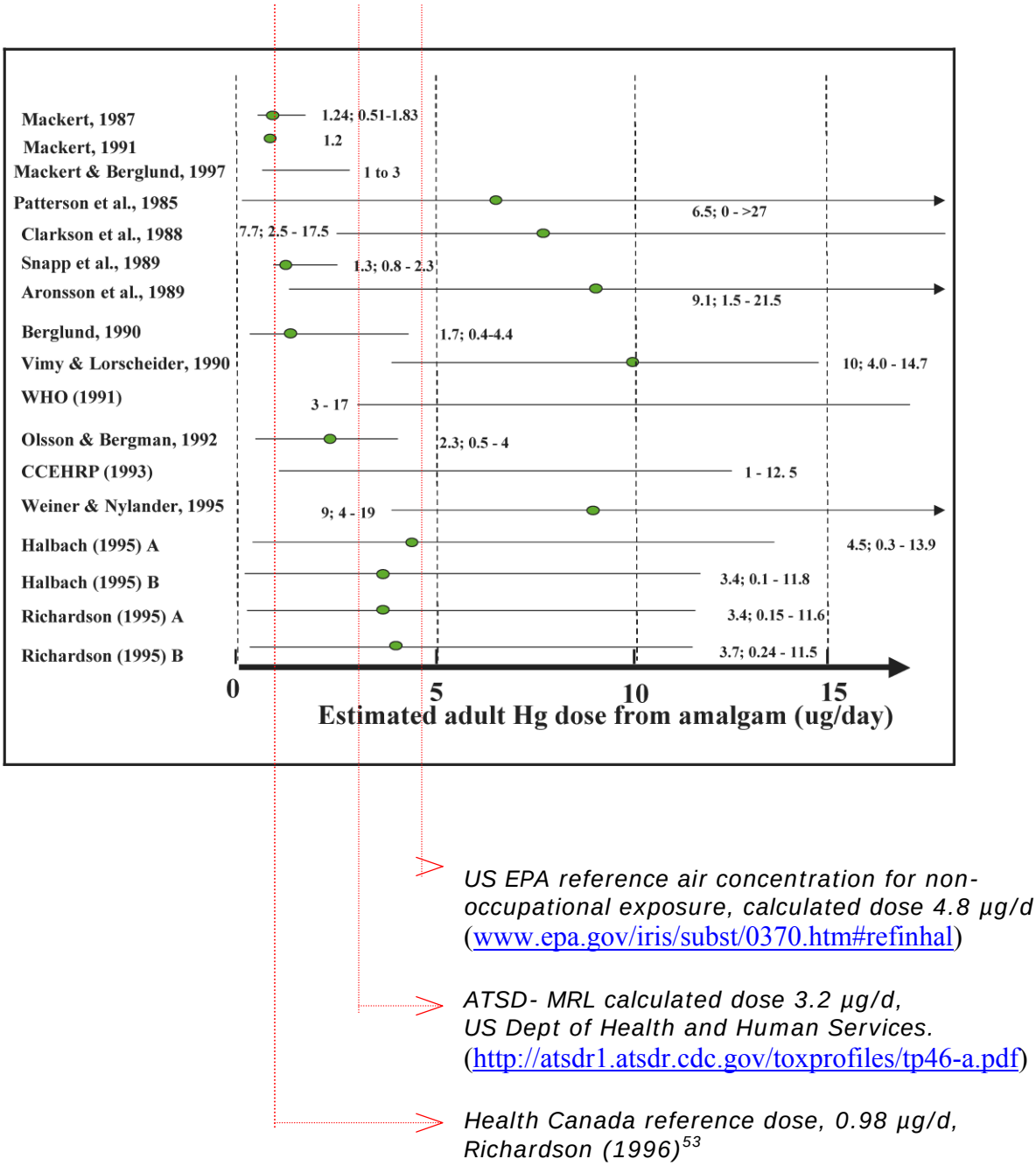

Figure 3 – Summary of seventeen literature citations estimating average mercury exposure in adults from amalgam fillings. The intersecting red lines show current allowable limits for non-occupational exposure to inorganic mercury from three different government agencies. The green dot in each horizontal bar represents the mean exposure found in that particular study. Adapted with publisher's permission from Richardson, GM; Human and Ecological Risk Assessment, 9: 1519-1531 (2003)

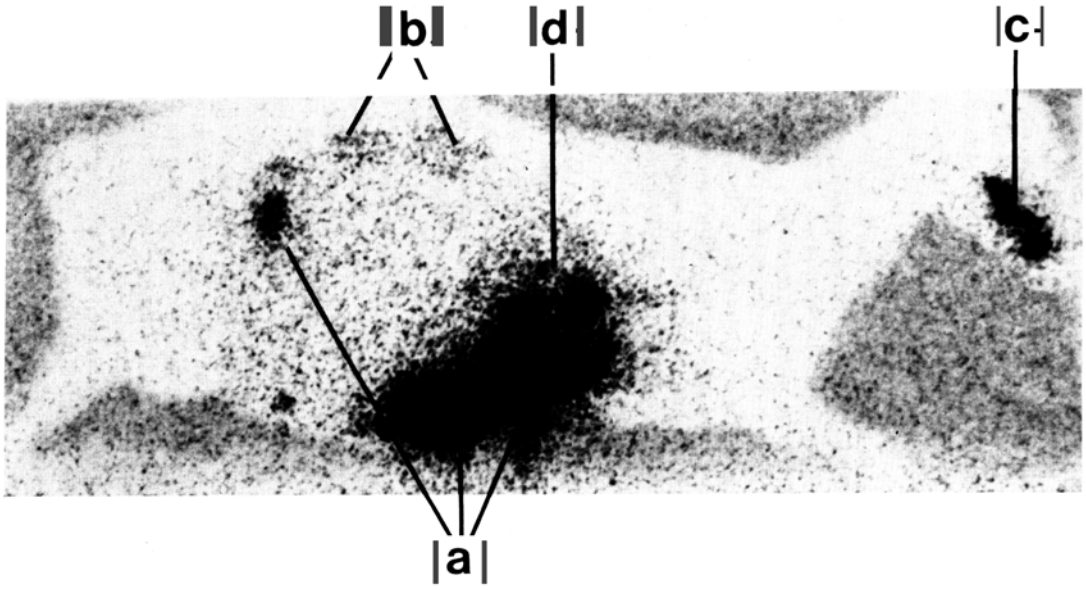

Figure 4 – Full body scan of a sheep 29 days after placement of 12 occlusal amalgams labeled with ^{203}Hg. The fillings were removed prior to the scan. (a) digestive tract. (b) kidneys. (c) gums and alveolar bone. (d) liver, partially obscured by the digestive tract. (From Hahn, et. al., 1989)

of the medical school's extensive animal program, and placed twelve occlusal fillings tagged with radioactive ^{203}Hg in the mouth of a sheep. The fillings were over-carved, not left high in the occlusion, as some have alleged, and the operators were careful to rinse all amalgam particles from the animal's mouth after placement. After twenty nine days, the sheep was killed, and the coronal portions of the teeth containing the radioactive fillings were removed. The sheep was placed in a full body gamma ray scanner, and the picture in figure 4 was the result.[18]

The graphic results are dramatic. Figure 4 is a full body gamma scan of the experimental sheep, showing translocation of radioactive mercury from the amalgam fillings into several organs. The teeth had been extracted prior to scanning, and the high concentration of radioactivity in the mouth region demonstrates movement of mercury into the jawbone from the fillings. The table below shows tissue concentrations of mercury that disseminated around the sheep's body. Control numbers would have been zero – all this mercury derived from the amalgam fillings, because the numbers were calculated from counts of radioactivity. In this experiment, the organ that accumulated the greatest amount of mercury was the kidneys, 7438 nanograms per gram of tissue (ng/g). The urine concentration was only 4.7 ng/g , demonstrating the inadequacy of plain urine samples as an indicator of mercury storage in internal organs. The order of magnitude of mercury accumulation in liver and kidney was confirmed by further studies using radioactive fillings in sheep.[19]

Tissue	ng Hg/g
Whole blood	9.0
Urine	4.7
Skeletal muscle (gluteus)	10.1
Fat (mesentery)	0.9
Cortical maxillary bone	3.6
Tooth alveolar bone	318.2
Gum mucosa	323.7
Mouth papilla	19.7
Tongue	13.0
Parotid gland	7.8
Ethmoturbinal (nasal) bone	10.7
Stomach	929.0
Small intestine	28.0
Large intestine	63.1
Colon	43.1
Bile	19.3
Feces	4489.3
Heart muscle (ventricle)	13.1
Lung	30.8
Tracheal lining	121.8
Kidney	7438.0
Liver	772.1
Spleen	48.3
Frontal cortex	18.9
Occipital cortex	3.5
Thalamus	14.9
Cerebrospinal fluid	2.3
Pituitary gland	44.4
Thyroid	44.2
Adrenal	37.8
Pancreas	45.7
Ovary	26.7

The dental establishment reacted with characteristic speed and determination. The "sheep experiment" was criticized for using an experimental animal that ate and chewed very differently from humans, and for not controlling for environmental factors, such as mercury in the diet. Of course, the experiment was not designed to look for mercury, but rather for radioactivity. There is no radioactive ^{203}Hg in nature, so any of it found could only have come from the fillings. The authors responded to the first criticism by saying that the sheep represents the "exacerbated case." If spread of mercury from amalgam could not be found in such a chewing machine as a sheep, the case would be closed, and the controversy over.

The same experiment was repeated using a monkey, which would eat much the same food and chew in much the same way as humans. The results were virtually identical to those found with the sheep.[20] Within twenty eight days, the radioactive mercury had spread

around the monkey's body, yielding tissue concentrations that were highly similar to the sheep's. The monkey experiment was confirmed by Danscher, et. al.[21] in Denmark. Figure 5 is the full body scan of the experimental monkey. Again, the teeth were sectioned and the coronal fillings removed prior to the scan.

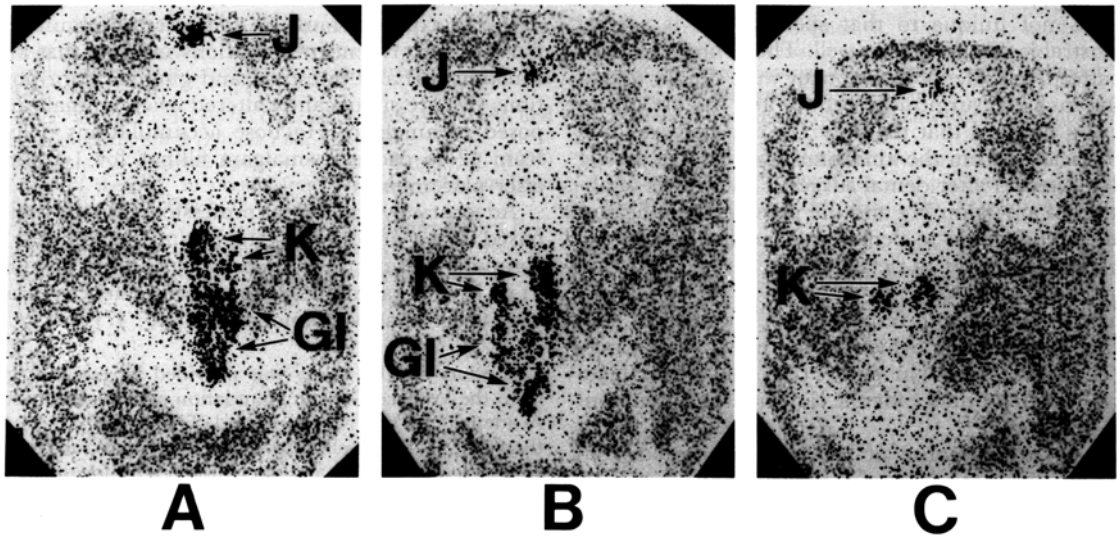

Figure 5 – Full body scan of a monkey 28 days after the placement of 16 occlusal fillings, labeled with ^{203}Hg, showing radioactivity in the jaws, kidneys and GI tract. (A) ventral view. (B) dorsal view. (C) dorsal view with the GI tract removed, clearly showing radioactive mercury accumulation in the kidneys. (From Hahn, et. al., 1990)

There is a large body of scientific literature that shows that amalgam–derived mercury spreads around the body, and that amalgam typically provides the greatest portion of the mercury to be found in the human body. Several autopsy studies showed a correlation between the mercury concentration in various tissues and organs of the human cadavers and the number of fillings or surfaces of amalgam present.[22 23 24 25 26] Blood levels of mercury correspond to amalgam exposure.[27 28 29] Subjects with amalgam excrete higher amounts of mercury in the feces.[30 31] Mercury in urine, blood, and feces declines after amalgam removal.[32 33 34]

Aposhian et. al.,[35] investigating the use of DMPS (2,3 dimercapto propane 1 sulfonic acid) as a chelating agent to remove toxic metals from the body, gave the drug to a group of subjects with amalgam fillings, and a control group of subjects who had never had amalgams. Urinary excretion of mercury in the non-amalgam group increased from 0.27 µg to 5.1 µg over a nine hour period, while among the amalgam subjects it went from 0.7 µg to 17.2 µg. They concluded that two thirds of the mercury excreted in the urine must derive from the amalgam fillings. They also reported a highly significant correlation between amalgam score and urinary excretion of mercury two hours after DMPS administration. Other labs report similar results.[36 37]

Maternal – fetal transfer of mercury.

Babies, with their still-developing nervous systems, are known to be more sensitive to the effects of mercury exposure than adults. Pediatric authorities say: "The developing fetus and young children are thought to be disproportionately affected by mercury exposure, because many aspects of development, particularly brain maturation, can be disturbed by the presence of mercury. Minimizing mercury exposure is, therefore, essential to optimal child health." And "Mercury in all of its forms is toxic to the fetus and children, and efforts should be made to reduce exposure to the extent possible to pregnant women and children as well as the general population."[38]

This was made tragically clear in the case of the Minamata Bay methyl mercury poisoning, in Japan in the 1960's, where children were born with profound developmental disturbances, while the adults suffered much less. There is a substantial experimental literature on the neuro–teratological effects of mercury, where both humans and animals exposed to low doses of mercury *in utero* and soon after birth show measurable deficits in intelligence, coordination, and other measures of neurological development [39] [40] [41] [42] [43] [44] [45] (and hundreds more). And now there is an added controversy about vaccines preserved with thimerosal, a form of ethyl mercury, possibly causing neurological damage in infants, including autism. [46] Does amalgam use in dentistry provide the unborn with a prenatal body burden of mercury?

Two more experiments by Vimy, Lorscheider and associates at the University of Calgary Medical School, supported by the IAOMT, provide some insight into the issue of amalgam–derived mercury exposure to the fetus and infant. In the first, [47] five pregnant ewes, at about 112 days of gestation, were fit with indwelling catheters that allowed the researchers to collect serial samples of maternal and fetal blood, amniotic fluid, plus maternal feces and urine. Each sheep received twelve occlusal amalgam fillings labeled with radioactive ^{203}Hg, as did the sheep in the original study. The various body fluid samples were collected for sixteen days, after which the sheep were sacrificed at intervals and tissue samples were analyzed for radioactive mercury. They found that the amalgam–derived mercury appeared in maternal and fetal fluids within two days of amalgam placement. Radioactive mercury was found in all post-mortem tissues studied. Tissue concentrations achieved steady state levels after about a month, levels that were maintained throughout the 140 day course of the experiment. The fact that tissue concentrations did not decline with time, as they would have with an acute, one time dose, implies that there was an ongoing exposure from the radioactive amalgam fillings. As before, the mothers concentrated the most mercury in the kidneys and liver, while the fetuses concentrated it in the liver and pituitary gland. Mercury concentration in the fetal blood was actually higher than in the maternal blood.

In the second study,[48] pregnant ewes received radioactive amalgams as before, and then nursed either their own lambs or foster lambs that had not been exposed to radioactive mercury in the womb. In the womb, the fetal lambs accumulated more mercury in the liver, while after birth the kidneys became the primary site of accumulation. Measurable quantities of radioactive mercury appeared in the tissues of both amalgam–bred lambs and those only nursed by amalgam–bearing ewes.

These studies are consistent with the work of other groups. For example, previous animal studies have shown that when the mother is exposed to Hg^0, the form of mercury that is emitted from amalgam, fetal tissues take up more mercury than when the mother is exposed to Hg^{2+}.[49] Drasch, et. al.[50] studied autopsy samples from human stillbirths and early post natal deaths. They found that the mercury concentration in the infants' kidneys, liver and cerebral cortex correlated significantly with the mother's amalgam scores. Two labs also found that mercury concentration in human breast milk correlated significantly with the mothers' amalgam scores.[51] [52]

Adverse physiological changes due to exposure to amalgam mercury.

So – all this exposure information is one thing, but as we have heard for years, "the dose makes the poison," and "no one has found dental amalgam to have caused any human disease, except for very rare allergic reactions."

Well, it's not exactly true. It is true that in the huge body of information on mercury toxicity the greatest number of papers concern acute doses. Relatively few experiments have been done on chronic trace level exposure to elemental mercury vapor, and fewer still made use of amalgam as the mercury source. But there are some very provocative indications in the literature. A picture emerges, not of overt disease, but of many subtle (and some not so subtle) biochemical and physiological events that together constitute the pathophysiology of chronic low level mercury poisoning from exposure to dental amalgam. Certainly there are many suggestions that chronic exposure to mercury can contribute to big–name diseases. [see www.bioprobe.com for a bibliography, or read *The Toxic Time Bomb,* available on that site] But that concept is not necessary to warrant caution in using mercury. After all, who would wait for proof that lead or arsenic caused a "disease" before avoiding these known poisons?

Risk assessment.

In the early 1990's, Health Canada was sued by a group of consumer activists over a law requiring an evaluation of safety and effectiveness for all medical devices. They eventually forced the agency to apply that standard to dental amalgam. A staff specialist in medical risk assessment, G. Mark Richardson, was assigned the task of evaluating the available literature on mercury and amalgam, and to make recommendations concerning the health impacts of amalgam use in Canada.[53] [54]

Richardson made detailed recalculations of mercury exposure from amalgams based upon the reported literature, and detailed recalculations of the level of mercury vapor exposure that would lead to "subclinical impairment of neurological and cognitive functions," based on the industrial hygiene literature. His general assessment was, in essence, that somewhere within the known range of mercury exposure from amalgam, there begins the known range of mercury exposure that produces neurological consequences. Based on his examination of the neurological data, he proposed a tolerable daily intake (TDI) of .014 μg Hg^0/kg-day, which was exceeded in all age groups by the average daily exposure

from amalgam in Canada. In order not to exceed the proposed TDI, the maximum number of amalgam fillings allowed would have to be:

Ages 3 – 11	0 – 1
12 – 19	1 – 3
20 – 59	2 – 4
60 +	2 - 4

If the US EPA non–occupational "reference concentration" of 0.3 μg Hg/m^3 in air were to be used, 9 – 11 amalgam fillings would be acceptable in an adult. On the other hand, the US Agency for Toxic Substances and Disease Registry (ATSDR) published a minimal risk level (MRL) for non–occupational exposure of .014 μg Hg^0/m^3 in air. If this standard were used, even one amalgam would expose the individual to more mercury than would be allowed by Richardson's proposed TDI. (see fig 3, above)

Richardson concluded that, "no clear threshold for subclinical neurological and cognitive function impairment is evident from published studies of the CNS effects of Hg vapor." In other words, no known safe level. Further, "the continued unconditional and unlimited use of amalgam as a dental restorative material, the placing of up to 25 amalgam fillings in one individual, is not supported by the available risk information."

The Canadian Dental Association called this report "unscientific," but later retracted that statement. Health Canada did not support a total ban on amalgam use, but, in 1996, did issue some restrictive recommendations: [55]

- Avoid using mercury to restore children's teeth.
- Avoid placing or removing amalgam in the teeth of pregnant women.
- Avoid using dental amalgams in patients suffering from kidney ailments.
- Use methods and equipment to reduce the risks of exposure to mercury vapor to protect their patients and their staff. [This is the subject of a later chapter in this on-line book.]
- Avoid using amalgams in patients who risk suffering from allergic hypersensitivity (5 to 15% of the population).
- On the advice of a physician, remove amalgams from a patient who has become sensitive.
- Avoid placing amalgam in contact with other metal appliances in the mouth (orthodontic appliances, etc).
- Fully inform patients of the risks and benefits involved.
- Recognize the patient's right to refuse treatment using a "specific material."

Immune System:

The "allergic hypersensitivity" to mercury issue is interesting. It is not very, very rare, as certain dental authorities would have us believe. The North American Contact Dermatitis Group, in 1972, determined that 5 - 8% of the US population demonstrates allergy to mercury by skin patch testing. [56] By using antibody – antigen flocculation tests on blood serum, the number is over 90%.[57] Djerassi and Berova [58] patch tested 180 subjects with amalgam fillings, and found that 16.1% of those without allergic disease, and 22.5% of those with allergic disease, tested positive for mercury allergy. Of sixty subjects without amalgam fillings, none tested positive for mercury allergy. In a study of 29 patients with oral lichen planus, 62% were positive for mercury allergy. [59] And at Baylor College of Dentistry, of 171 dental students patch tested, 32% were positive for mercury allergy. The percentage of positive tests correlated with the students' own amalgam scores, and with the length of time they had been in dental school. [60]

Mercury exposure is know to induce autoimmune reactions in susceptible animals,[61] [62] [63] and one investigation shows the same for amalgam. Hultman et. al.[64] implanted gelatin coated particles of either finished amalgam or unmixed silver alloy in the peritoneal cavity of mice known to be genetically susceptible to mercury–induced autoimmune reactions. Over the course of the experiment, both groups displayed their characteristic reactions of hyperimmuno-globulinemia, serum autoantibodies targeting nucleolar proteins, and systemic immune complex deposits. The authors ascribed the reactions in the alloy–only group to the silver component.

Think of the outbred human population, with its plethora of autoimmune diseases. We dentists have developed no method of screening our patients for contact dermatitis or for their susceptibility to metal–sensitive autoimmune responses. Knowing these mechanisms exist, how many such problems are we creating by using mercury – or nickel, for that matter?

Renal System:

Mercury, we now know, concentrates in the kidneys, and experimental evidence shows that it can inhibit kidney function. [65] But can mercury deriving from amalgam fillings have a direct effect upon kidney function? Once again in Calgary, six sheep received amalgam fillings, although they were not radioactive this time. Two control sheep received glass ionomer fillings. Renal clearance tests were performed before the fillings were placed and again at thirty and sixty days following. All six of the experimental sheep had a statistically significant decrease in their inulin clearance at both thirty and sixty days relative to the controls, with an average decline of 54%, $p < .01$. (see figure 6) They also had a significant increase in urinary sodium, and a decrease in urinary albumin as compared to the controls. The kidney tissue showed no structural change upon microscopic examination.[66] Molin, et. al.[67] reported that urinary albumin increased in humans one year after removal of amalgams. Mercury is known to concentrate in the proximal tubules, which are the primary site of sodium reuptake, so it makes sense that urinary sodium excretion increased if the mercury is inhibiting the function of those cells.

Although these effects could be described as "subclinical," in that overt disease was not induced, it demonstrates how much stress is placed upon the kidneys by the presence of amalgam, and suggests how patients with kidney malfunction may be endangered by amalgam fillings.

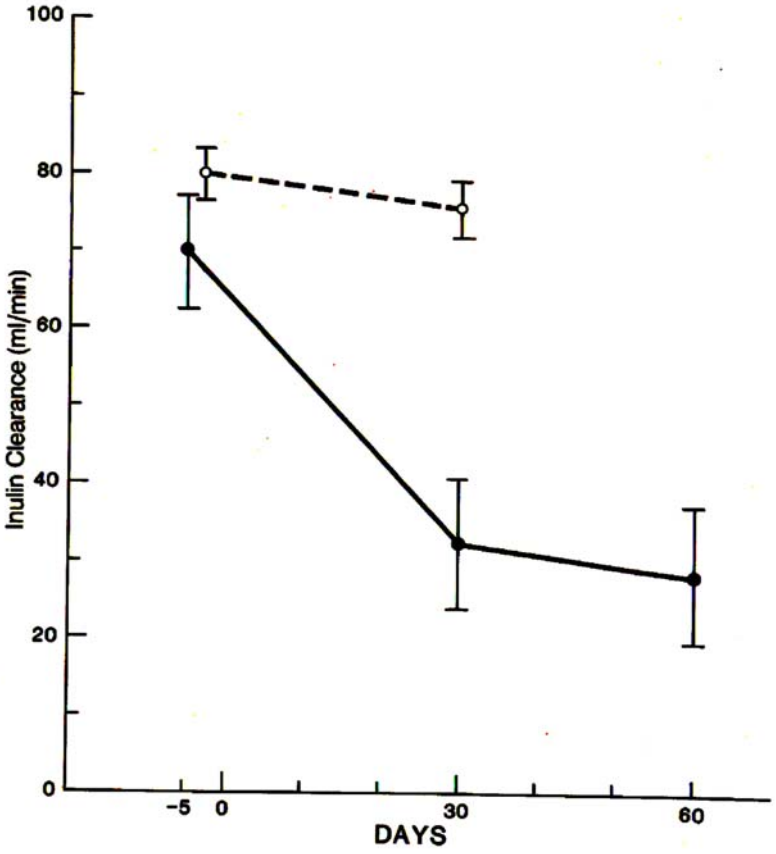

Figure 6 – Plasma inulin clearance (+/- SEM) of six sheep with twelve occlusal amalgam fillings (solid line) and two controls with glass ionomer fillings (dashed line). (from Boyd, et. al., 1991)

Intestinal Flora:

Anne Summers and her group in the Department of Microbiology, University of Georgia, were investigating resistance to antibiotics among intestinal bacteria when they discovered an unexpectedly high percentage of resistance in the flora of individuals who had had no recent exposure to antibiotics. They found that the genes for antibiotic resistance in these bugs were linked, on plasmids, to a gene for resistance to mercury toxicity. Therefore, subjects with a high percentage of mercury resistant bacteria in their intestines were significantly more likely to have bacteria with multiple antibiotic resistance as well. It was ecological pressure for mercury resistance that seemed to be maintaining the high prevalence of resistance in these gut flora samples. But where was the mercury coming from? [68]

To test the hypothesis that dental amalgam could provide enough mercury exposure to drive this ecological selection, monkeys were given amalgam fillings. Their intestinal

flora showed a marked increase in the proportion of mercury resistant bacteria, and the increase was maintained until the amalgams were removed. Most of the mercury resistant microbes also possessed resistance to one or more antibiotics. [69]

The implication of this finding for human medicine is unproven, but disturbing to contemplate. At least it shows again that amalgam, while perhaps not causing overt disease, has a detectable effect upon the homeostasis of the body that is not benign.

Are we dentists harming ourselves?

One of the mantras chanted in support of amalgam has been that dentists' health status is not different from that of the general population, despite the fact that we are exposed in our work to mercury. Perhaps, one might say, that's due to the mercury hygiene rules promulgated by the profession – don't touch mixed amalgam with the hands while you pack it into patients' teeth, store scrap amalgam in tightly closed containers under various liquids to prevent vapors from escaping in the office, dispose of it with licensed hazardous waste handlers, etc. Even so, there is some evidence that mercury–exposed dentists and staff do suffer various effects.

In one study, dentists with high baseline urinary mercury levels showed neuropsychological and motor control deficits.[70] In another, dentists and staff with high mercury levels, proven by DMPS challenge, had altered porphyrin (hemoglobin) metabolism, as well as neurobehavioral changes, including impairment of attention, motor and perceptual skills, and increased irritability.[71] [72]

The urinary mercury levels of 4272 dentists were measured at random at dental conventions by Naleway,[73] et. al., between 1975 to 1983. They found that dentists *on average* did not have urinary mercury concentrations outside "acceptable limits" and came to the conclusion that there was no problem with their occupational exposure due to amalgam. However, the urinary concentrations correlated significantly ($p<.001$) with the number of amalgams each dentist placed per week, and the range was tremendous. The general population has a range of 0 – 5 μg Hg per liter of urine, while 10.9% of the dentists in this study had over 30 μg per liter, including 1.3% with over 100 μg per liter! If the proportionality of mercury in urine to total body burden, as shown by the sheep and the monkey studies, holds true for humans, the dentists who use the most amalgam are storing prodigious quantities of mercury in their bodies.

In a survey of 7,000 female dental assistants, a subgroup of 418 women who placed over 30 amalgams per week, and had poor mercury hygiene habits, had a fertility rate of 63% that of control women not exposed to mercury. [74] Many other studies point to a negative effect of mercury vapor exposure on reproductive outcomes. [75] [76] [77] [78]

Depression and mood alteration is a known feature of chronic mercury toxicity.[79] Dare we speculate that occupational mercury exposure plays a part in the suicide rate of dentists, which is higher than the population average?

The unique neurotoxicity of mercury, and the Alzheimer's connection.

The scene shifts to the Sanders-Brown Center on Aging at the University of Kentucky, which has a very active program for the study of Alzheimer's disease (AD). Autopsy specimens of the AD brain show certain diagnostic lesions – deposition of amyloid protein plaques, and neurofibrillar tangles, remnants of degenerated axons. There are characteristic biochemical lesions as well, including phosphorylation of tau protein, depletion of intracellular glutathione and creatine kinase, excess production of glutamine synthetase, and disruption of tubulin formation. Most of the research that we hear about in the press in the last few years has concentrated on the amyloid plaques, although amyloid deposition is found in many diseases, in other organs. The neurofibrillar tangle is more unique to AD, but there hasn't been an experimental system with which to study it until recently.

Following one track, Markesbury, Ehmann, Vance, and associates published a series of papers in which they described a variety of trace mineral changes in AD brain as compared to controls from patients with other psychiatric diseases or normal brains. They consistently found elevated concentrations of mercury, in various regions and subcellular fractions in the AD brain samples. [80] [81] [82] [83] Other labs found elevated mercury in the blood and cerebrospinal fluid of AD patients. [84] [85]

An examination of the same topic that was published with great fanfare in the Journal of the American Dental Association, along with press releases heralding the exoneration of amalgam, showed no correlation between amalgam history and AD, nor differences in mercury concentration between AD brains and controls.[86] This is the only paper in existence that presents such a position, contradicting those mentioned above, and the other human autopsy studies quoted earlier.

Meanwhile, Boyd Haley, a protein biochemist and chairman of the chemistry department at the University of Kentucky, was working on the tubulin synthesis defect in AD with his associate Kurt Pendergrass and their group. Haley had developed a chemical probe for the active site of an enzyme that he called "photo-affinity labeling," which has since become a standard tool in biochemical research. The technique involves a photoreactive chemical bridge between the substrate molecule and a radioactive $^{32}PO_4$ group. In the test tube, the target enzyme is allowed to react with the prepared substrate, and then exposed to light. The light causes the photoreactive bridge to disintegrate, allowing the highly active $^{32}PO_4$ to staple itself to the protein. If the enzyme's active site is not available, blocked by a mercury atom or other inhibitor, the photo-labeling will not take place. To summarize – if the active site is open, the protein becomes radioactive. If the active site is blocked, the protein is there, but does not become radioactive.

Haley, Pendergrass and associates used this technique to work out the biochemical mechanism behind the tubulin synthesis defect in AD, and linked it firmly to mercury. Tubulin is a structural protein in all cells, forming the girders and beams of the cytoskeleton. It is a large polymer made up of dimeric units, each having an α and β subunit. In order for the two to join, the β-subunit must bind a GTP molecule. The researchers found that the β-tubulin from AD brain could not bind photolabelled $^{32}PO_4$-GTP. The protein was there, but the active site was blocked! [87]

Taking a hint from their colleagues at the Sanders Center, they investigated the possibility that toxic minerals could be blocking the GTP binding site on β-tubulin. To make a long story short, it turns out that the binding site on β-tubulin is uniquely blocked by mercury, at extremely low concentrations in the 10^{-7} M range. Cadmium has a smaller effect, by orders of magnitude, and aluminum and lead have no effect at all. Excess zinc had a slight effect, but greatly increased the inhibitory action of the low concentrations of mercury. [88] [89] [90]

The mercury story is making its way in the laboratory, if not yet in the press. Recently, Olivieri, et. al.[91] reported that adding a very low concentration of mercury, 36 x 10^{-9} M, to neuroblastoma cells in tissue culture caused them to exhibit all the biochemical lesions of AD – inhibited tubulin synthesis, drop in intracellular glutathione, excretion of phosphorylated tau protein, and finally, excretion of β-amyloid. If most contemporary researchers think that amyloid is the cause of AD, here we have vanishingly small quantities of mercury causing amyloid in turn. The authors of this study suggest that mercury is the ultimate cause of these events.

Closer to our world, research shows that this test tube phenomenon can be induced in living animals. Mercury chloride has been shown to get into rat brains and inhibit the binding of GTP to β-tubulin,[92] and the same for elemental mercury vapor. Rats breathing 300 μg Hg^0 per cubic meter of air, a concentration that has been found in the mouths of people with lots of amalgam, for just four hours a day for fourteen days, had 75% inhibition of the photolabeling of β-tubulin with $^{32}PO_4$–GTP.[93] [94] Did the rats become demented? That question was not asked. Perhaps this was a subclinical effect, one that did not cause overt disease. But is it not an effect we would wish to avoid?

The mercury story correlates with an epidemiological feature of AD. The age of onset of AD in the population is associated with the genetic variation of apolipoprotein–E, a "housekeeping" protein in the brain and cerebrospinal fluid. Its usual function appears to be transport of cholesterol. However, it comes in three genotypes, apo-E2, apo-E3, and apo-E4. Those individuals with apo-E2/2 almost never get AD, while those with apo-E4/4 tend to have early onset of the disease. Apo-E3 is intermediate. What's the difference among the genotypes? At amino acid position 112 and 158, apo-E2 has two of the sulfhydryl containing cysteine molecules. Apo-E3 has arginine at position 158, and apo-E4 has arginine at both places. In other words, apo-E2 has the most capacity to bind and remove divalent toxic metal atoms such as mercury as it moves from the brain into the cerebrospinal fluid, and out into the blood. Apo-E3 has less, and apo-E4 has none, at least by this mechanism.[95]

Dentists, we can be certain, have never screened patients for their apo-E genotype before exposing them to mercury in fillings.

Neurite growth inhibition on video.

What is it about Calgary? One of the few labs in the world that has the capacity to maintain growing neurons in tissue culture is at the University of Calgary Medical School. Very recently, a group there, supported in part by the IAOMT, published a paper and an accompanying video that shows how very low concentrations of mercury chloride, at 10^{-7} M again, causes the tubulin in the growth cones of young neurites to fall apart. [96] The subject cells were the large Pedal A neurons from the central ring ganglia of the snail *Lymnaea stagnalis*. The amino acid sequence of tubulin is at least 97% the same throughout the animal kingdom, so there is no difficulty comparing snail tubulin with human. Figure 7 is a series of still photographs from this experiment, which shows first the intact growth cone. Then the mercury solution is applied with a micropipette. Finally, seventeen minutes later, the growth cone has degenerated, leaving behind a tangle of neurofibrillar protein, reminiscent of those seen in AD brains. In another trial, growth-phase neurons in a culture medium containing 10^{-7} M mercury chloride failed to initiate growth cones. Other elements, aluminum, lead, cadmium and manganese were tried, but they produced neither effect.

The authors state: "Hg ions markedly disrupted membrane structure and linear growth rates of imaged neurites in 77% of all nerve growth cones. When growth cones were stained with antibodies specific for both tubulin and actin, it was the tubulin/microtubule structure that disintegrated following Hg exposure."

The complete paper is available on-line at this URL:
http://ipsapp002.lwwonline.com/J=1860&I=88&A=21&U=1&T=0

If you have a fast internet connection, you can view the video of this experiment at:
http://movies.commons.ucalgary.ca/mercury/.

It is a miracle of nature and evolution that we are so elaborately protected from diseases and toxins. We have, in the case of mercury and the other divalent metal toxins, essential metabolic systems such as reduced glutathione, metallothionines, and apolipoprotein-E which double as protective elements. But, as we have seen in the case of apo-E, there are genetic variations and polymorphisms that inevitably leave some individuals more vulnerable to assault. We dentists may never have a perfect understanding of biocompatibility. We may always be forced into biological compromises with our need to implant synthetic materials in our patients' mouths. But let us at least minimize that risk where the science is firm. Amalgam has got to go. And if the mercury–Alzheimer's disease connection holds up, our profession is going to need some heavy rain gear.

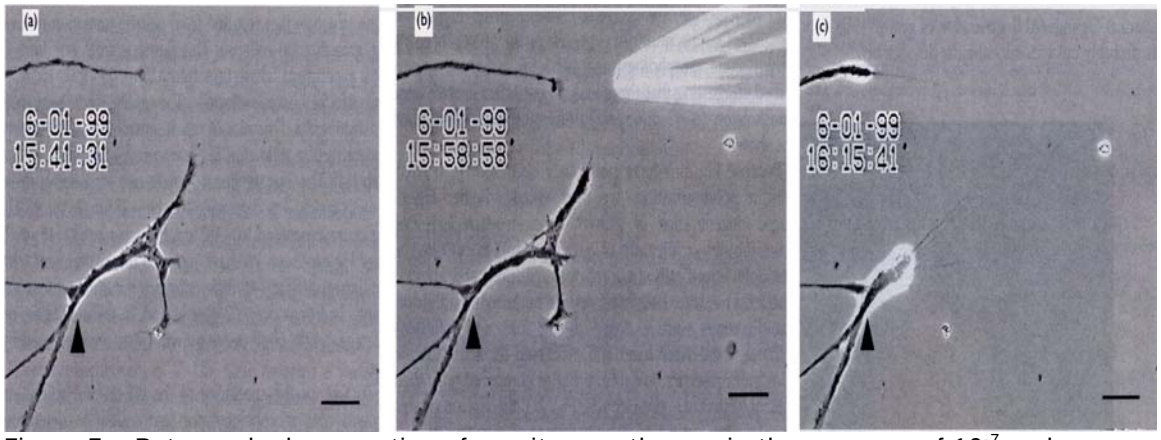

Figure 7 – Retrograde degeneration of neurite growth cone in the presence of 10^{-7} molar mercury chloride. Note the triangle reference mark. (From Leong, et. al. 2000)

The anecdotes

The world and the world wide web are full of anecdotes from people who claim their health improved once their amalgam fillings were replaced with other materials. These are real people with real life experiences, though their stories do not constitute scientific cause and effect evidence. Nevertheless, the scientific method requires that we observe natural phenomena, so as to gather ideas which we can try to develop into testable hypotheses. Where there's smoke there just might be fire.

The following is a summary of the subjective reports of 1569 patients who participated in six different surveys of health effects of replacing amalgam fillings. [97]

Symptom Reported	Percentage of patients claiming substantial relief
Allergy	89 %
Anxiety	93
Bad temper	89
Bloating	88
Blood pressure problems	54
Chest pains	87
Depression	91
Dizziness	88
Fatigue	86
Gastrointestinal problems	83
Gum problems	94
Headaches	87
Migraine	87
Insomnia	78
Irregular heartbeat	87
Irritability	90
Lack of concentration	80
Lack of energy	97

Memory loss	73
Metallic taste	95
Multiple sclerosis	76
Muscle tremor	83
Nervousness	83
Numbness	82
Skin disturbances	81
Sore throat	86
Tachycardia	70
Thyroid problems	79
Oral ulcers	86
Urinary tract problems	76
Vision problems	63

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Working for Mercury-Free Dentistry

Dental Amalgam is a Known Health Risk to Children, Fetuses, and Individuals with Impaired Kidney Function

Numerous governments have concluded that dental amalgam is a health risk to children, fetuses, and individuals with impaired kidney function. The Swedish government has banned amalgam altogether with no exceptions for children. Norway has acted similarly. Over a decade ago, Health Canada directed dentists to stop using amalgam in children, pregnant women, and people with impaired kidney function.

Despite the consensus that **dental amalgam places children, pregnant women, and people with impaired kidney function at risk**, intense pressure from the dental industry has blocked some governments from acting to protect these vulnerable populations even in developed countries where non-mercury alternative filling materials are widely available.

For example, in the United States, the U.S. Food and Drug Administration (FDA) released a rule on dental amalgam in July of 2009. FDA admits that scientists have not concluded that amalgam is safe for children under six or for pregnant women's unborn babies: "Very limited to no clinical information is available regarding long-term health outcomes in pregnant women and their developing fetuses, and children under the age of six, including infants who are breastfed." In fact, FDA admits that amalgam could cause severe harm: "The developing neurological systems in fetuses and young children may be more sensitive to the neurotoxic effects of mercury vapor." Additionally, many children are already affected by a high mercury bioburden from sources other than amalgam (such as tuna and vaccines) – a condition that makes them even more susceptible to amalgam's bioaccumulative effects – but FDA's rule did not consider this fact: "This type of comprehensive analysis of exposure to multiple species of mercury from multiple sources was beyond the scope of the review." Ignoring its own scientific advisory panels' vocal concerns for children and the unborn, FDA failed to take any steps to protect these most vulnerable populations.

We invite participants to join the growing number of countries that are protecting children, pregnant women, and individuals with impaired kidney function from dental amalgam, an unnecessary source of mercury exposure.

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Zogby Polls Indicate That Dentists Force Amalgam on Patients Even When Alternatives Are Available and Affordable

While the World Dental Federation claims that dentists need amalgam in order to address the oral health needs of the "disadvantaged," Zogby polls indicate that dentists make no effort to limit their amalgam use to populations in need. In fact, dentists in developed countries implant amalgam in many patients regardless of their socioeconomic status – after all, it's easier for the dentist.

The polls indicate that after they are informed that amalgam contains mercury, the vast majority of these patients (77%) in developed countries, such as the United States, are willing to pay more for alternative filling materials in order to avoid unnecessary mercury exposure.

However, pro-mercury dentists withhold from their patients the facts they need to make an informed decision – even though 92% of patients polled want their dentists to inform them that non-mercury filling materials are available. As a result, most patients do not even know that dental amalgam is composed primarily of mercury, much less are they aware of the risks to children, fetuses, and people with kidney impairments.

The Interstate Mercury Education and Reduction Clearinghouse (IMERC) predicts that increased consumer awareness of mercury in fillings will achieve future declines in the use of dental amalgam. Of course, the more patients choose non-mercury filling materials, the less mercury goes into our environment. If the World Dental Federation's real concern is preserving access to amalgam for disadvantaged patients, then it should not promote withholding information about environmentally-friendly alternatives from patients who can afford them. If the World Dental Federation is truly taking responsibility for dental mercury in our environment, then it will work to decrease the amount of mercury being released into everybody's wastewater and air by at least informing patients that they can choose non-mercury alternatives where they are available. The resulting decrease in mercury pollution will benefit the disadvantaged and the advantaged alike.

We invite participants to accelerate the phase out of dental mercury by implementing patient awareness building programs (1) to inform patients that amalgam contains mercury that poses environmental and health risks and (2) to encourage patients to choose alternatives where alternatives are available so as to lessen the burden of mercury pollution everywhere.

Mercury- a hazard to human health

Angela Kilmartin.
Patients Against Mercury Amalgams. United Kingdom.
www.angelakilmartin.com

HUMAN HEALTH.

Mercury has always been known to be hazardous to human health.

From early mining of cinnabar, gold and other ores, miners' lives were limited to six months and Bartholomew Anglicus in 1260 wrote that " it bredeth the palsey, shakyng, quaking, softening of the sinews and is bad for the mouth."
Leonardo da Vinci demonstrated its palsy effect from using it on a pet lizard which shook unceasingly.

More recently, "The Toxicity of Industrial Metals" textbook lists many brain illnesses caused by mercury which include depression, drowsiness, insomnia, headaches, fatigue, memory loss, personality disorders and many more.

In a 1970's Soviet study group of 650 patients with mercury fillings, results showed abnormal blood pressures, abnormal ECG patterns, altered hormonal and neuronal heart regulators, damaged heart muscle tissue in valves, arteries and capillaries, rapid pulse, fatigue, anaemia and lowered red blood cell counts.

Neurological disorders in many Studies and research papers show mercury as the main causal factor in Parkinson's, Multiple Sclerosis, Autism, Asthma and Alzheimer's Disease. Dr Boyd Haley's work on Alzheimer's is confirmed at autopsy showing mercury alone interfering with enzymes which control and inhibit brain neurofibrillar tangles and amyloid plaques. Other metals present at autopsy do not make such brain tissue changes or cause such damage.

Mercury is used in contraceptives to disable sperm action. When even four amalgam fillings were removed in a 24 yr old man, his sperm activity rose from 52% disabled to only 2% disabled. Sperm motability is reduced in the presence of mercury. Sperm merely 'head-bang'.

Mercury from amalgams shows a daily stored whole-body nanogram level of 29000 if 12 or more amalgams are present. With 5 fillings, stored mercury falls to around 8000 nanograms. Daily intake of mercury from amalgams varies between 3.8 to 21 micrograms.

Mercury vapour and particulate cross blood capillary walls both in lungs, brain, intestines travelling to every muscle, organ, enzyme system.

Irritable Bowel Syndrome, stomach cramps, diarrhoea, constipation are amongst many individualised symptoms.

Mercury attracts calcium and magnesium from bone structures including spine, gums, joints, hips, shoulders. Loss of magnesium depletes 78% of the whole-body enzyme system function leading to hormone loss, kidney and liver malfunction and sexual hormone malfunction.

An infant is immediately at risk of ill health from conception when parental mercury from sperm and egg is present. Placental mercury transference starts from egg division and continues for nine months as mercury from the pregnant mother transfuses and is taken up by the foetus. At birth, coloestrum and milk carry mother's mercury within the prime nutrition thus making breastmilk harmful and powdered milk safer. At six weeks the infant is assaulted with vaccines, many of which do still contain Thimerosal / Thiomersal as the preservative. With soft fish and then Tuna sandwiches the child intakes mercury for itself. Childhood teeth fillings conclude the attack and ingestion of mercury.

Children's kidneys are unable to cope with an overwhelming mercury input and many childhood illnesses result- autism, allergies, asthma, ADD, anger, personality changes, bladder control, weight gain, and much more.

By the end stages of life, mercury has wreaked havoc; living longer is no longer something we long for, quite the reverse.

THE GRAVE

At death, teeth fillings and presence of mercury in the body influence the environment, the land, the water. Cremation processes simply evaporate the mercury in teeth fillings into the air around the crematorium which is breathed in and, when cooled, drops into the city, the farm, the rivers, lakes and sea all again in turn to be up-taken back into the human food chain.

In the grave, breakdown of coffin, bone, soft tissues, all containing stored mercury will eventually return the elemental mercury to earth.
(see attachment and acknowledgements.)

PERSONAL ACCOUNT

For me, nine years in bed with more bewildering illnesses starting when gold was added to my mouthful of mercury showed me the devastation and dangers from mercury amalgam teeth fillings. Upon safe removal I was well inside three months when no medications had previously helped though many had been tried.
I was three months off killing myself. I have all my family now on file showing many symptoms, three generations including two autopsies. Its frightening what modern dentistry has done for cheapness and ease of placement.

CURRENT DENTAL TRAINING in UK.

In Great Britain, such has been the public demand for white fillings following many years of publicity by my organisation that Guys Dental training School in London has had to listen to trainees entering school asking to be taught placement of white fillings above mercury fillings.

White filling placement has now overturned the position of mercury filling placement training in UK to favour prime training in composites.

White fillings are the way forward and amalgam must be abandoned.

Mercury should not be placed in the human body- and isn't- except when big money is involved!

British Government Position on mercury from cremation

"Mercury emission from the dead through crematoria has to be halved by the end of 2012". In 2002 there were 437,124 cremations emitting 1.31 tonnes of mercury into air, 3 grams per body.

Mercury is toxic, accumulates in air and water, can harm the brain, kidneys, nervous system and unborn children.

Up to 16% of all mercury emitted in UK comes from crematoria because of fillings in teeth. This percentage is expected to rise to 25% by 2020 *unless* Cremator units achieve the 50% lowered mercury emission levels as required by DEFRA (Dept of Environment, Food and Rural Affairs) by 2012.

(50% reduction is the balanced figure obtained when environmental and realistic achievement of change rather than a 100% ban on mercury emissions from crematoria could be met.)

PROFITEERS from MERCURY AMALGAMS.

Money is the name of the game; dental pricing structures have varied; payment to the dentist per tooth filling saw an unprecedented rise in fillings and dentist's pockets in the 1950's with unnecessary placements in children's teeth. That generation is now polluting the environment in death.

Great profits ensue for the amalgam manufacturers and their shareholder-lobbyists in governmental departments particularly in America. The amalgam and dental trades federations worldwide also encourage promotion of sweet foods and drinks to entice gullible public consumption. Necessary dental cavity-filling completes the whole conspiratorial trade in tooth destruction and commercialism.

LOSERS.

This increase in mercury emissions from the dead is because dentistry has insisted upon saving teeth by using mercury fillings and has thereby influenced patients to remain 'dentate'. As a result, many teeth are merely blackened stumps of mercury fillings with little surrounding enamel structure. In previous generations teeth were removed much earlier or dropped out when diseased.

Continued and increasing use of commercial mercury worldwide is contributing a toxic waste to air, land, water and food supplies. It should not be put in the human body. Millions suffer costly illnesses.

UPDATING CREMATOR UNITS.

All new-build crematoria have to conform to new regulations for containing mercury droplets from the teeth of the dead by preventing vapourised and heated mercury from ascending the flues out into the air. New cremators now have internal mercury-gathering machinery so that this hazardous waste can be contained within the building ready for hazardous waste disposal to regulated toxic waste-disposal sites.

Costs to reduce mercury emissions from existing and ageing individual crematoria buildings varies between £150,000 and £450,000 PER UNIT! PER INDIVIDUAL CREMATOR!

Cremation buildings already vary in age and so will vary in the costs required for update and environmental safety. More will need to be spent on cremators in built-up areas with less being spent on those in countryside so that humans gain immediate greater safety.

Mercury when cooled will still drop onto surrounding landscapes/rivers and seascapes. Up-taken by cattle and fish, mercury will still gain access to the human food chain

elevating individual human Hg burden.
Costs of required cremator updates are not only immense to the cremation industry but will add another £30-00 or more to family funeral costs at death.

Crematoria with insufficient funds are encouraged by CAMEO (The Crematoria Abatement of Mercury Emissions Organisation, in UK) and the UK Government to join CAMEO and contribute annual funds to assist reduction in those areas less able to pay the enormous amounts per cremator to upgrade and reduce mercury emission.

A SIMPLER, CHEAPER WAY FORWARD.

It would be more effective to ban mercury from use as a dental filling both in terms of better health for the living and in stopping mercury vapour from entering the food chain.

Mercury emittance from the teeth of the dead injuriously affects the health of the living, is extremely costly to crematoria processes, links into the animal, air, water and land environment seriously contaminating them all.

It is hazardous WITHIN a human tooth, is classed a Hazardous waste during dental removal and as Hazardous waste AFTER body cremation. IT IS HAZARDOUS.

The Cremation Society of Great Britain, www.cremation.org.uk and
Patients Against Mercury Amalgams in UK (www.angelakilmartin.com)
and all the below as acknowledged, earnestly appeal to this UN conference and its resulting Treaty to:

BAN entirely the use of mercury as a tooth filling material in the living and that dental mercury amalgam should be INCLUDED in the list of products no longer allowed to use or include mercury.

Acknowledgements

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Some health papers citing mercury exposure as cause for illness

The Mercury Fillings Compilation. e-book. Patients Against Mercury Amalgams. UK

"Toxicology" pub: Pergammon Press 1991.

Dr. Boyd Haley, www.altcorp.com/amalgam.htm

Chronic low-level mercury exposure and neuropsychological Functioning; Uzell and Oler University of Pennsylvania, USA.

"Retrograde degeneration of Neurite membrane Structural Integrity of nerve growth Cones following exposure to mercury". Leong, Syed, Lorscheider. Journal; NeuroReport 12(4): 733-737, 2001.

The Toxicity of Mercury in Dental Amalgam. Bauer and First, CDA Journal. 47-51

Birth Defects. Work by Huggins, Chang, Vimy, Stortebecker, Hanson and many others.

Autism- a novel form of mercury poisoning. Autism research Institute, USA. www.autism.com/ari/mercury.html

There are tens of thousands of verifiable science papers and textbooks showing the toxicity of mercury within the human body in all body systems.

Mercury emissions from crematoria:

DEFRA (Dept of Environment, Food and Rural Affairs. Air quality notes AQ1 (05) AQ13(05), AQ24 (05), AQ10(07) www.defra.gov.org.uk)

Office of Fair Trading. Cost of a funeral. http:www.oft.gov.org.uk

CAMEO (Crematoria Abatement of Mercury Emissions Organisation (update January 2006)www.cameoonline.org.uk

Secretary of State's Guidance for Crematoria. Guidance Note 5/2/04.

The Federation of Burial and Cremation Authorities. www.fbca.org.uk

The Cremation Society of Great Britain. www.cremation.org.uk

Mercury emission abatement – the true costs. Westerleigh group,plc www.westerleighgroup.co.uk

ABATEMENT – THE REAL CHALLENGE-THE TRUE COSTS

ADRIAN BRITTON. www.westerleighgroup.co.uk

Hilary, thank you. Ladies and gentlemen good afternoon. I would like to take this opportunity to thank Duncan McCallum for suggesting that I come and speak to you today.

I do not want to dwell for any period of time on abatement equipment and cremator technology. This has been well documented over the last 3 to 4 years and there have been numerous seminars relating to this subject matter. Indeed cremator manufacturers are well represented at this conference and there will be further opportunities to have discussions with them over the next 2 days. Equally I do not want to discuss abatement legislation. Brendan Day will be covering this topic tomorrow and he will be informing you of some particularly important changes that have taken place in recent weeks. I do however need to mention the background and would like to start by just reminding you of the conference that took place at St. John's in Solihull in 2001.

Following that conference, when we had just been advised that abatement was to be introduced, a questionnaire was distributed by the Federation and it was discovered that should the equipment have to be fitted, 23% of UK crematoria may well close. This figure has been debated at length throughout the industry since 2001 and people appear to have very different opinions as to its accuracy, but I think that currently the majority of people are agreed that around 20% of sites will close if abatement had to be fully fitted.

Within two years DEFRA had reconsidered the situation and a 50% abatement target was introduced. It was agreed that by 1st January 2013 abatement equipment should be installed or burden sharing should take place. The burden sharing idea suggested by DEFRA had been used elsewhere in industry for sometime. Within a short period, as a result of this legislation, the Federation started CAMEO (Cremation Abatement and Mercury Emissions Organisation) and I would remind you very briefly of its four major functions.

Firstly it has the function of reporting annually to DEFRA in order that Mike Etkind can monitor the position at any given time against the targets set. Where we are in relation to those targets Brendan Day will be informing you during his paper later in the week.

Secondly at the creation of CAMEO the Board decided that it was important that due to the complexity of the subject matter that there was an education function. With this in mind the Stratford Conference was held last year and since that date a series of seminars has been arranged looking at different installations throughout the country. I do encourage you to attend these as there is a great deal to be learned from the practical experiences of others. Furthermore CAMEO will operate a burden sharing scheme from January 2013 and it is has been authorised to oversee other burden sharing schemes. It was planned that abatement would be based on 2003 cremations but negotiations are currently taking place in an effort to change this as in a developing market it does not

appear fully equitable. The other issue that should be covered at this point is cost. What is the cost to the individual who is going to burden share? Clearly this is impossible to say at this moment, until we know the numbers of sites who are actually going to abate. As we get closer to 2013 we will get more idea of the abated numbers and be able to make a more accurate assessment, but suffice it to say for the moment that we are probably talking of a figure between £25 and £35 per cremation.

My final slide by way of background shows the cost to the industry of abatement. If we were to end up with 50% abatement I believe that this will cost a minimum of £125m, approximately £1m per site, but it could be as much as £200m, depending on the amount of Civil Works to be carried out.

I am going to talk around the subject of fitting Abatement Equipment this afternoon and each step of the way I will endeavour to give you some estimates of appropriate costs. So what is the cost of the Equipment itself? These are pure estimates but to give people a guideline can we say that the cost of a new cremator is circa £150,000 the cost of single abatement is around £400,000, two into one £450,000 and three into one £500,000. So for a two cremator installation we are talking in terms of £700,000 to £750,000 for the equipment alone.

I think however it is important to understand that not many people are going to fit abatement equipment into their crematoria as they currently stand. For the majority of people the major problem will be to create sufficient space. I have visited a large number of sites throughout the country and I can assure you that there are very few where equipment can be installed without some major structural changes or reducing the number of cremators. Of people I have spoken to, I know that a number are considering reducing four cremators to three and from three to two. Equally a number are thinking of replacing double ended cremators with single ended. I know from the sites represented here that one or two of you have fairly cavernous crematories but the majority are not that lucky. If you are extending your crematory you are talking in terms of a minimum of £2000 per square metre, but if you were to have a listed building it could be considerably more. The other major consideration with the crematory is the base. The weight of the equipment which you are installing as opposed to the existing. Secondly as there may be a great deal of rearrangement in your crematory you need to consider the areas that are reinforced. If you are unsure you need to do some core work before you go too far with the process as parts of it may be reinforced and parts not. Certainly I have come across situations where this has been the case.

The other fundamental capital improvement that may need to be considered is the ease of access to the site and to the crematory itself. Entrances to the crematory are often very restricted. In some cases you may be able to fit larger double doors, in others it may require a wall to be taken out or even the roof to be removed.

The above are some of the more obvious costs of installation but what of those not so apparent. What is the cost of disposal of your old cremators? I think it is likely to be somewhere between £7k and £10k per unit. I have witnessed the job myself over recent

weeks and it is a particularly dirty job. An old cremator will fill six or seven skips and will require an extended weekend to dismantle. If there is any asbestos involved in the building that price may rise considerably and we may be talking in the realms of £15k - £20k per unit.

We've talked of access to the building, but how about access to the site. We know that a large number of crematoria are situated in the middle of old Victorian Cemeteries and often the width of access drives may be a problem. Often trees may need pruning or at worst removing. That may be large cost and planning. There may also be the need to increase the volume and pressure of your gas and you may need to consider the electrical capacity. It is important that you consider this and plan at an early stage as planning with Public Utility Companies can take an inordinate period of time. The changes that we have mentioned will vary in cost dramatically according to the circumstances of your site.

A one off cost to consider is the stack emission test. On commissioning this is likely to be in the region of £5k - £6k in addition to the annual testing. Costs of the latter will generally be slightly higher than they are currently. Other related issues include the hard standing for crane usage and installation. Also scaffolding for testing purposes.

In financial terms possibly the most important consideration is downtime during the installation. This is such a complicated topic that it could be the subject of a separate paper. If you can actually install abatement equipment, making the appropriate changes whilst loosing only a few operational days, then that is a massive saving in the overall cost. There have recently been a number of crematoria which have closed for considerable periods of time and this can be avoided by meticulous planning.

The easiest installations are in new buildings. You can see on this slide that we have a low loader carrying a cremator weighing 13-14 tonnes. Here the entrance next to the stack at the back of the building has been made of sufficient size to slide the equipment into the crematory. Just one consideration here as you are dealing with such heavy weights is to consider where your mains services are positioned to ensure that these are not damaged on entrance to the site. I have witnessed one installation where the mains drainage was badly damaged by the weight of the delivery lorries and although it wasn't recognised immediately it resulted in some very considerable expenditure 2-3 years down the line. So before the work is started some detailed surveying is essential.

We've talked about capital costs, so how about operational costs? When abatement was first muted, people generally thought that operational costs were going to be particularly high but I actually think that the figures bounded around have been greatly exaggerated. I only currently have the gas costs for two new installations, but both of these have experienced savings of up to 40%. There will be additional electrical costs; you have a further I D fan, motors etc. which may result in an additional cost of 10-15%. Purchase and disposal of reagent will only cost a couple of pounds per cremation. Servicing costs will be probably two extra days per year, so approximately £600 - £800. When abatement was introduced people said that labour costs would increase dramatically but in my experience to date they haven't. So in general the operational costs have risen

minimally and to weigh against this there are some gas savings. In terms of costs therefore we are talking primarily about Capital Expenditure and the interest costs therein.

From CAMEO's latest survey 65% of sites have said that they are looking to install abatement equipment. The survey does not however include some forty crematoria. Now it could be of those forty that a high proportion are considering abatement in which case the mentioned figure could be as high as 70%. There have been installations in the last year at Manchester, Kings Lynn, Bath, Westerleigh, North East Surrey and Carlisle. There have also been new installations at South Lanarkshire and Ollerton, and I am aware of at least eleven further projects to be completed in the coming year. While the process to date has been slightly slower than initially hoped, it is gaining speed all the time.

I'd like to look at some individual examples, I apologise for showing you Westerleigh first, a site dear to my heart since it was opened in 1992. It was planned in the early 1990's to complete 2,000 cremations per year, and space was made in the crematory for three cremators. It is now doing the 2,000 but only with the use of two cremators and hence we had spare space available. The first phase was to put a new cremator in the area marked yellow on the plan. Phase II was to remove the two old cremators and install a second piece of equipment and phase III saw the insertion of abatement equipment with an air blast cooler on the back wall of the building. From the slides you can see the original equipment together with the fan room next to it. Following on you see the two new cremators and the abatement equipment fitting like a glove. I must say this was more luck than judgement and there are not many people in this position. Space is normally at a premium.

The next example I would like to use is Torbay which is typical of many crematoria in the UK having been constructed in the 1950's, 1960's and early 1970's. You will see that at the front of the building there is a very large access road together with hard standing to the side of the building to the edge of the crematory. There is a little room to extend the crematory but in quite close proximity there are areas of lawn with interred cremated remains. This is a common scenario, but in many situations the position is even more difficult as remains are immediately next to the back wall of the building. You can see from the attached slide of Arnos Vale how close burials may have taken place to the crematory. This together with the very small entrance and the listed building status can make the changes required impossible.

A less extreme example is Haycombe in Bath built in the 1950's. You can see from the slide that the access is very narrow. A corridor four feet wide and some twists and turns before entering the crematory. The solution was to dig 250 tonnes of chalk away from the side of the crematory, build new retaining walls, safety fences and a new access through double doors in the crematory. The air blast cooler is on the hard standing at the back wall of the building. An interesting solution to a difficult problem.

The next slide is of West Bromwich, another 1960's building. Again a very dated building which has particular problems in accommodating the flow of people and traffic

around the site. There is no separate road to the Port-Cochere which is situated only fifty yards from the public highway and is therefore very difficult to change. It was originally decided to totally refurbish the building but when this was costed together with the replacement of the cremators the figures were around £3m. That being the case it was decided that Soundwell Council could have a new crematorium on the site for approximately the same price. When enquiries were made it was found that the Council owned the adjoining land so it was decided to position the new building some 100 yards to the east. This in turn has created the space for further parking and enables the flow of traffic to be improved. This plan will result in very little downtime as the existing building can continue to be used while the new one is constructed.

In conclusion when installing Abatement we have discussed operational costs and they are not a major issue. The capital costs however are very significant and an average installation may cost from £ ¾ m to £1¼m. We have examined cases where costs may be significantly more however, as in the Bath scenario where they have had to spend £300k - £400k before installing the equipment.

A large number of you will spend the minimum possible in installing abatement, whilst many of you may use this as an opportunity to improve you facility in the way that Brendan is at West Bromwich.

My suggestion would be that if you have a particularly difficult set of circumstances, with insufficient access, burial or cremated remains interred close to the building and a facility that is in need of upgrading you ask yourself the question whether or not it is worth spending £1m on abatement. We believe that up to 70% of sites are considering installation and hence the national target for the industry should not be a problem. In these circumstances in the short term, many people will be better off burden sharing and actually saving their money, ring fencing their profits for a future date five or ten years down the line when they will be able to build themselves a new crematorium worthy of the 21st century.

Ladies and Gentleman, thank you very much.
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