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# Public Comment to the FDA Proposed Classification of Mixed Encapsulated Dental Amalgams

## Submitted 7-28-08

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U.S. Food and Drug Administration
Division of Dockets Management (HFA-305)
5630 Fishers Lane, Rm. 1061
Rockville, MD 20852

Re: *Public Comment*
*Docket No. FDA-2008-N-0163 (formerly Docket No. 2001-N-0067)*

Dear FDA:

The International Academy of Oral Medicine and Toxicology ("IAOMT") is an organization of dentists, physicians, and research professionals who are committed to improving the biocompatibility of dental materials and procedures by examining the record of scientific literature, and by sponsoring relevant research where the literature is lacking. Our 620 active, dues paying members are the vanguard, the forerunners of opinion of the progressive dental community. For many years, our position has been that dental amalgam has never been (and cannot be) proven safe, that its use indiscriminately exposes dental patients and dental staff to unacceptable levels of mercury exposure, without regard to the myriad of individual factors that affect a person's ability to withstand such toxic exposures. Moreover, mercury-laden dental office waste and human excretion of amalgam-derived mercury place an unnecessary burden on the environment and on wastewater treatment facilities.

Dental mercury-silver amalgam must not be classified in Class II, which would effectively confer "generally regarded as safe" status. It is not safe. The IAOMT position is that amalgam should be banned, removed from the market just as every other mercurial medical device and substance has been. At the very least, it should be placed in Class III, and let the advocates prove that it is safe. We are confident that such proof is not available. Mercurial wound disinfectants are gone, mercurial diuretics are gone, mercury thermometers are gone, and so are all mercurial veterinary substances. There is no magic that makes dental mercury safer than those obsolete products of the past. In this era when the public worries about the mercury they are ingesting through fish consumption, the FDA should do the right thing and ban amalgam dental fillings as the time-release mercury exposure devices they are.

The IAOMT Scientific Advisory Committee, a blue-ribbon panel of toxicologists and biochemists, has separately submitted a brief detailing the scientific evidence against the concept of "amalgam safety." This letter will attempt to address more of the clinical topics involved, and answer the specific questions posed by the FDA staff, as they defined this public comment period.

## I. Pertinent FDA Regulations

### A. Regulation of Dental Amalgam

To date, dental amalgam is not an FDA approved dental device. There is no FDA notification of approval, no 510K, and no classification of dental amalgam in the Federal Register.

In 1976, the President and the Congress of the United States directed FDA to evaluate all medical (including dental) devices intended for human use and to classify them according to their safety and effectiveness. [FR 41(157):34099, 12 Aug 1976.] To this day, "dental amalgam" is not listed as an accepted and classified dental device, even though it has been the most widely utilized of all dental devices.

FDA has ruled that mercury is not GRAS (Generally Recognized to be Safe). [FR 63(77): 19799-19802, 22 Apr 1998.] Contradictory to this finding, the FDA Dental Device Division accepted and classified "Dental Mercury" as a Class I device, implicitly concluding that this material is safe and effective as a dental device. [FR 52(155):30082-30108, 12 Aug 1987.]

FDA Rules clearly define the evidence that is acceptable and worthy of consideration in determining the safety and effectiveness of dental devices. [FR 43(146):32988-32999, 28 Jul 1978.] Expressly excluded from consideration are "random experience, reports lacking the details to permit scientific evaluation, or unsubstantiated opinion." [Pg. 32995.] Further, FDA Rules define the "valid scientific evidence" required to accept devices. [Pg. 32990-32996.] Notwithstanding these requirements, the Dental Division of FDA voted to classify "Dental Mercury" and "Amalgam Alloy" as safe and effective dental devices without providing valid scientific documentation as required by FDA Rules.

**B. FDA's Regulation of Implants**

When utilized as a dental filling material and placed in living tissue in a human body, mercury amalgam implants are a medical/dental device and as such, under existing law, must be classified. By definition, it must be classified as an implant and automatically placed in Class III, requiring scientific proof of safety. The FDA definition of an implant appears in FR 43 32994, July 28, 1978: "Implant means a device that is placed into a surgically or naturally formed cavity of the human body. A device is regarded as an implant for the purpose of this part only if it is intended to remain implanted continuously for a period of 30 days or more, unless the commissioner determines otherwise in order to protect human health."

In 1978, the FDA Dental Device Panel requested that dental amalgam be exempted from the FDA Rule definition for "implant" ("a device that is placed into surgically or naturally formed cavities of the human body." [FR 42(177):46035, 13 Sep 1977.]) The FDA Commissioner denied that request [FR 43(146):32988, 28 Jul 1978.] The FDA Dental Device Panel ruled that dental amalgam was ***not*** an implant, in direct contravention of the ruling of the FDA Commissioner. [FR 45(251):85964, 30 Dec 1980.] We believe the ruling of the Dental Device Panel is a legal nullity because it is contrary to the ruling of the Commissioner.

**C. Mercury Amalgam Implants Must be Classified in Class III**

FDA Rules further state: "Although no device can be regulated adequately in Class I or Class II unless there are adequate data and information establishing its safety and effectiveness, a device for which there are such data and information may nevertheless require regulation in Class III because of the public health concerns posed by its use." [FR 42(177):46030, 13 Sep 1977.] The Dental Device Division violated this requirement in their final ruling in 1987. [FR 52(155):30082-30108, 12 Aug 1987.]

On February 20, 2002, FDA announced a proposed rule entitled: "Dental Devices: Classification of Encapsulated Amalgam Alloy and Dental Mercury and Reclassification of Dental Mercury; Issuance of Special Controls for Amalgam Alloy." The FDA's announced intention was to reclassify Dental Mercury into Class II and accept a "capsule" containing dental mercury on one side and amalgam alloy on the other as a "safe and effective" dental device. However, H.R.Rep. No. 853 94th Cong., 2d Sess. (1976), as well as the agency's own regulation, 21 C.F.R. § 860.93, require dental amalgam to be classified into Class III. To be classified in any other class, the Commissioner must file a full statement of the reasons for such classification, including "supporting documentation and data satisfying the requirements of sec. 860.7." 21 C.F.R. § 860.95(b). As dental amalgam is an implant, it must be classified in Class III unless and until the Commissioner enters written findings concluding that the product may generally be regarded as safe and may therefore be classified in Class II. However, the FDA has already ruled that mercury cannot be generally be regarded as safe, and amalgam therefore must be classified in Class III. Given the considerable body of science demonstrating the health risks associated with the use of this product, we do not believe that dental amalgam can be properly classified in Class II.

### D. Risk of Allergic Reactions

In the Federal Registry, Volume 52(155):30089, August 12, 1987, the FDA changed the classification of dental mercury, a component part of mercury fillings, from the proposed Class II to Class I, stating, "...warnings under the misbranding provisions (21 U.S.C. 352) of the general controls of the act would warn dentists about the rare risk of allergic reactions among patients and the risk of toxicity to dental health professionals."

Arriving at its conclusion that the risk of allergic reaction was "rare," the FDA relied on three (3) case reports, ignoring several other scientific studies clearly within the criteria set out in 21 C.F.R. 860.3, 860.7 for valid scientific evidence which showed that the risk of hypersensitivity (allergic) reaction to mercury effects at least five (5%) to eleven (11%) percent, and perhaps more, of those individuals receiving mercury fillings. Since August 12, 1987, most manufacturers have failed to warn of the risk of allergic reaction as required by 21 U.S.C. § 352 and the FDA has failed to force them to do so under 21 U.S.C. 334 and 21 C.F.R. § 800.55.

## II. Mercury Fillings are Potentially Harmful

### A. Mercury is not Locked into Dental Amalgam

Mercury is not locked into the amalgam matrix, but is continuously released as a vapor and inhaled into the lungs of the dental patient. On average, eighty percent of the mercury

inhaled into the lungs is absorbed into the bloodstream.[1] Elemental mercury is continuously emitted from dental amalgam fillings and absorbed by the patients in whom the fillings are implanted. Studies demonstrate that two-thirds of the mercury absorbed by non-occupationally exposed populations is derived from amalgam fillings.[2] [3] [4] [5] [6] Other studies have confirmed a correlation between the number of fillings and the mercury found in cadaver brains.[7] A recent peer-reviewed article that analyzes the relationship between maternal dental amalgam fillings and exposure of the developing fetus to mercury concludes that there is a strong positive correlation between maternal and cord blood mercury levels; and, the cord blood mercury levels were significantly associated with the number of maternal amalgam fillings. The investigators concluded, "Dental amalgam fillings in girls and women of reproductive age should be used with caution, to avoid increased prenatal mercury exposure."[8] Holmes found that mercury levels in the baby's first haircut correlated well with the maternal mercury/silver-filling burden unless the child suffered from Autism.[9] In the latter case the baby's hair had almost no mercury thus indicating in the authors view that the child was a non-excreter for mercury and was harmed inter-utero by maternal mercury burden.

Mercury is a very toxic substance-- more toxic than lead, cadmium, or arsenic.[10] The material has been banned in thermometers, thermostats, fluorescent lamp alternatives, mercury-containing components in vehicles, batteries, paint, medical waste. Steps are being taken to reduce human exposure to mercury in all contexts but not in the context of tooth restoration. One

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[1] Kudsk, F.N., *Absorption of Mercury Vapour from the Respiratory Tract in Man*, Acta Pharmacol. et Toxicol. 23:250-262 (1965.)

[2] Aposhian, H.V., et al., "*Urinary mercury after administration of 2,3-dimercaptopropaane-1-sufonic acid: correlation with dental amalgam score*," FASEB J, vol. 6 (April 1992), pp. 2472-2476.

[3] *See also*, Sandborgh-Englund, et al., "*Mercury in Biological Fluids After Amalgam Removal*," J Dent Res, 77(4): 615-24 (Apr. 1998);

[4] World Health Organization, "Environmental Health Criteria 118: Inorganic Mercury," (1991) p. 36;

[5] Clarkson, T.W.; et al., "*Biological Monitoring of Toxic Metals: The Prediction of Intake of Mercury Vapor From Amalgams*," (1988) p. 256. ("The release of mercury from dental amalgams makes the predominant contribution to human exposure to inorganic mercury including mercury vapor in the general population.");

[6] Lorscheider, FL; et al. *"Mercury Exposure from Silver Tooth Fillings: Emerging Evidence Questions a Traditional Dental Paradigm."* FASEB J., 9:504-8 (1995.) ("[D]ental amalgam tooth fillings are ***the*** major source of Hg exposure for the general population.") [Emphasis added.]

[7] Eggleston, et al., "*Correlation of dental amalgam with mercury in brain tissue*," J Prosth Dent, 58(6) (1987.)

[8] Palkovicaova, L., "*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.

[9] Amy S. Holmes, Mark F. Blaxill, Boyd E. Haley, "*Reduced Levels of Mercury in First Baby Haircut of Autistic Children*," International Journal of Toxicology 22:277-285 (2003.)

[10] Sharma, RP; Obersteiner, EJ., "*Metals and Neurotoxic Effects: Cytotoxicity of Selected Metallic Compounds on Chick Ganglia Cultures*," J Comp Pathol, 91(2):235-44 (1981.)

of the most dangerous metals known to mankind is mercury because it vaporizes into an extremely poisonous volatile gas at room temperature. There are a number of well-conducted studies that have found injury to dental employees and dentists exposed to low dose mercury. The injuries were both neurological and physical to dental personnel, especially females. [11] [12] [13] Certain dental procedures, such as mixing carving and polishing mercury/silver fillings, release so much mercury that they are inherently dangerous.[14] [15] [16] [17] [18] [19] [20] [21] Many of these procedures release sufficient mercury to exceed the California Maximum Allowable Concentration (MAC) of 100µg/m3. This level has been determined to be immediately hazardous to health and is never to be exceeded under any circumstances.

The U.S. Environmental Protection Agency released a study concluding that 630,000 newborns had unsafe levels of mercury in their blood between 1999 and 2000 — almost twice the original estimate of 322,000. The CDC reports that one in 12 women of childbearing age have mercury levels above EPA's recommended threshold.[22]

Advocates for the continued use of mercury/silver implants claim that the mercury in a

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[11] Linda Jones et al., "*A 30-year follow-up of residual effects on New Zealand School Dental Nurses, from occupational mercury exposure*," Human & Experimental Toxicology, vol. 26, No.4, 367-375 (2007.)

[12] Bjørn Hilt, Kristin Svendsen, Inger Melø, "*Exposure to elemental mercury and cognitive symptoms in Norwegian Dental Nurses 2007*,"
http://www.badn.org.uk/Default.asp?c=11&sc=0&a=100456&rs=&apg=1&r=&rt

[13] Rowland AS, et. al., "*The effect of occupational exposure to mercury vapor on the fertility of female dental assistants*," Journal of Occupational Environmental Medicine 51,28-34 (1994.)

[14] Nixon GS, Rowbotham TC, "*Mercury hazards associated with high speed mechanical amalgamators,*" Brit Dent J 131:308-11 (1971.)

[15] Cutright DE, Miller RA, Battistone GC, Millikan LJ: "*Systemic mercury levels caused by inhaling mist during high-speed amalgam grinding,*" J Oral Med 28:100-4 (1973.)

[16] Gronka PA, Bobkoskie RL, Tomchick GJ, Back F, Rakow AB, "*Mercury vapor exposures in dental offices,*" J Amer Dent Assoc 8l:923-5 (1970.)

[17] Roydhous RH et al., "*Mercury in dental offices,*" J Can Dent 51(2):156-158 (1985.)

[18] Mantyla DG, Wright OD, "*Mercury Toxicity in the dental office: a neglected problem,*" JADA 92:1189-94 (1976.)

[19] Gordon HP, Gordon LD, "*Reduction in mercury vapor levels in Seattle dental offices*," J Dent Res Abstract #1092 57A:347 (1978.)

[20] Schulein, TM, Reinhardt JW & Chan KC, "*Survey of Des Moines area dental offices for mercury Vapor,*" Iowa Dent J. 70(1):35-36 (1984.)

[21] Ochoa, R & Miller R.W., "*Report on independent survey of American dental offices for mercury contamination,*" Tex Dent J. 100(1):6-9 (1983.)

[22] MMWR *Blood Mercury Levels in Young Children and Childbearing-Aged Women* November 5, 2004/ 53(43);1018-1020.

silver filling has never been proven to have harmed anyone.[23] This is the equivalent of declaring that a bullet in a gun never harmed anyone. We are not concerned about the mercury in the filling but rather the mercury that continuously exits the filling and accumulates in distant organs or a fetus.[24] [25]

**B. Risk Assessment Studies**

Risk assessment studies for mercury demonstrate that the quantity of mercury absorbed by people with amalgam exceeds the mercury doses established as safe by the Environmental Protection Agency, the Agency for Toxic Substances & Disease Registry, and Health Canada. The U.S. and Canada have developed minimum risk levels for mercury through government sponsored risk and exposure assessments. Through the development of formal toxicological profiles, the ATSDR establishes "Minimal Risk Level (MRL)" exposure standards for the general population in the United States. The ATSDR has also published its Toxicological Profile for Mercury, in which it established a "minimum risk level" for mercury. The MRL for mercury established in this publication set the chronic inhalation MRL for mercury at 0.0002 mg/m³. The daily dose resulting from such an exposure would be 2.4 µgs/day. In Canada, Health Canada commissioned its own risk assessment to evaluate general population exposure to amalgam mercury. This formal assessment was presented to Health Canada in August of 1995 [26] and was later published in a peer-reviewed risk assessment journal.[27] This report established a tolerable daily intake (a/k/a reference dose) for mercury 0.014 µgs/kg/day, which would equal 1.4 µgs/day.

The EPA also conducted a formal risk assessment for mercury and determined a sub-chronic (short-term) reference dose of 0.3 µgs/m³ with an equivalent absorbed daily dose of 3.84 µgs. U.S. EPA. "Health Effects Assessment Summary Tables: FY-1997 Update" (1997). *Id*. Absorption of mercury in excess of these doses presents increasing risk of neurological harm. A dental patient with amalgam fillings may (and probably does) absorb mercury in excess of these published toxicological thresholds.

In reviewing scientific literature published on this subject between 1997 and 2002, the Swedish Dental Material Commission concluded in its risk assessment study that "[t]he lowest exposure, in terms of urinary mercury secretion, that has been found to give rise to a

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[23] Jendresen MD, "*Mercury in dental amalgam: is it safe?*," J Calif. Dent Assoc 10:3l-2 (1982.)

[24]Abraham JE, Svare CW, Frank CW, "*The effect of dental amalgam restorations on blood mercury levels.*" J Dent Res 63:71-3 (1984.)

[25] Svare CW, Peterson LC, Reinhardt JW, Boyer DB, Frank CW, Gay DD, Cox RD, "*The effect of Dental Amalgams on mercury levels in expired air,*" J Dent Res. 60:1668-71 (1981.)

[26] Health Canada. "*Assessment of Mercury Exposure and Risks From Dental Amalgam: Final Report*." Richardson, G.M., Ph.D., Medical Devices Bureau, Environmental Health Directorate.

[27] Richardson and Allan (1996), "*A Monte Carlo Assessment of Mercury Exposure and Risks from Dental Amalgam,*" Journal of Human and Ecological Risk Assessment (HERA Vol. 2, No. 4.)

demonstrable toxic effect has fallen from 30-50 µg/l to 10-25 µg/l. Accordingly, the safety margin that it was thought existed with respect to mercury exposure from amalgam has been erased."[28] The Commission further concluded that "[a]dditional facts have come to light that may indicate that mercury vapour can affect human foetal development."

This dose is of even greater concern where the patient is young. "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."[29] 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.[30]

About eight percent of U.S. women of childbearing age have enough mercury in their blood to be at risk. The National Academy of Sciences estimates that 60,000 newborns a year could be at risk of learning disabilities because of mercury their mothers absorbed during pregnancy. Significantly, mercury in the tissues of fetuses and infants (11-50 weeks of life) correlates significantly with the number of dental amalgam fillings of the mother.[31] Ninety percent of blood mercury is derived from time-release *in situ* mercury/silver implants.[32]

### C. Occupational Exposures and Adverse Effects

Using exposure assessment methods that he published[33], Dr. G. Mark Richardson, the author of the 1996 Health Canada risk assessment study on dental amalgam[34], estimated that a dentist who removes four amalgams per day will inhale 38 milligrams of mercury derived from amalgam particulate. Dr. Richardson was an expert witness for Dr. David Barnes in litigation against amalgam manufacture Kerr Corp. (discussed below). In assessing Dr. Barnes's

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[28] Berlin, M., "*Mercury in dental-filling materials—an updated risk analysis in environmental medical terms,*" The Dental Material Commission—Care and Consideration.

[29] Goldman LR, Shannon MW, "*Technical Report: Mercury in the Environment: Implications for Pediatricians,"* American Academy of Pediatrics: Committee on Environmental Health. Pediatrics (2001) Jul;108(1):197-205.

[30] Lynn R. Goldman, MD, MPH; Michael W. Shannon, MD, MPH, "*Technical Report: Mercury in the Environment: Implications for Pediatricians*" (RE109907), American Academy of Pediatrics and the Committee on Environmental Health, Pediatrics, Volume 108, Number 1, July 2001, pp 197-205.

[31] Drasch et. al., "*Mercury Burden of Human Fetal and Infant Tissues*," European Journal of Pediatrics (August 1994.)

[32] Snapp K.R. Svare C.W. and Peterson L.D., "*Contribution of Dental Amalgams to Blood Mercury Levels*," J Dent Res 65:311, 1981 Abstract #1276, Special issue.

[33] Richardson, G.M., *Inhalation of Mercury-Contaminated Particulate Matter by Dentists: An Overlooked Occupational Risk*, Human and Ecological Risk Assessment, 9:1519-1531 (2003.)

[34] Health Canada. "*Assessment of Mercury Exposure and Risks From Dental Amalgam: Final Report*." Richardson, G.M., Ph.D., Medical Devices Bureau, Environmental Health Directorate.

occupational exposure to mercury, Dr. Richardson estimated that Dr. Barnes was absorbing between 8019 and 8779 micrograms ("µgs") of mercury into his blood stream every workday. [Exhibit 1.]

A number of studies demonstrating neurobehavioral deficits in dental personal have been published.[35] [36] [37] [38] [39] [40] Dentists with occupational exposure to mercury score below normal on neurobehavioral tests of motor speed, visual scanning, verbal and visual memory, and visuomotor coordination.[41]

Studies demonstrate the neurobehavioral effects of elemental mercury on dentists.[42] One study detected "significant [central nervous system] effects" among dentists and dental assistants at very low levels of Hg $^0$ exposure (i.e. urinary $Hg^0$ ‹ 4 µgs/liter). Significantly, the authors concluded that "[t]he pattern of results, comparable to findings previously reported among subjects with urinary $Hg^0$ › 50 µgs/liter, presents convincing new evidence of adverse CNS effects associated with low $Hg^0$ exposures within the range of that received by the general population." This finding demonstrates adverse neurobehavioral deficits in dentists and dental assistants at urine mercury levels essentially equivalent to the urine mercury levels of those people in whom amalgam has been placed.

In Germany in 1994 the Department of Health conducted a peer-reviewed scientific assessment of the safety of dental amalgam. Arguments for amalgam safety were based upon occupational workplace standards. The reviewers concluded that arguments for safety failed to establish their point for two reasons. Some individuals who are outliers in exposure exceed these standards. Furthermore, occupational standards are for a 40-hour week and when converted to a 24/7 (168-hour) basis an even greater percentage of the population is exposed to mercury at and

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[35] Ngim, CH; et al., "*Chronic Neurobehavioral Effects of Elemental Mercury in Dentists*," Brit J Indust Med, 49:782-90, 1992.

[36] Gonzalez-Ramirez, D; et al., "*Sodium 2,3-Dimercaptopropane-1-Sulfonate Challenge Test for Mercury in Humans: II. Urinary Mercury, Porphyrins and Neurobehavioral Changes of Dental Workers in Monterrey, Mexico*," J Pharmocol Exper Therap, 272(1):264-74 (1995.)

[37] Shapiro, I.M., et al., "*Neurophysiological and neuropsychological function in mercury-exposed dentists*," Neurotoxicol Teratol, 17(2):161-8 (1995.)

[38] Standard medical textbooks also recognize this phenomenon. *See* Harrison's Principles of Internal Medicine, 14th Edition.

[39] Echeverria, D, et al., *"Behavioral Effects of Low-Level Exposure to $Hg^0$ Among Dentists*," J Pharmocol Exper Therap, 272(1):264-74 (1995.)

[40] Uzzell, B.P., et al., *Chronic low-level mercury exposure and neuropsychological functioning*. J of Clin and Exper Neuropsych. 8, 581-593.

[41] Harrison's Principles of Internal Medicine, 14th Edition at 2567.

[42] Echeverria, et al., "*Neurobehavioral Effects from Exposure to Dental Amalgam $Hg^0$: New Distinctions Between Recent Exposure and Hg Body Burden*," FASEB J. 12, 971-980 (1998.)

above this standard, which clearly was never intended to protect vulnerable subsets of the population.[43]

The current scientific data indicates that female dental personnel are severely impacted by occupational exposure to mercury. The Occupational Safety and Health Act (OSHA) has recommended no exposure of fertile women to amounts of mercury greater than 10 micrograms per cubic meter of air, and pregnant women should be occupationally exposed to no mercury. These recommendations are not being followed by the dental industry, and there is substantial scientific evidence that even these modest measures would not fully protect dental workers. Research has shown that mercury even in extremely small amounts has toxic effects on the neurological system, including cytotoxicity to nerve tissue.[44] [45] [46] [47] [48] [49] [50] [51] [52] [53] [54] [55]

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[43] Friberg, LT; Schrauzer, G.N., "*Status Quo and Perspectives of Amalgam and other Dental Materials*," International Symposium Proceedings Georg Thieme Verlag Stuttgart - ISBN 3-13-102471-2 New York (1995.)

[44] Sharma, R.P., Obersteiner, E.J., "*Metals and Neurotoxic Effects: Cytotoxicity of Selected Metallic Compounds on Chick Ganglia Cultures*," Journal of Comp. Pathology Vol. 91 (1981.)

[45] Leirskar, J., "*On the mechanism of cytotoxicity of silver and copper amalgams in a cell culture system*," Scand J Dent Res 82:74-81 (1974.)

[46] Wedeen, R.P., "*Lead, Mercury and cadmium nephropathy*." Neurotoxicology (Park Forrest Ill, 4(3): 134-146 (1983.)

[47] Weening, J.J. et al., "*Autoimmune reactions and glomerulonephritis caused by heavy metals and other toxins*," Dev Toxicol Environ Sci, 11: 211-216 (1983.)

[48] Weening, J.J. et al., "*Mercury induced immune complex glomerulopathy: an experimental study*." Chapter 4: pp 36-66. VanDendergen (1980.)

[49] Koller, L.D., "*Immunotoxicology of heavy metals*," Int J Immunopharmacol, 2:269-279 (1980.)

[50] Koller, L.D., "*Immunosuppression produced by lead, cadmium, mercury*," Am J Vet Res. 34:1457-1458 (1973.)

[51] Koller, L.D. et al., "*Immuno response in rats supplemented with selenium*," Clin Exp Immunol. 63 (3) :570-576, (1986.)

[52] Fiskesjo, G., "*The effect of two organic mercury compounds on human leukocytes in vitro*," Hereditas. 64:142-146 (1970.)

[53] Gerstner, H.B., Huff JE., "*Clinical Toxicology of Mercury*," Journal of Toxicology and Environmental Health. Vol 2, Issue 3 (491-526) (1977.)

[54] Verschaeve, L. et al., "*Genetic Damage induced by Occupational Low Mercury Exposure,*" Environmental Research. Vol 12, (306-316) (1976.)

[55] Nordberg, G.F., ed., "*Effects and Dose Response Relationships of the Toxic Metals*," New York: Scientific Publishing Co (1976.)

Dentists' exposure to mercury is associated with many health problems, most notably birth defects and neurological disorders.[56] [57] [58] [59] [60] A 1987 study by Sikorski identified a significant positive correlation between mercury levels in the hair of occupationally exposed women and the occurrence of reproductive failures and menstrual cycle disorders.[61] Recently reported in the literature is the case of a young dentist, professionally exposed to mercury for 35 weeks during her pregnancy, who delivered a severely brain-damaged mercury-poisoned infant.[62]

The textbook Occupational Hazards in the Health Professions cautions against comprehensive amalgam work during pregnancy.[63] Koos and Lango stated as early as 1970 that their research indicated that fertile women should be exposed to no more than 10 Hg µg/m3, and pregnant women should be exposed to no mercury at all.[64] It is likely that the use of mercury in dentistry makes exposure inevitable.[65]

Clearly, women in dentistry are not only at the greatest risk from exposure to mercury, but they are not being adequately protected. An assistant's death was reported in 1969 from kidney failure.[66] The United States Environmental Protection Agency states that, "Women chronically exposed to mercury vapor experience increased frequency of menstrual disturbances

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[56] Gordon H., "*Pregnancy in female dentists - A mercury hazard*," In proceedings of the International Conference on Mercury Hazards in Dental Practice Gloscow, Scotland 2-4 (Sept 1981.)

[57] Panova Z et al., "*Ovarian function in women having professional contact with mercury,*" Akusherstvoi Ginekologiya 13(1) : n29-34 (1974.)

[58] Noe FE, "*Mercury as a potential hazard in medical laboratories,*" New Eng J Med 261:1002-6( 1959.)

[59] Cook, T Yates P., "*Fatal mercury intoxication in a dental surgery assistant*," British Dent J. 127(12):553-555 (Dec 1969.)

[60] Marinova G et al., "*A study of the reproductive function in women working with mercury*," Problemi na akuserstvoto i Ginekologiyata 1:75-77 (1973.)

[61] Sikorski R., et al., "*Women in dental surgeries: reproductive hazards in occupational exposure to metallic mercury.*" Int Arch Occup Environ Health. 59 (6):551-557 (1987.)

[62] Gelbier S, Ingram J., "*Possible fetotoxic effects of mercury vapor: a case report*," Public Health 103(1):35-40 1/1989.

[63] Brune, D.K.. Edling, C., "*Occupational Hazards in the Health Professions*," Chapter 16, 315-316 Boca Raton Fl: CRC Press, Inc. (1989.)

[64] Koos BJ and Longo LD., "*Mercury Toxicity in the pregnant woman, fetus, and newborn infant*," Am J Obstetrics and Gynocology 126(3):390-409 (1976.)

[65] Eggleston DW., "*Dental Amalgam -- To Be or Not To Be*," Pacific Coast Society of Prosthodontists Newsletter 9(2):4-10 10 (1989.)

[66] Cook, T.A., *op. cit.*

and spontaneous abortions; also a high mortality rate was observed among infants born to women who displayed symptoms of mercury poisoning."[67]

The kidney filters the blood and, as a result, chronic exposure to chemicals might eventually induce kidney damage. A 1988 study by Verschoor, et al. evaluated the kidney function of 68 dentists (63 men, 5 women) and 64 female assistants who were apparently healthy, not pregnant, and taking no drugs. They compared the results of their kidney function analysis to 250 workers known to be exposed through the workplace to lead, cadmium, or chromium. Their conclusion was that, "Dentists and dental assistants appear to have a higher potential risk of kidney function disturbances than the workers in these industries. Although this study did not present evidence for changes of renal function parameters in dental practice in relation to Hg-urine levels below 20 µg/l, it certainly suggests that dental practice may carry a risk of renal dysfunction. There is a need to assess the renal hazard of the potential nephrotoxic chemicals used in dental practice."[68]

Kuntz followed 57 prenatal patients with no known exposure to mercury for changes in whole blood from initial prenatal examination to delivery and postpartum hospitalization. The mothers' whole blood total mercury increased during pregnancy from .79 ppb at initial examination to 1.16 ppb at delivery. This represents a 46% increase during pregnancy. Mercury has previously been recognized for its particular ease of crossing the placental membrane. The umbilical cord blood was also sampled at birth and found to have even higher levels of mercury at 1.5 ppb.[69] After careful analysis of the data, Kuntz concluded: "Previous stillbirths, as well as history of birth defects, exhibited significant positive correlation with background mercury levels." He further stated that patients with large numbers of dental fillings exhibited a tendency to higher maternal blood levels, which agrees with both Ott and Abraham. [70]

Vimy has confirmed the transport of mercury from fillings to the fetus in experimental animals (sheep and monkey), and the additional exposure through mothers milk.[71] Berlin has shown the fetal blood content of mercury was raised dramatically at the end of pregnancy exceeding that of the mother at delivery by a factor of at least five. Early abortion, premature birth, low birth weight with a perinatal death, have been observed in monkeys.[72]

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[67] U.S.E.P.A. Mercury Health Effects Update. Final Report (1984) EPA-600/8-84-019F United States Environmental Protection Agency, Office of Health and Environment Assessment. Washington, D.C. 20460.

[68] Verschoor MA, Herbert RFM, Zielhuis RL., "*Urinary Mercury Levels and Early Changes in Kidney Function in Dentists and Dental Assistants*," Community Dentistry and Oral Epidemiology, Vol. 16 #3 (June 1988.)

[69] Pitkin RM, Bahns JA, Filer LJ Jr, Reynolds WA., "*Mercury in human maternal and cord blood, placenta and milk*," Soc Exper Biol Med Proc 1976: 151: 565-7.

[70] Kuntz WD, Pitkin RM, Bostrom AW, Hughes MS, "*Maternal and Cord Blood Background Mercury Levels: a longitudinal surveillance*," Am J Obstet Gynecol 143(4):440-3 (1982.)

[71] Vimy, M.J.; Takahashi, T.; Lorscheider, F.L., "*Maternal-Fetal Distribution of Mercury (203 Hg) Released from Dental Amalgam Fillings*," Journal of American Physiological Society (April 1990.)

[72] Berlin, M Hua, J Logdberg, and Warvinge University of Lund, Institute of Environmental Medicine, Lund Sweden (Abstract The Toxicologist 31st Annual Meeting Vol 12 #1 February 1992.)

Mikhailova, et al. found that 26.8% of women working in a mercury polluted atmosphere suffered from menstrual disturbances. Marinova, et al. found that 29% had hypermenorrhea.[73] The controls found only 0.3% with the same condition. Hypomenorrhea occurred in 15.3% of the exposed group and only 0.6% of the nonexposed group. This could mean that more than 44% of female dental personnel working under these conditions will suffer from reproductive disorders due to mercury in the dental office. This hypothesis is corroborated by two other studies of women occupationally exposed to mercury, which found that 36% to 45% will develop these types of disorders within 6 months of employment, a proportion that increases to 67% within 3 years of employment.[74] [75]

This hypothesis has been further confirmed in a recent study of 418 women working in dentistry who became pregnant during the previous four years. Detailed information was collected on mercury-handling practices and the number of non-contracepting menstrual cycles it took the women to become pregnant. Dental assistants not working with amalgam served as unexposed controls. Women working in offices with poor mercury hygiene factors took longer to become pregnant. The fecundability (probability of conceiving in any given menstrual cycle) of this high exposure group was only 50% of that for unexposed women after controlling for age, smoking, race, frequency of intercourse, history of pelvic inflammatory disease, year the attempt began, and occupational exposure to cold sterilants, x-rays, and unscavanged nitrous oxide.[76]

The most common symptoms were dysmenorrhea (painful menstruation), hypermenorrhea, anovulation (infertility >40%), and hypomenorrhea. These symptoms are known to increase in populations additionally exposed to lead.[77] The relationship between spontaneous abortion, stillborn infants, and mercury has also been confirmed.[78]

Problems that may develop in the fetus from maternal exposure are not always evident at birth. Prenatal exposure to mercury vapor has been shown to have an effect on brain development.[79] Such delayed problems include diminished learning capacity, muscle spasms,

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[73] Mikhailova LM et al., "*The influence of occupational factors on disease of the female reproductive organs*," Pediatriya Akusherstvoi Ginekologiya. 33(6)56-58 (1971.)

[74] Panova Z and Dimitrov G, "*Ovarian function in women having professional contact with metallic mercury*," Akusherstvoi Ginekologiya 13(1):29-34 (1974.)

[75] Goncharuk GA, "*Problems relating to occupational hygiene of women in production of mercury*," Gigiena Truda i Professional nye Zabolevaniya. 5:17-20 (1977.)

[76] Rowland A, Baird D, Weinberg C, Shore D Shy C and Wilcox, "*A Reduced Fertility Among Dental Assistants With Occupational Exposure to Mercury*," National Institute of Environmental Health Sciences, Research Triangle, NC (Abstract The Toxicologist 31st Annual Meeting Vol 12 #1, February 1992.)

[77] Yang S., "*Influence of lead on female reproductive function*," Chung Hua Fu Chan Ko Tsa Chih, 21(4):208-210, Jun 1986 (English abstract p 252.)

[78] Koos BJ and Longo LD*., "Mercury Toxicity in the pregnant woman, fetus, and newborn infant."* A review Am J Obstetrics and Gynecology 126(3):390-409 (1976.)

[79] Berlin, *op cit*.

and altered electroencephalograms.[80] Exposure continues to increase if the infant is nursed, since mercury concentrates eight-fold in breast milk.[81] [82]

In a study of 298 dentists, Shapiro measured their mercury levels by X-ray fluorescence. Of those dentists with greater than 20 µg Hg/liter tissue levels, 30% had polyneuropathies, while those dentists with no detectable mercury levels had no polyneuropathies. Shapiro concluded that these findings suggest that the use of mercury as a restorative material is a health risk for dentists.[83]

In a series of experiments utilizing neutron activation analysis (NAA) to study the mercury content of brain tissues of amalgam bearers, non-amalgam bearers, and dentists, Dr. Magnus Nylander found in the cases of seven dentists and one dental nurse that all had a surprisingly high pituitary mercury content, totally out of proportion to the content found in other parts of the brain. Values ranged from 135 to 4,000 nanograms Hg per gram tissue.[84] [85] He also found in a related study of dentists and dental assistants in Sweden that they have twice the incidence of brain tumors as nondental personnel.[86]

## D. Adverse Effects Associated with Exposure to Mercury

Mercury, we now know, concentrates in the kidneys, and experimental evidence shows that it can inhibit kidney function.[87] Hahn et al. demonstrated distribution of mercury derived from dental amalgam to the kidney.[88] In this experiment, the organ that accumulated the greatest amount of mercury following amalgam placement was the kidney.

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[80] Dencker, et al.,University of Uppsala (Abstract The Toxicologist 31st Annual Meeting Vol 12 #1 February 1992.)

[81] Pierce P., et al., "*Alkyl mercury poisoning in humans. Report of an outbreak.*" JAMA 220:1439-1442 (1972.)

[82] Snyder RD., "*Congenital mercury poisoning*," N Eng J Med. 18:1014-1016 (1971.)

[83] Shapiro IM, Sumner AJ, Spitz LK, Cornblath DR, Uzzell B, Ship II, Bloch P., "*Neurophysiological and neuropsychological function in mercury-exposed dentists*," Lancet 8282:1147-50 (1982.)

[84] Nylander M., "*Mercury in pituitary glands of dentists,*" Lancet 442 (Feb 22, 1986.)

[85] Friberg L, Kullman L, Lind B, Nylander M., "*Kvicksilver i centrala nervsystemet i relation till amalgamfyllningar* (*Mercury in the central nervous system in relation to dental amalgam*)," Lakartidningen 83:519-22 (1986.)

[86] Ahlbom, A., Norell, S., Rodvall, Y., and Nylander, M., "*Dentists, dental nurses, and brain tumors*," Br. Med. J., 292, 662 (1986.)

[87] Boyd, N.D., et al., *Mercury from dental "silver" tooth fillings impairs sheep kidney function*. American J. Physiol, 261 (RICP 30): R1010-4 (1991.)

[88] Hahn, L.J., et al., *Dental "silver" tooth fillings: a source of mercury exposure revealed by whole body scan and tissue analysis*. FASEB J, 3:2641-6 (1989.)

Scientists are concluding that dental amalgam is an unsuitable restorative material because of its effects on the kidneys. "From the nephrotoxicity point of view, dental amalgam is an unsuitable filling material, as it may give rise to mercury toxicity. In these exposure conditions, renal damage is possible and may be assessed by urinary excretions of albumin, NAG, and gamma-GT."[89] [*See* sheep scan, Exhibit 2.] Additional studies found harm to sheep's ability to clear inulin a measure of kidney function (black line) in just 60 days after implanting mercury/silver fillings.[90] [*See* sheep kidney scan, Exhibit 3.]

Critics of the sheep studies claimed that sheep chew too much. Similar studies were conducted on primates (monkeys) fed twice daily and the same distribution pattern for mercury was observed.[91] [*See* monkey scan, Exhibit 4.] Scientific studies have suggested associations between mercury and neurological disease. These studies justify avoiding unnecessary mercury exposure. For example, one epidemiologic study correlates body mercury levels with increased risk of idiopathic Parkinson's disease.[92] Animal studies demonstrate exposure to mercury vapor and autoimmunity.[93] One such study showed that dental silver amalgam and silver alloy implanted in the physiological milieu of the peritoneal cavity released enough metals to adversely affect the immune system.[94]

John Pearlman, MD reported that a 50 year-old athletic female patient had mercury/silver fillings removed and suddenly developed permanent neurological impairment that was ultimately diagnosed as Parkinson's disease and is now in a wheelchair.[95] Manufacturers of mercury/silver fillings warn that removal can be dangerous.[96] Mercury has even been linked to Alzheimer's disease.[97] Professor Boyd Haley concludes that "mercury and other blood-brain permeable

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[89] Mortada WL, Sobh MA, El-Defrawy MM, Farahat SE. Urology and Nephrology Center, Mansoura University, Faculty of Science, Egypt. J Nephrol 2002 Mar-Apr;15(2):171-6.

[90] Vimy M.J., Boyd N.D., Hooper D.E. and LorscheiderF.L., "*Glomerular filtration impairment by mercury released from dental "silver" fillings in sheep*," Department of Medicine, Pathology, and Physiology, University of Calgary, Alberta, Canada. The Physiologist August 15 (1990.)

[91] Hahn, Leszek J.; Kloiber, Reinhard; Leininger, Ronald W.; Vimy, Murray J.; & Lorscheider, Fritz L., "*Whole-body imaging of the distribution of mercury released from dental fillings into monkey tissues*." FASEB, Vol. 4, Nov. 1990, pp. 3256-3260.

[92] Ngim, C., "*Epidemiologic Study on the Association between Body Burden Mercury Level and Idiopathic Parkinson's Disease*," Neuroepidemiology, 8:128-141 (1989.)

[93] Warfvinge, et al., "*Systemic Autoimmunity Due to Mercury Vapor Exposure in Genetically Susceptible Mice: Dose-Response Studies,"* Toxicol Appl Pharmacol, 132:299-309 (1995.)

[94] Hultman, P; et al., "*Adverse Immunological Effects and Autoimmunity Induced by Dental Amalgam and Alloy in Mice*," FASEB J, 8:1183-90 (1994.)

[95] Smoking Teeth Interviews. DVD furnished FDA panel in 2006.

[96] DISPERSALLOY® DISPERSED PHASE ALLOY Tablets, Powder MATERIAL SAFETY DATA SHEET by Dentsply Caulk 38 West Clarke Avenue, Milford DE 19963-0359 Date prepared 9/20/95 Dated Revised 9/24/97.

[97] Ehmann, et al., "*Brain Trace Elements in Alzheimer's Disease*," Neurotoxicology, 7(1):195-206 (Spring 1986); Thompson, et al., "*Regional Brain Trace-element Studies in Alzheimer's Disease*," Neurotoxicology, 9(1):107 (Spring 1988); Vance, "*Trace Element Imbalances in Hair and Nails of Alzheimer's Disease Patients*,"

toxicants that have enhanced specificity for thiol-sensitive enzymes are the etiological source of AD. Included in this category are other heavy metals such as lead and cadmium that act synergistically to enhance to toxicity of mercury and organic-mercury compounds."[98] Cultured neurons exposed to low levels of mercury have degenerated in a manner indicative of lesions observed in Alzheimer's brain.[99]

Dr. Richardson conservatively estimated that a dental patient may receive between of 1730 micrograms ("µgs") during the removal of a single amalgam filling. Another 93 µgs of mercury may be absorbed during the placement of an amalgam filling. [Exhibit 10.] Published accounts have documented adverse reactions immediately following these dental procedures.[100]

Other adverse health effects associated with mercury exposure are well documented. [Published references associating adverse effects with mercury exposure are attached hereto as Exhibit 5.] Professor Matts Berlin, the World Health Organization's leading expert on the risks of mercury, recently concluded that: "Regarding the risk for retardation of brain development it is not according to science and standard of care to place amalgam fillings in children and fertile women."

In 1995, an important review article summarized some of the scientific documentation concerning dental amalgam was published in the highly prestigious scientific publication, the FASEB Journal. The authors detailed the scientific data and conclusions from scores of peer-reviewed articles documenting the deleterious effects of mercury vapor on the immune, renal, reproductive, and central nervous systems. The authors noted that "[r]esearch evidence does not support the notion of amalgam safety." In their conclusion, the authors admonished that:

> The collective results of numerous research investigations over the past decade clearly demonstrate that the continuous release of $Hg^0$ from dental amalgam tooth fillings provides the major contribution to Hg body burden. The experimental evidence indicates that amalgam Hg has the potential to induce cell or organ pathophysiology. At the very least, the traditional dental paradigm, that amalgam is a chemically stable tooth restorative material and that the release of Hg from this material is insignificant, is without foundation. One dental authority states that materials are presently available that are suitable alternatives to Hg fillings.

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Neurotoxicology, 9(2):197-208 (Summer 1988); Wenstrup, et al., "*Trace Element Imbalances in Isolated Subcellular Fractions of Alzheimer's Disease Brains*," Brain Res, 12;533(1): 125-31 (Nov. 1990); Cornett, et al., "*Imbalances of Trace Elements Related to Oxidative Damage in Alzheimer's Disease Brain*," Neurotoxicology, 19(3):339-45 (June 1998); Mutter, "*Alzheimer Disease: Mercury as a Pathogenetic Factor and Apolipoprotein E as a Moderator*," Neuroendocrinol Lett. 2004; 25(5):275-283 ("Inorganic mercury (found in dental amalgam) may play a major role [in the pathogenesis of Alzheimer's Disease."])

[98] Haley, B., *The Relationship of the Toxic Effects of Mercury to Exacerbation of the Medical Condition Classified as Alzheimer's Disease*, The Nordic Journal of Biological Medicine (June-July 2003.)

[99] How Mercury Causes Brain Neuron Degeneration (video) http://www.youtube.com/watch?v=VImCpWzXJ_w

[100] *See, e.g.*, Taskinen, H; et al. "*A Possible Case of Mercury Related Toxicity Resulting From the Grinding of Old Amalgam Restorations*," Scand J. Work Environ. Health, 15:302-4 (1989.)

> It would seem that now is the time for dentistry to use composite (polymeric and ceramic) alternatives and discard the metal alchemy bestowed on its profession from a less enlightened era. Although human experimental evidence is incomplete at the present time, the recent medical research findings presented herein strongly contradict the unsubstantiated opinions pronounced by various dental associations and related trade organizations, who offer assurances of amalgam safety to dental personnel and their patients without providing hard scientific data, including animal, cellular and molecular evidence, to support their claims.

On March 15, 1991, the Dental Products Panel of the FDA convened to consider the issue whether mercury/silver fillings were safe or toxic. In discussing the issue, the Panel seemed to conclude that mercury was safe unless proven otherwise, and that there was not sufficient proof of its harm to consider taking any action to insure safety to the public. In so doing, the Panel ignored the testimony of many, including the testimony of one of the world's foremost and eminent scientific authorities on mercury. Lars Friberg, M.D., Ph.D. of the Karolinska Institute, Stockholm, Sweden, testified that: "[i]n conclusion, we consider that dental amalgam, from the strictly toxicological point of view, is an unsuitable dental filling material. It is our opinion that, in the future, steps should be taken to use, as far as possible other material than amalgam. *** In the interim we find it highly appropriate to classify the mercury used in dentistry as a class III device."[101]

In September 2006, a meeting of the Dental Products Panel and the Peripheral and Central Nervous System Drugs Advisory Committee convened to consider, *inter alia*, whether the conclusions in the FDA's position statement on amalgam (the "White Paper") should be deemed "reasonable." The Joint Panels rejected the FDA contention that the use of dental amalgam may be considered safe. [*See* FDA Questions for Committee Consideration, Exhibit 6.]

We agree with these investigators. The IAOMT prepared its own compilation of literature entitled "*The Case Against Amalgam*" [Exhibit 7], and we encourage the FDA to review this document and the other materials on the Academy website.[102] We believe that the only hurdle to the elimination of this dangerous product from the marketplace is the political influence of the ADA. Certainly, the scientific evidence clearly requires FDA to act responsibly by protecting the public from this hazardous material.

## III. The Purported Proof of Amalgam Safety

### A. The Saxe Study Published in the Journal of the American Dental Association

In an article published in the February 1999 issue of the Journal of the American Dental Association, researchers reported finding "no significant association of Alzheimer's disease with the number, surface area or history of having dental amalgam restorations." This research was led by a dentist, Dr. Saxe. The research was submitted to, but rejected by, the Journal of the

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[101] FDA Transcript 3/15/91, p. 81.

[102] www.iaomt.org.

American Medical Association. The Saxe team then submitted their article to the New England Journal of Medicine; however, the article was again rejected. The article was then submitted to the Journal of the American Dental Association ("JADA"), which agreed to publish the article. JADA even called a "press conference" announcing the release of the article.

At this press conference, two of the authors made statements that were not supported by any of the data in the article and conflicted with numerous major scientific reports, including a 1998 NIH study. The ancillary comments by some of the authors and the results of the JADA publication are at odds with the vast majority of research published that looks at elevated mercury levels in subjects with amalgam fillings. For example, an NIH study on military men demonstrated a very significant elevation of mercury in the blood that correlated with the number of dental amalgams.[103] Another recent publication demonstrated elevated mercury in the blood of living AD patients in comparison to age-matched controls.[104] These studies clearly demonstrate that there should be increased mercury in the blood of amalgam-bearers, especially if you have AD and amalgams. It remains a mystery how the amalgam-bearing group did not have elevated levels of mercury in their blood. Even cadavers have brain mercury levels that correlate with the number of amalgam fillings they had on death.[105]

Further, in addressing the contribution of amalgams to brain mercury and AD, it would be important to divide the AD and control subjects into those with and without existing amalgams upon death. In the Saxe article, this was not done. This singular publication supportive of the ADA position on amalgams, brain mercury levels, and AD represents a weak attempt to control the preconceptions of well-meaning dentists, scientists, physicians and medical research administrators.

The JADA article represents that no statistically significant differences in brain mercury levels exist between the subjects with Alzheimer's disease and the control subjects. Using straightforward statistics, a comparison of the level of mercury in the AD versus controls showed a significant difference in the mercury levels borne by these subjects. This allows one to invoke a Bon-Feroni statistical manipulation. With Bon-Feroni you include the comparison of one pair of data (that may be statistically significantly different taken alone, e.g. mercury levels in the brains of AD versus control subjects) with several other pairs of data rendering the difference statistically insignificant. One known weakness of the Bon-Feroni treatment of several coupled pairs of comparisons is that one very likely will miss a single comparison that is significantly different, and clever people know this. The application of the Bon-Feroni manipulation is what happened in this JADA study that reversed the previous significance of the mercury levels in AD

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[103] Kingman A, Albertini T, Brown LJ, "*Mercury concentrations in urine and whole blood associated with amalgam exposure in a US military population*," *J Dent Res* 1998 Mar;77(3):461-71.

[104] Hock, C. Drasch, G. Golombowski, S., Muller-Spahn, F., Willershausen-Zonnchen, B., Schwarz, P., Hock, U. Growdon, J.H. and Nitsch, R.M. "*Increased Blood Mercury Levels in Patients with Alzheimer's disease*." J. of Neural Transmission v105L1) 59-68, 1998; Gerhardsson L, Lundh T, Minthon L, Londos E., "*Metal Concentrations in Plasma and Cerebrospinal Fluid in Patients with Alzheimer's Disease*." Dement Geriatr Cogn Disord. 2008 May 5;25(6):508-515.

[105] Eggleston DW, Nylander M, Suffin SC, Martinoff JT, Rieders, MF., "*Correlation of dental amalgam with mercury in brain tissue*," J Pros Dent 58:704-7 (1988.)

versus control brain previously reported. Research previously reported by some of the very same researchers involved in the JADA study consistently indicated that mercury levels were higher in AD versus age-matched control brains (14a,b, 15). Only when an ADA dentist became involved did the results change to being insignificant. The data used in this JADA article and funded by NIH needs to be re-evaluated by a different statistician if we are to determine whether the mercury levels in the AD brains differed significantly from controls.

Dentists are fond of quoting trade associations declarations of safety as gospel while the scientific literature emphatically does not support their claims. One particularly disturbing claim often attributed to the dental division of the FDA is that there is absolutely NO scientific evidence linking mercury from amalgam to any medical disorder. This is in stark contrast to the before mentioned research linking mercury from mercury/silver filling to periodontal disease, inflammation and bone loss. In addition, research has linked mercury to idiopathic dilated cardiomyopathy (IDCM)[106]. Victims of this disorder usually suffer cardiac arrest usually at an early age and die. Their hearts have 22,000 times more mercury that comparable hearts that suffered secondary cardiac dysfunction.[107]

Snapp in 1981 carefully removed mercury/silver implants and his experimental subjects experienced a dramatic 90% decline in blood mercury to 10% of baseline. [108] The only logical conclusion is that their mercury/silver implants contributed substantially to their blood mercury. Snapp found a dramatic decline in blood mercury while Molin caused a dramatic increase followed by a slow drop in blood mercury over the next 12 months to 50% of baseline.[109] [110] The IAOMT criticized the careless approach to mercury removal so when she repeated her study she provided adequate protections and confirmed Snapp's earlier finding.[111]

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[106] Frustaci A, Magnavita N, Chimenti C, et al., "*Marked elevation of myocardial trace elements in idiopathic dilated cardiomyopathy compared with secondary cardiac dysfunction*," J American College of Cardiology 33(6) 1578 (1999.)

[107] Dr. John Wilson of Ashville, North Carolina recently discussed a physician colleague's health collapse following an unprotected amalgam removal. Even manufacturers of amalgam caution that mercury/silver filling removal can be dangerous. Apparently Dr. Wilson's 52 year-old physician colleague was not given adequate protection. He voluntarily asked his regular dentist to remove his old mercury/silver fillings and replace them with composite. Within two weeks the physician became seriously ill and was diagnosed with IDCM. Had he not had an emergency heart transplant he would have died of this mercury-related illness. The IAOMT has for over 24 years cautioned against unprotected removal of mercury fillings.

[108] Snapp K.R. Svare C.W. and Peterson L.D., "*Contribution of Dental Amalgams to Blood Mercury Levels*," J Dent Res 65:311, 1981 Abstract #1276, Special issue.

[109] Molin, M, "*Mercury Released from Dental Amalgam in Man Swedish*," Dental J. Suppl. 71 (1990.)

[110] Molin M, Bergman B, Marklund S L, Schütz A & Skerfving S, "*Mercury, selenium, and glutathione peroxidase before and after amalgam removal in man*." Acta Odontol Scand 48:189-202 (1990.)

[111] Molin M, Berglund JR & Mackert Jr, "*Kinetics of mercury in blood and urine after amalgam removal*," J Dent Res 74:420 IADR Abstract 159 (1995.)

"The National Institute of Dental and Craniofacial Research is currently supporting two very large clinical trials on the health effects of dental amalgam. Studies underway for several years each in Portugal and the Northeastern United States involve not only direct neurophysiological measures but also cognitive and functional assessments." Do we really think that the NIDCR and associated ADA personnel are going to deliver up a conclusion to American parents saying "we put a mercury containing toxic material in your child's mouth that lowered his/her I.Q. and made him more susceptible to neurological problems in comparison to the children whom we selected to not get exposed to this toxic material"? Unfortunately, the results from this study will likely follow previously ADA supported research, i.e. no significant results.

Since the NIDCR started this project only four years ago one has to wonder why it took so long for them to get involved in this issue. The "amalgam wars" have been going on for scores of years. Was it the overwhelming amount of modern science showing mercury from amalgams being a major part of the daily exposure that forced their hand and they had to develop a defense? May we trust the conclusions of this study without knowing who put it together and who did the statistics? Not any more than we may trust the assertions of the JADA article concluding that mercury from dental amalgams does not get into the brain.

The NIDCR study excluded children with neurological disease therefore, since autism has an onset at or before age 2 they excluded the principle target for mercury injury from study. In addition, data from CPOX positive children and was collected but inexplicably after more than two years has yet not been published. Porphyrinuria is linked to childhood autistic disorder.[112]

One follow up study in 2008 identified pathophysiology (harm) to kidneys of these children.[113] Another found boys were unable to excrete any mercury through their urine after 7 years.[114] Furthermore the data from the study does not support the author's biased conclusions. Their graph of urinary excretion clearly shows that urine mercury decline while exposure and intake increased. This is a well documented phenomena found frequently in cases of mercury poisoning.

As was proven by the tobacco debate, trying to find any significant negative effect of one product (amalgams) related to any disease through epidemiological studies is very difficult and complex. To do this with mercury would be difficult because of the synergistic effect two or more toxic metals or compounds (e.g., cadmium from smoking) may have on the toxicity of the mercury emitted from amalgams. For example, one publication showed that combining mercury

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[112] Robert Nataf a, Corinne Skorupkab, Lorene Ametb, Alain Lama, Anthea Springbettc, Richard Lathed, "*Porphyrinuria in Childhood Autistic Disorder: Implications for Environmental Toxicity*," Toxicology and Applied Pharmacology Volume 214, Issue 2, 15 July 2006, Pages 99-108.

[113] Lars Barregard, et al., "*Renal Effects of Dental Amalgam in Children: The New England Children's Amalgam Trial,* Environmental Health Perspectives Volume 116, Number 3 (March 2008.)

[114] Woods James, et al., "*The Contribution of Dental Amalgam to Urinary Mercury Excretion in Children*," Environmental Health Perspectives Vol. 115, #10 (October 2007.)

and lead both at LD1 levels caused the death rate to go to 100% or to an LD100 level.[115] At a LD1 level, the mercury or the lead alone was only modestly toxic (i.e., killed less than one percent of the exposed rats.) Thus, the metals were synergistically toxic. The mixing of two non-lethal doses of mercury and lead resulted in an amazingly toxic mixture. Thus, one cannot define a "safe level of mercury" unless the nature of the other absorbed toxicants is determined. The combined toxicity of various materials, such as mercury, thimerosal, lead, aluminum, formaldehyde, etc., is unknown. The effects of various combinations of these toxicants have not been precisely determined, except that a combination of these toxicants is more toxic than any of the toxicants alone.

The ADA was founded on the basis of the organization's belief that mercury-containing amalgams are safe and effective as dental fillings. This may have been an acceptable position in 1850. Today, modern science has proven that amalgams constantly emit unacceptable levels of mercury. Amalgams emit significant levels of neurotoxic mercury, and that mercury is injurious to human health. This mercury would certainly exacerbate (and may cause) the medical condition of those individuals with neurological diseases such as ALS, MS, Parkinson's, autism and AD. The synergistic effects of mercury with many of the toxicants commonly found in our environment make the danger of mercury unpredictable and possibly quite severe, especially any mixture containing elemental mercury, organic mercury, and other heavy metal toxicants such as aluminum.

### B. The Children's Amalgam Study

The major scientific problems with the studies are that the investigators:

1. Ignored measuring the amount of mercury exposure to children by first determining the amount of mercury emitted from an average sized amalgam outside of the mouth. No in vitro data is given to establish a possible dose.

2. Used urine and blood mercury levels, when 90% -plus of mercury is excreted in the feces. This obviates any conclusions they make, as urine mercury levels are unreliable with regards to exposure, which is exactly what the study's own data demonstrates.

3. Did not select the most sensitive clinical testing parameters for detecting mercury toxicity but instead used testing parameters that are known to fluctuate without known cause, or parameters that require much longer-term low level exposure to show an affect.

4. Did not state that their conclusions of amalgam safety should not include children with any prior neurodevelopmental or systemic illness, because that sensitive sub-population was excluded from the trials.

5. Ignored the drop in mercury excretion in the urine after the second year even though the mercury exposure from amalgams remained the same or increased. This is a certain sign of the subjects losing their ability to excrete mercury with continued exposure to this toxic metal.

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[115] Schubert, et al., "*Combined Effects in Toxicology—A Rapid Systematic Testing Procedure: Cadmium, Mercury & Lead*," J of Toxicology & Environmental Health, 4:763 (1978.)

6. Suppressed their porphyrin profile data, which was collected but not published, and dismissed the data with an offhand comment.

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

One of the study authors, James Wood, published an analysis of the data in 2007 that showed evidence of renal damage and differences between boys and girls in the ability to excrete mercury in the urine.[116] The study data demonstrate a declining ability for the male children's kidneys to excrete mercury via the urine after the second year of continued mercury exposure from their amalgam fillings.[117] This is consistent with increased kidney damage and not consistent with a conclusion of safety for dental amalgams as previously stated by the authors of the CAT studies.

Quoting Woods:

> In conclusion, in the present study we describe a strong, positive correlation between mercury exposure from dental amalgam fillings and urinary mercury excretion over a 7-year longitudinal course of amalgam treatment in children. However, significant differences in urinary mercury concentrations between boys and girls with comparable levels of amalgam treatment and times since placement suggest sex-related differences in mercury handling and, possibly, susceptibility to mercury toxicity. These findings are relevant within the context of children's health risk assessment and suggest directions for future research to determine whether differential sensitivities to mercury between boys and girls do exist.

At first, there was significant correlation between the number of implants placed and the urinary mercury but that is clearly not the case by the end of the experiment. Furthermore, urinary excretion of mercury declined as exposure to mercury and the number of mercury/silver fillings increased. This indicates harm to the children and firmly establishes greater accumulation and greater retention in boys.

Boys should be added to the FDA's list of known vulnerable subsets of the population along with females of child-bearing age, infants, small children, CPOX positive, APOe 4/4 and 4/3 genetic subsets, those who are hypersensitive, and especially those with even minimal lead exposure since lead acts synergistically with mercury.

## IV. Amalgam's Effect on the Environment

[116] Woods JS, Martin MD, Leroux BG, DeRouen TA, Leitao JG, Bernardo MF, "*The contribution of dental amalgam to urinary mercury excretion in children*," Environ Health Perspect 115:1527Ð1531 (2007.)

[117] Barregard L, Trachtenberg F, McKinlay S.0, "*Renal effects of dental amalgam in children: the New England children's amalgam trial*," Environ Health Perspect. 2008 Mar;116(3):394-9 (showed a "significantly increased prevalence of microalbuminuria in the children in the amalgam group (CAT study) in the years 3-5.")

For more than a decade the IAOMT has been concerned with the environmental impact of dental amalgam mercury because of its extreme toxicity. Environmental scientists label it a "Persistent Bioaccumulative Toxin" (PBT), and as such, it is important to reduce mercury in the environment by eliminating its source, wherever possible. Current estimates indicate that the dental profession uses about 40 tons of mercury per year in the fabrication of mercury ("silver" or amalgam) dental fillings. Even after the constituents of dental amalgam are mixed and hardened into tooth fillings, this product continues to emit mercury vapor continuously for decades. This product is the major contributor of mercury to human body burden. EPA requires that the excess unused newly mixed "scrap" amalgam be handled as a toxic waste disposal hazard, just as it does the amalgam particles resulting from the removal of old mercury fillings.

There are four major routes by which dental mercury may come into contact with the environment:

**A. Dental Clinic Wastewater [6.5 tons per year]**

Dental offices have been shown by many studies to be significant contributors of mercury to the environment. The process of either placing or removing mercury fillings generates a slurry of mercury-rich amalgam waste that is vacuumed into the chair-side suction unit. Most of it passes right through the chair-side screens or traps, which capture only the larger particles. Facing no other obstacles, this slurry passes directly into the dental office wastewater and out to the environment. According to a recent study commissioned by the American Dental Association 6.5 tons of mercury are released to waste water treatment plants annually by 133,059 dentists in the United States.[118] This equates to approximately 230 milligrams of mercury per dentist per workday and represents more than 50% of the total mercury entering wastewater treatment facilities. A report released in 2002 by the National Association of Clean Water Agencies (NACWA) indicated that dental clinics are the single largest source of mercury in our nation's wastewater.[119]

Levels of mercury measured in dental office wastewater far exceed local limits for discharge by "small quantity generators" of hazardous waste. Because wastewater treatment facilities are not designed to process or handle heavy metals, most of the mercury settles out into the sludge, or "bio-solids" as wastewater is treated. These bio-solids are usually incinerated or used as fertilizer, the mercury content going directly into the environment.

Amalgam separators exist which capture 95% to 99% of the mercury from the wastewater before leaving the dental office. Studies in the United States, Canada and Europe have shown that when such devices are installed in dental offices community-wide there is a significant drop in mercury entering the wastewater treatment plants. Such studies have led to recommendations or regulations in several European countries that all dental offices install mercury-separating equipment.

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[118] Vandeven, J. and McGinnis, S.L., "*An Assessment of Mercury in the Form of Amalgam in Dental Wastewater in the United States*," Water, Air and Soil Pollution, 2005, 164, 349-366.

[119] Association of Metropolitan Sewerage Agencies (now the National Association of Clean Water Agencies.) "*Mercury Source Control & Pollution Prevention Program Evaluation: Final Report*." March 2002 (amended July 2002.)

The San Francisco RWQCB reported during the Watson Burton congressional hearings at USC that the separators alone caused a 90% reduction in sewer mercury in just 30 days. The remaining 10% of sewer mercury is the mercury I have previously discussed going through the bowel and until amalgam is banned and all old fillings are removed is unavoidable.

One of the IAOMT's first efforts was to prevent this spike in blood mercury and share this technology with the profession. Unfortunately few dentists take mercury exposure seriously and thus many people who seek to have mercury removed are actually exposed to a bolus dose of mercury for their trouble. We do not think this is reasonable or ethical and should and can be prevented. To facilitate patient understanding and professional accountability we've placed the safe removal procedure on the home page and listed it on the top of featured articles at www.iaomt.org. Even the ADA and the amalgam manufacturers agree: the removal of amalgam fillings can expose everyone present to a high amount of mercury vapor and particulate amalgam.

The IAOMT, recognizing the scientific validity of the statements above, and recognizing that the dental profession has the opportunity and obligation to eliminate or reduce this environmental hazard, urges all general dentists to install effective mercury separator equipment. We support efforts by municipal agencies to make this a regulatory requirement, since efforts toward voluntary compliance by dental associations have failed. We have for years urged all our IAOMT Academy members to take a leadership role and install such equipment without delay.

**B. Air Discharge [1 ton per year]**

Mercury vapor has also been measured in air vented from the central turbine vacuum systems to the outside of the dental office.[120] An estimated one ton of mercury vapor per year finds its way into the atmosphere through this route in the US. There is currently no known technology to prevent this form of pollution.

**C. Amalgam in Human Cadavers [3 tons per year]**

If mercury fillings are not removed from a patient's mouth and disposed of ecologically before death, the mercury contained in them will eventually find its way into the environment. It may contaminate the earth and ground water if the person is buried and it will contaminate the atmosphere if the body is cremated. A mercury flow worksheet developed for EPA estimated that in 2005 over 3 tons (6,613 pounds) of mercury were released into the environment in the U.S. from crematoria.[121]

**D. Human Waste [5.7 tons per year]**

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[120] Rubin, P.G. and Yu, Ming-Ho "*Mercury Vapor in Amalgam Waste Discharged from Dental Office Vacuum Units*," Arch Env Health, 51:4 335-337 (1996.)

[121] Cain, A., Mercury Flow Workbook, US EPA, Region 5, Excel Spreadsheet, January 2006.

Published studies have concluded that each and every amalgam bearer excretes an average of 150 micrograms of mercury per day in his/her urine and feces.[122] That amounts to 8.5 tons of mercury entering the environment annually in the U.S. Assuming two-thirds of this mercury is derived from dental fillings then 5.7 tons of dental mercury annually are flushed directly into our wastewater.[123] There is no known technology to prevent this form of pollution nor are there any regulations over this domestic waste. One hundred percent of this mercury ends up in the environment.

The above four routes of mercury entering the environment combine for at least sixteen tons of mercury annually from dental fillings. Less than half of that total-- the 6.5 tons from dental office wastewater-- can be captured by Best Management Practices and Amalgam Separators in dental offices- and then only if mandatory. Of the total mercury currently used in all products in the U.S., our EPA estimates that mercury fillings comprise 55% of that total repository-- or over 1,000 tons of mercury implanted in the teeth of Americans nationwide! Assuming a ten to fifteen year average durability of those mercury fillings in patients' mouths, this enormous reservoir of mercury will be continuously flushed into the environment for decades to come. Amalgam separators and Dental Amalgam Best Management Practices Sewer utilities may be required to reduce the levels of heavy metals in their bio-solids, the material that is removed from the sewer as solids. These solids are sent to farmlands as a soil nutrient enhancer, are deposited in landfills, or they are incinerated. If the bio-solids contain too much mercury the farmers will not accept them and the utility company may have to pay to dispose of them. If it is incinerated it liberates mercury in its most toxic form - elemental mercury vapor. Therefore, these utilities, in order to operate economically and meet their own discharge limits, are looking to place the responsibility on the largest contributors, the dentists. Organized dentistry has responded by establishing 'Best Management Practices' or (BMP's) for managing hazardous waste. Historically, these practices have had disappointing participation by dentists when purely voluntary.

It should also be noted that many dental clinics are now using a different type of vacuum pump system than was used in the past. The new type ("dry" or "turbine") uses no extra water and does not have a vacuum filter at the pump. Therefore, when clinics do not install an amalgam separator, yet switch to a turbine vacuum pump, there will actually be an increase in the amount of amalgam and mercury discharged to the sewer.

FDA should consider information published in the Fall edition of the ADA Professional Product Review (2007, 4:1). There it was reported that a "survey of [ADA Clinical Evaluator

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[122] Skare I. & Engqvist A., "*Amalgam Restorations - An Important Source of Human Exposure of Mercury and Silver*." Lakartidningen, 1299-1301 (1992); Skare I., Engqvist A.: NIOH, Arch. Env. Health, 1994 Sep-Oct; 49(5): 384-94.

[123] Aposhian, H.V. et al, "*Urinary Mercury After Administration of 2,3-dimercaptopropane-1-sulfonic acid: Correlation with Amalgam Score*," FASEB Journal, Vol. 6, pp2472-2476 (April 1992.)

Panel] members shows that relatively few panel members own an amalgam separator or plan to purchase one."[124]

To place a mixture containing 50% mercury-- the most neuro-toxic element known on earth-- within inches of a child's brainstem and assume it to be harmless is at best counterintuitive. To release this same pollutant into the environment is irresponsible when simple and available technology exists to reduce that release by over 95%. But that still leaves more than half of the dental derived mercury that is dumped into the water supply that remains beyond our ability to capture.

Until dentistry joins the rest of the 21st century health care profession and abandons its use of mercury there will be no effective environmental solution to the dental mercury crisis.

## V. *Barnes v. Kerr Corp.*

In 1999, Dr. David Barnes, a dentist licensed in the State of Tennessee, sued Kerr Corporation, the nation's largest manufacturer of mercury amalgam[125], alleging that he was severely injured by his occupational exposure to mercury and amalgam particles.[126] Dr. Barnes alleged that he suffered a constellation of neurological and neuro-behavioral injuries caused by his occupational exposure to mercury. Qualified expert witnesses concluded that Dr. Barnes's symptoms and injuries were caused by his occupational exposure to mercury. Kerr filed comprehensive pretrial *Daubert*[127] motions challenging the admissibility of the opinions proffered by Dr. Barnes's causation experts. However, these motions were denied.[128]

Barnes claims that he never received any information from Kerr stating that mixing dental amalgam presents a health risk. The jars in which the capsules were sent, however, bore prominent warning labels. These labels stated, in capital letters, that the product "CONTAIN[ED] METALLIC MERCURY" and featured an image of a skull and crossbones next to the word "POISON." On a chart of different hazards, including flammability, reactivity,

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[124] The ADA Clinical Evaluator Panel is a volunteer group of ADA members who contribute feedback for the clinical input segments of the ADA Professional Product Review program. More on the ADA Clinical Evaluator Panel can be found at http://www.ada.org/prof/resources/pubs/ppr/ace.asp.

[125] Kerr's website formerly claimed it controlled 46% of the U.S. dental amalgam market. Kerr has since removed this information from its website.

[126] *Barnes v. Kerr Corp*., Case No. 4:99-CV-79, United States District Court for the Eastern District of Tennessee (Winchester Division.)

[127] *Daubert v. Merrill Dow Pharmaceuticals, Inc*., 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993.)

[128] In the case of *McReynolds v. Mindrup*, Case No. CV97-1891, Circuit Court of Jackson County, Missouri, a dental patient alleged that she was injured by mercury following the removal and replacement of her amalgam fillings. Comprehensive *Daubert* challenges brought the defendant dentist against the opinions of the plaintiff's expert witnesses were also denied. On the basis of these repeated findings, it appears there is a sufficient body of scientific literature to adequately support medical testimony on the issue of causation.

and health hazards, the labels warned that metallic mercury presents a "serious" health hazard. The labels also recommend using protective gear such as glasses, gloves, and a facemask when handling the amalgams, and warned that the ingestion of mercury could cause "Neurotoxic/Nephrotoxic effects," that the inhalation of mercury could cause "Bronchiolitis, Pneumonitis, [and] Pulmonary Edema," and that even skin contact with mercury could have harmful effects, including "redness and irritation to [the] eyes and skin."

Barnes argued that these warnings were not adequate because he was taught and understood that the mercury was locked into amalgam matrix when mixed with the other amalgam constituents. Moreover, Barnes demonstrated that the 81% and 89.3% of his daily exposure to mercury came from removing mixed dental amalgam. Despite conceding that its MSDS's addressed only the dangers of mercury and not mixed dental amalgam, Kerr successfully defended Dr. Barnes's lawsuit by arguing that Dr. Barnes was adequately warned of the health risks associated with using mercury ***and*** mixed dental amalgam. The Sixth Circuit Court of Appeals affirmed the dismissal of Dr. Barnes's lawsuit. In its published opinion, the Court noted that "one has difficulty 'imagin[ing] what more Kerr could have done to advise Barnes about the potential dangers... that can result from exposure to elemental mercury.'"[129] Moreover, the Court stated that "[Barnes] cited no Tennessee authority holding that a warning about a dangerous ingredient in a product must affirmatively state that the particular ingredient remains dangerous when it is combined or is being combined with the other ingredients."

The legal significance of the *Barnes* decision is that a manufacturer may escape liability for injuries caused by the mercury in dental amalgam by warning about the myriad of adverse effects associated with the use of the product. Meanwhile, the unwitting patient is actively prevented from receiving this information by proclamations of amalgam safety communicated by the American Dental Association, deceptive pamphlets by the same mercury advocates, the various state dental boards, the FDA, and the patient's own dentist.[130] The FDA cannot reasonably offer assurances of amalgam safety while manufacturers of dental amalgam are flatly contradicting these assurances in their warnings materials.

The prevailing standard of care in the practice of dentistry requires a dentist to advise patients of a manufacturers' warnings. As learned intermediaries, dentists are expected-- indeed required-- to pass along these warnings. *See e.g., McNeil v. Wyeth American Home Products Corp*., Slip Copy, 2005 WL 544222 (N.D. Tex. 2005) ("[I]t is reasonable for the manufacturer to rely on a physician to pass on its warnings to the ultimate consumer....") *See also, McKee v. Moore*, 648 P.2d 21 (Okla. 1982). ("The manufacturer's duty to warn the ultimate consumer of prescription drugs, or devices, as distinguished from those sold directly to the consumer, is limited to advising the prescribing or treating physician of the drug's or device's potential dangers in the absence of contrary FDA regulations. Once the physician is warned, the choice of treatment and the duty to explain the risk is incumbent on the physician."); *Koury v. Follo*, 272 N.C. 366, 158 S.E.2d 548 (1968.) ("[A manufacturer's label] is evidence of a warning which the

[129] *Barnes v. Kerr Corp*., 418 F.3d 583 (6th Cir. 2004.)

[130] The Kerr MSDS's and warnings do not distinguish between occupational exposure and exposure resulting from the implantation of dental amalgam in the teeth of a dental patient.

physician disregards at his peril, and his disregard of it is relevant upon the issue of his use of reasonable care....")

Despite the apparent legal obligation to pass along manufacturer warnings to he patient, the IAOMT is fully aware that this is ***not*** the prevailing practice around the country. Since its inception, the ADA has consistently proclaimed amalgam's safety. Furthermore, it has consistently quelled any dissension concerning the safety of amalgam.[131] As demonstrated in the next section of this paper, candid discussion between dentists and their patients concerning the warnings provided by manufacturers are discouraged, if not prohibited, by state dental boards around the country.

## VI. The Declaratory Rulings of the North Carolina State Board of Dental Examiners

In the February 2005 edition of its publication "*The Dental Forum*," the North Carolina Dental Board ("NCDB") published the following admonishments to North Carolina dentists, which forbade certain advertising practices. Specifically, the North Carolina Board recommended that dentists avoid advertising:

> [T]hat you practice 'Mercury-free Dentistry,' 'Metal-free dentistry,' or that you should 'Eliminate your exposure to Mercury,' or that 'Silver fillings contain mercury that may leak into your body and cause health problems,' or make any reference that mercury fillings are harmful.[132]

The position taken by the NCDB is typical of the position taken by dental boards around the country. In response to the admonishments appearing in *The Dental Forum*, one of our member dentists requested a declaratory ruling of the NCDB [Exhibit 8], and requested permission to

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[131] The ADA's efforts to quell dissension on this issue led one legal commentator to recently conclude that the "health risks and legal issues surrounding mercury amalgams paint, at worst, a bullying image, and at best, a less than flattering portrayal of the ADA, an organization that is supposed to represent an entire profession, promote the safety of overall dental health, and protect-- and respect-- the patients it serves." Chirba-Martin, "*An Uncertain Risk and an Uncertain Future: Assessing the Legal Implications of Mercury Amalgam Fillings*," Health Matrix Journal of Law Medicine, Vol. 14:293-324 (2004.)

[132] The District of Columbia Dental Board has similar rules:
4213.44 A dentist shall not remove amalgam restorations containing mercury from patients who are not allergic to mercury for the alleged purpose of removing toxic substances from the body, when such treatment is performed solely at the recommendation or suggestion of the dentist.
4213.45 A dentist shall not remove sound or serviceable amalgam restorations containing mercury, at the request of a patient who is not allergic to mercury, without first obtaining appropriate informed consent from the patient, which includes but is not limited to advising the patient that:
(a) The National Institutes of Health has determined that there are no verifiable systemic health benefits resulting from the removal of mercury amalgam restorations; and
(b) The removal of sound or serviceable mercury amalgam restorations may significantly affect the integrity of the tooth.
4213.46 A dentist shall not represent that dental treatment or diagnostic techniques recommended or performed by the dentist have the capacity to diagnose, cure or alleviate diseases, infections or other conditions, when such representations are not based upon accepted scientific knowledge or research.

advise his patients of the warnings reflected in Kerr Corp.'s MSDS's relating to dental amalgam.[133] The NCDB responded that, in disclosing these warnings to patients, the petitioner was improperly portraying "the alleged dangers of dental amalgam as established fact." The NCDB further advised that "[l]eading health organizations such as the Federal Drug (sic) Administration (FDA), World Health Organization (WHO), and American Dental Association (ADA) have rejected similar assertions as unsupported by peer-reviewed, controlled research." In order to avoid misleading patients and potential patients, the NCDB ruled that the petitioner was required to disclose to his patients that "the FDA, WHO and ADA have approved dental amalgam for use in non-allergic individuals." According to the NCDB, the failure of the dentist to make communicate this mandatory assurance of safety would violate the NCDB's published rules. By discouraging the unequivocal and uncontradicted disclosure of manufacturer warnings, and by requiring mandatory assurances of safety that directly contradict these manufacturer warnings, it is clear that dental patients are not clearly advised by the dental profession of the manufacturer's position on the identified health risks associated with the use of this product.

## VII. Questions Posed by the FDA

Preliminarily, we note that FDA question Nos. 1, 2, and 3 have no bearing on FDA's primary function, which is to determine whether a regulated product is safe and effective. Question Nos. 1, 2, and 3 appear to be designed to build a cost / benefit justification for the continued use of the product in spite of its demonstrable health risks. Any meaningful discussion of a justification for the continued use of the product should begin only after the known health risks are identified and acknowledged. In our view, FDA has been historically irresponsible in recognizing and acknowledging the documented health risks associated with the use of this product. Most recently, FDA has failed to effectively respond to the position of the 2006 Joint Panels that the FDA draft white paper on mercury amalgam was "not reasonable" in its conclusions.

To the extent that the continued use of this product may be justified based solely on modest cost considerations, (a position vehemently disputed by this Academy), we trust FDA will agree that effective patient informed consent would be imperative. At a minimum, this would require a dentist to inform a patient of the known health risks associated with exposure to mercury. As an organization whose membership is comprised primarily of dentists, we are aware that effective informed consent is not traditionally obtained from dental patients in connection with amalgam use. As discussed above, state dental boards have routinely prosecuted, or threatened to prosecute, dentists for making statements to their patients about the known potential health risks associated with amalgam placement, use, and removal.

Given the gravity of potential harm posed by continued amalgam use, we do not believe that any modest savings associated with the use of this material is a proper consideration for FDA in determining how amalgam should be classified. Without prejudice to the foregoing position, we will respond directly to the specific questions posed by FDA in connection with the reopening of the Public Comment Period.

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[133] The pertinent requests were request numbers 16, 18, 20, 22, and 24.

**1(a). How many annual procedures use mercury amalgams?**

Dentistry is second only to chloralkali production in the use of mercury and is one of the last remaining sources of unregulated mercury pollution to sewers and the environment. Annually approximately 40,000 pounds of mercury is implanted in American's teeth. Note that figures vary depending upon source.

If an amalgam lasts on average 10-15 years that means that 400,000 pounds of mercury resides in American's teeth. Craig's Textbook on Dental Materials estimated each amalgam filling to contain 750 mg and an average number of filling was 12 per person. Approximately 25% of Americans choose cremation.[134] In 2000 approximately 2,403,351 people died and 600,000 were cremated thus releasing on average 5,400,000,000 µgs of mercury into the environment.[135]

Roughly 100 million fillings are placed each year in the United States. Recent figures show that amalgam use has declined dramatically; it is currently used for about one-third of fillings. That implies that the dental profession is comfortable with the other filling materials available, especially the composites. Two surveys are illustrative. The independent and highly regarded Clinicians Report (formerly known as Clinical Research Associates) reports that in 2005, 32% of American dentists were not using amalgam at all, and more recently, the Wealthy Dentist newsletter reported a survey that found 52% of American dentists were not using it.

**1(b). What are the trends?**

The trends are clear. American consumers are increasingly aware of the mercury controversy surrounding amalgam, and they favor esthetic tooth-colored restorations. The dental profession is moving away from amalgam, and has largely embraced the newer materials. We are ready to leave amalgam on the shelf with other dangerous relics.

This is a remarkable development considering that a recent Zogby poll found that dentists have been very effective in deceiving the public as to the true nature of amalgam. Less than twenty-five percent of the general public even knew that there was mercury in an amalgam filling much less that it is the principle ingredient.

In addition advocates for continued use of mercury/silver implants use deceptive terminology calling the filling an alloy. Mercury/silver fillings are not alloys of mercury as mercury does not form covalent bonds with the other metals.

Quoting Dun,

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134 http://query.nytimes.com/gst/fullpage.html?res=9E03E7DD133CF931A25753C1A961958260&sec=&spon=&pagewanted=all

135 www.csdp.org/research/1238.pdf

It is a fallacy that mercury is neutralized when it is combined with other components of silver amalgam. The laws of physical chemistry are followed. Mercury is diluted by the other components of amalgam in what may be considered a solid solution. Although the vapor pressure of mercury is reduced, mercury is still given off. An identical situation arises when alcohol is diluted by water.

Heat drives mercury from a set amalgam filling causing it to emit deadly elemental mercury vapor. Elemental mercury vapor is one of the most toxic forms of mercury due to its high absorption rate from the lung and ability to cross the blood brain barrier.

The Smoking Teeth Video [www.iaomt.org and Exhibit 9] clearly shows that exposure to elemental mercury vapor is a known consequence of using mercury/silver filling implants both in patients and in the dental office.

**2. What are the differences in cost between amalgams and alternative materials (e.g., composite, other metals, ceramics, etc.)?**

As in the case of many other sources of pollution, the market price of an amalgam filling does not take into account the realities of its actual cost— the costs of environmental damage, the costs of safe disposal, and the system-wide costs of adverse health effects from indiscriminate mercury exposure. [*See,* Hansen, Exhibit 10.]

Proponents of dental amalgam incorrectly claim that mercury filling implants are safe and cost effective. Using national blood mercury prevalence data from the Centers for Disease Control and Prevention, it was determined that between 316,588 and 637,233 children each year have cord blood mercury levels > 5.8 µg/L, a level associated with loss of IQ.[136] That means that 8 to 16% of the newborns had levels of mercury in the umbilical cord blood sufficient to cause neurological impairment. Although we believe the authors incorrectly identified the source of the mercury to potentially be from fish, they estimated the lost productivity to be $8.7 billion annually (range, $2.2–43.8 billion). That makes the neurological harm from mercury alone greater than the annual cost of all amalgam fillings.

In a paper prepared as testimony before Congress, The Mercury Policy Project presents a cogent argument to the effect that each amalgam filling imposes an environmental cost ranging from $20 to $30. (www.mercurypolicy.org)

[136] Leonardo Trasande, Philip J. Landrigan, and Clyde Schechter, "*Public Health and Economic Consequences of Methyl Mercury Toxicity to the Developing Brain*," Vol. 113 #5 May 2005 Environmental Health Perspectives.

The actual material cost of an amalgam filling is $1-2.00, while a composite costs $2-4.00. The typical composite filling commands a fee that is approximately $30 higher than the typical amalgam, for a complex set of reasons. There is a perception among dentists that composites take longer to place, although many IAOMT members, experienced in composite placement, assert that a composite may be placed as quickly as an amalgam. There are also perceptions of added value with the more modern material, which may skew an examination of fee distribution. For example, the switch to mercury-free, composite-based practice has been led by independent-minded, fee-for-service dentists who charge more for all services, including fillings. That would tend to drive up the average fee for a composite. Many of the lower fee, discount practices have stayed with amalgam, thus driving down the average fee for amalgam.

The market price of an amalgam filling also does not take into account the widely acknowledged long-term tendency for amalgam-filled teeth to break at a higher rate than composite-filled teeth. This inevitable reality requires the eventual use of more expensive restorations, such as crowns.

In 1993 when Dr. Harold Löe was the head of the National Institute of Dental Research he stated for the record that;

> The first filling is a critical step in the life of a tooth. Using amalgam for the first filling requires removing a lot of the tooth substance, not only diseased tooth substance but healthy tooth substance as well. So in making the undercut you sacrifice a lot, and this results in a weakened tooth. The next thing you know the tooth breaks off, and you need a crown. Then you need to repair the crown . . . and so it continues to the stage where there is no more to repair and you pull the tooth.
> * * *
> With the first filling you should do something that can either restore the tooth or retain more health tooth substance. Use new materials-composites or materials you can bond to the surface without undercuts. You can do this with little removal of the tooth substance so that the core of the tooth is still there.

Placement of an amalgam filling weakens the strength of a tooth by 75%. The resulting mutilated tooth is no stronger with or without a mercury/silver implant. This weakness is due, as Dr. Löe pointed out, to the excessive drilling required to implant a very thick mixture of mercury and silver in a tooth.

Restoration of a cavity with a bonded composite filling actually increases the tooth's strength slightly. Furthermore, less of the natural the tooth is removed and so the vitality and structural integrity of the tooth is better preserved.

Placement of a mercury/silver filling in a tooth saturates the tooth and roots and jawbone with mercury. (*See,* Sheep Scan) The teeth have been removed at this point so all the dark jawbone is mercury that transferred from the fillings into bone in 30 days. This saturation is linked to tooth death and bone loss.

Mercury/silver fillings expand over time due to chewing compression (creep) and corrosion. This exerts lateral forces on the weakened tooth and results in tooth fracture. Fractures can open the pulp chamber and result in pulpal death. In addition, a silent fracture may allow recurrent decay to enter the dentin layer of the tooth and infect the pulp. A broken tooth at a minimum will require full coverage crown or onlay to restore the tooth. The drilling necessary to prepare a tooth for a full crown creates inflammation within the pulp chamber. This over time results in a substantial percentage of pulpal death.

According to a study by Whitsett and Schwartz the average life span of a single crown is approximately 7 years.[137] The lifespan of an endodontically treated tooth is significantly reduced. Repeated restoration of mercury/silver implanted teeth is the financial mainstay of the restorative dental practice.

Gordon Christensen states that;

> "Full-crown restorations are commonly placed today, and the revenue produced from this procedure makes up a significant portion of a typical general dentist's income, as I have observed in the past."[138] [139] [140]

Furthermore, there is no question that implanting mercury in teeth saturates jawbone and results in bone loss, produces inflammation and periodontal breakdown.[141] [142] [143] [144] [145] [146]

Thus, as early as 1973, a case can be made that the presence of dental mercury-amalgam

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[137] Schwartz NL, Whitsett LD, Berry TG, Stewart JL., "*Unserviceable crowns and fixed partial dentures: life-span and causes for loss of serviceability*," J Am Dent Assoc. 1970 Dec: 81(6):1395-401.

[138] Christensen GJ. "*Ceramic vs. porcelain fused to metal crowns: give your patients a choice*," JADA 1994;125(3):311–4.

[139] Christensen GJ., "*The confusing array of tooth-colored crowns*," JADA 2003;134(9): 1253–5.

[140] Christensen GJ., "*Why all-ceramic crowns?*," JADA 1997;128(10):1453–5.

[141] 1957, Zander (JADA, 55:11-15) reported "materials used in restorative dentistry may be a contributing factor in gingival disease."

[142] 1961, App (J Prosth Dent 11:522-532) suggested that there was greater chronic inflammation around amalgam sites than non-amalgam areas.

[143] 1964, Trott and Sherkat (J CDA, 30:766-770) showed that the presence of amalgam correlates with gingival disease. Such disease was not present at contralateral amalgam-free sites.

[144] 1969, Sanches Sotres et al (J. Periodo. l40: 543-546) confirmed Trott and Sherkat findings.

[145] 1972, Turgeon et al. (J CDA 37:255-256) reported the presence of very significant erythema around amalgam restorations that was not present at control non-amalgam sites.

[146] 1973, Trivedi and Talim (J. Prosth. Dentistry, 29:73-81) demonstrated that 62.5% of amalgam sites have inflammatory periodontal tissue reaction.

results in chronic inflammation and bleeding in the gingival tissue adjacent to it; in other words, *in situ* amalgam produced chronic gingivitis.[147]

In 1984, the year of the NIDR/ADA Workshop, Fisher et al.,[148] reported that at amalgam sites alveolar bone loss was very pronounced and statistically significant as compared to control non-amalgam sites. In other words, *in situ* amalgam produces chronic periodontitis. Periodontal disease is the principle reason for two-thirds of adult tooth loss in the U.S. and mercury from mercury/silver implants contributes substantially to this common disease.

Using a toxic material as a primary restorative that weakens teeth, contaminates pulp and gingiva with mercury, inflames soft tissue and deteriorates bone resulting in bone loss and gum disease and causes tooth fractures material drives up the cost of keeping natural teeth for a lifetime and makes mercury/silver implants the most expensive material ever invented to treat dental caries.

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Defenders of amalgam argue that eliminating amalgam for low-income clinics would drive up costs and reduce services for the poor. Notwithstanding these arguments, the NAACP, in their Health Policies and Resolutions document of 2004, called for "a ban on mercury-containing dental fillings being placed in young children, pregnant women, or nursing mothers," and "all Americans, including families on Medicaid and/or dental insurance plans be given a choice of dental fillings, including the right to choose alternatives to mercury fillings."

Given the gravity of amalgam's potential harm to the public health, the environment, and those employed in the field of dentistry, the IAOMT does not support any consideration of modest short-term cost savings associated with the use of amalgam. The health of the U.S. population should be the only concern.

**Are there differences in replacement lives?**

The experience of IAOMT members shows that there is no difference in the replacement life for composite fillings vs. amalgams. Many of our members were the early converters to mercury-free practice — ten, twenty years ago, or even longer. Their experience puts them in a position to make well-informed statements on this matter, and the consensus is that composites are as durable and long lasting as amalgam fillings.

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[147] 1976, Goldschmidt et al (J. Perio. Res., 11:108-115) demonstrated that amalgam corrosion products were cytotoxic to gingival cells at concentrations of 10-6; that is, micrograms/gram of tissue.

[148] J Oral Rehab, 11:399-405.

Statements made by dentists on this subject vary widely, because there are doubts about the durability of composites that derive from the state of materials science in the 1980's that persist today. However, ten years ago, Dr. Gordon Christensen, DDS, PhD, the author of the aforementioned Clinician's Report, noted that composites were fully acceptable for all fillings. Writing in the Journal of the American Dental Association, (December 1998, page 1757-9) he said that "it is time to accept [composite] resin restorations, … and to bring this concept into the mainstream of US dentistry."

Bonded composite restorations have the additional advantage of being repairable. As a result, they may not require total replacement. Furthermore they do not leak heavy metals into tissues and stimulate periodontal breakdown.

**3. What are the reimbursement rates for dental amalgam and the alternative materials?**

In general, insurance reimbursement for fillings of any type is made at the rate that applies to amalgams, with some notable exceptions. A small number of self-funded insurance programs, led by the Fortune-100 Parker Hannifin Corporation, have taken it upon themselves to eliminate reimbursement for mercury amalgam fillings, and to pay only for composite and other non-mercury materials. They are doing this in the interest of their employees' health, and with the conviction that other health costs will go down as mercury exposure is reduced.

**4. How would labeling describing the risks of amalgam for certain subpopulations (e.g., children under age 6, pregnant and lactating women, hypersensitive or immunocompromised individuals) affect the demand for, and use of, mercury amalgam?**

It is the position of this Academy that dental amalgam should be banned as a dental restorative material. Labeling cannot adequately protect the public from this toxic material. Notwithstanding this position, patients have a right to know what is being used in their treatment. Dentists have abridged that right for over a century by refusing to reveal that restoring teeth exposes patients to mercury. The FDA should require dentists to comply with the consumer protection laws and disclose to every recipient of these implants what exactly what substances are being implanted through the use of mercury/silver copper zinc and tin fillings.

Furthermore, dentists should be required to disclose that according to both the Environmental Protection Agency (EPA) and the World Health Organization (WHO) there is no safe level of exposure to mercury and that mercury from implanted fillings has been proven to be the predominant source of human and fetal exposure to elemental mercury vapor. See Table #2 . They did not include the fecal measurements by Skare that found 150 µg/day from stool, the most likely source of which was abrasion of mercury from the surface of fillings due to chewing.

The MSDS published by Kerr (and other amalgam manufacturers) and the risk assessments for mercury do not limit susceptibility to mercury intoxication to certain subpopulations. The IAOMT believes that the multiple avenues of risk posed by amalgam place all amalgam-bearing persons at risk. Moreover, the suggestion that only certain subpopulations

are at risk implicitly and falsely suggests that persons who are not members of those subpopulations are free from risk. Again, labeling is not an adequate means of protecting the public from the health risks posed by this dental material.

Certainly, there are groups at higher risk of mercury intoxication, including the grave risk of neurodevelopmental damage to a fetus and young child. However, other groups are obviously at immediate risk, including women who might become pregnant, people who are hypersensitive, those with apolipoprotein E4, people with CPOX4, and people with kidney disease. Subtle renal effects appear to occur in all males, as identified in the NIH Children's Amalgam Trials and our Scientific Advisory Committee submission.

Dentists do not engage in genetic screening prior to placing amalgam fillings and therefore cannot identify people with genetic polymorphisms known to make them more susceptible to the effects of mercury over the course of years. Person with apolipoprotein E4, present in over 25% of the population, or CPOX4, present in about 15% of the population, make them more vulnerable to mercury's toxic effects. How many more unknown genes can there be that code for increased susceptibility to mercury? Moreover, this list only scratches the surface of the enormous number of mechanisms of mercury toxicity, each of which can have individual variants that constitute subpopulations with increased susceptibility. [*see*, Summary of Low Level Mercury Toxicology, attached as Exhibit 12.]

If risk assessment performed by government agencies is to be given any credit, everyone with amalgam fillings is already overexposed to mercury. A chart (mercury exposure table, attached) showing seventeen estimates of mercury exposure in adults with typical amounts of amalgam fillings, drawn from the scientific literature, is cross-referenced with some of the exposure limits suggested by government agencies. [Exhibit 12.] Most of the exposure estimates exceed these standards.

Each of the seventeen referenced estimates of mercury exposure from amalgam may not fully estimate the true exposure borne by amalgam bearers.[149] Professor Haley measured mercury released from set dental amalgam under no stress and in distilled water.

> The International Academy of Science (IAOMT) supported a study of mercury release from different dental amalgams. Nine IAOMT dentists were sent identical Plexiglas molds with 10 holes that would hold 1 spill of amalgam. Each of the dentists was also sent amalgam material from different manufacturers. They placed 10 amalgams in the molds and sent them to Dr. Haley's lab at the

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[149] If Mackert's estimate of exposure were accurate, you should not be able to see any effect from one or two mercury/silver implants being placed. However, the Children's Mercury Exposure studies conducted by DeRouen shows a dramatic rise in urinary mercury to above 3 µg at year two from on average 3 fillings. Urine is not the primary excretion route for mercury; therefore, exposure was much greater. Mackert's low estimate is not supported by the empirical evidence from numerous studies. In the view of this Academy, his low mathematical estimate should not be considered in evaluating the exposure to mercury from mercury/silver fillings.

Of even greater concern is the dramatic drop in urinary mercury after two years so that the by the end of 7 years there was no statistical difference between those with a multitude of mercury/silver filling implants and those with no time-release mercury implants.

> University of Kentucky for testing of mercury release using a Nippon Direct Mercury Analyzer. The amalgams were first allowed to age until the mercury emissions leveled off and then the amount of mercury being released was measured for 25 continuous days. Some of the results are shown in Table 1.

This data shows that the level of mercury released by 1 cm2 of dental amalgam from a single spill filling (each filling was slightly less than 1 cm2) was between 4 to 22 micrograms per day. This result was obtained by leaving the amalgams setting in distilled water at room temperature, gently mixing the water without disturbing the amalgam and collecting 1 ml for analysis. We did some other experiments. For example, holding an amalgam under water and brushing it 15 strokes with a conventional medium bristle tooth brush lead to a 5 to 10 fold increase in the release of mercury. Professor Haley's measurements are in stark contrast to the often-quoted "low estimates" of amalgam advocates.

Those individuals who are lucky enough to have a constitution that would allow them to escape all the effects of amalgam mercury for a lifetime are, in fact, the subpopulation. We just don't have a practical method of identifying them. We can only conclude that amalgam must be banned, or at least placed in Class III.

The legal requirement for accurate informed consent has not been changed although it is widely ignored with regard to mercury/silver implants. Enforcement of the law would improve compliance with this long held tradition in medicine. In addition guidance from the FDA on what can be said legitimately would improve communication and compliance.

**5. How would the risks included in the labeling be communicated to those subpopulations?**

Dentists are fond of quoting the ADA's declarations of safety as gospel in lieu of the scientific literature, which emphatically does not support these claims. Historically, patients have not been advised of the warnings communicated to dentists by the dental amalgam manufacturers—despite a clear legal obligation to do so. Given the historical resistance of the ADA to the documented health risks posed by this material, there is little reason to believe that mandatory FDA labeling will now be communicated to these patients. Only a ban of the material will protect the public health.

One particularly disturbing claim often attributed to the dental division of the FDA is that there is absolutely NO scientific evidence linking mercury from amalgam to any medical disorder. This is in stark contrast to the before mentioned research linking mercury from mercury/silver filling to periodontal disease, inflammation and bone loss. In addition, research has linked mercury to idiopathic dilated cardiomyopathy (IDCM). Victims of this disorder usually suffer cardiac arrest usually at an early age and die. Their hearts have 22,000 times more mercury that comparable hearts that suffered secondary cardiac dysfunction.

The injection of this quantity of mercury into a heart would destroy the ability of that heart muscle to beat normally. We should begin to refer to mercury as causal with respect to IDCM disease.

In 1981, Snapp carefully removed mercury/silver implants and his experimental subjects experienced a dramatic 90% decline in blood mercury. The only logical conclusion is that the subjects' mercury/silver implants contributed substantially to their blood mercury. In the sheep and monkey experiments conducted by Vimy and Lorscheider, the experimental subjects never experienced high blood mercury, yet over the limited time of the experiment they found that mercury has accumulated in kidney, liver, heat and brain tissues. Thus, low blood levels over time result in substantial increases in tissue mercury. In addition, Vimy found that kidney function declined in sheep after placement of mercury/silver implants. The major difference between the sheep and the children's mercury exposure studies by Bellinger and DeRouen is that the sheep's kidneys revealed adverse effects within a few months while the children took a few years to experience these effects.[150] [151]

**6. What is the current exposure to mercury for patients? For professionals? What would be the reduction in exposure associated with the use of alternative materials?**

The predominant source of human exposure to mercury is from *in situ* mercury / silver implants (despite Mackert's and the ADA's unfounded claims to the contrary.[152] [Exhibit 13.]

Exposure to mercury for patients is addressed at page 35, *supra*. Dentists and their staff are exposed to mercury at a greater rate than their patients. This is discussed in Section II(C) of this Comment. While the catastrophic exposures from past practices such as hand-squeezing of fresh amalgam, where drops of liquid mercury would run over their hands, is gone, dangerous levels of mercury and amalgam particulate are generated in the dental workplace. Eighty-five percent of dentists have aberrant porphyrin metabolism, characteristic of low level mercury poisoning.[153]

Exposure to mercury for patients and dental staff does not exist where alternative materials are used for new fillings. However, there is a high risk of exposure when old fillings are drilled out, and the big challenge for the future will be to train dentists to be careful as over

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[150] The Office of Human Research Protections (OHRP) has ruled, in response to our [CDC's] petition, that University of Washington (UW) dental school professors Timothy DeRouen and Michael Martin refused to provide informed consent in their notorious mercury experiment at Casa Pia, a Portuguese orphanage. To quote from the OHRP letter to Consumers for Dental Choice: "OHRP found that the informed consent document for the research conducted by UW failed to adequately describe the reasonable foreseeable risks of the amalgams … ." This finding by OHRP – the federal agency policing experiments on children – was accepted by the University of Washington.

[151] The fact that in the Casa Pia study the investigators did not disclose to the guardians of the orphans that they were implanting time-release mercury/silver fillings violates the laws of this country and is unethical.

[152] WHO Environmental Health Criteria 118 (1991), section 5.1. General population exposure, Table 2, <http://www.inchem.org/documents/ehc/ehc/ehc118.htm>.

[153] Echeverria D, Woods JS, Heyer NJ, Rohlman DS, Farin FM, Bittner AC Jr, Li T, Garabedian C., "*Chronic low-level mercury exposure, BDNF polymorphism, and associations with cognitive and motor function.*" Echeverria D, Woods JS, Heyer NJ, Rohlman DS, Farin FM, Bittner AC Jr, Li T, Garabedian C (2005.)

the years they remove the thousands of tons of mercury stored in the amalgam fillings of the American population. We should not add to this burden by continuing to place additional fillings in the mouths of U.S. citizens.

## CONCLUSION

In his 1998 JADA paper, Dr. Gordon Christensen noted that there are dental schools in other countries that don't teach the amalgam technique, and that dentists trained in those schools have trouble using amalgam. The point is that amalgam is not necessary for good dental practice; it is just a choice among other choices, a habit, a tradition. It is possible for the FDA to choose to relegate dental amalgam to the same history museum as all the other mercurial devices and substances of the past, none of which are still on the market.

The International Academy of Oral Medicine and Toxicology has steadfastly studied this issue for the last twenty-three years. They have continuously invited lecturers from multiple scientific disciplines to lecture at its meetings. Based on this experience and expertise, the Academy strongly recommends the following:

1. That the FDA formally ban the use of dental amalgam as a dental restorative material.

2. Failing that, that the FDA place encapsulated dental amalgam into Class III, and seek strict proof of safety and effectiveness.

3. If the FDA decides to place encapsulated amalgam into Class III, that it place restrictions, not special controls, on its use in young children, women of childbearing age, males, patients with compromised kidney, immune, and neurological function, those who are hypersensitive to mercury, those who tests for apolipoprotein E4, and others within susceptible subpopulations as described herein. Neither "Class II controls" nor "Special Controls" can accomplish reasonable assurance of safety for all sectors of our general population. Reasonable assurance of safety can only be achieved by abolishing the use of dental amalgam or by placing it into Class III.

Sincerely yours,

Pierre Larose, DDS, President
Stephen M. Koral, DMD, Executive Vice President
Jack Kall, DDS, Chairman of the Board
Kimberly Smith, Executive Director
James M. Love, General Counsel

INTERNATIONAL ACADEMY OF
ORAL MEDICINE AND TOXICOLOGY

PUBLIC COMMENT

EXHIBIT 1

In The
# United States Court of Appeals
## For The Sixth Circuit

# DAVID E. BARNES, DR.,

*Plaintiff - Appellant /Cross-Appellee,*

v.

# THE KERR CORPORATION,

*Defendant - Appellee/Cross-Appellant.*

ON APPEAL FROM THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TENNESSEE
AT WINCHESTER

## JOINT APPENDIX
**Volume VII of VIII**


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**IN THE UNITED STATES DISTRICT COURT FOR THE EASTERN DISTRICT OF TENNESSEE, WINCHESTER DIVISION**

| | | |
|---|---|---|
| **DR. DAVID BARNES,** | ) | |
| | ) | |
| **Plaintiff,** | ) | |
| | ) | |
| **vs.** | ) | **Case No. 4:99-CV-79** |
| | ) | |
| **THE KERR CORPORATION,** | ) | |
| | ) | |
| **Defendant.** | ) | |

**AFFIDAVIT OF G. MARK RICHARDSON, Ph.D.**
**(VOLUME 1)**

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DATED: February 20, 2002

IN THE UNITED STATES DISTRICT COURT FOR THE
EASTERN DISTRICT OF TENNESSEE, WINCHESTER DIVISION

| | | |
|---|---|---|
| DR. DAVID BARNES, | ) | |
| | ) | |
| Plaintiff, | ) | |
| | ) | |
| vs. | ) | Case No. 4:99-CV-79 |
| | ) | |
| THE KERR CORPORATION, | ) | |
| | ) | |
| Defendant. | ) | |

## AFFIDAVIT OF G. MARK RICHARDSON, Ph.D.

I, G. Mark Richardson, Ph.D., being first duly sworn, declare and state:

1. I am a senior risk assessment specialist employed by Risklogic Scientific Services, Inc., a company specializing in environmental and human health risk assessments. The matters referred to in this declaration are based upon my personal knowledge, except where otherwise indicated, and if called as a witness I could and would testify competently thereto. True and correct copies of all exhibits identified herein are attached hereto and are listed in a summary set forth at the end of this affidavit.

2. I have conducted extensive research in the fields of human chemical exposure, (aquatic) toxicology and the environmental fate of mercury. I have also conducted extensive research into the environmental fate, metabolism and/or effects of environmental contaminants in aquatic biota and humans. I have authored over twenty articles in peer reviewed journals. I have also authored several government studies designed to assess risks associated with a number of contaminants.

3. I am the author of a government study entitled "Assessment of Mercury Exposure and Risks from Dental Amalgam." The study was prepared on behalf of the Bureau of Medical Devices, Health Protection Branch, Health Canada. That study is widely available and distributed, including an abridged version published in the peer reviewed scientific literature (Richardson and Allan, 1996), and the unabridged report available at two separate Government of Canada Internet sites: by Health Canada (http://www.hc-sc.gc.ca/hpb-dgps/therapeut/zfiles/english/publicat/richards-scan_e.pdf) and by the Canadian Government's official publisher, Public Works and Government Services Canada (http:// dsp-psd. pwgsc.gc.ca/ Collection/H46-1-36-1995E.pdf).

4. I have also authored two peer-reviewed published articles that pertain to exposure to mercury derived from dental amalgam (Richardson and Allen, 1996; Richardson et al., 1995) and have contributed to the risk assessment and evaluation of dental amalgam undertaken by the government of Sweden. My professional training and experience are set forth in greater detail in my curriculum vitae, a true and correct copy of which is attached hereto as Exhibit 1.

5. I am frequently invited to speak at professional meetings, conferences and symposia that cover current issues in risk assessment. I have been invited to Kuwait, Australia, the U.K. and the United States, as well as within Canada, to present and discuss issues of risk assessment. With respect to the exposure and risks posed by dental amalgam I have been sponsored to conferences and meetings in the U.K., France, Norway, Sweden, Germany, Australia, United States and within Canada. I have teach methods of risk

assessment to state regulatory agencies. I have teach a graduate level university course on risk assessment for the University of Ottawa, Ottawa, Canada. I also regularly confer with risk assessment specialists, both nationally and internationally, concerning the state of scientific knowledge in the field. I was an invited editor of a special issue of the International Journal Human and Ecological Risk Assessment dedicated to probabilistic methods in quantitative risk assessment. I am routinely called upon as a peer reviewer for manuscripts dealing with risk assessment, including exposures to and risks from mercury, which have been submitted to various journals including Human and Ecological Risk Assessment, the Journal of Soil Contamination, and others.

6. I am routinely called upon by Canadian regulatory agencies to conduct risk assessments of unique situations, contaminants and/or population groups, and to peer review and to comment upon risk assessments conducted by other experts in the field. I am currently supporting Canada's Department of National Defense (DND) by undertaking exposure and risk assessments in support of their battle theater environmental health and safety program. I provided sworn testimony before DND's formal Board of Enquiry on Croatia, having assessed the exposure and risks posed by a variety of contaminants encountered by Canadian troops while in that country on peace-keeping duties. I participate on behalf of Canada on International Task Force 40 (ITF-40), a tripartite (USA, Canada, UK) military working group tasked with evaluating the potential for industrial chemicals to function as weapons of mass destruction, either intentionally or as a result of inadvertent collateral damage to industrial installations.

7. I have reviewed the affidavits of Drs. Werley, Cohen, Clarkson, and Mackert submitted by Kerr in support of its request for summary judgment. The information presented in those affidavits, with respect to my assessment of mercury exposure in Dr. David Barnes, is incomplete, incorrect or misrepresented in order to suggest that the methods used by me, and the resulting exposures estimated for Dr. Barnes, are inaccurate or otherwise inadmissible as evidence in this case. The purpose of this affidavit is to set the record straight as to the general acceptability of the methods used, the appropriateness of assumptions and parameters employed with those methods to arrive at exposure estimates for Dr. Barnes, and to identify aspects of Kerr's expert affidavits that are factually in error.

## I THE GENERAL ACCEPTANCE OF MODELING AS A MEANS TO ESTIMATE DELIVERED DOSE

8. Exposure modeling as a way to estimate or predict the exposure dose of a toxic material is widely accepted and relied upon by people in government, academia, medicine, public health, and consulting. The United States Environmental Protection Agency (US EPA), the US authority in exposure assessment methods and guidance, provides a wide range of guidance on exposure and risk assessment. The methods used by me to estimate exposure for Dr. Barnes where wholly consistent with those methods and guidance. That guidance is too voluminous to append hereto, but can be found at a US EPA site titled "*TOOLS OF THE TRADE*" at: http://www.epa.gov/superfund/programs/risk/tooltrad.htm.

9. Modeling and exposure estimation are necessary because it would be unethical to recreate the conditions of exposure so that direct measurements on humans can be made.

10. Schools of Public Health at leading universities in Canada and the US teach courses at the graduate level that stress the principles and the methods of exposure modeling. Some medical schools teach such courses as well, sometimes in conjunction with an associated school of public health or biomedical engineering. The University of Waterloo has established the Institute for Risk Research, of which I am a member, whose mandate is to encourage research on exposure and risk assessment, and to educate industries, regulators, consultants and others on the appropriate methods of exposure and risk assessment. This Institute also routinely conducts exposure and risk assessments for industrial, regulatory and other clients.

11. There are several international peer-reviewed journals that focus either exclusively or very heavily on modeling at a key tool in quantitative exposure analysis:

- Journal of Environmental Toxicology and Chemistry
- Human and Ecological Risk Assessment,
- Journal of Risk Analysis
- Journal of Exposure Analysis and Environmental Epidemiology

The last two journals are the official publications of international professional societies devoted to the underlying issues of public health, exposure modeling, and risk assessment.

12. Modeling for exposure analysis has been the key tool for designing remedies at more than 1,000 Superfund hazardous waste sites in the United States. In fact, the US EPA develops and distributes risk assessment models of various types and encourages their distribution and use. Government agencies such as Health Canada and the U.S.

Environmental Protection Agency routinely employ exposure models to estimate concentrations of contaminants in drinking water, in air, in indoor dust and in soil. These agencies also routinely employ models of contaminant intake and absorption to establish regulatory limits for industrial chemical contaminants, pesticide residues and additives in foods. Therefore, modeling to establish chemical exposure doses is widely pervasive, accepted and encouraged in the regulatory community.

13. I have prepared an exposure assessment in connection with this lawsuit at the request of the plaintiffs and their attorneys. In this assessment, I conservatively estimated that Dr. David Barnes would receive a dose of mercury from all sources (vapor and particulate) amounting to between 8019 and 8779 $\mu$gs per day, on average, over the course of his professional career as a dentist (Exhibit 2). This dose is fully supported by the general acceptability of the methods used.

14. Kerr's experts (Cohen, Werley, Clarkson, Mackert) claim to provide evidence that the Richardson model and estimates of Dr. Barnes' mercury exposure derived therefrom, are invalid and further, that Dr. Barnes could not be toxicologically impaired by mercury largely because of measured low mercury concentrations in his urine. I offer the following information in rebuttal to those expert opinions.

## II. EVIDENCE OF NEUROLOGICAL IMPAIRMENT IN DENTISTS

15. Kerr's experts imply that Dr. Barnes could not be toxicologically impaired by his mercury exposure because of the low measurements of mercury levels in his urine. Irrespective of the likely loss of volatile mercury from his mercury samples due to improper

storage and long delays before mercury analysis (see paragraphs 16 to 19, below), in fact numerous studies have been published on neurological impairment in dentists. These studies are included as Exhibit 3. Several of those studies demonstrate measurable neurological impact at urine mercury concentrations down to the levels comparable to those of Dr. Barnes.

## III UNRELIABILITY OF URINE MERCURY MEASUREMENTS AS A QUANTITATIVE INDICATOR OF MERCURY TOXICITY

16. Albeit that studies investigating large numbers of dentists demonstrate neurological impairment at levels of mercury in urine similar to those measured in Dr. Barnes, urine mercury is considered to be very unreliable as a surrogate measure of potential toxicity. This is emphasized in studies published in the Journal of the American Dental Association (Langan et al., 1987; Exhibit 5). This large inter-individual variability is due to differences between one person and another in their toxic response to chemical exposure and due to wide variation between different people with respect to the way they metabolize and excrete mercury.

17. Studies demonstrate that "[b]lood and urine values may be used only on a group basis owing to gross individual variations." (Casaret & Doull, Exhibit 6 at 608.) As a result, although studies such as those included in Exhibit 4 show a general trend in large groups or populations for an increasing neurological impairment with increasing urine mercury concentration, this relationship cannot be directly translated to, or interpreted for, individuals. Therefore, Dr. Barnes' urine mercury concentration is only one of a number of

diagnostic tests that would be conducted by a qualified medical practitioner to determine what health impacts he might suffer from and what might cause those effects. Neither I, nor Kerr's expert witnesses Cohen, Werley, Clarkson, Mackert are qualified to interpret such diagnostic tests or draw conclusions respecting their meaning. Therefore, Kerr's experts' implied conclusion that, based on Dr. Barnes' urine mercury levels alone, he could not be suffering from any ill effects, is beyond their expertise to conclude.

18. The handling of urine samples and the timing of mercury analysis following urine collection are very critical. Samples must be chilled to prevent the escape of volatile mercury from the urine sample, and analysis should done soon after collection, likely within 10 minutes (Satoh, Hursh and Clarkson et al., 1981; Exhibit 7). Therefore, if the urine sample was shipped to the laboratory without benefit of refrigeration, as was the case for Dr. Barnes, and the mercury analysis took place days or weeks after sample collection, again as in the case of Dr. Barnes, then the mercury analysis will have little toxicological meaning; most of the volatile elemental mercury in the sample will have already escaped from the urine sample.

19. Therefore, the urine mercury data collected for Dr. Barnes is neither valid nor reliable, due to errors in sample storage, and delays in sample analysis. As a result, Dr. Barnes' urine mercury analysis results are an invalid basis for estimating his exposure or for diagnosing mercury toxicity.

## IV. DUST INHALATION IN THE DENTAL INDUSTRY

20. Kerr experts imply by their affidavits that dust inhalation in the dental industry

is non-existent and certainly that 19 mg of respirable particulate, as estimated for Dr. Barnes, is inconceivable. However, respirable dust inhalation is prevalent in the dental industry, as evidenced by articles by Le Gros, et al. (1988; Exhibit 8) and Stahlhofen and Moller (1993; Exhibit 9) to provide two examples. Stahlhofen and Moller (1993) report an average deposition of 22 mg of dust to the lungs of dental technicians whereas Le Gros et al. (1988) reported respirable dust deposition to the lungs of 4 dental technicians ranging from 725 mg to as much as 3,065 mg. Our estimate for Dr, Barnes is only 19 mg, far lower than these other measurements. However, those publications serve as clear evidence that respirable dust deposition to the lungs of dental workers does occur, and at levels similar to and well in excess of those estimated for Dr. Barnes.

## V.RESPONSE TO COMMENTS OFFERED BY COHEN

21. Dr. J. Cohen offers comment with respect to my exposure assessment for Dr. Barnes. Dr. Cohen is an industrial hygienist by training and, most recently, a senior manager in an occupational health and safety consulting company. The principal activity of occupational hygienists is not to conduct exposure assessments of the type performed for Dr. Barnes. Their function is to monitor contaminant levels in the air of occupational environments, and occasionally in urine and blood, for comparison to pre-published occupational hygiene standards. Those standards are published by the American Conference of Governmental Industrial Hygienists (ACGIH) and/or federal and state occupational safety and health agencies (OSHA).

22. The modeling of environmental fate and transport phenomena, the realm of

practitioners of exposure assessment, is not part of industrial hygiene nor part of their training. Dr. Cohen's résumé demonstrates no experience in modeling for purposes of exposure assessment. His resume indicates that his primary activity has been to monitor occupational environments for comparison to pre-published standards.

23. In paragraph 18 of his affidavit, Dr. Cohen indicates that the only way to assess Dr. Barnes' exposure to mercury is by direct monitoring in Dr. Barnes' dental operatory. This is false. The entire field of exposure assessment is directed at estimating exposures for situations that can not be repeated or recreated. It would be unethical to recreate the degree of contamination in Dr. Barnes' dental office. Therefore, modeling is the only suitable means available to estimate the levels of mercury contamination to which Dr. Barnes was exposed.

24. As for the type of mercury compounds present in Dr. Barnes' dental office (Cohen affidavit, paragraph 18), the only form of mercury present is elemental mercury (also known as metallic mercury and mercury vapor; all of these forms are, in fact, the same - chemically denoted as $Hg^0$). Elemental mercury does not oxidize at normal temperatures when exposed to air, as the metallic form is the more stable or noble form (Merck Index, 2001; Exhibit 10). It is elemental (metallic) mercury that is mixed with an alloy powder to create amalgam (Berry et al., 1994; Exhibit 11) and it is the elemental form that is emitted by set dental amalgam (WHO, 1991; Exhibit 12). Therefore, Dr. Cohen's reference to other forms of mercury is misdirection.

25. In paragraph 19 of Dr. Cohen's affidavit he indicates that the article by Nimmo

et al. (1990; Exhibit 13) can not be relied upon for exposure assessment in dentists as its primary purpose was only to evaluate the influence of using a rubber dam on the exposure of dental patients to amalgam particulate. This is false. If this were true, Nimmo et al. (1990; Exhibit 13) would not have created a surrogate dentist for which particulate exposure was measured. Also, the stated purpose of that article was to "...estimate the amount of particulate inhalation by... the dentist during the removal of existing amalgam restorations under varying conditions". Therefore, Dr. Cohen's statement in paragraph 19 of his affidavit is false.

26. In paragraph 20 of Dr. Cohen's affidavit, he states that the article of Nimmo et al. (1990; Exhibit 13) can not be used for exposure assessment because it did not use standard industrial hygiene methods. This is also false. The study employed an Anderson Impactor to quantify particulate exposures. The Andersen Impactor is among the most widely used instrument for the measurement of particle size and quantification of the mass of particulate, in industrial and other environments (Exhibit 14).

27. In paragraph 21, Dr. Cohen criticizes the use of the Andersen Impactor because he claims that the human breathing rate is "significantly less" than the device's operational air flow rate of 28.3 liters/minute. This is also false. Dr. Cohen reports no specific inhalation rate for a human to support his contention. Such information is summarized and reviewed by the US Environmental Protection Agency's Exposure Factors Handbook (US EPA, 1997; Exhibit 15), the US authoritative source for data on the inhalation rates for use in exposure assessments. Dr. Barnes was not inactive or at rest when inhaling the particulate.

He was involved in intricate work demanding intense concentration and light to moderate levels of physical exertion. The extensive inhalation rate data reviewed by the US EPA and presented in Exhibit 15 indicate that the range of breathing rates for adults involved in light to moderate exertion ranges up to 40.9 liters per minute, a range that encompasses the air flow rate of the Andersen Impactor and making it clearly suitable for quantifying particulate exposure in working dentists such as Dr. Barnes.

28. In paragraph 22 of his affidavit, Dr. Cohen suggests that less than 100% of particles of smaller diameter than 10 μm are not deposited in the lungs. However, Dr. Werley, another of the Kerr's experts in this case, repeatedly refers to particulate matter of less than 10 μm aerodynamic diameter as being "fully respirable" (Nimmo, Werley et al., 1988, Exhibit 16; Nimmo, Werley et al., 1989a, Exhibit 17; Werley et al., 1990, Exhibit 18; and Nimmo, Werley et al., 1990, Exhibit 13). Dr. Cohen offers no citation to support his contention that some of this particulate matter is not fully respirable. Be that as it may, it should be noted that the amalgam particulate inhaled by Dr. Barnes was not simply less than 10 μm in aerodynamic diameter. In fact, the vast majority of dental-generated particulate is less than 3 μm (Nimmo, Werley et al., 1990; Exhibit 13; Nimmo, Werley et al, 1989a, Exhibit 17; Brune et al., 1980, Exhibit 19), and at least 65% of particulate generated by dental drilling is less than 1 μm in aerodynamic diameter (Brune et al., 1980, Exhibit 19); one study determined that more than 63% of the amalgam particulate from a dental office passed through a 0.7 μm filter (Cupelin et al., 1986; Exhibit 20). Between 95% and 100% of inhaled particles of this size range are deposited directly to the alveoli of the lungs

(ACGIH, 2001, Exhibit 21; Owen et al., 1992, Exhibit 22). Therefore, the vast majority of inhaled amalgam particulate will be fully respirable, as determined by authoritative sources in occupational/industrial hygiene and others, and an assumption of 100% introduces very little if any error.

29. In paragraphs 23 and 24 of his affidavit, Dr. Cohen indicates that the Nimmo et al. study failed to account for the filtering of a dust mask and that the masks of the type used by Dr. Barnes have a filtering efficiency of at least 95%. This stated filtering efficiency is for particles greater than or equal to 3 μm in aerodynamic diameter (Christensen et al. 1991; Exhibit 23). However, their efficiency at filtering out particles of less than 3 μm is not stated, and their efficiency at filtering out particles that are less than or equal to 1 μm in diameter is very poor. A study undertaken by Kerr's expert Dr. Werley (Nimmo, Werley et al., 1989b; Exhibit 24) shows that, although larger particles are stopped, particles of 1 μm or less pass through the types of mask worn by Dr. Barnes. As previously stated, amalgam particles to which dentists are exposed are predominantly less than 3 μm (Nimmo et al., 1990; Exhibit 13) and a majority of these particles are even smaller than 1 μm in size (Exhibits 19 and 20).

30. The fact that the Nimmo et al. (1990; Exhibit 13) study did not use a mask to filter particulate exposure in fact best reflects the condition under which Dr. Barnes was exposed to dental amalgam particulate. Dr. Barnes has stated that he wore his mask loosely, regularly displacing it below his nose due to discomfort and to communicate with his patients. The filtering efficiency of a face mask is greatly compromised if it does not fit

tightly against the face and it is recommended not to rely on the mask if it is worn under conditions that prevent direct contact between the face and the edge of the mask (Exhibit 25). As previously indicated, the filtering efficiency for particles less than or equal to 1 μm is effectively zero (Nimmo, Werley et al., 1989b, Exhibit 24).

31. For the reasons outlined above, the comments of Dr. Cohen in no way impact, alter or invalidate the methods or assumptions employed by Richardson in his assessment of amalgam particulate exposure of Dr. Barnes.

## VI. RESPONSE TO COMMENTS OFFERED BY WERLEY

32. Dr. M.S. Werley was second author on the study by Nimmo, Werley et al. (1990; Exhibit 13) which was employed by Richardson in the assessment of Dr. Barnes' exposure to dental amalgam particulate matter. In paragraph 20 of his affidavit, Dr. Werley states that "It was never the purpose to the investigators or this study to assess the amount (mass) of amalgam particulate that could be inspired by a patient or dentist." It is notable that the stated purpose in that same study was to "...estimate the amount of particulate inhalation...by the dentist...". Also, in the methods section of that same paper, Nimmo, Werley et al. state that "inhaled particles were evaluated according to two parameters - mass and size." It is odd that some 12 years after its publication, the second author would now claim that the stated purpose was incorrect. It is also interesting to note that Dr. Werley, in other of his own publications on this body of research, identified the purpose varyingly as:

a) "...to characterize the size and mass of particulate generated during the removal of a series of single amalgam restorations" (Werley et al., 1990;

Exhibit 18);

b) "to determine wether amalgam removal...would produce significant yields of fully respirable particulates in the breathing zone of the patient and dentist." (Nimmo, Werley et al., 1988; Exhibit 16);

c) "...to evaluate the size and range of fully respirable amalgam particles..." (Nimmo, Werley et al., 1989a; Exhibit 17).

33. Therefore, even Dr. Werley considered this research to provide a direct measure of the mass (or yield) of amalgam particulate and the particulate size range to which dentists undertaking amalgam removals were exposed. He offers no explanation as to why the original stated purpose of this body of research should now be altered to omit consideration of the mass and size of particles generated by amalgam removal, particularly since the only reported measurements in those studies were of particle size and mass.

34. In paragraphs 21 and 22, Dr. Werley indicates that the study of Nimmo Werley et al. (1990; Exhibit 13) was meant to provide "a model" of the dentist removing amalgam, "to mimic generally a dentist's chair et cetera" and that the Andersen Cascade Impactor was chosen "as a reasonable simulation of the various levels of the human respiratory tract". With these statements I fully agree. It would be unethical to intentionally expose a person to a toxic substance such as mercury in dental amalgam for the simple purpose of research. Therefore, animal models and in vitro simulations are widely used. Dr. Werley's care in the design of the model and selection of the Andersen Impactor increase the usefulness and applicability of the results to estimating amalgam particulate exposure in Dr. Barnes.

35. In paragraph 22, Dr. Werley repeats his claim that the purpose of the research of Nimmo, Werley et al. (1990; Exhibit 13) was not to estimate the mass of amalgam particulate that might be inspired by a dentist. Again, I refer to paragraphs 32 and 32, above, that clearly indicates that the purpose of this body of research was, indeed, to quantify the mass of particulate matter deposited to the lungs.

36. In paragraph 23, Dr. Werley suggests that the air flow rate of the Anderson Impactor (28.3 liters per minute) was significantly greater than the human inhalation rate, a rate Dr. Werley claims would have been only 6 to 8 liters per minute. However, Dr. Werley fails to mention that the human inhalation rate he quotes is that for a resting, non-active individual. Far from being at rest, Dr. Barnes was involved in the treatment of his patients when inhaling amalgam particulate. This activity would involve a light to moderate level of physical exertion. As previously discussed, the US EPA indicates that the inhalation rate for an adult involved in light to moderate physical activity ranges up to 40.9 liters per minute (US EPA, 1997; Exhibit 15), which encompasses the air flow rate of the Andersen Impactor. Therefore, the amalgam particulate mass measurements determined with the Andersen Impactor are applicable to Dr. Barnes' exposure assessment.

37. In paragraph 25, Dr. Werley introduces a variety of hypothetical errors in his research that he claims invalidate that research. However, he offers no quantification or actual evidence of that error. He does indicate that the filters were dried prior to measurement of amalgam particulate mass. Thus, if any such error did exist, it would be negligible. Also, the research was published in a peer reviewed dental journal and it is very reasonable to

assume that the potential existence of such a basic error would be raised by one of those peer reviewers. In five separate reports of that research (Exhibits 13, 16, 17, 18 and 24) there was never a single mention of this hypothetical error. Given that his research was published in a peer reviewed journal and that such hypothetical error was not mentioned in a total of 5 separate reports of that research, it can be safely and validly assumed that this purported error is only hypothetical and should be ignored until such time as actual evidence of the error can be provided and its impact on reported data can be quantified.

38. Also in paragraph 25, Dr. Werley alludes to plastic as another source of particulate matter measured in the Nimmo, Werley et al. (1990) study. Again, this source of error is not mentioned in any of Dr. Werley's reports of this body of research nor does he provide any actual evidence or quantification of that error. In fact, Dr. Werley has only ever identified that particulate matter as "aerosolized amalgam restoration" (Werley et al., 1990; Exhibit 18), or "amalgam particles" (Nimmo, Werley et al., 1989a; Exhibit 17), and has stated that "When a high-speed handpiece is used to remove an existing restoration, the amalgam is aerosolized into minute particles" (Nimo, Werley et al., 1990; Exhibit 13). Therefore, until some evidence and quantitative measure of this error can be provided, it is very reasonable to interpret the data in the same manner as did Dr. Werley and his co-authors in the past, as providing a measure of the mass and particle size of amalgam particulate to which a dentist is exposed while removing amalgam fillings.

39. In paragraph 26 Dr. Werley states that dental masks as worn by Dr. Barnes "certainly afford some protection against inhalable particles". Within the same reference

cited by Dr. Werley to support this point, it also states "do not wear [masks under] conditions that prevent direct contact between the face and the edge of the respirator". (http://www.ehs.cornell.edu/ochs/rpp.htm, section 4.10; Exhibit 25). Dr. Barnes has testified that he wore his masks loosely and routinely dislodged them from his nose due to discomfort and to communicate with his patients, thereby wearing his masks in a manner that drastically compromises their performance efficiency for particle filtration. Also, as previously discussed, most amalgam particulate inhaled by dentists is less than or equal to 1 μm (Exhibits 19 and 20). Dr. Werley's own research demonstrates that particles less than or equal to 1 μm pass through face masks of the type worn by Dr. Barnes (Nimmo, Werley et al., 1989b; Exhibit 24).

40. Based on the forgoing information, particularly from Dr. Werley's own research reports, it is apparent that his criticisms offered in his affidavit are unfounded and in no way invalidate the exposure assessment performed by Richardson to estimate Dr. Barnes' particulate and mercury exposures. If anything, Dr. Werley's own research supports the use of the Nimmo et al. article as the basis for estimating exposure of a dentist to amalgam particulate matter, and supports my contention that the mask worn by Dr. Barnes provided no protection against inhalation of the amalgam particulate, particularly that which was less than or equal to 1 μm in aerodynamic diameter.

## VII. RESPONSE TO COMMENTS OFFERED BY CLARKSON

41. Dr. Thomas Clarkson criticizes the Richardson assessment of exposure of Dr. Barnes primarily on the grounds that the estimated exposure of 8019 to 8779 μg of mercury

per day does not correspond to Dr. Barnes' measured urine mercury levels. However, this is based on Dr. Clarkson's incorrect assumption that the collected urine was properly stored and transported to the laboratory for analysis, and that the analysis for mercury content was conducted sufficiently soon after urine collection that no mercury would be lost from the sample. However, all of these assumptions are false.

42. The handling of urine samples and the timing of mercury analysis following urine collection are very critical. According to Dr. Clarkson's own research, samples must be chilled to prevent the escape of volatile mercury from the urine sample, and analysis should be done soon after collection, likely within 10 minutes, or volatile mercury in the sample will be lost (Satoh, Hursh and Clarkson et al., 1981; Exhibit 7). Therefore, if the urine sample was shipped to the laboratory without benefit of refrigeration, as was the case for Dr. Barnes, and the mercury analysis took place days or weeks after sample collection, again as in the case of Dr. Barnes, then the mercury analysis will have little meaning toxicologically or with respect to actual mercury exposure. Most of the volatile elemental mercury in the sample will have already escaped from the urine sample prior to analysis.

43. Therefore, the urine mercury data collected from Dr. Barnes is neither valid nor reliable, due to errors in sample storage, and delays in sample analysis. As a result, Dr. Barnes' urine mercury analysis results are an invalid basis for estimating his exposure or for diagnosing mercury toxicity.

44. For these same reasons it is likely that the mercury concentrations reported for comparison to Dr. Barnes' urine mercury levels, from dentists who were attending dental

conferences, were also generally invalid. In particular, urine samples were not likely stored according to Dr. Clarkson's recommendations, nor analyzed within 10 minutes as recommended by Dr. Clarkson. For example, in the report of dental urine levels by Naleway et al. (1985; Exhibit 26), urine samples were refrigerated only within 8 hours and analyzed within one month of urine collection. Other studies of urinary mercury levels in dentists, included as exhibits to Dr. Clarkson's affidavit, are vague or uninformative regarding sample storage and speed of analysis. Therefore, this purported 'standard' for mercury levels in urine of dentists may be significantly below actual values, since samples had not been stored correctly and analyses were not done immediately after urine sampling, as required for valid results.

45. Dr. Clarkson also incorrectly assumes that all of the mercury to which Dr. Barnes was exposed was in the vapor form. Much of that exposure would not have been immediately as vapor, but rather the mercury would have dissolved out of the particulate amalgam by the alveolar fluid.

46. Dr. Clarkson correctly states that "there is no literature which addresses the issue of the absorption of mercury from amalgam particles", particularly in humans (Clarkson affidavit, paragraph 35). However, there is ample evidence that mercury readily dissolves from amalgam. Chew et al. (1991; Exhibit 27) reported that mercury dissolves into distilled water at a rate of 43 μg per $cm^2$ of amalgam surface area per day. Gross and Harrison (1989; Exhibit 28) determined the loss of mercury from amalgam to be 37.5 μg per $cm^2$ of amalgam per day into Ringers solution.

51. In paragraph 35, Dr. Clarkson suggests that studies involving the implantation of amalgam into the soft tissue of guinea pigs (Eley, 1990; Exhibit 34) are a better model for the absorption of amalgam particulate from the lungs. However, it is inconceivable how the study of Eley (1990) could be an appropriate model of lung absorption of amalgam particulate, or any particulate matter. First, the Eley study was undertaken in order to investigate the fate of amalgam particles contained in 'amalgam tatoos' that are amalgam particles embedded in the gums of dental patients. It was not designed or conceived to mimic the lungs. Second, the 'soft tissue' in no way mimics lung tissue. This soft tissue is anatomically, physiologically and microbiologically different from lung tissue. It is not perfused with capillaries and blood supply nearly to the same extent as lung alveoli (the deposition zone for amalgam particulate matter). Amalgam implanted into this soft issue can not contact the alveolar fluid that plays an important role in the dissolution of metallic particulate matter (Ansoborlo et al., 1999; Exhibit 35). Amalgam embedded in soft tissue will not be exposed to air as occurs in the lungs during normal respiration and, therefore, would not permit or encourage the volatilization of mercury vapor as would occur in the lungs. Third, amalgam embedded in soft tissue can not be contacted by or phagocytized by lung-associated macrophages, one of the key vectors for removal of particulate matter deposited to the lungs to the lymphatic system (Casarett and Doull, 1996; Exhibit 36). Fourth, the size of the particles implanted by Eley (1990) were much greater in size (less than 45 μm in diameter) than those that were inhaled by Dr. Barnes (mean diameter of 2 μm). Since absolute amount of mercury released from amalgam in any given amount of time

increases as the total surface area increases (see Exhibits 27 and 28), the smaller the particles, the larger the total surface area and the greater the amount of mercury released. Finally, the guinea-pigs in the study of Eley (1990) were fed selenium throughout the entire study. Selenium is known to inhibit and delay the rate of absorption and tissue clearance of mercury (Khayat and Dencker, 1983; Exhibit 37) thereby nullifying the validity of that study for use in any estimate of Dr. Barnes' mercury absorption and dose.

52. Despite Dr. Clarkson's involvement with the preparation of the World Health Organization's Environmental Health Criteria 118 on inorganic mercury, he failed to mention in his affidavit about the studies reviewed by WHO concerning the absorption of mercury aerosols (WHO, 1991; Exhibit 38). The WHO noted that macrophages can increase the solubility of metals. The WHO concluded that, for inhaled particulate mercury, "significant absorption must take place directly from the lung". Therefore, it is certain that elemental mercury delivered in an aerosol (particulate form) will undergo "significant absorption".

53. Perhaps another acceptable approach to determining the rate of absorption of mercury from the amalgam particulate deposited to Dr. Barnes' lungs would have been to apply the methods used in the assessment of exposure to air-borne, particulate radiological hazards. The occupational health area that has dealt most with the problem of inhaled particles is that of radiological protection; the inhalation of radio-active particles. The International Commission on Radiological Protection (ICRP) has established models for estimating internalized dose due to inhalation of respirable particulate matter. These models

conferences, were also generally invalid. In particular, urine samples were not likely stored according to Dr. Clarkson's recommendations, nor analyzed within 10 minutes as recommended by Dr. Clarkson. For example, in the report of dental urine levels by Naleway et al. (1985; Exhibit 26), urine samples were refrigerated only within 8 hours and analyzed within one month of urine collection. Other studies of urinary mercury levels in dentists, included as exhibits to Dr. Clarkson's affidavit, are vague or uninformative regarding sample storage and speed of analysis. Therefore, this purported 'standard' for mercury levels in urine of dentists may be significantly below actual values, since samples had not been stored correctly and analyses were not done immediately after urine sampling, as required for valid results.

45. Dr. Clarkson also incorrectly assumes that all of the mercury to which Dr. Barnes was exposed was in the vapor form. Much of that exposure would not have been immediately as vapor, but rather the mercury would have dissolved out of the particulate amalgam by the alveolar fluid.

46. Dr. Clarkson correctly states that "there is no literature which addresses the issue of the absorption of mercury from amalgam particles", particularly in humans (Clarkson affidavit, paragraph 35). However, there is ample evidence that mercury readily dissolves from amalgam. Chew et al. (1991; Exhibit 27) reported that mercury dissolves into distilled water at a rate of 43 μg per $cm^2$ of amalgam surface area per day. Gross and Harrison (1989; Exhibit 28) determined the loss of mercury from amalgam to be 37.5 μg per $cm^2$ of amalgam per day into Ringers solution.

47. We also know that elemental mercury is highly lipid soluble (Lorscheider et al., 1995; Exhibit 29) and that alveolar (lung) fluid contains lipids (Hamm, Fabel and Bartsch, 1992, Exhibit 30; Guthmann et al., 1995; Exhibit 31). Therefore, the ready solubility of mercury from amalgam, combined with the lipids contained in lung fluid, will rapidly and completely dissolve all of the mercury deposited to Dr. Barnes' lungs as a result of his dental practice.

48. The amalgam particulate inhaled by Dr. Barnes averages 2 μm ($2 \times 10^{-4}$ cm) in diameter (Nimmo et al., 1990; Exhibit 13). Based on a density of amalgam of 11.5 g/cm$^3$ (Letzel et al., 1997; Exhibit 32), each amalgam particle would weigh, on average, $4.82 \times 10^{-11}$ grams. With 0.019 g of amalgam particulate deposited to Dr. Barnes' lungs per working day (Richardson assessment of Dr. Barnes' exposure, Exhibit 3), this represents a total of $3.96 \times 10^8$ amalgam particles. That many particles, with an average diameter of 2 μm, will have a total surface area of 51.5 cm$^2$. Assuming a dissolution rate for mercury from set amalgam of 40 μg/cm$^2$-day (based on Exhibits 27 & 28), and ignoring the lipid content of the lung (to be conservative) which would enhance mercury dissolution, then in 1 day some 2060 μg of mercury could dissolve from those amalgam particles. This would mean that all of the mercury deposited to Dr. Barnes' lungs within amalgam particulate in a day (9500 μg) would dissolve within 4.6 days, again assuming that lung fluid were only distilled water or saline; the lipid content of lung fluid would make the mercury dissolve faster. Regardless, it is apparent that a rate of three days, as measured by Cutright et al. (1973), closely reflects this estimated dissolution time. The study of Eley (1990), where absorption peaked only

after a year and was not complete for four years, is obviously inappropriate. The solubility phenomena of mercury within amalgam, if not inhibited by selenium (as in the case of Eley (1990)), will cause the dissolution of that mercury in just a few days at most. It is apparent that the capacity for mercury to dissolve from amalgam in the lungs closely parallels the direct measurements made by Cutright et al. (1973; Exhibit 33). Therefore, there is ample evidence to suggest that all of the mercury deposited to Dr. Barnes' lungs in the form of particulate was absorbed. Richardson, however, assumed only 80%.

49. In paragraph 33 of his affidavit, Dr. Clarkson indicates that reliance on the study of Cutright et al. (1973; Exhibit 33), respecting the rate of clearance of 'amalgam mist' from the lungs of rats, is misplaced because the relative proportion of mercury vapor and amalgam particulate were not reported. In fact, when amalgam is subjected to grinding, both mercury vapor and particulate are generated and the particulate itself will also emit mercury vapor. Therefore, amalgam particulate and mercury vapor can never be separated; where there is amalgam particulate there will be mercury vapor.

50. In paragraph 34, Dr. Clarkson indicates that the 'amalgam mist' was generated by the dry grinding of an amalgam block. Therefore, the 'amalgam mist' can not be anything other than a combination of amalgam particulate and amalgam vapor. This is precisely what Dr. Barnes would have been exposed to during the removal of amalgam fillings from his dental patients. The fact that it is described as a 'mist' alludes to the fact that it was visible and indicates that much of that mist was, in fact, particulate matter as mercury vapor is invisible.

51. In paragraph 35, Dr. Clarkson suggests that studies involving the implantation of amalgam into the soft tissue of guinea pigs (Eley, 1990; Exhibit 34) are a better model for the absorption of amalgam particulate from the lungs. However, it is inconceivable how the study of Eley (1990) could be an appropriate model of lung absorption of amalgam particulate, or any particulate matter. First, the Eley study was undertaken in order to investigate the fate of amalgam particles contained in 'amalgam tatoos' that are amalgam particles embedded in the gums of dental patients. It was not designed or conceived to mimic the lungs. Second, the 'soft tissue' in no way mimics lung tissue. This soft tissue is anatomically, physiologically and microbiologically different from lung tissue. It is not perfused with capillaries and blood supply nearly to the same extent as lung alveoli (the deposition zone for amalgam particulate matter). Amalgam implanted into this soft issue can not contact the alveolar fluid that plays an important role in the dissolution of metallic particulate matter (Ansoborlo et al., 1999; Exhibit 35). Amalgam embedded in soft tissue will not be exposed to air as occurs in the lungs during normal respiration and, therefore, would not permit or encourage the volatilization of mercury vapor as would occur in the lungs. Third, amalgam embedded in soft tissue can not be contacted by or phagocytized by lung-associated macrophages, one of the key vectors for removal of particulate matter deposited to the lungs to the lymphatic system (Casarett and Doull, 1996; Exhibit 36). Fourth, the size of the particles implanted by Eley (1990) were much greater in size (less than 45 μm in diameter) than those that were inhaled by Dr. Barnes (mean diameter of 2 μm). Since absolute amount of mercury released from amalgam in any given amount of time

increases as the total surface area increases (see Exhibits 27 and 28), the smaller the particles, the larger the total surface area and the greater the amount of mercury released. Finally, the guinea-pigs in the study of Eley (1990) were fed selenium throughout the entire study. Selenium is known to inhibit and delay the rate of absorption and tissue clearance of mercury (Khayat and Dencker, 1983; Exhibit 37) thereby nullifying the validity of that study for use in any estimate of Dr. Barnes' mercury absorption and dose.

52. Despite Dr. Clarkson's involvement with the preparation of the World Health Organization's Environmental Health Criteria 118 on inorganic mercury, he failed to mention in his affidavit about the studies reviewed by WHO concerning the absorption of mercury aerosols (WHO, 1991; Exhibit 38). The WHO noted that macrophages can increase the solubility of metals. The WHO concluded that, for inhaled particulate mercury, "significant absorption must take place directly from the lung". Therefore, it is certain that elemental mercury delivered in an aerosol (particulate form) will undergo "significant absorption".

53. Perhaps another acceptable approach to determining the rate of absorption of mercury from the amalgam particulate deposited to Dr. Barnes' lungs would have been to apply the methods used in the assessment of exposure to air-borne, particulate radiological hazards. The occupational health area that has dealt most with the problem of inhaled particles is that of radiological protection; the inhalation of radio-active particles. The International Commission on Radiological Protection (ICRP) has established models for estimating internalized dose due to inhalation of respirable particulate matter. These models

are employed by the US Department of Energy, as part of their DOE Standard: Guide of Good Practices for Occupational Radiological Protection in Uranium Facilities (Exhibit 39). According to those ICRP models, for any particulate substance that is classified as being highly soluble, and/or where in vivo studies in living laboratory animals demonstrate absorption in less than 10 days, it is assumed to be rapidly absorbed, with 80% entering the blood and 20% of the deposited dose entering the lymphatic system (Parsont et al., 1972; Exhibit 40).

54. Dr. Clarkson's criticisms relate primarily to the fact that, in his opinion, the model offered by Cutright et al. (1973; Exhibit 33) does not deal specifically with particulate amalgam, and that an alternate model is required that can be confirmed to exclude mercury vapor, which is a component of the 'amalgam mist' described by Cutright et al. (1973). I have offered above an alternate model of mercury absorption from amalgam particulate in the lung that further corroborates my assumed absorption rate of 80%. However, a further corroborative model exists. That study is by Takenaka et al. (2001; Exhibit 41) who investigated the fate of silver particulate deposited to rat lungs. Amalgam is approximately 50% mercury by weight but is also approximately 35% metallic silver by weight (Berry et al., 1994; Exhibit 11). Silver is not volatile and, therefore, the study of Takenaka et al. provides perhaps the best model for the fate of non-volatilized amalgam particulate deposited to the lung.

55. Takenaka et al. reported that 96% of the silver particulate was removed from the lungs within 7 days, not greatly dissimilar to the 3 days for elimination of the 'amalgam

mist' from the lungs of rats in the Cutright et al. (1973; Exhibit 33) study, and far less that the 4 or more years reported in the Eley (1990; Exhibit 34) study for elimination of mercury amalgam embedded in low-vascularized soft tissue of selenium-fed guinea pigs.

56. Takenaka et al. reported that levels of silver in the blood increased immediately after inhalation exposure. This clearly indicates absorption of this particulate matter. What's more, silver is considered to be 'insoluble' (ATSDR, 1990; Exhibit 42). Given that mercury is much more soluble than silver (ATSDR, 1999; Exhibit 43), that mercury is readily dissolved out amalgam (Exhibits 27 and 28), and whereas silver dissolution out of amalgam can not be detected (Kozono et al., 1982; Exhibit 44), it would be impossible for silver to be absorbed but not mercury. Due to mercury's greater solubility, it would be absorbed at a much greater rate and percent than silver.

57. I have demonstrated above that the mercury in amalgam is highly soluble. There are no studies specifically investigating the clearance of amalgam particulate from the lungs of humans, as agreed with Dr. Clarkson, Kerr's expert. However, there are published studies that demonstrate that the two most significant components of amalgam, mercury and silver, are cleared from the lungs in less that 10 days; 3 days for mercury (Cutright et al., 1973, Exhibit 33) and 7 days for silver (Takenaka et al., 2001; Exhibit 41). ICRP methodology for assessing risks from inhaled particulate matter specifies an assumption of 80% absorption into the blood under such conditions (Exhibit 39). Therefore, Richardson's assumption of 80% of mercury absorbed from the inhaled particulate was correct.

58. We must rely on the most representative models for deducing the extent of

61. Clearance of amalgam particulate from the lung will not take longer than 4 years as suggested by Clarkson based on the study of Eley (1990) on low-vascularized soft tissue.. It will take days at most. The mercury contained in that amalgam particulate would be absorbed rapidly, demonstrably within a day, and at least 80% would be absorbed, as originally assumed by Richardson.

## VIII. RESPONSE TO COMMENTS OFFERED BY MACKERT

62. Dr. Mackert offers expert opinion concerning the exposure of Dr. Barnes to mercury vapor and particulate that occurred during Dr. Barnes practice of dentistry. However, Dr. Mackert has no expertise in this subject field of exposure assessment. He is a dental materials scientist. He has published no articles that have been peer reviewed by members of the exposure and risk assessment community. Refereed journals in that field include *Human and Ecological Risk Assessment*, the *Journal of Exposure Analysis and Environmental Epidemiology*, *Environmental Toxicology and Chemistry*, and *Risk Analysis*.

63. Dr. Mackert has published three articles regarding the exposure to mercury in dental patients (Mackert, 1987, Exhibit 45; Mackert, 1991, Exhibit 46; Mackert and Berglund, 1997, Exhibit 47). In those articles, Dr. Mackert is out of step with scientific opinion on the issue of mercury exposure in dental patients from dental amalgam. Exhibit 48 presents a summary of some 14 published estimates of mercury exposure from dental amalgam in patients, along with those estimates provided in Dr. Mackert's articles. His estimates are the extreme lowest values published.

64. Dr. Mackert correctly identifies himself as a peer reviewer of the Health Canada

risk assessment on dental amalgam (Mackert affidavit, paragraph 8). However, Dr. Mackert was included as a peer reviewer only at the request of Dr. Mackert himself, and legal counsel for Sybron Inc. (which included Kerr's counsel Mr. David Bartel), the parent company of Kerr Corporation. That request was made directly to Dr. Richardson when he was an employee of Health Canada and preparing that risk assessment. Dr. Mackert, as a paid consultant to Sybron Inc. and Kerr, as well as legal counsel for Kerr, requested and attended two meetings with Dr. Richardson while the Health Canada risk assessment was in preparation. The request to be included in the peer review process was made during those meetings. Dr. Mackert was not included in Health Canada's list of preferred peer reviewers (Exhibit 49). His inclusion as a peer reviewer was done as a courtesy, not due to any purported expertise relevant to the risk assessment.

65. Dr. Mackert has a track record of selectively interpreting available science toward minimizing exposure to mercury arising from dental amalgam. This is evident in his dental journal articles on this issue (see Exhibits 45, 46 and 47). This is also evident in the comments that he submitted to Health Canada (Exhibit 50). Those comments were reviewed and predominantly dismissed as being inappropriate, inadequate or biased toward minimizing the estimate of mercury exposure and the subsequent health risks posed by that exposure (Exhibit 51).

66. In his affidavit, Dr. Mackert alludes to 'well-established, peer reviewed methodology' provided in his Exhibit 3a (see Mackert affidavit, paragraph 20). However, what is offered in his Exhibit 3a is not well-established, peer reviewed methodology, but

merely Dr. Mackert's own model, based on alternate published literature (inappropriate as it may be for the application employed) that seemingly justifies the lower dose he advocates. As for Dr. Mackert's assertion that the dose of dental particulate would cause a urine mercury measurement of some 2000 to 3000 µg/liter, this is merely a repetition of comments provided by Dr. Clarkson and I refer you back to paragraphs 16 to 19, and paragraphs 41 to 61 above which address similar issues. The points will not be repeated here, for the sake of brevity.

67. Also in Exhibit 3 of Dr. Mackert's affidavit, he alludes to the fate of inhaled particulate and implies that once engulfed by macrophages, amalgam would somehow be isolated and mercury could no longer dissolve out of that particulate and enter the body. However, the WHO (1991; Exhibit 38) noted that macrophages can increase the solubility of metals.

68. When particles are inhaled, one of the first defensive mechanisms of the body is the engulfing of particles by lung macrophages (Casarett and Doull, 1996; Exhibit 36). However, this does not mean that the particulate in general, or the mercury within amalgam particulate in particular, is actually cleared from the lungs. The mechanism for clearance of ultra fine (sub-micron) particulate matter from the lung is very inefficient (Casarett and Doull, 1996; Exhibit 36). Within the first 24 hours only about 20% of deposited submicron particles will be cleared, irrespective of the mechanism (Casarett and Doull, 1996; Exhibit 36). Richardson did assume that 20% of the mercury in amalgam particles would not be available for absorption.

69. Once engulfed by macrophages, elemental mercury will continue to be released by amalgam particles and that mercury will escape from the macrophages. Elemental mercury is highly lipid soluble and readily crosses all biological membranes (Lorscheider et al., 1995). In fact elemental mercury also readily crosses the blood-brain barrier and the placenta into the fetus (WHO, 1991; Exhibit 52), demonstrating the ease and speed with which it moves across membranes and throughout the body. What macrophages do escape the lungs will carry a large proportion of their mercury and amalgam particulate load into the lymphatic system (Casarett and Doull, 1996; Exhibit 36). This is not removing it from the body, but rather segregating it to the lymphatic system, which also ultimately returns it to the circulatory system (Casarett and Doull, 1996; Exhibit 36).

70. As previously mentioned, the fate of inhaled particulate and the role of macrophage phagocytosis is considered within the inhalation dose models developed for quantifying doses of inhaled radioactive particles. The ICRP model assumes that, for highly soluble particles that have *in vivo* evidence of lung clearance in less than 10 days, 80% of those particles are absorbed into the blood and 20% enters the lymphatic system via macrophages (ICRP, 1994; Exhibit 39). Richardson assumed that only 80% of the inhaled mercury would be absorbed, omitting consideration of the possible absorption of additional mercury from the lymphatic system.

71. Richardson assumed that 80% of the particulate-borne mercury was absorbed. It is quite apparent that this is consistent with Casarett and Doull (1996; Exhibit 36) that indicates only 20% clearance of sub-micron particles from the lungs in 24 hours. This

assumption is also consistent with the ICRP model for quantifying doses of inhaled particulate matter. We have demonstrated above (paragraph 42) that the solubility of elemental mercury from the inhaled amalgam particulate is sufficiently high to dissolve all mercury contained therein within the same time frame (just a few days) observed in direct study by Cutright et al. (1973; Exhibit 33). If contained within macrophages, that elemental mercury will escape the macrophage cell due to its lipid solubility (Lorscheider et al., 1995; Exhibit 29) and enter the blood stream due to the high perfusion of alveolar tissue with blood capillaries.

72. The study by Cutright et al. (1973; Exhibit 33) is the single most suitable basis for demonstrating that the mercury contained in amalgam particulate is absorbed within very short time. It clearly demonstrated increased blood mercury levels immediately following inhalation of 'amalgam mist' in rats, and the majority of the mercury associated with that inhaled particulate matter was absorbed from the lung in no more than three days. Since mercury vapor is rapidly absorbed immediately upon inhalation, the mercury contained in the lungs of rats investigated by Cutright et al. (1973) could not have been mercury vapor, as it would all have disappeared from the lungs within minutes of the end of the exposure. For the mercury to remain in the lungs for up to 3 days, that mercury had to be contained within particulate matter. Calculations provided above in paragraph 42 match with Cutright's observations. The study of Takenaka et al. (2001) clearly demonstrates that non-volatile and non-soluble metal particulate (silver, the next most significant component of dental amalgam particles) is also rapidly absorbed into the blood stream with this 'insoluble'

silver cleared within 7 days.

73. The available evidence very clearly indicates that the absorption of the mercury in amalgam particulate matter will occur, certainly faster than 4 years as suggested by Dr. Clarkson, and will continue to completion, within a few days. Therefore, claims by Kerr experts to the contrary are unfounded and not supported by the most relevant science. To imply that macrophages somehow stop, or even impede, the mercury absorption simply by particle engulfment without ***any*** scientific evidence of such a phenomenon, is simply to ignore the vast scientific evidence to the contrary. Dr. Richardson's assumption of 80% absorption is strongly supported by the published scientific evidence.

74. In paragraphs 22 through 25 of his affidavit, Dr. Mackert attempts to extrapolate Dr. Barnes' amalgam particulate exposure to his urine mercury levels and claims that since they don't correspond, the estimates of Richardson are in error. Urine testing has been previously discussed and will be omitted in this discussion for the sake of brevity.

75. Dr. Mackert claims that the methodology employed by Richardson is not realistic or scientifically-based (Mackert affidavit, paragraph 24). The regulatory authority on exposure assessment in the United States is the Environmental Protection Agency (US EPA). That agency provides extensive and detailed guidance on the conduct of exposure assessments for quantifying exposure to vapors (such as mercury vapor) and particulate matter (such as amalgam particulate). Those methods and guidance documents are too voluminous to reproduce herein. However, they can be found at the following INTERNET addresses: http://www.epa.gov/superfund/programs/risk/tooltrad.htm#gp The US EPA

titles this web site "*TOOLS OF THE TRADE*".

76. Guidance offered by the US EPA was followed in the conduct of Dr. Barnes', exposure assessment, with one exception. One aspect of US EPA's direction on risk assessment was not followed, that being the method of deriving the concentration of mercury in air or on surfaces from which exposure estimates were determined. The US EPA recommends the use of the upper 95$^{th}$ percentile concentration from all the concentration data collected. For example, if 100 concentration measurements were made, when ordered from lowest to highest the 95$^{th}$ percentile value would be the 95$^{th}$ concentration, nearly the maximum concentration measured. To ensure that exposure estimates were realistic, Richardson selected the arithmetic average concentration in order not to overestimate Dr. Barnes' likely exposure to mercury vapor and amalgam particulate.

77. In paragraphs 29 through 31 of his affidavit, Dr. Mackert complains that Richardson did not use specific information supplied by Dr. Barnes respecting the number of filling removals performed in a year. In fact, the number of days assumed (220 days per year) and the number of fillings removed per year (1000 amalgams per year) were provided by Dr. Barnes or determined during consultation with him. In fact, Dr. Mackert has applied the number of amalgams placed in 1996 and 1998 with the number of days worked in 1997. These numbers do not correspond and are totally mismatched. For example, in 1998 Barnes went to a 4 day week, 8 hour day, down from a 5 day week, 10 hour day from the previous years. Therefore, Dr. Mackert is using a minimal number of fillings from a period of short work weeks, with a maximal days worked from a period of full work weeks. It must also be

noted that the purpose of my exposure assessment was to estimate the average daily exposure for Dr. Barnes over his working career (since early 1980's) not just for one particular day or year. Therefore, the numbers of days worked and number of amalgam procedures performed, as reported by Dr. Barnes, were employed correctly.

78. In paragraphs 32 through 35, Dr. Mackert criticizes the use of the study of Richards & Warren (1985; Exhibit 53) as the basis for estimating breathing zone mercury vapor levels during an amalgam procedure, claiming that the Richards & Warren study over-estimates breathing zone mercury vapor levels. Instead, Dr. Mackert suggests the study of Powell et al. (1994; Exhibit 54) as being more representative. What Dr. Mackert fails to recognize is that Powell et al. performed only discrete occasional mercury measurements whereas Richards & Warren employed continuous (non-stop) mercury monitoring. This latter technique is crucial for the accurate characterization of mercury vapor levels throughout the entire amalgam removal procedure. To suggest that spot measurements made at intervals separated by several minutes will accurately reflect mercury levels throughout a prolonged period of time is to misunderstand the importance of continuous air quality monitoring for purposes of exposure assessment.

79. Dr. Mackert also identifies the study of Bergman & Pohl (1995; Exhibit 55) as preferable to that of Richards & Warren (1985) for estimating Dr. Barnes' exposure to mercury vapor. However, that study is unreliable as a basis for exposure assessment. First, there is no description of mercury hygiene practices in the dental office where the study was conducted, or by the single dentist who was studied. The study was conducted in Sweden,

where awareness of the hazards posed by mercury vapor inhalation have been well known and well publicized for many years. Dr. Barnes was not the beneficiary of such awareness.

80. Also, the personal sampling device used was operated at a volumetric air flow rate of 8 liters per minute. As previously discussed, this air flow rate falls well below the measured inhalation rates of persons involved in light to moderate physical exertion and is almost 3 times below US EPA's recommended inhalation rate for exposure assessment of adults involved in light to moderate activity.

81. Finally, the sampling equipment was equipped "with a Y-formed orifice at nose level". Given that air was drawn through this orifice at a constant rate, it would be drawing air from the dentist's own exhalations, not just from the air that he/she would be breathing it. When inhaled, mercury vapor is rapidly and almost completely absorbed (WHO, 1991, Exhibit 38). Therefore, during exhalations, the device was sampling virtually mercury-free air.

82. Based on comments offered above, it is apparent that the most preferable approach to estimating exposure to mercury vapor by Dr. Barnes is to rely on continuous measurements of mercury vapor in the vicinity of the dentist during amalgam removal. That data best reflects the environment to which Dr. Barnes was exposed, and can then be combined with information on inhalation rate and mercury vapor absorption to properly estimate Dr. Barnes' mercury vapor exposure. This is precisely what was done by Richardson.

83. In paragraph 36, Dr. Mackert attempts to further claim that the measurements

made by Richards & Warren were inaccurate due to deficiencies in the mercury vapor detector employed by Richards & Warren. The device they used, the Bacharach MV2 mercury vapor detector, is a recommended device by the Occupational Safety and Health Administration, US Department of Labor (Exhibit 56). Therefore, I fail to see the problem with the device, particularly since Kerr's other expert, Mr. Cohen, emphasized the need to employ approved industrial hygiene measurement methods. Therefore, the measurements of Richards & Warren were made using standard, approved industrial hygiene equipment and are fully valid and reliable.

84. With respect to concentration of mercury vapor reported by Richards & Warren, that mercury vapor level actually exceeded 100 $\mu g/m^3$ during amalgam removal. However, Richardson set an arbitrary maximum limit of 100 $\mu g/m^3$ in order to be conservative in the estimates of mercury exposure in Dr. Barnes.

85. In paragraph 39, Dr. Mackert states "contrary to Dr. Richardson's assertions ... it is not common practice in the scientific community to ignore other published data and cite only one source as a representative example..". Quite to the contrary, instructions to contributors for the journals *FASEB* and *Nature* are attached as Exhibits 57 and 58, respectively. These are just two examples of journals' desire to keep papers brief and succinct.

86. Instructions from *FASEB* state "For **Review Articles**, literature citations of earlier findings should be selective rather than encyclopedic.". *Nature* specifies that the maximum number of references permitted is 50 for Articles and 30 for Letters.

87. The nature of scientific writing is to be concise. Therefore, from Exhibits 57 and 58, it is very apparent that the normal scientific writing style is to use citations with economy, avoiding the unnecessary citation of each and every possible example when one or two will suffice. This is precisely the style of writing that Richardson practiced with respect to Dr. Barnes' exposure assessment report.

88. In paragraphs 41 and 42, Dr. Mackert asserts that Richardson somehow misquoted or misrepresented the results of an article by Haikel et al. (1990; Exhibit 59). This was not the case. The fact that an amalgam placement releases mercury vapor does not appear to be disputed by Dr. Mackert. The article by Haikel et al. was cited only as qualitative evidence of vapor release during placements and not as a basis for quantifying those mercury vapor levels. Dr. Mackert is quite correct in stating that the Haikel et al. article would be inappropriate for that purpose.

89. In paragraphs 44 through 51, Dr. Mackert is simply repeating the same arguments offered by Kerr's expert Dr. Werley. Responses to Dr. Werley's comments were provided above in paragraphs 32 to 40. For the sake of brevity, those comments will not be repeated here. In essence, Dr. Werley's own research supports the manner in which Richardson employed the data from the Nimmo, Werley et al. (1990) study.

90. In paragraphs 52 through54 of his affidavit, Dr. Mackert suggests that a study by Musajo et al. (1988; Exhibit 60) is a preferable alternative basis for assessing the intake of particles. However, if Dr. Mackert were familiar with exposure assessment methods and authoritative sources, particularly pertaining to human inhalation, he would know that the

air flow rate of 3 liters per minute used by Musajo et al. is far too low to represent or mimic adult inhalation rate. In fact, extensive inhalation rate data reviewed by the US EPA (Exhibit 15) suggests that an inhalation rate of at least 10 liters per minute (15 $m^3$/24 hours) is required just to support basal metabolism in an adult male. As indicated by the US EPA (Exhibit 15), and discussed earlier in paragraph 27, the inhalation rates for adult males involved in light to moderate activity range up to 41 liters per minute, a rate that is far in excess of the air flow rate employed by Musajo et al. (1985). Therefore, the Musajo et al. study is certainly faulty with respect to providing representative particulate intakes for dentists.

91. It is also noted that, in the Musajo et al. study, particulate samples were collected on filters which had a limited capacity to collect submicron particulate matter. The filters collected particulate matter in the range of 0.5 to 5 μm in aerodynamic diameter. Because the air flow rate of the collection system was so low, it would preferentially collect only the smallest particles, since the flow rate could only overcome the gravitational force on these smallest particles. Gravity causes particles to settle out of the air and must be overcome by particle collection systems. With a large proportion of amalgam particulate being less than 0.5 μm in size (Brune et al., 1980, Exhibit 19; Cupelin et al., 1986, Exhibit 20) and with the Musajo et al. collection method failing to collect particles of less than 0.5 μm, then the measurements grossly under-estimate the true air-borne particulate concentration. This problem is not encountered with an Andersen Impactor, however, (as used by Nimmo et al., 1990; Exhibit 13) which collects all particles less than 10 μm, irrespective of particle size.

For these reasons, the Musajo et al. study is totally inappropriate for estimating particulate inhalations in dentists.

92. It is appropriate to point out at this stage that Dr. Mackert agrees with the methods employed by Dr. Richardson in the assessment of particulate exposure. He has applied the same basic methods. Therefore, his only complaint appears to be the selection of the study from which to obtain data to input to the model, not the model itself.

93. In paragraphs 55 through 60 of his affidavit, Dr. Mackert merely repeats the same criticisms offered by Dr. Clarkson, another of Kerr's expert witnesses. As Dr. Mackert is not an expert in the absorption or pharmacokinetics of contaminants in general nor mercury specifically, nor is he a physiologist or toxicologist, his comments fall well outside his area of expertise. Please refer to paragraphs 41 to 61 above which address all of the issues raised by Dr. Clarkson that were repeated by Dr. Mackert.

94. In paragraphs 61 to 67, Dr. Mackert again offers comments in areas well outside his area of expertise. The comments offered merely repeat those offered by Kerr's expert Mr. Cohen. Those criticisms have been commented on above in paragraphs 21 to 31, and will not be repeated here, for the sake of brevity. However, there are three specific points that require comment.

95. First, Dr. Mackert does make the comment in paragraph 61 of his affidavit that Richardson cited no established procedure for estimating air-borne mercury vapor concentrations in Dr. Barnes' dental office based in surface area contamination. Therefore, I have appended Exhibit 61 an article by Sverdrup, Warfvinge and Sverdrup (1990) which

employs essentially the same methodology to estimate mercury levels in the air of a hypothetical bedroom as a result of spilled mercury. This methodology is not unique or non-existent. Such models and calculations are common practice to exposure and risk assessors.

96. Second, Dr. Mackert claims in paragraph 64 that the Tennessee Occupational Safety and Health Agency (TOSHA) failed to detect mercury vapor in Dr. Barnes' dental operatory when they visited the office following Dr. Barnes' thorough clean up of his office. The TOSHA method for attempting to measure mercury vapor used a Sensidine-Gastec mercury detection tube connected to a hand-operated pump. TOSHA personnel operated that hand pump for only 3 pumps of air through the detection tube. The detection limit for this device with only 3 pump strokes is 150 μg of mercury vapor in 1 cubic meter of air ($ug/m^3$) (Exhibit 62). However, the post-clean up levels of mercury vapor in Dr. Barnes' office would only have been approximately 3 $\mu g/m^3$ to 8 $\mu g/m^3$ (Exhibit 3). Therefore, the detection limit of the TOSHA mercury vapor sampling method was some 20 to 50 times too high to detect the probable level of mercury in Dr. Barnes' office at the time of TOSHA sampling. Therefore, this TOSHA result in no way suggests that no mercury vapor was present after Dr. Barnes decontaminated his office; it simply indicates that mercury levels were below 150 $\mu g/m^3$, a fact with which I agree.

97. Third, Dr. Mackert claims in paragraph 62 of his affidavit that the highest concentration of mercury measured on the surfaces of Dr. Barnes' office should be omitted as "an outlier". To support this notion, Dr. Mackert attempts to conduct a statistical test known as Dixon's test (presented in Mackert affidavit, exhibit 3a). Unfortunately, Dr.

Mackert has applied this test incorrectly. The underlying assumption for this test for outliers is that the remaining data (all other data excluding the purported outlier) are normally distributed (see Sokal and Rohlf, 1984, pp 412-413; Exhibit 63). Where those data are not normally distributed, they must first be mathematically 'transformed' to achieve normality.

98. The data in question, the measurements of surface area mercury contamination in Dr. Barnes' office, are not normal (Lilliefors test, $p<<0.0005$; a value of $p \leq 0.05$ indicates that the data are not normal). However, when the logarithms of the data are determined, which is the customary statistical transformation for all concentration data, the data are now normally distributed (Lilliefors test, $p=0.073$; since $p>0.05$, then the hypothesis that the data are different from a normal distribution is rejected and the data are concluded to be normal). It is essential that Dixon's test be performed on these logarithmically-transformed values for the test to be valid statistically. When this is done, the Dixon test achieves a value for the parameter $r_{11} = 0.237$. Since this value for $r_{11}$ is less than 0.677, the maximum value measured is not an outlier and, therefore, it must be retained in the calculation of the arithmetic average surface area mercury contamination level.

99. In paragraph 67 of his affidavit, Dr. Mackert asserts that the Warfvinge (1995; Exhibit 64) article is an inappropriate basis from which to judge the pre-clean up concentrations of mercury in Dr. Barnes' dental office. In fact, this may be true because Dr. Barnes' efforts at clean up far exceeded those reported by Warfvinge (1995) (see Exhibit 65) and, therefore, using the Warfvinge article likely under-estimates the pre-clean up levels in Dr. Barnes' office. As a result of relying on the Warfvinge article I likely underestimated

Dr. Barnes' mercury exposure, not overestimated it.

## IX. CONSERVATISM IN THE ASSESSMENT OF DR. BARNES' EXPOSURE

100. Far from grossly over-estimating Dr. Barnes' mercury exposure, as suggested by Kerr's experts, Dr. Richardson's exposure assessment for Dr. Barnes observed an number of conservative assumptions that would serve to under-estimate his true exposure. First, as mentioned in paragraph 77, above, the concentration of mercury vapor reported by Richards & Warren (1985; Exhibit 53) during amalgam removal was actually greater than 100 $\mu g/m^3$, but Richardson set an arbitrary maximum limit of 100 $\mu g/m^3$ in order to be conservative.

101. Richardson ignored the venting of the chair-side evacuation system to the building mechanical room in Dr. Barnes' office building. Evacuation systems (both high volume and low volume (saliva extractor)) collect amalgam particles from the mouth during amalgam removals. The air drawn by these evacuation systems must be vented and is known to contain mercury vapor at measured levels averaging 92 $\mu g/m^3$ and ranging up to 10 times the occupational health limit of 25 $\mu g/m^3$ (Rubin and Yu, 1996, Exhibit 66; Stonehouse and Newman, 2001, Exhibit 67). If this air is vented to the interior of the clinic building, it would contribute significantly to general office mercury vapor levels. Omitting this from the exposure assessment would under-estimate total in-office mercury exposure.

102. Richardson assumed that the average amalgam filling removed by Dr. Barnes was the same size as those removed by Nimmo et al. (1990; Exhibit 13). The standard filling used by Nimmo et al. (1990) contained less than 100 mg of amalgam, whereas the average amalgam filling is much larger. Reinhardt et al. (1983; Exhibit 68) indicate an average

filling size exceeding 1000 mg. Assuming an average amalgam filling size of less than 100 mg under-estimated the mass of particulate generated during a removal procedure and thereby under-estimated Dr. Barnes' actual mercury exposure.

103. Richardson ignored micro-environmental aspects of mercury vapor exposure when estimating exposure from room air contamination. Research (Stopford et al., 1978; Exhibit 69) demonstrates that breathing zone mercury vapor levels are greater than general room air mercury levels due to emissions from contaminated clothing close to the face. Omitting this micro-environmental consideration would under-estimate Dr. Barnes' total in-office mercury exposure.

104. Richardson assumed that Dr. Barnes' inhalation rate was equivalent to the rate of typical, non-occupationally involved adult males. It is recommended for the exposure assessment adults involved in light to moderate levels of exertion that an inhalation rate of 1.0 to 1.6 $m^3$ of air per hour (U.S.EPA, 1997; Exhibit 15). Dr. Barnes' breathing rate was assumed to be only 0.73 $m^3$ per hour, or equivalent to the rate for an adult male involved in non-occupational activities. Use of this lower breathing rate under-estimated Dr. Barnes' actual exposure.

## X. CONCLUSIONS

105. From the forgoing, it is evident that experts for Kerr have not offered any information that demonstrates that the methods employed by me are invalid. They do offer their own models and assumptions to arrive at alternate conclusions, often relying on questionable or misrepresented data and information. Obviously, in their opinion, Dr. Barnes

received virtually no mercury dose at all. Generally, to support their arguments, Kerr's experts employ the same basic methods used by me, thus recognizing their necessity and validity in this case. However, their analysis and interpretation of available data is subjective, they obviously lack familiarity with normal exposure assessment methods, assumptions and their authoritative sources, they fail to point out or recognize the limitations (for exposure assessment purposes) of their own preferred source studies, and even fail to identify key recent articles (such as Exhibit 41 on the absorption of non-volatile metal particulate matter from the lung) that directly contradict their stated conclusions.

106. This all suggests a lack of understanding of exposure assessment methods and the process in general, and at the least merely reflects a differing opinion on the interpretation of that science. However, it does not attack exposure assessment methods or their utility in determining the exposure of an individual to mercury from a severely contaminated environment, said environment no longer existing and not reproducible for ethical reasons.

107. Dr. Werley's failure to identify his own published research that contradicts his stated opinion on the article by Nimmo, Werley et al. (1990; Exhibit 13), and the nature and disposition of particulate matter generated during the removal of amalgam fillings, clearly demonstrates both bias and misdirection with respect to the opinion that has been offered.

108. Dr. Clarkson's opinion that amalgam embedded in soft tissue (that is neither anatomically, physiologically nor microbiologically similar to the lung) is a better model for the fate of inhaled particulate than the study of Cutright et al. (Exhibit 33) is unfounded, and

is not supported by the work of Takenaka et al. (2001) (Exhibit 41) on inhaled silver particles, silver being the most significant component of dental amalgam after mercury. The work of Takenaka et al. clearly supports the results of Cutright et al. in that inhaled metallic particulate matter enters the blood, and will be absorbed in a few days, not 4 years or more. The elemental mercury in dental amalgam, being far more soluble than silver, it is therefore apparent that the mercury contained in that amalgam particulate will be rapidly absorbed into the blood, likely at a rate of 80% or more within three days, as measured by Cutright et al., (Exhibit 33).

109. It is interesting that Mr. Cohen, an industrial hygienist, offers extensive comment on exposure assessment, an area for which he demonstrated no formal training or experience, but offered no opinion on the shortcomings of the TOSHA assessment of mercury vapor in Dr. Barnes' office. Surely, as a trained industrial hygienist, he must have recognized that the Sensidine-Gastec system used by TOSHA, and the method in which it was operated, was inappropriate for measuring the expectedly low mercury vapor levels in Dr. Barnes' office, or any dental office for that matter.

110. Whereas Kerr's other experts do at least provide some comment within their specific areas of expertise, Dr. Mackert, a dental materials scientist, offers comment on all subjects — exposure assessment, industrial hygiene methods for measuring both vapors and particulate matter, and mercury pharmacokinetics and toxicology — except his area of expertise. In fact, his resume provides no evidence of expertise in any of these disciplines. His citations of literature preclude any attempt to ascertain the appropriateness of those

studies for his intended purpose nor the appropriateness of their methods vis-a-vis exposure assessment. He seems primarily intent on suggesting that Dr. Barnes had virtually no exposure at all, similar to his opinion on exposure of dental patients to mercury from amalgam, an opinion which is also out of step with all other scientists that have published on that issue. The available, reliable science does not support Dr. Mackert's opinions.

I declare under penalty of perjury under the laws of the State of Tennessee that the foregoing is true and correct and that this affidavit was executed in Ottawa, Ontario, Canada.

Dated: February 8, 2002

G. Mark Richardson, Ph.D.

Province of Ontario, Canada

_____ ss.

Subscribed and sworn to before me this 8th day of February, 2002.

My commission expires unlimited.

Notary Public within and for

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# EXHIBITS

Exhibit 1: Resume of Dr. G. Mark Richardson

Exhibit 2: Department of Health and Human Services, 42 CFR Part 82. Federal Register, 66(194): 50978 - 50991. Friday, October 5, 2001.

Exhibit 3: Richardson, G. M. 2000. Derivation of Mercury Exposure for Dr. David Barnes. Dated May 12, 2000.

Exhibit 4: Echeverria, D. H.V. Apposhian, J.S. Woods, N.J. Heyer, M.M. Aposhian, A.C. Bittner Jr. and R. K. Mahurin. 1999. Neurobehavioral effects from exposure to dental amalgam $Hg^0$: new distinctions between recent exposure and Hg body burden. In: Amalgam and Health - *New Perspectives on Risks*, FORSKNINGSRÅDSNÄMNDEN (FRN; Swedish Council for Planning and Coordination of Research), Report 99:1, Stockholm, Sweden.

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Exhibit 5: Langan, D.C., P.L. Fan and A.A. Hoos. 1987. The use of mercury in dentistry: a critical review of the recent literature. JADA, 115: 867-880.

Exhibit 6: Casarett and Doull. 1996. Page 608.

Exhibit 7: Satoh H, Hursh JB, Clarkson TW, Suzuki T. 1981. Selective determination of elemental mercury in blood and urine exposed to mercury vapor in vitro. J Appl Toxicol, 1(3):177-181

Exhibit 8: Le Gros, V., D. Lemaigre, C. Suon, J.P. Pozzi, P. Berthaud and F. Liot. 1988. Estimation of pulmonary dust load using magnetic pneumography. Rev. Mal. Respir., 5(6): 601-608.

Exhibit 9: Stahlhofen, W. and W. Moller. 1993. Behavior of magnetic meiro-particles in the human lung. Radiat. Environ. Biophys., 32(3): 221-238.

Exhibit 10: Anonymous. 2001. Mercury, In: The Merck Index, 13th Edition. An Encyclopedia of Chemicals, Drugs, and Biologicals.

Exhibit 11: Berry, T.G., Nicholson, J., Troendle, K. 1994. Almost two centuries with amalgam: Where are we today? *JADA*, 125: 392-399.

Exhibit 12: World Health Organization (WHO). 1991. Inorganic Mercury. Environmental Health Criteria 118. WHO, Geneva. pp. 35-40.

Exhibit 13: Nimmo, A., M.S. Werley, J.S. Martin and M.F. Tansy. 1990. Particulate inhalation during the removal of amalgam restorations. J. Prosthet. Dent., 63: 228-233.

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Exhibit 48: Figure: Comparison of Published estimates of mercury exposure from dental amalgam.

Exhibit 49. List of Health Canada's preferred peer reviewers for the Assessment of Mercury Exposure and Risks from Dental Amalgam.

Exhibit 50. Comments provided by R.J. Mackert to Health Canada regarding the Assessment of Mercury Exposure and Risks from Dental Amalgam.

Exhibit 51. Tabulation of responses to comments provided by R.J. Mackert to Health Canada regarding the Assessment of Mercury Exposure and Risks from Dental Amalgam.

Exhibit 52: World Health Organization (WHO). 1991. Inorganic Mercury. Environmental Health Criteria 118. WHO, Geneva. pp. 49-52.

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Exhibit 62: Performance specifications for the Sensidine-Gastec mercury detection tube, and covering e-mail message from G.M. Richardson to Mr. J. Love, dated May 5, 2000.

Exhibit 63: Sokal, R.R. and F.J. Rohlf. 1981. Biometry: The Principles and Practice of Statistics in Biological Research, 2nd Edition. W.J. Freeman and Company, New York. pp. 412-414.

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Exhibit 67: Stonehouse, CA; Newman, AP. 2001. Mercury Vapour Release From a Dental Aspirator. Brit Dental J. 190(10):558-560.

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# G. MARK RICHARDSON, Ph.D., Risklogic Scientific Services Inc.

EDUCATION B.Sc. (Biology) 1980; M.Sc. (Biology) 1983; Ph.D. (Biology) 1994

## EXPERTISE

Dr. Richardson has extensive experience in risk assessment and has conducted, assisted or peer reviewed a variety of high profile assessments. These include risk assessments for Wawa, Ontario (arsenic), Yellowknife, NWT (arsenic), Port Colborne, Ontario (nickel), Sydney, N.S. Tar Ponds (as an advisor to Health Canada), as well as the 1998 mine tailings catastrophe at Aznacollar, Spain (Boliden, Inc.), considered by some to be the worst mine tailings disaster in European history. He was commissioned by the Department of National Defense to conduct the assessment of risks posed by contaminated soils to Canadian Peace Keepers, as part of the DND Board of Inquiry on Croatia. Risk assessments have been conducted for specific contaminated sites, specific and unique exposure scenarios, and for medical devices and materials. On a number of occasions, he has served as an expert witness on risk assessment for legal proceedings.

Dr. Richardson published the first probabilistic risk assessment of chemical exposures arising from medical devices and materials, having authored Health Canada's *Assessment of Mercury Exposure and Risks from Dental Amalgam*. He also completed and published the first assessment of risks posed by components and degradation products of the family of dental materials known as composite resins. Contributions were also solicited by the Government of Sweden in their review of the potential hazards posed by mercury released from dental materials, and he has lectured in Canada, the U.S.A., Australia and Europe on chemical risks posed by dental materials.

Dr. Richardson was one of six North Americans invited to Kuwait in 1995 to address a special workshop convened to discuss risks and problems posed by the severe crude oil contamination in that country following the Gulf War. Also in that year he toured major cities of Australia, under sponsorship of the Australian federal Department of Human Services and Health, to advise state and federal regulatory agencies on risk assessment methods and programs established in Canada to manage contaminated sites.
Dr. Richardson has trained staff of Health Canada, Environment Canada, Public Works and Government Services Canada, Indian and Northern Affairs Canada, the Louisiana Department of Environmental Quality, other regulatory agencies and private sector consultants in risk assessment methods. Since 1997, Dr. Richardson has instructed graduate level courses in risk assessment for the University of Ottawa's School of Graduate Studies and their Ecotoxicology Program. He has also guest edited a special issue of the journal *Human and Ecological Risk Assessment (HERA)* on the subject of risk assessment.

A significant proportion of Dr. Richardson's work involves risk assessment support to regulatory agencies. For example, he provided primary consulting support to the CCME Development Committee that was charged with establishing human health-based soil quality guidelines for the CCME Canada Wide Standard for petroleum hydrocarbons. Other projects involving government agencies have examined human and/or environmental risks posed by mercury, arsenic, various other heavy metals, PAH, PCB, DDT, petroleum hydrocarbons, as well as human and veterinary drugs.

Between 1989 to 1995, Dr. Richardson was Head of Health Canada's Air and Waste Section, developing procedures for, and conducting, quantitative risk assessments of environmental contaminants, as well as contributing to the CCME National Contaminated Sites Remediation Program, developing national risk-based soil quality guidelines. He is also routinely called upon to provide third party peer review of risk assessments conducted for regulatory and private sector clients for sites and issues throughout Canada.

Dr. Richardson has research experience is a broad range of topics including: risk assessment methods; toxicology, pharmacokinetics; environmental fate of pesticides and environmental contaminants; the biogeochemistry of mercury; dental materials; contaminated soils; and human chemical exposure. He has published the *Compendium of Canadian Human Exposure Factors for Risk Assessment*. This risk assessment resource is the first and only one of its kind in Canada, presenting basic statistics and probability distributions for numerous physical and behavioural characteristics of the Canadian population; data essential to valid and defensible risk assessment, but never before available to the risk assessment community in Canada. This *Compendium* is now being distributed by the University of Waterloo's, Institute for Risk Research.

Dr. Richardson has also established an expertise in biostatistics, metanalysis, and quantitative trend analysis of large and complex data sets. These skills have been developed through the quantitative evaluation and analysis of Canadian population data respecting human tissue chemical analysis (such as mercury levels in blood and hair samples), food consumption statistics, and time-activity patterns. Dr. Richardson has also served as an external reviewer and as a member of site visit committees on behalf of the Natural Sciences and Engineering Research Council of Canada (NSERC), to evaluate the merits of academic research grant proposals in Canada pertaining to mercury.

EXPERIENCE

Nov/98 - present **Risklogic Scientific Services Inc., 14 Clarendon Ave., Ottawa, Ontario CANADA K1Y 0P2**

Director and Risk Assessment Specialist

Founder and director of the company, which specializes in human health and ecological risk assessment, environmental guidelines, data analysis and biostatistics. Conducts risk assessments for chemical exposures arising from medical devices and materials, contaminated sites, and other sources of exposure, as well as multi-media human health exposure assessments for contaminants generally dispersed in the environment. Provides contract support to environmental regulatory programs at the federal and provincial levels. Provides biostatistical consulting and support, particularly relating to statistical analysis and probability density functions for human exposure factors. Conducts training courses in probabilistic risk assessment to regulatory and industrial clients.

Aug/95-Aug/00 **O'Connor Associates Environmental Inc., 14 Clarendon Ave., Ottawa, Ontario CANADA K1Y 0P2**

Senior Risk Assessment Specialist

Responsible for conducting and reviewing risk assessments for chemical exposures arising from medical devices and materials, contaminated sites, and for conducting multi-media human health exposure assessments for contaminants in the environment. Develops and presents, on behalf of OAEI, training courses in probabilistic risk assessment to regulatory and industrial clients. Provides biostatistical support to other risk assessors in the company, particularly relating to probability density functions for human exposure factors. Also responsible for the development and recommendation of environmental regulatory policies and procedures to O'Connor Associates' regulatory clients, both nationally and internationally.

94-Aug/95 **Medical Devices Bureau, Health Protection Branch**
**Health Canada, Ottawa, Ontario CANADA**

Special Assignment

Responsible for preparing the Department's assessment of the exposure and risks posed by mercury arising from dental amalgam.

3-1995 **Air and Waste Section**
1989 - 1991 **Health Canada, Ottawa, Ontario CANADA**

Section Head

Responsible for the development and management of procedures for quantitative risk assessment of environmental contaminants and contaminated soils; provision of expertise in human exposure assessments during chemical fires, spills and other environmental emergency situations; direction of Health Canada's contribution to the National Contaminated Sites Remediation Program; evaluation of the adequacy of risk assessments prepared in support of megaprojects under the Federal Environmental Assessment and Review Process; assessment of risks posed by incinerator stack emissions; and, establishment of a new program within Health Canada on the health risks of hazardous waste, contaminated soils and soil cleanup technologies. This position also involved financial management, project management and management and supervision of professional and support staff

1-1993 **Full-time doctoral candidate, University of Ottawa, Department of Biology**

) **Environmental Health Services, Medical Services Branch**

**Health Canada**

Special Assignment

Responsible for providing expertise in human health risk assessment associated with chemical exposur in northern environments and native diets.

1987-1989 **Environmental Health Directorate**
**Health and Welfare Canada**

Science Advisor

Responsible for providing input, advice and recommendations on the direction, conduct a management of federal R & D programs in environmental and occupational health. Duties includ participation on Federal/Provincial Advisory Committees related to environmental and occupatio health, specifically pertaining to chemical and radiation contamination of air, drinking water, soils a occupational environments.

1985-1987 **Environmental Analysis Branch**
**Environment Canada**

Assessment Specialist

Reviewed and assessed the potential environmental hazards posed by industrial, municipal a institutional activities.

1982-1985 **Urea Formaldehyde Foam Insulation Assistance Program**
**Consumer and Corporate Affairs Canada**

Biologist

Managed contracts for health, biological and chemical research on urea formaldehyde foam insulatio analysed and interpreted indoor air quality and health-related data collected through the UFFI Assistan Program.

## OTHER EXPERIENCE

Other experience includes an extensive knowledge and working ability in: biostatistics; methods of regulatory toxicology, ri assessment and risk management; an extensive knowledge of human exposure factors used in risk assessment; chemical exposur and risks posed by dental materials and other medical devices; development of regulatory risk management processes a environmental policy.

Extensive knowledge and experience in risk assessment and regarding metals in the environment has led to participation as external reviewer and as a site visit committee member on behalf of the Natural Sciences and Engineering Research Council Canada to evaluate the merits of academic research grant proposals in Canada.

## PROFESSIONAL AFFILIATIONS

- Institute for Risk Research, University of Waterloo
- Society for Environmental Toxicology and Chemistry, Laurentian Chapter
- Sigma-Xi Scientific Research Society, Ottawa-Kingston Chapter
- Visiting Scientist, Geologic Survey of Canada

PUBLISHED JOURNAL ARTICLES, BOOK CONTRIBUTIONS AND BOOK REVIEWS

Richardson, G.M., K.E. Clark and D.R. Williams. 1999. Preliminary estimates of adult exposure to bisphenol-a from dental materials, food and ambient air. In: *Environmental Toxicology and Risk Assessment: Standardization of Biomarkers for Endocrine Disruption and Environmental Assessment: Eighth Volume, ASTM STP 1364*, D.S. Henshel, M.C. Black and M.C. Harrass, Eds., American Society for Testing and Materials, West Conshohocken, PA. pp. 286-301.

Richardson, G.M. 1999. Mercury Exposure From Dental Amalgam: Re-evaluation of the Richardson Model, Standardization by Body Surface Area, and Consideration of Recent Occupational Studies. In: Chapter VI. Expert Commissions, *Amalgam and Health - New Perspectives on Risks*, FORSKNINGSRÅDSNÄMNDEN (FRN; Swedish Council for Planning and Coordination of Research), Report 99:1, Stockholm, Sweden.

Richardson, G.M. 1999. Dental Amalgam and Mercury Exposure: Potential Patient Risks and the Basis for Ristrictions on Use. In: Chapter 3, *Amalgam and Health - New Perspectives on Risks*, FORSKNINGSRÅDSNÄMNDEN (FRN; Swedish Council for Planning and Coordination of Research), Report 99:1, Stockholm, Sweden.

Richardson, G.M. 1998. Book Review: How to Control Costs in Your Pollution Prevention Program, by J.A. Cichowicz. *Water, Air and Soil Pollution*, 106: 199-200.

Clark, K.E. and G.M. Richardson. 1998. Debate/Commentary - Fate and exposure models: Selecting the appropriate model for a specific application, Introduction. *J. Soil Contam.*, 7(3): 267-274.

Allan, M. and Richardson, G.M. 1998. Probability Density Functions Describing 24-hour Inhalation Rates for Use in Human Health Risk Assessments. *Human and Ecological Risk Assessment*, Vol. 4(2), 379-408.

Richardson, G.M. 1997. Assessment of adult exposure and risks from components and degradation products of composite resin dental materials. *Human and Ecological Risk Assessment*, 3(4): 683-697.

Richardson, G.M. 1997. Invited Debate/Commentary: Is bioremediation a green technology? Introduction. *J. Soil Contam.*, 6(3): 205-206.

Richardson, G.M. 1997. Invited Debate/Commentary: What research is needed on indoor infiltration of volatile organic contaminants? Introduction. *J. Soil Contam.*, 6(1): 1-2.

Richardson, G.M. and M. Allan. 1996. A Monte Carlo Assessment of Mercury Exposure and Risks from Dental Amalgam. *Human and Ecological Risk Assessment*, 2(4):709-761.

Richardson, G.M. and D.Burmaster (Eds.). 1996. Theoretical, Toxicological and Biostatistical Foundations for Deriving Probability Density Functions Describing Variability and Uncertainty in References Doses, Benchmark Doses and Slope Factors. *Human and Ecological Risk Assessment*, 2(1).

Richardson, G. M. 1996. Deterministic versus Probabilistic Risk Assessment: Strengths and Weaknesses in a Regulatory Context. *Human and Ecological Risk Assessment*, 2(1), 44-54.

Bartlett, S., G.M.Richardson, D. Krewski, S.N.Rai and M.Fyfe. 1996. Commentary: Characterizing uncertainty in risk assessment - conclusions drawn from a workshop. *Human and Ecological Risk Assessment*, 2(1), 221-231.

Williams, D.R., J.C.Paslawski and G.M. Richardson. 1996. Development of a screening relationship to describe migration of contaminant vapours into buildings. *J. Soil Contam.*, 5(2): 141-156.

Richardson, G.M. and D.J. Currie. 1995. Using empirical methods to assess the risks of mercury accumulation in fish from lakes receiving acid rain. *Human and Ecological Risk Assessment*, 1(3): 306-322.

Richardson, G.M., M.Egyed and D.J. Currie. 1995. Does acid rain increase human exposure to mercury? A review and analysis of recent literature. *Environ. Toxicol. Chem.*, volume 14 (5), 809-813.

Richardson, G.M., M.Egyed and D.J. Currie. 1995. Human exposure to mercury may decrease as acidic deposition increases. *Water, Air and Soil Pollution*, 80: 31-39.

Richardson, G.M., M.Mitchell, S.Coad and R.Raphael. 1995. Exposure to mercury in Canada: a multimedia assessment. *Water, Air and Soil Pollution*, 80: 21-30.

Richardson, G.M. and A. Myers. 1993. Risk Assessment and its Application to Risk Management in Environmental Health. *Environmental Health Review*, 37(2): 48-55.

Richardson, G.M. and D.J. Currie. 1993. Estimating Fish Consumption Rates for Ontario Amerindians. *J. Expos. Anal. Environ. Epidemiol.*, 3(1): 23-38.

Murray, W.D. and M. Richardson. 1993. Development of Biological and Process Technologies for the Reduction and Degradation of Pulp Mill Wastes That Pose a Threat to Human Health. *Critical Reviews in Environmental Science and Technology*, 23(2): 157-194.

Murray, W. and M. Richardson. 1993. Progress Toward the Biological Treatment of C1 and C2 Halogenated Hydrocarbons. *Critical Reviews in Environmental Science and Technology*, 23(3): 195-217.

Jessiman, B., G.M. Richardson, C. Clark and B. Halbert. 1992. A Quantitative Evaluation of Ten Approaches to Setting Site-Specific Cleanup Objectives. *J. Soil Contamin.*, 1(1): 39-59.

Richardson, G.M. 1991. Contaminated Sites: An Update of Health Protection Activities at the National Level. *Environ. Health Rev.*, 35(4): 88-93.

Bourdeau, P., E. Somers, G.M. Richardson and J.R. Hickman (Eds.) 1990. *Short-Term Toxicity Tests for Non-Genotoxic Effects*. SCOPE 41, SGOMSEC 4, IPCS Joint Symposia 8. John Wiley and Sons, Chichester.

Richardson, G.M. and S.U. Qadri. 1987. Extraction of Aminocarb and a Metabolite from Whole Fish and Derivatization For Electron-Capture Gas Chromatography. *J. Agric. Food Chem.*, 35(6): 877-878.

Richardson, G.M. and S.U. Qadri. 1986. Tissue distribution of $^{14}$C-Labelled Residues of Aminocarb in Brown Bullhead (*Ictalurus nebulosus* Le Sueur) Following Acute Exposure. *Ecotoxicol. Environ. Safety*, 12: 180-186.

Richardson, G.M., S.U. Qadri and B. Jessiman. 1983. Acute toxicity, Uptake and Clearance of Aminocarb by the Aquatic Isopod, *Caecidolea racovitzai racovitzai*. *Ecotoxicol. Environ. Safety*, 7: 552-557.

Richardson, G.M. and S.U. Qadri. 1982. Acute Toxicity, Kinetics and Metabolism of Aminocarb in the Brown Bullhead (*Ictalurus nebulosus*). *Water Poll. Res. J. Canada*, 17: 153-158.

**INVITED LECTURES, COURSES, CONFERENCE PRESENTATIONS AND PROCEEDINGS**

Richardson, G.M. 2001. Arsenic-contaminated soil: an overview of arsenic's potential health risks and the risk assessment process in Canada. Presented on behalf of the Yellowknife Arsenic Soil Remediation Committee, Yellowknife, NWT, May 15, 2001.

Richardson, G.M. 2001. Human health-based soil quality guidelines for PHC: an overview of their basis and derivation. Presented at: CCME Workshop on the Canada-wide Standards for Petroleum Hydrocarbons in Soil (PHC CWS), Calgary, AB, May 10-11, 2001.

Richardson, G.M. 2001. Human health risk assessment: an overview of methods applied in Canada. 3rd Annual Conference/Workshop on Realty Asset and Environment, Department of National Defense, ADM(IE), Cornwall, ON, February 18-23, 2001.

Garrett, R.G., J. Pacyna, **G. M.Richardson,** and M. Mah-Paulson. 2000. Industrial and Atmospheric Sources of Cadmium into the Food Chain. Presented at: SCOPE Workshop on Environmental Cadmium in the Food Chain: Sources, Pathways, and Risks, International Council for Science (ICSU), Brussels on 13-16 September, 2000

Richardson, G.M. 2000. Patient exposures to toxic chemicals from dental materials. Presented at Odenth 2000: 5ème Congrès International, la Baule, France, June 1-3, 2000.

Richardson, G.M. and D. Currie. 2000. Will reduced atmospheric mercury emissions lead to reduced fish contamination in Canadian lakes? Presented at: Annual meeting of the Laurentian Chapter, Society of Environmental Toxicology and Chemistry, Carleton University, Ottawa, ON, May 12-13, 2000.

Richardson, G.M., M. Mah-Paulson, K.E. Clark and D.R. Williams. 2000. Boxes, cylinders and parallel plates: an examination of current methods employed to predict indoor infiltration of volatile soil contaminants. Presented at: Atlantic Canada Environmental Business and Municipal Expo, Halifax, N.S. May 25 - 28, 2000.

Richardson, G.M. 1999. Human Health and Ecological Risk Assessment: A Short Course on Methods Applied in Canada. Principle instructor for course offered in association with the University of Ottawa, Ecotoxicology Program. University of Ottawa, Ottawa, Ontario. August 16-19, 1999.

Richardson, G.M. 1999. Mercury, still with us after all these years; or Dental amalgam, mercury exposure and dental materials safety. Invited lecture presented at the 5th Annual Environmental Symposium, Queen's University, Kingston, Ontario. January 30, 1999.

Richardson, G.M. 1998. Instructor: Environmental Risk Assessment. BIO8100C, School of Graduate Studies, University of Ottawa. Ottawa, Ontario. Sept.-Dec., 1998.

Richardson, G.M. 1996.Should there be concern regarding mercury exposure from dental amalgam? Seminar offered by O'Connor Associates Environmental Inc., Toronto, Ontario. December 4, 1996.

Richardson, G.M. 1996.Should there be concern regarding mercury exposure from dental amalgam? Seminar organized by the Association of Holistic Dentists of Quebec, Montreal, Quebec. November 19, 1996.

Richardson, G.M. 1996. An Introduction to Probabilistic Exposure Assessment. Presented at the 11th Annual Conference on Contaminated Soils, Association for the Environmental Health of Soils (AEHS), Amherst, MA, October 21-24, 1996.

Richardson, G.M. 1996. Should there be concern regarding mercury exposure from dental amalgam. Invited lecture to the International Academy of Oral Medicine and Toxicology. Houston, Texas, September 28-30, 1996.

Richardson, G.M. 1996. Introduction to deterministic and probabilistic risk assessment. Presented at Contaminated and Hazardous Waste Site Management, GOwen Environmental Ltd. and the Association for Environmental Health of Soils. Toronto, Ontario. September 26, 1996.

Richardson, G.M. 1996.Should there be concern regarding mercury exposure from dental amalgam? Seminar offered by O'Connor Associates Environmental Inc., Ottawa, Ontario. August 28, 1996.

Richardson, G.M. 1996. Comparative dietary risks: Risks in perspective. Presented at the Federal Forum on Contaminants in Fish. Convened by the U.S.EPA and the American Fisheries Society, Alexandria, VA. June 9-11, 1996.

Richardson, G.M., K.E. Clark, J.C. Paslawski, and D.R. Williams. 1996. Assessing the uncertainty in uncertainty analysis: application of stochastic uncertainty analysis to exposure assessment. Proceedings of the 3rd National Hazardous and Solid Waste Convention, Sydney, Australia, May 26-30, 1996, p.730-735.

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Richardson, G.M., M. Mitchell, S. Coad and R. Raphael. 1994. Exposure to Mercury in Canada: a Multimedia Assessment. Presented at the International Conference on Mercury as a Global Pollutant, Whistler, B.C. July 10-14, 1994.

where awareness of the hazards posed by mercury vapor inhalation have been well known and well publicized for many years. Dr. Barnes was not the beneficiary of such awareness.

80. Also, the personal sampling device used was operated at a volumetric air flow rate of 8 liters per minute. As previously discussed, this air flow rate falls well below the measured inhalation rates of persons involved in light to moderate physical exertion and is almost 3 times below US EPA's recommended inhalation rate for exposure assessment of adults involved in light to moderate activity.

81. Finally, the sampling equipment was equipped "with a Y-formed orifice at nose level". Given that air was drawn through this orifice at a constant rate, it would be drawing air from the dentist's own exhalations, not just from the air that he/she would be breathing it. When inhaled, mercury vapor is rapidly and almost completely absorbed (WHO, 1991, Exhibit 38). Therefore, during exhalations, the device was sampling virtually mercury-free air.

82. Based on comments offered above, it is apparent that the most preferable approach to estimating exposure to mercury vapor by Dr. Barnes is to rely on continuous measurements of mercury vapor in the vicinity of the dentist during amalgam removal. That data best reflects the environment to which Dr. Barnes was exposed, and can then be combined with information on inhalation rate and mercury vapor absorption to properly estimate Dr. Barnes' mercury vapor exposure. This is precisely what was done by Richardson.

83. In paragraph 36, Dr. Mackert attempts to further claim that the measurements

made by Richards & Warren were inaccurate due to deficiencies in the mercury vapor detector employed by Richards & Warren. The device they used, the Bacharach MV2 mercury vapor detector, is a recommended device by the Occupational Safety and Health Administration, US Department of Labor (Exhibit 56). Therefore, I fail to see the problem with the device, particularly since Kerr's other expert, Mr. Cohen, emphasized the need to employ approved industrial hygiene measurement methods. Therefore, the measurements of Richards & Warren were made using standard, approved industrial hygiene equipment and are fully valid and reliable.

84. With respect to concentration of mercury vapor reported by Richards & Warren, that mercury vapor level actually exceeded 100 $\mu g/m^3$ during amalgam removal. However, Richardson set an arbitrary maximum limit of 100 $\mu g/m^3$ in order to be conservative in the estimates of mercury exposure in Dr. Barnes.

85. In paragraph 39, Dr. Mackert states "contrary to Dr. Richardson's assertions ... it is not common practice in the scientific community to ignore other published data and cite only one source as a representative example..". Quite to the contrary, instructions to contributors for the journals *FASEB* and *Nature* are attached as Exhibits 57 and 58, respectively. These are just two examples of journals' desire to keep papers brief and succinct.

86. Instructions from *FASEB* state "For **Review Articles**, literature citations of earlier findings should be selective rather than encyclopedic.". *Nature* specifies that the maximum number of references permitted is 50 for Articles and 30 for Letters.

87. The nature of scientific writing is to be concise. Therefore, from Exhibits 57 and 58, it is very apparent that the normal scientific writing style is to use citations with economy, avoiding the unnecessary citation of each and every possible example when one or two will suffice. This is precisely the style of writing that Richardson practiced with respect to Dr. Barnes' exposure assessment report.

88. In paragraphs 41 and 42, Dr. Mackert asserts that Richardson somehow misquoted or misrepresented the results of an article by Haikel et al. (1990; Exhibit 59). This was not the case. The fact that an amalgam placement releases mercury vapor does not appear to be disputed by Dr. Mackert. The article by Haikel et al. was cited only as qualitative evidence of vapor release during placements and not as a basis for quantifying those mercury vapor levels. Dr. Mackert is quite correct in stating that the Haikel et al. article would be inappropriate for that purpose.

89. In paragraphs 44 through 51, Dr. Mackert is simply repeating the same arguments offered by Kerr's expert Dr. Werley. Responses to Dr. Werley's comments were provided above in paragraphs 32 to 40. For the sake of brevity, those comments will not be repeated here. In essence, Dr. Werley's own research supports the manner in which Richardson employed the data from the Nimmo, Werley et al. (1990) study.

90. In paragraphs 52 through54 of his affidavit, Dr. Mackert suggests that a study by Musajo et al. (1988; Exhibit 60) is a preferable alternative basis for assessing the intake of particles. However, if Dr. Mackert were familiar with exposure assessment methods and authoritative sources, particularly pertaining to human inhalation, he would know that the

air flow rate of 3 liters per minute used by Musajo et al. is far too low to represent or mimic adult inhalation rate. In fact, extensive inhalation rate data reviewed by the US EPA (Exhibit 15) suggests that an inhalation rate of at least 10 liters per minute (15 $m^3$/24 hours) is required just to support basal metabolism in an adult male. As indicated by the US EPA (Exhibit 15), and discussed earlier in paragraph 27, the inhalation rates for adult males involved in light to moderate activity range up to 41 liters per minute, a rate that is far in excess of the air flow rate employed by Musajo et al. (1985). Therefore, the Musajo et al. study is certainly faulty with respect to providing representative particulate intakes for dentists.

91. It is also noted that, in the Musajo et al. study, particulate samples were collected on filters which had a limited capacity to collect submicron particulate matter. The filters collected particulate matter in the range of 0.5 to 5 μm in aerodynamic diameter. Because the air flow rate of the collection system was so low, it would preferentially collect only the smallest particles, since the flow rate could only overcome the gravitational force on these smallest particles. Gravity causes particles to settle out of the air and must be overcome by particle collection systems. With a large proportion of amalgam particulate being less than 0.5 μm in size (Brune et al., 1980, Exhibit 19; Cupelin et al., 1986, Exhibit 20) and with the Musajo et al. collection method failing to collect particles of less than 0.5 μm, then the measurements grossly under-estimate the true air-borne particulate concentration. This problem is not encountered with an Andersen Impactor, however, (as used by Nimmo et al., 1990; Exhibit 13) which collects all particles less than 10 μm, irrespective of particle size.

For these reasons, the Musajo et al. study is totally inappropriate for estimating particulate inhalations in dentists.

92. It is appropriate to point out at this stage that Dr. Mackert agrees with the methods employed by Dr. Richardson in the assessment of particulate exposure. He has applied the same basic methods. Therefore, his only complaint appears to be the selection of the study from which to obtain data to input to the model, not the model itself.

93. In paragraphs 55 through 60 of his affidavit, Dr. Mackert merely repeats the same criticisms offered by Dr. Clarkson, another of Kerr's expert witnesses. As Dr. Mackert is not an expert in the absorption or pharmacokinetics of contaminants in general nor mercury specifically, nor is he a physiologist or toxicologist, his comments fall well outside his area of expertise. Please refer to paragraphs 41 to 61 above which address all of the issues raised by Dr. Clarkson that were repeated by Dr. Mackert.

94. In paragraphs 61 to 67, Dr. Mackert again offers comments in areas well outside his area of expertise. The comments offered merely repeat those offered by Kerr's expert Mr. Cohen. Those criticisms have been commented on above in paragraphs 21 to 31, and will not be repeated here, for the sake of brevity. However, there are three specific points that require comment.

95. First, Dr. Mackert does make the comment in paragraph 61 of his affidavit that Richardson cited no established procedure for estimating air-borne mercury vapor concentrations in Dr. Barnes' dental office based in surface area contamination. Therefore, I have appended Exhibit 61 an article by Sverdrup, Warfvinge and Sverdrup (1990) which

employs essentially the same methodology to estimate mercury levels in the air of a hypothetical bedroom as a result of spilled mercury. This methodology is not unique or non-existent. Such models and calculations are common practice to exposure and risk assessors.

96. Second, Dr. Mackert claims in paragraph 64 that the Tennessee Occupational Safety and Health Agency (TOSHA) failed to detect mercury vapor in Dr. Barnes' dental operatory when they visited the office following Dr. Barnes' thorough clean up of his office. The TOSHA method for attempting to measure mercury vapor used a Sensidine-Gastec mercury detection tube connected to a hand-operated pump. TOSHA personnel operated that hand pump for only 3 pumps of air through the detection tube. The detection limit for this device with only 3 pump strokes is 150 μg of mercury vapor in 1 cubic meter of air (ug/$m^3$) (Exhibit 62). However, the post-clean up levels of mercury vapor in Dr. Barnes' office would only have been approximately 3 μg/$m^3$ to 8 μg/$m^3$ (Exhibit 3). Therefore, the detection limit of the TOSHA mercury vapor sampling method was some 20 to 50 times too high to detect the probable level of mercury in Dr. Barnes' office at the time of TOSHA sampling. Therefore, this TOSHA result in no way suggests that no mercury vapor was present after Dr. Barnes decontaminated his office; it simply indicates that mercury levels were below 150 μg/$m^3$, a fact with which I agree.

97. Third, Dr. Mackert claims in paragraph 62 of his affidavit that the highest concentration of mercury measured on the surfaces of Dr. Barnes' office should be omitted as "an outlier". To support this notion, Dr. Mackert attempts to conduct a statistical test known as Dixon's test (presented in Mackert affidavit, exhibit 3a). Unfortunately, Dr.

Mackert has applied this test incorrectly. The underlying assumption for this test for outliers is that the remaining data (all other data excluding the purported outlier) are normally distributed (see Sokal and Rohlf, 1984, pp 412-413; Exhibit 63). Where those data are not normally distributed, they must first be mathematically 'transformed' to achieve normality.

98. The data in question, the measurements of surface area mercury contamination in Dr. Barnes' office, are not normal (Lilliefors test, $p<<0.0005$; a value of $p \leq 0.05$ indicates that the data are not normal). However, when the logarithms of the data are determined, which is the customary statistical transformation for all concentration data, the data are now normally distributed (Lilliefors test, $p=0.073$; since $p>0.05$, then the hypothesis that the data are different from a normal distribution is rejected and the data are concluded to be normal). It is essential that Dixon's test be performed on these logarithmically-transformed values for the test to be valid statistically. When this is done, the Dixon test achieves a value for the parameter $r_{11} = 0.237$. Since this value for $r_{11}$ is less than 0.677, the maximum value measured is not an outlier and, therefore, it must be retained in the calculation of the arithmetic average surface area mercury contamination level.

99. In paragraph 67 of his affidavit, Dr. Mackert asserts that the Warfvinge (1995; Exhibit 64) article is an inappropriate basis from which to judge the pre-clean up concentrations of mercury in Dr. Barnes' dental office. In fact, this may be true because Dr. Barnes' efforts at clean up far exceeded those reported by Warfvinge (1995) (see Exhibit 65) and, therefore, using the Warfvinge article likely under-estimates the pre-clean up levels in Dr. Barnes' office. As a result of relying on the Warfvinge article I likely underestimated

Dr. Barnes' mercury exposure, not overestimated it.

## IX. CONSERVATISM IN THE ASSESSMENT OF DR. BARNES' EXPOSURE

100. Far from grossly over-estimating Dr. Barnes' mercury exposure, as suggested by Kerr's experts, Dr. Richardson's exposure assessment for Dr. Barnes observed an number of conservative assumptions that would serve to under-estimate his true exposure. First, as mentioned in paragraph 77, above, the concentration of mercury vapor reported by Richards & Warren (1985; Exhibit 53) during amalgam removal was actually greater than 100 $\mu g/m^3$, but Richardson set an arbitrary maximum limit of 100 $\mu g/m^3$ in order to be conservative.

101. Richardson ignored the venting of the chair-side evacuation system to the building mechanical room in Dr. Barnes' office building. Evacuation systems (both high volume and low volume (saliva extractor)) collect amalgam particles from the mouth during amalgam removals. The air drawn by these evacuation systems must be vented and is known to contain mercury vapor at measured levels averaging 92 $\mu g/m^3$ and ranging up to 10 times the occupational health limit of 25 $\mu g/m^3$ (Rubin and Yu, 1996, Exhibit 66; Stonehouse and Newman, 2001, Exhibit 67). If this air is vented to the interior of the clinic building, it would contribute significantly to general office mercury vapor levels. Omitting this from the exposure assessment would under-estimate total in-office mercury exposure.

102. Richardson assumed that the average amalgam filling removed by Dr. Barnes was the same size as those removed by Nimmo et al. (1990; Exhibit 13). The standard filling used by Nimmo et al. (1990) contained less than 100 mg of amalgam, whereas the average amalgam filling is much larger. Reinhardt et al. (1983; Exhibit 68) indicate an average

filling size exceeding 1000 mg. Assuming an average amalgam filling size of less than 100 mg under-estimated the mass of particulate generated during a removal procedure and, thereby under-estimated Dr. Barnes' actual mercury exposure.

103. Richardson ignored micro-environmental aspects of mercury vapor exposure when estimating exposure from room air contamination. Research (Stopford et al., 1978; Exhibit 69) demonstrates that breathing zone mercury vapor levels are greater than general room air mercury levels due to emissions from contaminated clothing close to the face. Omitting this micro-environmental consideration would under-estimate Dr. Barnes' total in-office mercury exposure.

104. Richardson assumed that Dr. Barnes' inhalation rate was equivalent to the rate of typical, non-occupationally involved adult males. It is recommended for the exposure assessment adults involved in light to moderate levels of exertion that an inhalation rate of 1.0 to 1.6 $m^3$ of air per hour (U.S.EPA, 1997; Exhibit 15). Dr. Barnes' breathing rate was assumed to be only 0.73 $m^3$ per hour, or equivalent to the rate for an adult male involved in non-occupational activities. Use of this lower breathing rate under-estimated Dr. Barnes' actual exposure.

## X. CONCLUSIONS

105. From the forgoing, it is evident that experts for Kerr have not offered any information that demonstrates that the methods employed by me are invalid. They do offer their own models and assumptions to arrive at alternate conclusions, often relying on questionable or misrepresented data and information. Obviously, in their opinion, Dr. Barnes

received virtually no mercury dose at all. Generally, to support their arguments, Kerr's experts employ the same basic methods used by me, thus recognizing their necessity and validity in this case. However, their analysis and interpretation of available data is subjective, they obviously lack familiarity with normal exposure assessment methods, assumptions and their authoritative sources, they fail to point out or recognize the limitations (for exposure assessment purposes) of their own preferred source studies, and even fail to identify key recent articles (such as Exhibit 41 on the absorption of non-volatile metal particulate matter from the lung) that directly contradict their stated conclusions.

106. This all suggests a lack of understanding of exposure assessment methods and the process in general, and at the least merely reflects a differing opinion on the interpretation of that science. However, it does not attack exposure assessment methods or their utility in determining the exposure of an individual to mercury from a severely contaminated environment, said environment no longer existing and not reproducible for ethical reasons.

107. Dr. Werley's failure to identify his own published research that contradicts his stated opinion on the article by Nimmo, Werley et al. (1990; Exhibit 13), and the nature and disposition of particulate matter generated during the removal of amalgam fillings, clearly demonstrates both bias and misdirection with respect to the opinion that has been offered.

108. Dr. Clarkson's opinion that amalgam embedded in soft tissue (that is neither anatomically, physiologically nor microbiologically similar to the lung) is a better model for the fate of inhaled particulate than the study of Cutright et al. (Exhibit 33) is unfounded, and

is not supported by the work of Takenaka et al. (2001) (Exhibit 41) on inhaled silver particles, silver being the most significant component of dental amalgam after mercury. The work of Takenaka et al. clearly supports the results of Cutright et al. in that inhaled metallic particulate matter enters the blood, and will be absorbed in a few days, not 4 years or more. The elemental mercury in dental amalgam, being far more soluble than silver, it is therefore apparent that the mercury contained in that amalgam particulate will be rapidly absorbed into the blood, likely at a rate of 80% or more within three days, as measured by Cutright et al., (Exhibit 33).

109. It is interesting that Mr. Cohen, an industrial hygienist, offers extensive comment on exposure assessment, an area for which he demonstrated no formal training or experience, but offered no opinion on the shortcomings of the TOSHA assessment of mercury vapor in Dr. Barnes' office. Surely, as a trained industrial hygienist, he must have recognized that the Sensidine-Gastec system used by TOSHA, and the method in which it was operated, was inappropriate for measuring the expectedly low mercury vapor levels in Dr. Barnes' office, or any dental office for that matter.

110. Whereas Kerr's other experts do at least provide some comment within their specific areas of expertise, Dr. Mackert, a dental materials scientist, offers comment on all subjects — exposure assessment, industrial hygiene methods for measuring both vapors and particulate matter, and mercury pharmacokinetics and toxicology — except his area of expertise. In fact, his resume provides no evidence of expertise in any of these disciplines. His citations of literature preclude any attempt to ascertain the appropriateness of those

studies for his intended purpose nor the appropriateness of their methods vis-a-vis exposure assessment. He seems primarily intent on suggesting that Dr. Barnes had virtually no exposure at all, similar to his opinion on exposure of dental patients to mercury from amalgam, an opinion which is also out of step with all other scientists that have published on that issue. The available, reliable science does not support Dr. Mackert's opinions.

I declare under penalty of perjury under the laws of the State of Tennessee that the foregoing is true and correct and that this affidavit was executed in Ottawa, Ontario, Canada.

Dated: February 8, 2002

G. Mark Richardson, Ph.D.

Province of Ontario, Canada

_____ ss.

Subscribed and sworn to before me this 8th day of February, 2002.

My commission expires unlimited.

Notary Public within and for

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# EXHIBITS

Exhibit 1: Resume of Dr. G. Mark Richardson

Exhibit 2: Department of Health and Human Services, 42 CFR Part 82. Federal Register, 66(194): 50978 - 50991. Friday, October 5, 2001.

Exhibit 3: Richardson, G. M. 2000. Derivation of Mercury Exposure for Dr. David Barnes. Dated May 12, 2000.

Exhibit 4: Echeverria, D. H.V. Apposhian, J.S. Woods, N.J. Heyer, M.M. Aposhian, A.C. Bittner Jr. and R. K. Mahurin. 1999. Neurobehavioral effects from exposure to dental amalgam $Hg^0$: new distinctions between recent exposure and Hg body burden. In: Amalgam and Health - *New Perspectives on Risks*, FORSKNINGSRÅDSNÄMNDEN (FRN; Swedish Council for Planning and Coordination of Research), Report 99:1, Stockholm, Sweden.

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Ritchie, K.A., E.B. Macdonald, R. Hammersley, J.M. O'Neil, D.A. McGowan, I.M. Dale and K. Wesnes. 1995. A pilot study of the effect of low level exposure to mercury on the health of dental surgeons. *Occup. Environ. Med.*, 52, 813-817.

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Exhibit 5: Langan, D.C., P.L. Fan and A.A. Hoos. 1987. The use of mercury in dentistry: a critical review of the recent literature. JADA, 115: 867-880.

Exhibit 6: Casarett and Doull. 1996. Page 608.

Exhibit 7: Satoh H, Hursh JB, Clarkson TW, Suzuki T. 1981. Selective determination of elemental mercury in blood and urine exposed to mercury vapor in vitro. J Appl Toxicol, 1(3):177-181

Exhibit 8: Le Gros, V., D. Lemaigre, C. Suon, J.P. Pozzi, P. Berthaud and F. Liot. 1988. Estimation of pulmonary dust load using magnetic pneumography. Rev. Mal. Respir., 5(6): 601-608.

Exhibit 9: Stahlhofen, W. and W. Moller. 1993. Behavior of magnetic meiro-particles in the human lung. Radiat. Environ. Biophys., 32(3): 221-238.

Exhibit 10: Anonymous. 2001. Mercury, In: The Merck Index, 13th Edition. An Encyclopedia of Chemicals, Drugs, and Biologicals.

Exhibit 11: Berry, T.G., Nicholson, J., Troendle, K. 1994. Almost two centuries with amalgam: Where are we today? *JADA*, 125: 392-399.

Exhibit 12: World Health Organization (WHO). 1991. Inorganic Mercury. Environmental Health Criteria 118. WHO, Geneva. pp. 35-40.

Exhibit 13: Nimmo, A., M.S. Werley, J.S. Martin and M.F. Tansy. 1990. Particulate inhalation during the removal of amalgam restorations. J. Prosthet. Dent., 63: 228-233.

Exhibit 14: Andersen Instruments. http://www.anderseninstruments.com/

Exhibit 15: US Environmental Protection Agency. 1997. Exposure Factors Handbook, Chapter 5: Inhalation. US EPA report EPA/600/P-95/002Fa. http://www.epa.gov/ncea/pdfs/efh/sect5.pdf

Exhibit 16: Nimmo, A., M.S. Werley, J.S. Marin and M.F. Tansy. 1988. Particulate inhalation during the removal of amalgam restorations. Abstract 1776, In: Annual Meeting of the American Association for Dental Research, Abstract of Papers, Journal of Dental Research, 1988.

Exhibit 17: Nimmo, A., M.S. Werley, M.F. Tansy and J.S. Marin. 1989a. Profile of respirable particulates produced during amalgam removal. Abstract 334, In: Annual Meeting of the American Association for Dental Research, Abstract of Papers, Journal of Dental Research, 1989.

Exhibit 18: Werley, M.S., E. Holland Jr., A. Nimmo, and M.F. Tansy. 1990. Profile of respirable particles produced during the removal of amalgam restorations. Abstract 4C.3, In: Annual Meeting of the American Association for Aerosol Research, Abstract Book, June 18-22, 1990.

Exhibit 19: Brune, D., H. Beltesbrekke and G. Strand. 1980. Dust in dental laboratories. Part II: Measurement of particle size distributions. Journal of Prosthetic Dentistry, 44(1): 82-87.

Exhibit 20: Cupelin, F., M. Callmander, J.-C. Landry, and J.M. Meyer. 1986. Rejets d'amalgame dans les eaux usées d'un cabinet dentaire. [Amalgam discharge into wastewater from a dental cabinet]. *Trav. chim. aliment. hyg.*, 77: 39-47.

Exhibit 21: American Conference of Governmental Industrial Hygienists (ACGIH). 2001. TLVs and BEIs. Tables on particle deposition by particle size.

Exhibit 22: Owen, M.K., D.S. Ensor and L.E. Sparks. 1992. Airborne particle sizes and sources found in indoor air. Atmospheric Environment, 26A(12): 2149-2162.

Exhibit 23: Christensen, R.P., R.A. Robison, D.R. Robinson, B.J. Ploeger and R.W. Leavitt. 1991. Efficiency of 42 brands of face masks and 2 face shilds in preventing inhalation of airborne debris. General Dentistry, 39(6): 414-421.

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Exhibit 26: Naleway, C., R. Sakaguchi, E. Mitchell, T. Muller, W.A. Ayer and J.J. Hefferren. 1985. Urinary mercury levels in US dentists, 1975-1983: review of health assessment program. JADA, 111: 37-42.

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Exhibit 28: Gross, M.J. and J. A. Harrison. 1989. Some electrochemical features of the in vivo corrosion of dental amalgam. J. Appl. Electrochem., 19: 301-310.

Exhibit 29: Lorscheider,F.L., Vimy, M.J. and Summers, A.O. 1995. Mercury exposure from "silver" tooth fillings: emerging evidence questions a traditional dental paradigm. *FASEB J.*, 9, 504-508.

Exhibit 30: Hamm, H., H. Fabel and W. Bartsch. 1992. The surfactant system of the adult lung: physiology and clinical perspectives. Clin. Investig., 70(8): 637-657.

Exhibit 31: Guthmann, F., R. Haupt, M. Schlame, P.A. Stevens and B. Rustow. 1995. Alveolar surfactant subfractions differ in their lipid composition. Int. J. Biochem. Cell. Biol., 27(10): 21-26.

Exhibit 32: Letzel, H., F.A. de Boer and M.A. Van'T Hof. 1997. An estimation of the size distribution of amalgam particles in dental treatment waste. *J. Dent. Res.*, 76(3): 780-788.

Exhibit 33: Cutright, D.E., R.A. Miller, G.C. Battistone and L.J. Millikan. 1973. Systemic mercury levels caused by inhaling mist during high-speed amalgam grinding. *J. Oral Med.*, 28(4): 100-104.

Exhibit 34: Eley, B.M. 1990. A study of mercury redistribution, excretion and renal pathology in guinea-pigs implanted with powdered dental amalgam for between 2 and 4 years. J. Exp. Path., 71: 375-393.

Exhibit 35: Ansoborlo, E., M.H. Hengé-Napoli, V. Chazel, R. Gibert and R.A. Guilmette. 1999. Review and critical analysis of available in vitro dissolution tests. Health Physics, 77(6): 638-645.

Exhibit 36: Cassarett and Doull's Toxicology: The Basic Science of Poisons, 5th Edition. 1996. McGraw-Hill, New York. pp. 97-99, 447-450, 709-712, 870.

Exhibit 37: Khayat, A. and L. Dencker. 1983. Interactions between selenium and mercury in mice: marked retention in the lung after inhalatiuon of metallic mercury. Chem. Biol. Interact., 46(3): 283-298.

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Exhibit 39: US Department of Energy. 2000. DOE Standard: Guide of Good Practices for Occupational Radiological Protection in Uranium Facilities. The document is too voluminous to supply but may be found at http://tis.eh.doe.gov/whs/rhmwp/utotal.pdf

Exhibit 40: Parsont, M.A., W.L. Holley and W.D. Burnett. 1972. The effect of particle size on organ distribution of radioactive material deposited in the lungs. Health Physics, 22: 143-148.

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Exhibit 42: US Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Silver; Table 3-7: Chemical and Physical Properties of Silver.

Exhibit 43: US Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for Mercury, Table 3-2.

Exhibit 44: Kozono, Y., B.K. Moore, R.W. Phillips and M.L. Swartz. 1982. Dissolution of amalgam in saline solution. J. Biomed. Mater. Res., 16: 767-774.

Exhibit 45: Mackert, J.R. Jr. 1987. Factors affecting estimation of dental amalgam mercury exposure from measurements of mercury vapor levels in intra-oral and expired air. J. Dent. Res., 66(12): 1775-1780.

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Exhibit 47: Mackert, J.R. Jr. and A. Berglund. 1997. Mercury exposure from dental amalgam fillings: absorbed dose and the potential for adverse health effects. Crit Rev Oral Biol Med., 8(4):410-436.

Exhibit 48: Figure: Comparison of Published estimates of mercury exposure from dental amalgam.

Exhibit 49. List of Health Canada's preferred peer reviewers for the Assessment of Mercury Exposure and Risks from Dental Amalgam.

Exhibit 50. Comments provided by R.J. Mackert to Health Canada regarding the Assessment of Mercury Exposure and Risks from Dental Amalgam.

Exhibit 51. Tabulation of responses to comments provided by R.J. Mackert to Health Canada regarding the Assessment of Mercury Exposure and Risks from Dental Amalgam.

Exhibit 52: World Health Organization (WHO). 1991. Inorganic Mercury. Environmental Health Criteria 118. WHO, Geneva. pp. 49-52.

Exhibit 53: Richards and Warren. 1985. Mercury vapour released during the removal of old amalgam restorations. British Dental Journal, 159(7): 231-232.

Exhibit 54: Powell, L.V., G. H. Johnson, M. Yashar and D.J. Bales. 1994. Mercury vapor release during insertion and removal of dental amalgam. Operative Dentistry, 19: 70-74.

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Exhibit 56: Occupational Safety and Health Administration (OSHA). Chemical Sampling Information - Mercury (Vapor) (as Hg).
http://www.osha-slc.gov/dts/chemicalsampling/data/CH_250510.html

Exhibit 57: *FASEB Journal*, Instructions to Authors: Technical Requirements - References
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Exhibit 62: Performance specifications for the Sensidine-Gastec mercury detection tube, and covering e-mail message from G.M. Richardson to Mr. J. Love, dated May 5, 2000.

Exhibit 63: Sokal, R.R. and F.J. Rohlf. 1981. Biometry: The Principles and Practice of Statistics in Biological Research, 2nd Edition. W.J. Freeman and Company, New York. pp. 412-414.

Exhibit 64: Warfvinge, K. 1995. Mercury exposure of a female dentist before pregnancy. *Br. Dent. J.*, 178: 149-152.

Exhibit 65: David Barnes, History of Mercury Clean-up.

Exhibit 66: Rubin, P.G. and M-H., Yu. 1996. Mercury vapour in amalgam waste discharged from dental office vacuum units. Arch. Environ. Health, 51(4): 335-337.

Exhibit 67: Stonehouse, CA; Newman, AP. 2001. Mercury Vapour Release From a Dental Aspirator. Brit Dental J. 190(10):558-560.

Exhibit 68: Reinhardt, Boyer, Svare, Frank, Cox and Gay. 1983. Exhaled mercury following removal and placement of amalgam restorations. J. Prosthet. Dent., 49(5): 652-656

Exhibit 69: Stopford, W., S.D. Bundy, L.J. Goldwater and J.A. Bittikofer. 1978. Microenvironmental exposure to mercury vapor. Am. Ind. Hug. Assoc. J., 39(5): 378-384.

# G. MARK RICHARDSON, Ph.D., Risklogic Scientific Services Inc.

EDUCATION B.Sc. (Biology) 1980; M.Sc. (Biology) 1983; Ph.D. (Biology) 1994

## EXPERTISE

Dr. Richardson has extensive experience in risk assessment and has conducted, assisted or peer reviewed a variety of high profile assessments. These include risk assessments for Wawa, Ontario (arsenic), Yellowknife, NWT (arsenic), Port Colborne, Ontario (nickel), Sydney, N.S. Tar Ponds (as an advisor to Health Canada), as well as the 1998 mine tailings catastrophe at Aznacollar, Spain (Boliden, Inc.), considered by some to be the worst mine tailings disaster in European history. He was commissioned by the Department of National Defense to conduct the assessment of risks posed by contaminated soils to Canadian Peace Keepers, as part of the DND Board of Inquiry on Croatia. Risk assessments have been conducted for specific contaminated sites, specific and unique exposure scenarios, and for medical devices and materials. On a number of occasions, he has served as an expert witness on risk assessment for legal proceedings.

Dr. Richardson published the first probabilistic risk assessment of chemical exposures arising from medical devices and materials, having authored Health Canada's *Assessment of Mercury Exposure and Risks from Dental Amalgam*. He also completed and published the first assessment of risks posed by components and degradation products of the family of dental materials known as composite resins. Contributions were also solicited by the Government of Sweden in their review of the potential hazards posed by mercury released from dental materials, and he has lectured in Canada, the U.S.A., Australia and Europe on chemical risks posed by dental materials.

Dr. Richardson was one of six North Americans invited to Kuwait in 1995 to address a special workshop convened to discuss risks and problems posed by the severe crude oil contamination in that country following the Gulf War. Also in that year he toured major cities of Australia, under sponsorship of the Australian federal Department of Human Services and Health, to advise state and federal regulatory agencies on risk assessment methods and programs established in Canada to manage contaminated sites.
Dr. Richardson has trained staff of Health Canada, Environment Canada, Public Works and Government Services Canada, Indian and Northern Affairs Canada, the Louisiana Department of Environmental Quality, other regulatory agencies and private sector consultants in risk assessment methods. Since 1997, Dr. Richardson has instructed graduate level courses in risk assessment for the University of Ottawa's School of Graduate Studies and their Ecotoxicology Program. He has also guest edited a special issue of the journal *Human and Ecological Risk Assessment (HERA)* on the subject of risk assessment.

A significant proportion of Dr. Richardson's work involves risk assessment support to regulatory agencies. For example, he provided primary consulting support to the CCME Development Committee that was charged with establishing human health-based soil quality guidelines for the CCME Canada Wide Standard for petroleum hydrocarbons. Other projects involving government agencies have examined human and/or environmental risks posed by mercury, arsenic, various other heavy metals, PAH, PCB, DDT, petroleum hydrocarbons, as well as human and veterinary drugs.

Between 1989 to 1995, Dr. Richardson was Head of Health Canada's Air and Waste Section, developing procedures for, and conducting, quantitative risk assessments of environmental contaminants, as well as contributing to the CCME National Contaminated Sites Remediation Program, developing national risk-based soil quality guidelines. He is also routinely called upon to provide third party peer review of risk assessments conducted for regulatory and private sector clients for sites and issues throughout Canada.

Dr. Richardson has research experience is a broad range of topics including: risk assessment methods; toxicology, pharmacokinetics; environmental fate of pesticides and environmental contaminants; the biogeochemistry of mercury; dental materials; contaminated soils; and human chemical exposure. He has published the *Compendium of Canadian Human Exposure Factors for Risk Assessment*. This risk assessment resource is the first and only one of its kind in Canada, presenting basic statistics and probability distributions for numerous physical and behavioural characteristics of the Canadian population; data essential to valid and defensible risk assessment, but never before available to the risk assessment community in Canada. This *Compendium* is now being distributed by the University of Waterloo's, Institute for Risk Research.

Dr. Richardson has also established an expertise in biostatistics, metanalysis, and quantitative trend analysis of large and complex data sets. These skills have been developed through the quantitative evaluation and analysis of Canadian population data respecting human tissue chemical analysis (such as mercury levels in blood and hair samples), food consumption statistics, and time-activity patterns. Dr. Richardson has also served as an external reviewer and as a member of site visit committees on behalf of the Natural Sciences and Engineering Research Council of Canada (NSERC), to evaluate the merits of academic research grant proposals in Canada pertaining to mercury.

EXPERIENCE

Nov/98 - present — **Risklogic Scientific Services Inc., 14 Clarendon Ave., Ottawa, Ontario CANADA K1Y 0P2**

Director and Risk Assessment Specialist

Founder and director of the company, which specializes in human health and ecological risk assessment, environmental guidelines, data analysis and biostatistics. Conducts risk assessments for chemical exposures arising from medical devices and materials, contaminated sites, and other sources of exposure, as well as multi-media human health exposure assessments for contaminants generally dispersed in the environment. Provides contract support to environmental regulatory programs at the federal and provincial levels. Provides biostatistical consulting and support, particularly relating to statistical analysis and probability density functions for human exposure factors. Conducts training courses in probabilistic risk assessment to regulatory and industrial clients.

.ug/95-Aug/00 — **O'Connor Associates Environmental Inc., 14 Clarendon Ave., Ottawa, Ontario CANADA K1Y 0P2**

Senior Risk Assessment Specialist

Responsible for conducting and reviewing risk assessments for chemical exposures arising from medical devices and materials, contaminated sites, and for conducting multi-media human health exposure assessments for contaminants in the environment. Develops and presents, on behalf of OAEI, training courses in probabilistic risk assessment to regulatory and industrial clients. Provides biostatistical support to other risk assessors in the company, particularly relating to probability density functions for human exposure factors. Also responsible for the development and recommendation of environmental regulatory policies and procedures to O'Connor Associates' regulatory clients, both nationally and internationally.

:t/94-Aug/95 — **Medical Devices Bureau, Health Protection Branch**
**Health Canada, Ottawa, Ontario CANADA**

Special Assignment

Responsible for preparing the Department's assessment of the exposure and risks posed by mercury arising from dental amalgam.

)3-1995
1 1989 - 1991 — **Air and Waste Section**
**Health Canada, Ottawa, Ontario CANADA**

Section Head

Responsible for the development and management of procedures for quantitative risk assessment of environmental contaminants and contaminated soils; provision of expertise in human exposure assessments during chemical fires, spills and other environmental emergency situations; direction of Health Canada's contribution to the National Contaminated Sites Remediation Program; evaluation of the adequacy of risk assessments prepared in support of megaprojects under the Federal Environmental Assessment and Review Process; assessment of risks posed by incinerator stack emissions; and, establishment of a new program within Health Canada on the health risks of hazardous waste, contaminated soils and soil cleanup technologies. This position also involved financial management, project management and management and supervision of professional and support staff

1-1993 — **Full-time doctoral candidate, University of Ottawa, Department of Biology**

9 — **Environmental Health Services, Medical Services Branch**

**Health Canada**

Special Assignment

Responsible for providing expertise in human health risk assessment associated with chemical exposur in northern environments and native diets.

1987-1989 **Environmental Health Directorate**
**Health and Welfare Canada**

Science Advisor

Responsible for providing input, advice and recommendations on the direction, conduct a management of federal R & D programs in environmental and occupational health. Duties includ participation on Federal/Provincial Advisory Committees related to environmental and occupatio health, specifically pertaining to chemical and radiation contamination of air, drinking water, soils a occupational environments.

1985-1987 **Environmental Analysis Branch**
**Environment Canada**

Assessment Specialist

Reviewed and assessed the potential environmental hazards posed by industrial, municipal a institutional activities.

1982-1985 **Urea Formaldehyde Foam Insulation Assistance Program**
**Consumer and Corporate Affairs Canada**

Biologist

Managed contracts for health, biological and chemical research on urea formaldehyde foam insulatio analysed and interpreted indoor air quality and health-related data collected through the UFFI Assistan Program.

## OTHER EXPERIENCE

Other experience includes an extensive knowledge and working ability in: biostatistics; methods of regulatory toxicology, ri assessment and risk management; an extensive knowledge of human exposure factors used in risk assessment; chemical exposur and risks posed by dental materials and other medical devices; development of regulatory risk management processes a environmental policy.

Extensive knowledge and experience in risk assessment and regarding metals in the environment has led to participation as a external reviewer and as a site visit committee member on behalf of the Natural Sciences and Engineering Research Council Canada to evaluate the merits of academic research grant proposals in Canada.

## PROFESSIONAL AFFILIATIONS

- Institute for Risk Research, University of Waterloo
- Society for Environmental Toxicology and Chemistry, Laurentian Chapter
- Sigma-Xi Scientific Research Society, Ottawa-Kingston Chapter
- Visiting Scientist, Geologic Survey of Canada

PUBLISHED JOURNAL ARTICLES, BOOK CONTRIBUTIONS AND BOOK REVIEWS

Richardson, G.M., K.E. Clark and D.R. Williams. 1999. Preliminary estimates of adult exposure to bisphenol-a from dental materials, food and ambient air. In: *Environmental Toxicology and Risk Assessment: Standardization of Biomarkers for Endocrine Disruption and Environmental Assessment: Eighth Volume, ASTM STP 1364*, D.S. Henshel, M.C. Black and M.C. Harrass, Eds., American Society for Testing and Materials, West Conshohocken, PA. pp. 286-301.

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Richardson, G.M. 1999. Dental Amalgam and Mercury Exposure: Potential Patient Risks and the Basis for Ristrictions on Use. In: Chapter 3, *Amalgam and Health - New Perspectives on Risks*, FORSKNINGSRÅDSNÄMNDEN (FRN; Swedish Council for Planning and Coordination of Research), Report 99:1, Stockholm, Sweden.

Richardson, G M. 1998. Book Review: How to Control Costs in Your Pollution Prevention Program, by J.A. Cichowicz. *Water, Air and Soil Pollution*, 106: 199-200.

Clark, K.E. and G.M. Richardson. 1998. Debate/Commentary - Fate and exposure models: Selecting the appropriate model for a specific application, Introduction. *J. Soil Contam.*, 7(3): 267-274.

Allan, M. and Richardson, G.M. 1998. Probability Density Functions Describing 24-hour Inhalation Rates for Use in Human Health Risk Assessments. *Human and Ecological Risk Assessment*, Vol. 4(2), 379-408.

Richardson, G.M. 1997. Assessment of adult exposure and risks from components and degradation products of composite resin dental materials. *Human and Ecological Risk Assessment*, 3(4): 683-697.

Richardson, G.M. 1997. Invited Debate/Commentary: Is bioremediation a green technology? Introduction. *J. Soil Contam.*, 6(3): 205-206.

Richardson, G.M. 1997. Invited Debate/Commentary: What research is needed on indoor infiltration of volatile organic contaminants? Introduction. *J. Soil Contam.*, 6(1): 1-2.

Richardson, G.M. and M. Allan. 1996. A Monte Carlo Assessment of Mercury Exposure and Risks from Dental Amalgam. *Human and Ecological Risk Assessment*, 2(4):709-761.

Richardson, G.M. and D.Burmaster (Eds.). 1996. Theoretical, Toxicological and Biostatistical Foundations for Deriving Probability Density Functions Describing Variability and Uncertainty in References Doses, Benchmark Doses and Slope Factors. *Human and Ecological Risk Assessment*, 2(1).

Richardson, G. M. 1996. Deterministic versus Probabilistic Risk Assessment: Strengths and Weaknesses in a Regulatory Context. *Human and Ecological Risk Assessment*, 2(1), 44-54.

Bartlett, S., G.M.Richardson, D. Krewski, S.N.Rai and M.Fyfe. 1996. Commentary: Characterizing uncertainty in risk assessment - conclusions drawn from a workshop. *Human and Ecological Risk Assessment*, 2(1), 221-231.

Williams, D.R., J.C.Paslawski and G.M. Richardson. 1996. Development of a screening relationship to describe migration of contaminant vapours into buildings. *J. Soil Contam.*, 5(2): 141-156.

Richardson, G.M. and D.J. Currie. 1995. Using empirical methods to assess the risks of mercury accumulation in fish from lakes receiving acid rain. *Human and Ecological Risk Assessment*, 1(3): 306-322.

Richardson, G.M., M.Egyed and D.J. Currie. 1995. Does acid rain increase human exposure to mercury? A review and analysis of recent literature. *Environ. Toxicol. Chem.*, volume 14 (5), 809-813.

Richardson, G.M., M.Egyed and D.J. Currie. 1995. Human exposure to mercury may decrease as acidic deposition increases. *Water, Air and Soil Pollution*, 80: 31-39.

Richardson, G.M., M.Mitchell, S.Coad and R.Raphael. 1995. Exposure to mercury in Canada: a multimedia assessment. *Water, Air and Soil Pollution*, 80: 21-30.

Richardson, G.M. and A. Myers. 1993. Risk Assessment and its Application to Risk Management in Environmental Health. *Environmental Health Review*, 37(2): 48-55.

Richardson, G.M. and D.J. Currie. 1993. Estimating Fish Consumption Rates for Ontario Amerindians. *J. Expos. Anal. Environ. Epidemiol*, 3(1): 23-38.

Murray, W.D. and M. Richardson. 1993. Development of Biological and Process Technologies for the Reduction and Degradation of Pulp Mill Wastes That Pose a Threat to Human Health. *Critical Reviews in Environmental Science and Technology*, 23(2): 157-194.

Murray, W. and M. Richardson. 1993. Progress Toward the Biological Treatment of C1 and C2 Halogenated Hydrocarbons. *Critical Reviews in Environmental Science and Technology*, 23(3): 195-217.

Jessiman, B., G.M. Richardson, C. Clark and B. Halbert. 1992. A Quantitative Evaluation of Ten Approaches to Setting Site-Specific Cleanup Objectives. *J. Soil Contamin.*, 1(1): 39-59.

Richardson, G.M. 1991. Contaminated Sites: An Update of Health Protection Activities at the National Level. *Environ. Health Rev.*, 35(4): 88-93.

Bourdeau, P., E. Somers, G.M. Richardson and J.R. Hickman (Eds.) 1990. *Short-Term Toxicity Tests for Non-Genotoxic Effects*. SCOPE 41, SGOMSEC 4, IPCS Joint Symposia 8. John Wiley and Sons, Chichester.

Richardson, G.M. and S.U. Qadri. 1987. Extraction of Aminocarb and a Metabolite from Whole Fish and Derivatization For Electron-Capture Gas Chromatography. *J. Agric. Food Chem.*, 35(6): 877-878.

Richardson, G.M. and S.U. Qadri. 1986. Tissue distribution of $^{14}$C-Labelled Residues of Aminocarb in Brown Bullhead (*Ictalurus nebulosus* Le Sueur) Following Acute Exposure. *Ecotoxicol. Environ. Safety*, 12: 180-186.

Richardson, G.M., S.U. Qadri and B. Jessiman. 1983. Acute toxicity, Uptake and Clearance of Aminocarb by the Aquatic Isopod, *Caecidolea racovitzai racovitzai*. *Ecotoxicol. Environ. Safety*, 7: 552-557.

Richardson, G.M. and S.U. Qadri. 1982. Acute Toxicity, Kinetics and Metabolism of Aminocarb in the Brown Bullhead (*Ictalurus nebulosus*). *Water Poll. Res. J. Canada*, 17: 153-158.

**INVITED LECTURES, COURSES, CONFERENCE PRESENTATIONS AND PROCEEDINGS**

Richardson, G.M. 2001. Arsenic-contaminated soil: an overview of arsenic's potential health risks and the risk assessment process in Canada. Presented on behalf of the Yellowknife Arsenic Soil Remediation Committee, Yellowknife, NWT, May 15, 2001.

Richardson, G.M. 2001. Human health-based soil quality guidelines for PHC: an overview of their basis and derivation. Presented at: CCME Workshop on the Canada-wide Standards for Petroleum Hydrocarbons in Soil (PHC CWS), Calgary, AB, May 10-11, 2001.

Richardson, G.M. 2001. Human health risk assessment: an overview of methods applied in Canada. 3rd Annual Conference/Workshop on Realty Asset and Environment, Department of National Defense, ADM(IE), Cornwall, ON, February 18-23, 2001.

Garrett, R.G., J. Pacyna, **G. M.Richardson,** and M. Mah-Paulson. 2000. Industrial and Atmospheric Sources of Cadmium into the Food Chain. Presented at: SCOPE Workshop on Environmental Cadmium in the Food Chain: Sources, Pathways, and Risks, International Council for Science (ICSU), Brussels on 13-16 September, 2000

Richardson, G.M. 2000. Patient exposures to toxic chemicals from dental materials. Presented at Odenth 2000: 5ème Congrés International, la Baule, France, June 1-3, 2000.

Richardson, G.M. and D. Currie. 2000. Will reduced atmospheric mercury emissions lead to reduced fish contamination in Canadian lakes? Presented at: Annual meeting of the Laurentian Chapter, Society of Environmental Toxicology and Chemistry, Carleton University, Ottawa, ON, May 12-13, 2000.

Richardson, G.M., M. Mah-Paulson, K.E. Clark and D.R. Williams. 2000. Boxes, cylinders and parallel plates: an examination of current methods employed to predict indoor infiltration of volatile soil contaminants. Presented at: Atlantic Canada Environmental Business and Municipal Expo, Halifax, N.S. May 25 - 28, 2000.

Richardson, G.M. 1999. Human Health and Ecological Risk Assessment: A Short Course on Methods Applied in Canada. Principle instructor for course offered in association with the University of Ottawa, Ecotoxicology Program. University of Ottawa, Ottawa, Ontario. August 16-19, 1999.

Richardson, G.M. 1999. Mercury, still with us after all these years; or Dental amalgam, mercury exposure and dental materials safety. Invited lecture presented at the 5th Annual Environmental Symposium, Queen's University, Kingston, Ontario. January 30, 1999.

Richardson, G.M. 1998. Instructor: Environmental Risk Assessment. BIO8100C, School of Graduate Studies, University of Ottawa. Ottawa, Ontario. Sept.-Dec., 1998.

Richardson, G.M. 1998. Patient and occupational exposures to toxic chemicals from dental materials. Presented at: Conference on Bio-Compatible Dentistry, Australasian Society of Oral Medicine and Toxicology, Sydney, Australia, September 5-6, 1998.

Richardson, G.M. 1998. Risk assessments: dental amalgam and polymer filling materials. Presented at: Odontologiske biomaterialer - Bruks og- risikoaspekter ved tannfyllings, Norwegian Dental Association, Oslo, Norway, August 19-20, 1998.

Richardson, G.M. 1998. Discussion Paper on the technical feasibility for the landfilling of building materials coated with PCB-amended paint. Appendix D in: *The Technical Feasibility of Landfilling PCB-Amended Painted Materials - Workshop Proceedings.* Convened by the Department of National Defence, the Department of Indian Affairs and Northern Development, and Environment Canada, Edmonton, Alberta, June 8-9, 1998. Environmental Protection Service, Manuscript Series, no. M-379.

Richardson, G.M. and D.J. Currie. 1998. *Assessing Net Impacts: Apportioning Hg Contamination in Fish Between Natural and Anthropogenic Factors.* Presented at: Environmental and Biomedical Toxicology: Educating Each Other, 1998 ANNUAL MEETING OF THE LAURENTIAN CHAPTER OF SETAC, BioSciences Complex, Queen's University, Kingston, Ontario, May 29-30.

Richardson, G.M. 1998. *Chronic Chemical Exposures from Dental Materials: Mercury, Bisphenol-a and Formaldehyde.* Presented at: Environmental and Biomedical Toxicology: Educating Each Other, 1998 ANNUAL MEETING OF THE LAURENTIAN CHAPTER OF SETAC, BioSciences Complex, Queen's University, Kingston, Ontario, May 29-30.

Richardson, G.M. 1998. So How Much Fish Do You Assume People are Eating When Assessing the Risks Posed by Fish Consumption? Presented at the *5th Annual International Conference on the St. Lawrence River Ecosystem.* Cornwall, ON, April 30 - May 2, 1998.

Richardson, G.M., K.E. Clark and D.R. Williams. 1998. Preliminary Estimates of Adult Exposure to Bisphenol-a from Dental Materials, Food and Ambient Air. Presented at the *Eighth Symposium on Environmental Toxicology and Risk Assessment: Standardization of Biomarkers for Endocrine Disruption and Environmental Assessment.* Atlanta, GA, April 20-22, 1998.

Richardson, G.M. 1998. Applying Biostatistics To Environmental Risk Assessment. Invited lecture presented to the Society of Environmental Chemistry and Toxicology, Laurentian Chapter. Ottawa, Ontario. March 3, 1998.

Richardson, G.M. 1997. Dental Amalgam, Mercury Exposure and Dental Materials Safety. Presented at the Centre for Metal Biology, Uppsala University, Uppsala, Sweden. November 21, 1997.

Richardson, G.M. 1997. Assessment of mercury exposure and risks from dental amalgam and other dental materials. Presented at "*Perspectives on Risks - Cases: Lead, Smoking and Amalgam*", A Swedish Council for Planning and Coordination of Research conference, Stockholm, Sweden. November 20, 1997.

Richardson, G.M. 1997. Assessment of risks from dental amalgam and other dental materials. Presented at the University of Bergen, Biomaterials Adverse Reaction Unit, Bergen, Norway. November 14, 1997.

Richardson, G.M. 1997. An Introduction to Probabilistic Risk Assessment. Presented at the 12th Annual Conference on Contaminated Soils, Association for the Environmental Health of Soils (AEHS), Amherst, MA, October 20-23, 1997.

Richardson, G.M. 1997. An Introduction to Risk Assessment. Presented at Western New England College, Springfield, MA, October 20, 1997.

Richardson, G.M. 1997. Patient and Occupational Risks of Chemical Exposures from Dental Materials. Invited lecture to the International Academy of Oral Medicine and Toxicology. Toronto, Ontario, Canada, September 18-20, 1997.

Richardson, G.M. 1997. Instructor: Environmental Risk Assessment. BIO8100C, School of Graduate Studies, University of Ottawa. Ottawa, Ontario. Sept.-Dec., 1997.

Richardson, G.M. 1997. Human Health and Ecological Risk Assessment: A Short Course on Methods Applied in Canada. Principle instructor for course offered in association with the University of Ottawa, Ecotoxicology Program. University of Ottawa, Ottawa, Ontario. June 16-19, 1997.

Richardson, G.M. 1997. Mercury, still with us after all these years; or Dental amalgam, mercury exposure and dental materials safety. Invited lecture presented to the Sigma-Xi Research Society, Ottawa, Ontario. June 18, 1997.

Clark, K.E., G.M. Richardson and D.R. Williams. 1997. How Much Site Characterization is Necessary? Presented at the Air and Waste Management's 90th Annual Meeting and Exhibition, Toronto, Ontario, CANADA, June 8-13, 1997.

Richardson, G.M., K.E. Clark and D.R. Williams. 1997. Is 'Reasonable Maximum Exposure' Actually Reasonable? Presented at the Society of Environmental Toxicology and Chemistry, St. Laurent Chapter Conference, Montreal, Quebec, May 30-31, 1997.

Richardson, G.M. 1996.Should there be concern regarding mercury exposure from dental amalgam? Seminar offered by O'Connor Associates Environmental Inc., Toronto, Ontario. December 4, 1996.

Richardson, G.M. 1996.Should there be concern regarding mercury exposure from dental amalgam? Seminar organized by the Association of Holistic Dentists of Quebec, Montreal, Quebec. November 19, 1996.

Richardson, G.M. 1996. An Introduction to Probabilistic Exposure Assessment. Presented at the 11th Annual Conference on Contaminated Soils, Association for the Environmental Health of Soils (AEHS), Amherst, MA, October 21-24, 1996.

Richardson, G.M. 1996. Should there be concern regarding mercury exposure from dental amalgam. Invited lecture to the International Academy of Oral Medicine and Toxicology. Houston, Texas, September 28-30, 1996.

Richardson, G.M. 1996. Introduction to determinisitic and probabilistic risk assessment. Presented at Contaminated and Hazardous Waste Site Management, GOwen Environmental Ltd. and the Association for Environmental Health of Soils. Toronto, Ontario. September 26, 1996.

Richardson, G.M. 1996.Should there be concern regarding mercury exposure from dental amalgam? Seminar offered by O'Connor Associates Environmental Inc., Ottawa, Ontario. August 28, 1996.

Richardson, G.M. 1996. Comparative dietary risks: Risks in perspective. Presented at the Federal Forum on Contaminants in Fish. Convened by the U.S.EPA and the American Fisheries Society, Alexandria, VA. June 9-11, 1996.

Richardson, G.M., K.E. Clark, J.C. Paslawski, and D.R. Williams. 1996. Assessing the uncertainty in uncertainty analysis: application of stochastic uncertainty analysis to exposure assessment. Proceedings of the 3rd National Hazardous and Solid Waste Convention, Sydney, Australia, May 26-30, 1996, p.730-735.

Clark, K.E., G.M. Richardson and D.R. Williams. 1996. Computer models for developing remediation guidelines: a comparison of MEPAS and CalTOX. Proceedings of the 3rd National Hazardous and Solid Waste Convention, Sydney, Australia, May 26-30, 1996, p.184-189.

Clark, K.E., G.M. Richardson and D.R. Williams. 1996. The relative importance of site-specific and exposure parameters in developing generic remediation guidelines. Proceedings of the 3rd National Hazardous and Solid Waste Convention, Sydney, Australia, May 26-30, 1996, p.736-740.

Williams, D., J. Paslawski and G.M. Richardson. 1995. Stochastic Exposure Assessment Training Course. Presented to Louisiana Department of Environmental Quality, Baton Rouge, Louisiana, December 4 to 8, 1995.

Richardson, G.M. 1995. An Introduction to Probabilistic Exposure Assessment. Presented at the 10th Annual Conference on Contaminated Soils, Association for the Environmental Health of Soils (AEHS), Amherst, MA, October 23-26, 1995.

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| ICD–9 code | Cancer description |
|---|---|
| 189 | Malignant neoplasm of kidney and other and unspecified urinary organs. |
| 190 | Malignant neoplasm of eye. |
| 191 | Malignant neoplasm of brain. |
| 192 | Malignant neoplasm of other and unspecified parts of nervous system. |
| 193 | Malignant neoplasm of thyroid gland. |
| 194 | Malignant neoplasm of other endocrine glands and related structures. |
| 195 | Malignant neoplasm of other and ill-defined sites. |
| 196 | Secondary and unspecified malignant neoplasm of the lymph nodes. |
| 197 | Secondary malignant neoplasm of the respiratory and digestive organs. |
| 198 | Secondary malignant neoplasm of other tissue and organs. |
| 199 | Malignant neoplasm without specification of site. |
| 200 | Lymphosarcoma and reticulosarcoma. |
| 201 | Hodgkin's disease. |
| 202 | Other malignant neoplasms of lymphoid and histiocytic tissue. |
| 203 | Multiple myeloma and other immunoproliferative neoplasms. |
| 204 | Lymphoid leukemia. |
| 205 | Myeloid leukemia. |
| 206 | Monocytic leukemia. |
| 207 | Other specified leukemia. |
| 208 | Leukemia of unspecified cell type. |

[1] The International Classification of Diseases Clinical Modification (9th Revision) Volume I&II. [1991] Department of Health and Human Services Publication No. (PHS) 91–1260, U.S. Government Printing Office, Washington, DC.

Dated: September 21, 2001.

**Tommy G. Thompson,**

*Secretary, Department of Health and Human Services.*

[FR Doc. 01–24878 Filed 10–4–01; 8:45 am]

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## DEPARTMENT OF HEALTH AND HUMAN SERVICES

42 CFR Part 82

RIN 0920–ZA00

## Methods for Radiation Dose Reconstruction Under the Energy Employees Occupational Illness Compensation Program Act of 2000; Interim Final Rule With Request for Comments

**AGENCY:** Department of Health and Human Services.

**ACTION:** Interim final rule with request for comments.

**SUMMARY:** This rule implements select provisions of the Energy Employees Occupational Illness Compensation Program Act of 2000 ("EEOICPA" or "Act"). The Act requires the promulgation of methods, in the form of regulations, for estimating the dose levels of ionizing radiation incurred by workers in the performance of duty for nuclear weapons production programs of the Department of Energy and its predecessor agencies. These "dose reconstruction" methods will be applied by the National Institute for Occupational Safety and Health, which is responsible for producing the radiation dose estimates that the U.S. Department of Labor will use in adjudicating certain cancer claims under the Act.

**DATES:** *Effective Date:* This interim final rule is effective October 5, 2001. *Compliance Dates:* Affected parties are not required to comply with the information collection requirements in § 82.10 until the Department of Health and Human Services publishes in the **Federal Register** the control numbers assigned by the Office of Management and Budget (OMB) to these information collection requirements. Publication of the control numbers notifies the public that OMB has approved these information collection requirements under the Paperwork Reduction Act of 1995.

*Comments:* The Department invites written comments on the interim final rule from interested parties. Comments on the rule must be received by November 5, 2001. Comments on the collection of information requirements should be received by October 22, 2001.

**ADDRESSES:** Address written comments on the interim final rule to the NIOSH Docket Officer. Submit comments electronically by e-mail to NIOCINDOCKET@CDC.GOV. See **SUPPLEMENTARY INFORMATION** for file formats and other information about electronic filing. Alternatively, submit printed comments to the following address: NIOSH Docket Office, Robert A. Taft Laboratories; M/S C34, 4676 Columbia Parkway, Cincinnati, OH 45226.

Written comments on the collection of information requirements should be sent to Anne O'Connor, CDC Assistant Reports Clearance Officer, 1600 Clifton Road, MS–D24, Atlanta, GA 30333.

**FOR FURTHER INFORMATION CONTACT:** Larry Elliott, Director, Office of Compensation Analysis and Support, National Institute for Occupational Safety and Health, 4676 Columbia Parkway, MS–R45, Cincinnati, OH 45226, Telephone 513–841–4498 (this is not a toll-free number). Information requests may also be submitted by e-mail to OCAS@CDC.GOV.

**SUPPLEMENTARY INFORMATION:**

### I. Comments Invited

Interested persons or organizations are invited to participate in this rulemaking by submitting written views, arguments, recommendations, and data. Comments are invited on any topic related to this rulemaking. Some generic topics for comment include the following questions:

(1) Does the interim rule make appropriate use of current science for conducting dose reconstructions to be used in an occupational illness compensation program?

(2) Does the interim rule appropriately balance the potential precision of dose reconstructions and the necessary efficiency of the dose reconstruction process?

(3) Does the interim rule implement an appropriate process for involving the claimant in the dose reconstruction?

Comments should identify the author(s), return address, and phone number, in case clarification is needed. Comments can be submitted by e-mail to: NIOCINDOCKET@CDC.GOV. If submitting comments by e-mail, they should be provided as a Microsoft Word or Word Perfect file attachment. Printed comments can be submitted to the NIOSH Docket Office at the address above. The Secretary will consider all communications received on or before the closing date for comments before taking action on the interim final rule. All comments submitted will be available for examination in the Rule Docket both before and after the closing date for comments. A report summarizing each substantive public contact with personnel involved in this rulemaking will be filed in the docket. An electronic docket containing all comments submitted by e-mail will be available over the Internet from the National Institute for Occupational Safety and Health (NIOSH) homepage at www.cdc.gov/niosh.

### II. Final Rule

The Department of Health and Human Services ("HHS") expects to issue a final rule within six months of publication of this interim final rule. Upon publication of the final rule, dose reconstructions completed under this interim final rule will be reviewed and revised, as necessary, to conform with any substantive changes that might be included in the final rule.

## III. Background

### A. Statutory Authority

The Energy Employees Occupational Illness Compensation Program Act of 2000 ("EEOICPA"), Public Law 106–398, 114 Stat. 1654, 1654A–1231 (October 30, 2000), was enacted as Title XXXVI of the Floyd D. Spence National Defense Authorization Act for Fiscal Year 2001. EEOICPA established a compensation program to provide a lump sum payment of $150,000 and medical benefits as compensation to covered employees suffering from designated illnesses incurred as a result of their exposure to radiation, beryllium, or silica while in the performance of duty for the Department of Energy and certain of its vendors, contractors, and subcontractors. This law also provided for payment of compensation to certain survivors of covered employees.

EEOICPA instructed the President to designate one or more federal agencies to carry out the compensation program. Pursuant to this statutory provision, the President issued Executive Order 13179, titled Providing Compensation to America's Nuclear Weapons Workers, which assigned primary responsibility for administering the compensation program to the Department of Labor ("DOL"). 65 FR 77487 (Dec. 7, 2000). DOL published an interim final rule governing DOL's administration of EEOICPA on May 25, 2001 (see 66 FR 28948).

The executive order directed HHS to perform several technical and policymaking roles in support of the DOL program:

(1) HHS is to develop methods to estimate radiation doses ("dose reconstruction") for certain individuals with cancer applying for benefits under the DOL program. These methods are the subject of this rule. HHS is also to apply these methods to conduct the program of dose reconstructions required by EEOICPA. This program will be delegated to the National Institute for Occupational Safety and Health ("NIOSH"), an institute of the Centers for Disease Control and Prevention.

(2) HHS is also to develop guidelines to be used by DOL to assess the likelihood that an employee with cancer developed that cancer as a result of exposure to radiation in performing his or her duties at a DOE facility or atomic weapons facility. These guidelines are being published simultaneously with this interim final rule as a notice of proposed rulemaking under 42 CFR part 81 in this issue of the **Federal Register**.

(3) HHS is to staff the Advisory Board on Radiation and Worker Health and provide it with administrative and other necessary support services. The Board, a federal advisory committee, will advise HHS in implementing its roles under EEOICPA described here.

(4) Finally, HHS is to develop and apply procedures for considering petitions by classes of employees to be added to the *Special Exposure Cohort* established under EEOICPA. Employees included in the *Special Exposure Cohort* who have a specified cancer and meet other conditions, as defined by DOL regulations (66 FR 28948), qualify for compensation under EEOICPA. HHS procedures for considering *Special Exposure Cohort* petitions are under development. HHS expects to issue these procedures within the next six months.

As provided for under section 3625 of EEOICPA, HHS is implementing its responsibilities with the assistance of NIOSH.

### B. What Legal Requirements Are Specified by EEOICPA for Dose Reconstruction?

Section 3623(d) of EEOICPA requires that HHS establish, by regulation, methods for arriving at reasonable estimates of the radiation doses incurred by covered employees seeking compensation for cancer, other than as members of the *Special Exposure Cohort* seeking compensation for a *specified cancer*. These methods will be applied to estimate radiation doses for the following covered employees seeking compensation for cancer under EEOICPA: (1) An employee who was not monitored for exposure to radiation at a DOE or Atomic Weapons Employer facility; (2) an employee who was monitored inadequately for exposure to radiation at such a facility; or (3) an employee whose records of exposure to radiation at such facility are missing or incomplete.

EEOICPA requires the Advisory Board on Radiation and Worker Health to independently review the methods established by this rule and to verify a reasonable sample of dose reconstructions established under these methods. The Advisory Board is a federal advisory committee established and appointed by the President to advise HHS on its major responsibilities under EEOICPA.

Sections 3623(e) and 3626(c) of EEOICPA require that DOE provide HHS with relevant information on worker radiation exposures necessary for dose reconstructions and require DOE to inform covered employees with cancer of the results of their dose reconstructions. NIOSH, which will be conducting the dose reconstructions, will inform covered employees of the results of these dose reconstructions on behalf of DOE.

Subject to provisions of the Privacy Act (5 U.S.C. 552a), HHS will also make available to researchers and the general public information on the assumptions, methodology, and data used in estimating radiation doses, as required by Section 3623(e)(2) of EEOICPA.

Finally, HHS notes that EEOICPA does not authorize the establishment of new radiation protection standards through the promulgation of these methods, and these methods do not constitute such new standards.

### C. What Is the Purpose of Dose Reconstruction?

Dose reconstructions are used to estimate the radiation doses to which individual workers or groups of workers have been exposed, particularly when radiation monitoring is unavailable, incomplete, or of poor quality. Originally dose reconstructions were conducted for research on the health effects of exposure to radiation. In recent decades, dose reconstruction has become an integral component of radiation illness compensation programs in the United States and internationally.

### D. How Are Radiation Doses Reconstructed?

The procedures and level of effort involved in dose reconstructions depend in part on the quantity and quality of available dose monitoring information, the conditions under which radiation exposure arose, and the forms of radiation to which the individual was exposed. If individuals for whom dose estimates are needed were monitored using present day technology and received only external radiation doses, dose reconstruction could be very simple. It might only require summing the radiation doses recorded from radiation badges and adding estimated potential "missed" doses resulting from the limits of detection of monitoring badges.

Dose reconstruction can require extensive research and analysis. Such work is required if radiation doses were not monitored or there is uncertainty about the monitoring methods involved; if there was potential for internal doses through the ingestion, inhalation or absorption of radioactive materials; or if the processes and circumstances involved in the radiation exposures were complex. For the most complex dose reconstructions, research and analyses may include determining or

assuming specific characteristics of the monitoring procedures; identifying events or processes that were unmonitored; identifying the types and quantities of radioactive materials involved; evaluating production processes and safety procedures employed; identifying the locations and activities of exposed persons; identifying comparable exposure circumstances for which data is available to make assumptions; and conducting a variety of complex analyses to interpret the data compiled or estimated.

*E. How is Dose Reconstruction Conducted in a Compensation Program?*

An additional, critical factor affecting how doses are reconstructed is the amount of time available. For health research studies dose reconstructions may take from months to years to complete. In compensation programs, however, a balance must be struck between efficiency and precision. Section 3611 of EEOICPA specifically states that one of the purposes of the compensation program is to provide for "timely" compensation. As applied under EEOICPA, dose reconstruction must rely on information that can be developed on a timely basis and on carefully developed assumptions.

When conducting dose reconstruction for a compensation program, our primary concern will be to ensure the assumptions used to estimate doses are fair, consistent, and well grounded in the best available science. To address fairness, the Defense Threat Reduction Agency ("DTRA"), which conducts dose reconstructions for veterans and Department of Defense civilian personnel who participated in U.S. atmospheric nuclear testing and in the occupation forces of Hiroshima and Nagasaki, applies certain assumptions that err reasonably on the side of overestimating exposures (see 32 CFR part 218). These assumptions substitute for more detailed information that would be time-consuming and costly to develop. HHS will take an approach similar to that of DTRA by using reasonable, fair, and scientifically based assumptions as substitutes for additional research and analysis to achieve an efficient dose reconstruction process.

*F. How Will Dose Reconstruction Methods Under EEOICPA Differ From Dose Reconstruction for Veterans?*

The major differences for the HHS methods for dose reconstructions arise from characteristics that distinguish the radiation exposure experiences of nuclear weapons production workers from those of veterans. Whereas veterans were primarily exposed to external sources of radiation over brief periods in acute doses, employees covered by EEOICPA frequently may have received both acute and chronic exposures to internal and external radiation over periods as long as three to four decades. Further, nuclear weapons production workers experienced more diverse exposures and circumstances of exposure, on an individual basis and as a group than did veterans. As a result, many HHS dose reconstructions will be more complex than those conducted by DTRA, making it necessary that HHS place a high premium on any efficiencies that can be achieved.

Addressing the need for efficiency, HHS is establishing a dose reconstruction process that limits the work performed in cases where it is evident the outcome of the compensation claim will be unaffected. HHS will rely on less detailed or precise estimates for claims for which compensation would clearly be due based on the more limited dose reconstruction, and for claims for which additional work clearly would not result in compensation. In the former case, if it is evident from limited dose reconstruction that the estimated cumulative dose is sufficient to qualify the claimant for compensation, no additional work will be performed. In the latter case, limited dose reconstructions will be conducted only for claims for which it is evident that further research and dose reconstruction is extremely unlikely to produce a compensable level of radiation dose, because the use of worst-case assumptions does not produce a compensable level of radiation dose. In these latter cases, the decisive factors that result in NIOSH deciding to limit the dose reconstruction process will be clearly set forth in the draft of the dose reconstruction results reported to the claimant under § 82.25, and in the dose reconstruction results reported to the claimant under § 82.26.

A second important aspect of the HHS dose reconstruction process is that it will involve interaction with the covered employee or survivor. NIOSH will use information provided by the claimant to evaluate the completeness and adequacy of dose information available, to locate additional exposure or dose-related information, and to estimate unmonitored doses.

*G. How Will HHS Incorporate Scientific Methods Established by the Radiation Safety Scientific Community in Internal Dose Estimation Under EEOICPA?*

The methods for calculating internal dose in this rule use current models published by the International Commission on Radiological Protection (ICRP). Specifically, NIOSH will use the new ICRP respiratory tract model for assessing doses due to inhalation of radioactive particles.[1] In addition, NIOSH will use the new biokinetic models for the radionuclides contained in publications 56,[2] 67[3] and 69[4] in place of those described in previous ICRP publications. These models provide the most widely accepted methods for mathematically describing the uptake, transport and retention of radionuclides in the body.

*H. What Elements Underlying the Dose Reconstruction Process Are Expected to Change With Scientific Progress?*

ICRP periodically updates the models used to evaluate internal doses, based on new research on the metabolic properties of radioactive materials (radionuclides). These ICRP updates reflect the current state of scientific knowledge on the uptake, transport, and retention of radionuclides in the human body.

In addition, technological advances in the areas of retrospective detection of radiation exposure or radiation exposure and dose biomarkers (detectable changes in human tissues and/or physiologic processes resulting from radiation exposure) may make it possible to add new analyses to the dose reconstruction process in the future.

As outlined below, NIOSH will address the need to update the scientific elements underlying dose reconstructions in a process that permits input from the public.

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[1] International Commission on Radiological Protection (ICRP). 1994. Human Respiratory Model for Radiological Protection. ICRP Publication 66, Annals of the ICRP 24(1–4). Elsevier Scientific Ltd., Oxford.

[2] International Commission on Radiological Protection (ICRP). 1989. Age Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 1. ICRP Publication 56, Annals of the ICRP 20(2). Pergamon Press, Oxford.

[3] International Commission on Radiological Protection (ICRP). 1993. Age Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 2. ICRP Publication 67, Annals of the ICRP 23(2/3). Pergamon Press, Oxford.

[4] International Commission on Radiological Protection (ICRP). 1995. Age Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 3: Ingestion Dose Coefficients. ICRP Publication 69, Annals of the ICRP 25(1). Elsevier Scientific Ltd., Oxford.

*I. How Will NIOSH Inform the Public of Any Plans to Change Scientific Elements Underlying the Dose Reconstruction Process to Maintain Methods Reasonably Current With Scientific Progress?*

Periodically, NIOSH will publish a notice in the Federal Register notifying the public of plans to change scientific elements underlying the dose reconstruction process under EEOICPA to reflect scientific progress. Notice will include a summary of the planned changes and the expected completion date for such changes.

*J. How Can the Public Recommend Changes to Scientific Elements Underlying the Dose Reconstruction Process, as Scientific Progress Makes Substantive Improvements in Methods Possible?*

At any time, the public can submit written recommendations to NIOSH for changes to scientific elements underlying the dose reconstruction process, based on relevant new research findings and technological advances. Recommendations will be provided to the Advisory Board on Radiation and Worker Health and may be addressed at a public meeting of the Advisory Board, with notification provided to the source of the recommendations. Recommendations should be addressed to: Director, Office of Compensation Analysis and Support, National Institute for Occupational Safety and Health, 4676 Columbia Parkway, MS–R45, Cincinnati, Ohio 45226.

The public can also submit recommendations by e-mail. Instructions will be provided on the NIOSH Internet homepage at *www.cdc.gov/niosh*.

*K. How Will NIOSH Make Changes in Scientific Elements Underlying the Dose Reconstruction Process, Based on Scientific Progress?*

Proposed changes will be presented to the Advisory Board on Radiation and Worker Health prior to implementation. These proposed changes will be summarized in the notice of the board meeting published in the Federal Register. The public will have the opportunity to comment on proposed changes at the meeting of the Advisory Board and/or in written comments submitted for this purpose. NIOSH will fully consider the comments of the Advisory Board and of the public before deciding upon any changes.

*L. How Will NIOSH Inform the Public of Changes to the Scientific Elements Underlying the Dose Reconstruction Process?*

NIOSH will publish a notice in the Federal Register informing the public of changes and the rationale for the changes. This notice will also provide a summary of the recommendations and comments received from the Advisory Board and the public, as well as responses to the comments.

## IV. History of Rule Development

*A. What Experience Does HHS Have in Dose Reconstruction?*

NIOSH, an Institute of the Centers for Disease Control and Prevention, has conducted a program of federally sponsored health research on DOE employees since 1991. Dose reconstructions are an integral element of this research. In fact, NIOSH will draw substantially on records it has developed through its research on DOE employees in conducting the program of dose reconstructions under EEOICPA.

*B. Did HHS Consult With Outside Experts and Interested Parties During the Development of This Notice of Proposed Rulemaking?*

HHS consulted individually with a wide variety of experts and interested parties to help ensure the quality and practicality of these methods. Reports on these consultations are available in the regulatory docket for public review. While these consultations provided less opportunity for initial public input than generally desired for rulemaking, they served the purpose of ensuring that this interim final rule was developed with reasonable information on the points of view of individual experts and members of public directly affected by the rule. HHS will fully consider comments from the public and from the Advisory Board on Radiation and Worker Health in producing a final rule.

## V. Summary of the Interim Rule

Congress, in enacting EEOICPA, created a new Energy Employees Occupational Illness Compensation Program to ensure an efficient, uniform, and adequate compensation system for certain employees. Under Executive Order 13179, the President assigned primary responsibility for administering the program to DOL. The President assigned various technical responsibilities for policymaking and assistance to HHS. Included among these is promulgation of this rule to establish methods NIOSH will apply to conduct dose reconstructions for covered employees seeking compensation for cancer, other than as members of the Special Exposure Cohort seeking compensation for a specified cancer. NIOSH dose reconstructions will be used by DOL to estimate the probability that the cancers of these covered employees were related to radiation exposures at covered facilities.

*Introduction*

Sections 82.0 and 82.1 briefly describe how these regulations relate to DOL authorities under EEOICPA and the assignment of authority for these regulations to HHS. In § 82.2, HHS provides a general introduction to dose reconstruction and describes the hierarchy of information to be relied upon for dose reconstructions. This hierarchy gives preference to individual radiation monitoring data, if complete and adequate, and provides for use of information on the workplace environment and radiation exposures for interpretation and as a secondary source of data, and provides for use of reasonable and scientific assumptions in lieu of certain data when the workplace environment cannot be fully characterized. HHS believes this approach would give due weight to the potentially most precise data, but would take into account the limitations of such data and its availability.

Section 82.3 summarizes the specific provisions of EEOICPA directing HHS in the development of this regulation and NIOSH in the conduct of dose reconstructions under this regulation. Section 82.4 describes how DOL will use the results of NIOSH dose reconstructions for the adjudication of claims.

*Definitions*

Section 82.5 defines the principal terms used in this part. It includes terms specifically defined in EEOICPA that, for the convenience of the reader of this part, are repeated in this section. It clarifies the definition of radiation. Section 3621(16) of EEOICPA defines radiation as ionizing radiation in the form of alpha or beta particles, neutrons, gamma rays, or accelerated ions or subatomic particles from accelerator machines. The rule elaborates upon this definition, specifically including x rays, protons and other particles capable of producing ions in the body, which are components of ionizing radiation exposures experienced by nuclear weapons production workers. In addition, for clarity the definition in this rule explicitly excludes non-ionizing forms of radiation, such as radio-frequency radiation and microwaves.

*Dose Reconstruction Process*

Section 82.10 provides an overview of the major elements of the dose reconstruction process that NIOSH will implement under EEOICPA. It describes the steps in the process, the sources and types of information that will be collected and analyzed, the role of the claimants in developing a factual basis for dose reconstruction, the types of analyses, and criteria that will direct NIOSH to ensure dose reconstructions produce reasonable dose estimates and serve claimants efficiently.

NIOSH will obtain available monitoring data and information on the workplace environment and practices from DOE and other sources. NIOSH will interview the claimant to obtain information and to report to the claimant on dose reconstruction results and the methods and data used to produce the results. NIOSH will take measures to produce results as efficiently as possible, so that adjudication of the claim by DOL can be resumed and completed in a timely fashion. These measures include limiting the dose reconstruction process to use less detailed or precise estimates for claims for which it is evident that further research and analysis will not affect the outcome of the claim.

For example, under these proposed regulations, if it is evident from the record of external radiation dose alone that an employee incurred a sufficiently high level of dose to have the claim accepted by DOL for compensation (a dose that would result in a probability of causation of 50% or higher), NIOSH would conclude the process without continuing with time consuming research and analysis to estimate internal dose. Instead, NIOSH would immediately report the limited dose estimate, based on external dose only, to the claimant and DOL, along with an explanation of the reason for limiting the dose reconstruction process.

Similarly, if, for example, records and information establish that an employee incurred radiation doses evidently below a level that could result in compensation, NIOSH would substitute worst-case assumptions for additional research and analysis, to complete and report on the dose reconstruction without delay.

This approach will provide more timely compensation for claims for which it is evident the claimant will qualify for compensation, and more timely results and adjudication for claims for which it is evident further research and analysis is extremely unlikely to produce a compensable level of radiation dose. The Department seeks public comment on all aspects of this process.

Section 82.11 defines the subset of claimants under EEOICPA for whom NIOSH will conduct dose reconstructions. NIOSH will attempt to conduct dose reconstructions for all claims forwarded to NIOSH from DOL. This includes all covered employees seeking compensation for cancer, other than as members of the *Special Exposure Cohort* seeking compensation for a specified cancer, as determined by DOL.

Section 82.12 describes NIOSH procedures for notifying any claimants for whom a dose reconstruction cannot be completed because of insufficient information to reasonably estimate the dose potentially incurred by the covered employee. NIOSH will notify the claimant and DOL that a dose reconstruction cannot be completed and describe the basis for this finding. In these cases, the claimant would have the opportunity to seek administrative review of this result after DOL produces a recommended decision to deny the claim, based on the report from NIOSH that there is insufficient evidence to complete a dose reconstruction. For a claim in which the employee has a specified cancer, the claimant might still be eligible for compensation under EEOICPA. Classes of covered employees have the option to petition HHS to be added to the Special Exposure Cohort. HHS will establish procedures to consider such petitions, as required under section 3626 of EEOICPA and § 2(b) of E.O. 13179. HHS expects to establish the procedures within six months of publication of this rule.

Sections 82.13 and 82.14 describe in detail the sources and examples of the types of information NIOSH will use in dose reconstructions. DOE and claimants will be the primary sources of information. Information types include: Subject and employment information, worker monitoring data, monitoring program data, workplace monitoring data, workplace characterization data, and process descriptions for each work location. The actual use of this wide range of information will be determined for each claim individually, based on the types of information available and necessary.

Sections 82.15–82.17 describe how NIOSH will evaluate the completeness and adequacy of monitoring data and how NIOSH would remedy limitations, applying the general approach described in § 82.2 and making use of the data sources and types described in §§ 82.13 and 82.14. NIOSH will evaluate the completeness and adequacy of monitoring data by various means, such as evaluating associated information on the workplace environment and practices, evaluating the monitoring technology, and evaluating other sources of information. NIOSH will remedy data limitations using established dose reconstruction practices, such as interpolating from recorded doses to estimate unrecorded doses, and substituting monitoring data from comparably exposed workers. HHS seeks public comments suggesting alternative approaches that NIOSH should consider.

Sections 82.18–82.19 describe how NIOSH will address salient technical issues of calculating internal dose and taking into account uncertainty with respect to dose information. Internal dose is the radiation dose received by radioactive materials taken into the body, such as by inhalation or ingestion. It is important because it accumulates year after year, increasing the risk of certain cancers over time. NIOSH will use current ICRP models for calculating internal dose, and will accompany dose estimates with uncertainty distributions. DOL will use these distributions with appropriate statistical methods to take into account uncertainty about the dose when calculating probability of causation for a claim.

*Reporting and Review of Dose Reconstruction Results*

Sections 82.25 and 82.26 describe in detail NIOSH procedures for reporting the results of dose reconstructions to claimants and DOL, specifying the timing, content, and form of the dose reconstruction reports.

Section 82.27 describes how and when claimants can obtain reviews of NIOSH dose reconstructions. NIOSH will review dose reconstructions upon request by DOL under DOL procedures for claimants seeking review of dose reconstructions. These procedures also allow for DOL to request reviews of dose reconstruction upon its own initiative; for example, to request review of previously completed dose reconstructions to reflect updated scientific methods.

**VI. Regulatory Procedures**

The Department of Health and Human Services (HHS) follows the Administrative Procedure Act ("APA") rulemaking procedures specified in 5 U.S.C. 553 in the development of its regulations. In most circumstances, the APA requires a public notice and comment period and consideration of the submitted comments prior to promulgation of a final rule having the effect of law. However, the APA provides for exceptions to its notice and

comment procedures when an agency finds that there is good cause for dispensing with such procedures on the basis that they are impracticable, unnecessary, or contrary to the public interest. In the case of this interim final rule, HHS has determined that under 5 U.S.C. 553(b)(B), good cause exists for waiving the notice and comment procedures. For these same reasons, HHS has also determined good cause exists under 5 U.S.C. 553(d)(3) for these interim rules to become effective immediately.

A number of courts have considered the circumstances under which an agency can conclude that good cause exists for issuing regulations without prior notice and comment. *In American Transfer & Storage Co., et al* v. *Interstate Commerce Commission*, 719 F.2d 1283, 1295 (5th Cir. 1983), the Fifth Circuit described the impracticability test as requiring "analysis in practical terms of the particular statutory-agency setting and the reasons why agency action could not await notice and comment." Similarly, the Seventh Circuit noted that the "legislative history of the impracticability standard reveals that Congress intended this exemption to operate when the regular course of rulemaking procedure would interfere with the agency's ability to perform its functions with the time constraints imposed by Congress." *United States Steel Corporation* v. *United States Environmental Protection Agency*, 605 F.2d 283, 287 (7th Cir. 1979). Courts have also recognized that while strict deadlines alone do not justify dispensing with notice and comment, "deviation from APA requirements has been permitted where congressional deadlines are very tight and the statute is particularly complicated." *Methodist Hospital of Sacramento* v. *Shalala*, 38 F.3d 1225, 1236 (D.C. Cir. 1994).

Precisely such an "analysis in practical terms" demonstrates that in this case, as with respect to changes in the Aid to Families with Dependent Children program at issue in *Philadelphia Citizens in Action* v. *Schweiker*, 669 F.2d 887, 894 (3rd Cir. 1982), "Congress, by setting an effective date so close to the date of enactment, expressed its belief that implementation * * * was urgent." Legislation enacting EEOICPA was signed by the President on October 30, 2000, and responsibility for implementing EEOICPA was assigned to specific agencies by Executive Order on December 7, 2000. In sections 3628 and 3629 of EEOICPA, however, Congress authorized the Secretary of Labor to begin providing compensation to qualified claimants on July 31 2001. To ensure qualified claimants who have cancer or survive employees who had cancer caused by exposure to radiation in their employment by DOE or its contractors or subcontractors receive the compensation to which they are entitled as soon as possible after July 31, 2001, HHS has determined it is necessary to implement the dose reconstruction methods set forth here on an interim final basis.

Under Executive Order 13179, the President assigned HHS three primary responsibilities in assisting the Department of Labor to make determinations on claims for cancer. First, HHS must promulgate methods for estimating the radiation doses incurred in the performance of duty by covered employees who submit claims or are the subject of claims submitted by their survivors. Second, pursuant to the methods established by this interim final regulation, HHS must perform individual dose reconstructions to determine the radiation dose incurred by each covered employee for whom a claim is made. Third, HHS must promulgate guidelines for DOL to use in determining whether the cancers presented by the employees were "as least as likely as not" caused by the radiation doses they incurred. HHS is publishing these probability of causation guidelines simultaneously with this interim final rule as a notice of proposed rulemaking (NPRM) in this issue of the **Federal Register.**

Completion of HHS work on dose reconstructions is a prerequisite for DOL to begin using the HHS probability of causation guidelines to make individual determinations. HHS has determined to publish the methods for dose reconstruction as an interim final rule so that HHS can initiate the lengthy process of dose reconstructions for individual claimants. HHS must identify and gather relevant records, evaluate their adequacy, and interact with the claimant in completing each dose reconstruction. By publishing the dose reconstruction methods as an interim final rule, HHS will be able to complete dose reconstruction work to allow DOL to complete the adjudication of claims as soon as possible after the HHS probability of causation guidelines are published as final rules.

If HHS were to issue an NPRM proposing dose reconstruction methods, HHS would be delayed in processing dose reconstructions for individual claimants by at least 150 days, until a final regulation could be issued.

HHS believes good cause exists to waive the notice and comment procedures under the APA for the promulgation of these interim final rules. There is a strong public interest in the expeditious adjudication of claims that these workers, who served in this nation's nuclear weapons programs, were harmed in the performance of their duties. This public interest is clearly reflected in the mandate given by Congress to swiftly initiate this program. Moreover, qualified claimants should be given the opportunity to obtain their benefits, including medical benefits, as soon as possible. This is especially material given that many of the covered workers eligible to make claims under this Act are elderly and ill. An undue delay in the processing of their claims would result in real harm to these claimants.

With the publication of this interim final rule, HHS can begin the labor intensive process of reconstructing the radiation doses of employees covered by these claims. Once the probability of causation guidelines are finalized, DOL will be able to expeditiously adjudicate cancer claims requiring dose reconstructions.

Although HHS is adopting these dose reconstruction rules on an interim final basis, it requests public comment on this rule. After full consideration of public comments, HHS will publish a final rule with any necessary changes. HHS expects to issue a final rule within six months of the publication of this interim final rule, at the same time as it expects to issue final guidelines regarding the probability of causation. Since dose reconstructions completed under the interim final rule cannot be used to finally adjudicate claims until those guidelines are issued in final form, HHS will be able to review and revise dose reconstructions completed under this interim final rule, as necessary, to conform with any substantive changes that might be included in the final dose reconstruction rule before any final action is taken on a particular claim. By issuing the dose reconstruction regulation as an interim final regulation, however, substantial time can be saved and many more claims can be timely adjudicated, based on the final regulation and guidelines, enabling covered employees or their survivors to receive benefits to which they may be entitled as expeditiously as possible.

### VII. Significant Regulatory Action (Executive Order 12866)

This rule is being treated as a "significant regulatory action" within the meaning of Executive Order (E.O.) 12866 because it raises novel or legal policy issues arising out of the legal mandate established by EEOICPA. The rule is designed to establish practical

methods, grounded in current science, to fairly and efficiently assist claimants and support DOL in the adjudication of applicable claims seeking compensation for cancer under EEOICPA. NIOSH will apply the methods to produce reasonable, scientifically supported estimates of the radiation doses incurred by covered employees subject to the claims, as permitted by available data and information. The financial cost to the federal government of producing these estimates is expected to be several thousand dollars per claim, on average.

The rule carefully explains the manner in which the regulatory action is consistent with the mandate for this action under § 3623(d) of EEOICPA and implements the detailed requirements concerning this action under this section of EEOICPA. The rule does not interfere with State, local, and tribal governments in the exercise of their governmental functions.

The rule is not considered economically significant, as defined in section 3(f)(1) of the Executive Order 12866. It has a subordinate role in the adjudication of claims under EEOICPA, serving as one element of an adjudication process administered by DOL under 20 CFR parts 1 and 30. DOL has determined that its rule fulfills the requirements of Executive Order 12866 and provides estimates of the aggregate cost of benefits and administrative expenses of implementing EEOICPA under its rule (see FR 28948, May 25, 2001). OMB has reviewed this rule for consistency with the President's priorities and the principles set forth in E.O. 12866.

## VIII. Regulatory Flexibility Act

The Regulatory Flexibility Act (RFA), 5 U.S.C. 601 *et seq.*, requires each agency to consider the potential impact of its regulations on small entities including small businesses, small governmental units, and small not-for-profit organizations. We certify that this rule will not have a significant economic impact on a substantial number of small entities within the meaning of the RFA. This rule affects only DOL, DOE, HHS, and some individuals filing compensation claims under EEOICPA. Therefore, a regulatory flexibility analysis as provided for under RFA is not required.

## IX. What Are the Paperwork and Other Information Collection Requirements (Subject to the Paperwork Reduction Act) Imposed Under This Rule, and How Are Comments Submitted?

Under the Paperwork Reduction Act of 1995, a Federal agency shall not conduct or sponsor a collection of information from ten or more persons other than Federal employees unless the agency has submitted a Standard Form 83, Clearance Request, and Notice of Action, to the Director of the Office of Management and Budget (OMB), and the Director has approved the proposed collection of information. A person is not required to respond to a collection of information unless it displays a currently valid OMB control number. The Paperwork Reduction Act is applicable to the data collection aspects of this rule.

In compliance with the requirement of Section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995 for opportunity for public comment on proposed data collection projects, the Centers for Disease Control and Prevention (CDC) will publish periodic summaries of projects. To request more information on this project or to obtain a copy of the data collection plans and instruments, call the CDC Reports Clearance Officer at (404) 639–7090.

Comments are invited on: (a) Whether the proposed collection of information is necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (b) the accuracy of the agency's estimate of the burden of the proposed collection of information; (c) ways to enhance the quality, utility, and clarity of the information to be collected; and (d) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques or other forms of information technology.

NIOSH is requesting an emergency clearance from the Office of Management and Budget (OMB) to collect data under EEOICPA. Send comments to Anne O'Connor, CDC Assistant Reports Clearance Officer, 1600 Clifton Road, MS–D24, Atlanta, GA 30333. Written comments should be received within 14 days of this notice. OMB is expected to act on the request of HHS within 21 days of publication of this notice.

In performance of its dose reconstruction responsibilities under the Act, NIOSH will interview claimants individually and provide them with the opportunity, through a structured interview, to assist NIOSH in documenting the work history of the employee (characterizing the actual work tasks performed), identifying incidents that may have resulted in undocumented radiation exposures, characterizing radiation protection and monitoring practices, and identifying co-workers, radiation protection management and staff, line managers, and other witnesses, if NIOSH determines this is necessary, to confirm undocumented information. In this process, NIOSH will use a computer assisted telephone interview (CATI) system, which will allow interviews to be conducted more efficiently and quickly than would be the case with a paper-based interview instrument.

NIOSH will use the data collected in this process to complete an individual dose reconstruction that accounts for radiation dose, including unmonitored or inadequately monitored dose, incurred by the employee in the performance of duty for DOE nuclear weapons production programs. After dose reconstruction, NIOSH will provide a draft of the dose reconstruction report to the claimant and perform a brief follow-up interview with the claimant to explain the results and to allow the claimant to confirm or question the record NIOSH has compiled. This will also be the final opportunity for the claimant to supplement the dose reconstruction record.

At the conclusion of the dose reconstruction process, the claimant will be requested to submit to NIOSH a form (OCAS–1) to confirm that the claimant has completed providing information to NIOSH for the dose reconstruction. The form will notify the claimant that signing the form allows NIOSH to provide a final dose reconstruction report to DOL and closes the record on data to be used for the dose reconstruction. DOL will use data from the dose reconstruction report to determine the probability that the cancer(s) of the covered employee may have been caused by radiation doses incurred in the performance of duty at a DOE or AWE facility.

There will be no cost to respondents for this data collection. This is a new data collection. The estimated burden of this data collection is described in the table below.

| Respondents | Number of respondents | Number of responses | Avg. burden per response (hrs.) | Total hours |
|---|---|---|---|---|
| Initial interview | 22,500 | 1 | 60/60 | 22,500 |
| Conclusion form | 22,500 | 1 | 5/60 | 1,875 |
| Total | | | | 24,375 |

**X. Small Business Regulatory Enforcement Fairness Act**

As required by Congress under the Small Business Regulatory Enforcement Fairness Act of 1996 (5 U.S.C. 801 *et seq.*), the Department will report to Congress promulgation of this rule prior to its effective date. The report will state that the Department has concluded that this rule is not a "major rule" because it is not likely to result in an annual effect on the economy of $100 million or more. However, this rule has a subordinate role in the adjudication of claims under EEOICPA, serving as one element of an adjudication process administered by DOL under 20 CFR parts 1 and 30. DOL has determined that its rule is a "major rule" because it will likely result in an annual effect on the economy of $100 million or more.

**XI. Unfunded Mandates Reform Act of 1995**

Title II of the Unfunded Mandates Reform Act of 1995 (2 U.S.C. 1531 *et seq.*) directs agencies to assess the effects of Federal regulatory actions on State, local, and tribal governments, and the private sector, "other than to the extent that such regulations incorporate requirements specifically set forth in law." For purposes of the Unfunded Mandates Reform Act, this rule does not include any Federal mandate that may result in increased annual expenditures in excess of $ 100 million by State, local or tribal governments in the aggregate, or by the private sector.

**XII. Executive Order 12988 (Civil Justice)**

This rule has been drafted and reviewed in accordance with Executive Order 12988, Civil Justice Reform and will not unduly burden the Federal court system. Dose reconstruction may be an element in reviews of DOL adverse decisions in the United States District Courts pursuant to the Administrative Procedure Act. However, DOL has attempted to minimize that burden by providing claimants an opportunity to seek administrative review of adverse decisions, including those involving dose reconstruction. This rule provides a clear legal standard for HHS and DOL to apply regarding dose reconstruction. This rule has been reviewed carefully to eliminate drafting errors and ambiguities.

**XIII. Executive Order 13132 (Federalism)**

The Department has reviewed this rule in accordance with Executive Order 13132 regarding federalism, and has determined that it does not have "federalism implications." The rule does not "have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government."

**XIV. Executive Order 13045 (Protection of Children From Environmental, Health Risks and Safety Risks)**

In accordance with Executive Order 13045, HHS has evaluated the environmental health and safety effects of this rule on children. The agency has determined that the rule will not affect children.

**XV. Executive Order 13211 (Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use)**

In accordance with Executive Order 13211, HHS has evaluated the effects of this rule on energy supply, distribution or use, and has determined that this rule is not likely to have a significant adverse effect on them.

**List of Subjects in 42 CFR Part 82**

Cancer, Dose reconstruction, Government employees, Occupational safety and health, Nuclear materials, Radiation protection, Radioactive materials, Workers' compensation.

**Text of the Rule**

For the reasons discussed in the preamble, the Department of Health and Human Services amends 42 CFR to add Part 82 to read as follows:

**PART 82—METHODS FOR CONDUCTING DOSE RECONSTRUCTION UNDER THE ENERGY EMPLOYEES OCCUPATIONAL ILLNESS COMPENSATION PROGRAM ACT OF 2000**

**Subpart A—Introduction**

Sec.



**Subpart B—Definitions**



**Subpart C—Dose Reconstruction Process**



**Subpart D—Reporting and Review of Dose Reconstruction Results**



Authority: 42 U.S.C. 7384n; E.O. 13179, 65 FR 77487.

## Subpart A—Introduction

### § 82.0 Background information on this Rule.

The Energy Employees Occupational Illness Compensation Program Act (EEOICPA), Public Law 106–398, provides for the payment of compensation benefits to covered employees and, where applicable, survivors of such employees, of the United States Department of Energy, its predecessor agencies and certain of its contractors and subcontractors. Among the types of illnesses for which compensation may be provided are cancers. There are two categories of covered employees with cancer under EEOICPA for whom compensation may be provided. The regulations that follow under this part apply only to the category of employees described under (a) of this section.

(a) One category is employees with cancer for whom a dose reconstruction must be conducted, as required under 20 CFR 30.115.

(b) The second category is members of the Special Exposure Cohort seeking compensation for a specified cancer, as defined under EEOICPA. The U.S. Department of Labor (DOL) which has primary authority for implementing EEOICPA, has promulgated regulations at 20 CFR 30.210 and 30.213 that identify current members of the Special Exposure Cohort and requirements for compensation. Pursuant to section 3626 of EEOICPA, the Secretary of HHS is authorized to add additional classes of employees to the Special Exposure Cohort.

### § 82.1 What is the purpose of this rule?

The purpose of this rule is to provide methods for determining a reasonable estimate of the radiation dose received by a covered employee with cancer under EEOICPA, through the completion of a dose reconstruction. These methods will be applied by the National Institute for Occupational Safety and Health (NIOSH) in a dose reconstruction program serving claimants under EEOICPA, as identified under § 82.0.

### § 82.2 What are the basics of dose reconstruction?

The basic principle of dose reconstruction is to characterize the radiation environments to which workers were exposed and to then place each worker in time and space within this exposure environment. Then methods are applied to translate exposure to radiation into quantified radiation doses at the specific organs or tissues relevant to the types of cancer occurring among the workers. A hierarchy of methods is used in a dose reconstruction, depending on the nature of the exposure conditions and the type, quality, and completeness of data available to characterize the environment.

(a) If found to be complete and adequate, individual worker monitoring data, such as dosimeter readings and bioassay sample results, are given the highest priority in assessing exposure. These monitoring data are interpreted using additional data characterizing the workplace radiation exposures. If radiation exposures in the workplace environment cannot be fully characterized based on available data, default values based on reasonable and scientific assumptions may be used as substitutes. For dose reconstructions conducted in occupational illness compensation programs, this practice may include use of assumptions that represent the worst case conditions. For example, if the solubility classification of an inhaled material can not be determined, the dose reconstruction would use the classification that results in the largest dose to the organ or tissue relevant to the cancer.

(b) If individual monitoring data are not available or adequate, dose reconstructions may use monitoring results for groups of workers with comparable activities and relationships to the radiation environment. Alternatively, workplace area monitoring data may be used to estimate the dose. As with individual worker monitoring data, workplace exposure characteristics are used in combination with workplace monitoring data to estimate dose.

(c) If neither adequate worker nor workplace monitoring data are available, the dose reconstruction may rely substantially on process description information to analytically develop an exposure model. For internal exposures, this model includes such factors as the quantity and composition of the radioactive substance (the source term), the chemical form, particle size distribution, the level of containment, and the likelihood of dispersion.

### § 82.3 What are the requirements for dose reconstruction under EEOICPA?

(a) Dose reconstructions are to be conducted for the following covered employees with cancer seeking compensation under EEOICPA: An employee who was not monitored for exposure to radiation at Department of Energy (DOE) or Atomic Weapons Employer (AWE) facilities; an employee who was monitored inadequately for exposure to radiation at such facilities; or an employee whose records of exposure to radiation at such facility are missing or incomplete. Technical limitations of radiation monitoring technology and procedures will require HHS to evaluate each employee's recorded dose. In most, if not all cases, monitoring limitations will result in possibly undetected or unrecorded doses, which are estimated using commonly practiced dose reconstruction methods and would have to be added to the dose record.

(b) Section 3623(e) of EEOICPA requires the reporting of radiation dose information resulting from dose reconstructions to the covered employees for whom claims are being adjudicated. DOE is specifically charged with this responsibility but the Department of Health and Human Services (HHS), which will be producing the dose reconstruction information, will implement this reporting responsibility on behalf of DOE. HHS will also make available to researchers and the general public information on the assumptions, methodology, and data used in estimating radiation doses, as required by EEOICPA.

### § 82.4 How will DOL use the results of the NIOSH dose reconstructions?

Under 42 CFR part 81, DOL will apply dose reconstruction results together with information on cancer diagnosis and other personal information provided to DOL by the claimant to calculate an estimated probability of causation. This estimate is the probability that the cancer of the covered employee was caused by radiation exposure at a covered facility of DOE or an Atomic Weapons Employer (AWE).

## Subpart B—Definitions

### § 82.5 Definition of Terms Used in this Rule.

(a) *Atomic weapons employer* (AWE) means any entity, other than the United States, that:

(1) Processed or produced, for use by the United States, material that emitted radiation and was used in the production of an atomic weapon, excluding uranium mining and milling; and,

(2) Is designated by the Secretary of Energy as an atomic weapons employer for purposes of EEOICPA.

(b) *Bioassay* means the determination of the kinds, quantities, or concentrations, and in some cases, locations of radioactive material in the human body, whether by direct measurement or by analysis, and

evaluation of radioactive material excreted or eliminated by the body.

(c) *Claimant* means the individual who has filed with the Department of Labor for compensation under EEOICPA.

(d) *Covered employee* means, for the purposes of this rule, an individual who is or was an employee of DOE, a DOE contractor or subcontractor, or an atomic weapons employer, and for whom DOL has requested HHS to perform a dose reconstruction.

(e) *Covered facility* means any building, structure, or premises, including the grounds upon which such building, structure, or premise is located:

(1) In which operations are, or have been, conducted by, or on behalf of, the DOE (except for buildings, structures, premises, grounds, or operations covered by Executive Order 12344, dated February 1, 1982, pertaining to the Naval Nuclear Propulsion Program); and,

(2) With regard to which the DOE has or had:

(i) A proprietary interest; or,

(ii) Entered into a contract with an entity to provide management and operation, management and integration, environmental remediation services, construction, or maintenance services; or

(3) A facility owned by an entity designated by the Secretary of Energy as an atomic weapons employer for purposes of EEOICPA that is or was used to process or produce, for use by the United States, material that emitted radiation and was used in the production of an atomic weapon, excluding uranium mining or milling.

(f) *DOE*: The U.S. Department of Energy, includes predecessor agencies of DOE, including the Manhattan Engineering District.

(g) *DOL*: The U.S. Department of Labor.

(h) *EEOICPA* means the Energy Employees Occupational Illness Compensation Program Act of 2000, Public Law 106–398, as amended.

(i) *Equivalent dose* is the absorbed dose in a tissue multiplied by a radiation weighting factor to account for differences in the effectiveness of the radiation in inducing cancer.

(j) *External dose* means that portion of the equivalent dose that is received from radiation sources outside of the body.

(k) *Internal dose* means that portion of the equivalent dose that is received from radioactive materials taken into the body.

(l) *NIOSH*: the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services.

(m) *Primary cancer* means a cancer defined by the original body site at which the cancer was incurred, prior to any spread (metastasis) resulting in tumors at other sites in the body.

(n) *Probability of causation* means the probability or likelihood that a cancer was caused by radiation exposure incurred by a covered employee in the performance of duty. In statistical terms, it is the cancer risk attributable to radiation exposure divided by the sum of the baseline cancer risk (the risk to the general population) plus the cancer risk attributable to the radiation exposure. This concept is further explained under 42 CFR part 81, which provides guidelines by which DOL will determine probability of causation under EEOICPA.

(o) *Radiation* means ionizing radiation, including alpha particles, beta particles, gamma rays, x rays, neutrons, protons and other particles capable of producing ions in the body. For purposes of this rule, radiation does not include sources of non-ionizing radiation such as radio-frequency radiation, microwaves, visible light, and infrared or ultraviolet light radiation.

(p) *Specified cancer* is a term defined in section 3621(17) of EEOICPA and 20 CFR part 30.5(dd) that specifies types of cancer that, pursuant to 20 CFR part 30, may qualify a member of the Special Exposure Cohort for compensation. It includes leukemia (other than chronic lymphocytic leukemia), multiple myeloma, non-Hodgkin's lymphoma, and cancers of the lung (other than carcinoma in situ diagnosed at autopsy), thyroid, male breast, female breast, esophagus, stomach, pharynx, small intestine, pancreas, bile ducts, gall bladder, salivary gland, urinary bladder, brain, colon, ovary, liver (not associated with cirrhosis or hepatitis), and bone. Pursuant to section 2403 of Public Law 107–20, this definition will include renal cancer.

(q) *Uncertainty distribution* is a statistical term meaning a range of discrete or continuous values arrayed around a central estimate, where each value is assigned a probability of being correct.

(r) *Worst-case assumption* is a term used to describe a type of assumption used in certain instances for certain dose reconstructions conducted under this rule. It assigns the highest reasonably possible value, based on reliable science, documented experience, and relevant data, to a radiation dose of a covered employee.

## Subpart C—Dose Reconstruction Process

### § 82.10 Overview of the Dose Reconstruction Process.

(a) Upon receipt of a claims package from the Department of Labor, as provided under 20 CFR part 30, NIOSH will request from the Department of Energy (DOE) records on radiation dose monitoring and radiation exposures associated with the employment history of the covered employee. Additionally, NIOSH may compile data, and information from NIOSH records that may contribute to the dose reconstruction. For each dose reconstruction, NIOSH will include records relevant to internal and external exposures to ionizing radiation, including exposures from medical screening x rays that were required as a condition of employment.

(b) NIOSH will evaluate the initial radiation exposure record compiled to: Reconcile the exposure record with the reported employment history, as necessary; complete preliminary calculations of dose, based upon this initial record, and prepare to consult with the claimant. Any discrepancies in the employment history information will be reconciled with the assistance of DOE, as necessary.

(c) NIOSH will interview the claimant. The purpose of the interview is to:

(1) Explain the dose reconstruction process;

(2) Confirm elements of the employment history transmitted to NIOSH by DOL;

(3) Identify any relevant information on employment history that may have been omitted;

(4) Confirm or supplement monitoring information included in the initial radiation exposure record;

(5) Develop detailed information on work tasks, production processes, radiologic protection and monitoring practices, and incidents that may have resulted in undocumented radiation exposures, as necessary;

(6) Identify co-workers and other witnesses with information relevant to the radiation exposures of the covered worker to supplement or confirm information on work experiences, as necessary.

(d) NIOSH will provide a report to the claimant summarizing the findings of the interview, titled: "NIOSH Claimant Interview under EEOICPA." The report will also notify the claimant of the opportunity to contact NIOSH if necessary, by a specified date, to make any written corrections or additions to

information provided by the claimant during the interview process.

(e) Information provided by the claimant will be accepted and used for dose reconstruction, providing it is reasonable, supported by substantial evidence, and is not refuted by other evidence. In assessing whether the information provided by the claimant is supported by substantial evidence, NIOSH will consider:

(1) Consistency of the information with other information in the possession of NIOSH, from radiation safety programs, research, medical screening programs, labor union documents, worksite investigations, dose reconstructions conducted by NIOSH under EEOICPA, or other reports relating to the circumstances at issue;

(2) Consistency of the information with medical records provided by the claimant;

(3) Consistency of the information with practices or exposures demonstrated by the dose reconstruction record developed for the claimant; and,

(4) Confirmation of information by co-workers or other witnesses.

(f) NIOSH will seek to confirm information provided by the claimant through review of available records and records requested from DOE.

(g) As necessary, NIOSH will request additional records from DOE to characterize processes and tasks potentially involving radiation exposure for which dose and exposure monitoring data is incomplete or insufficient for dose reconstruction.

(h) NIOSH will review the adequacy of monitoring data and completeness of records provided by DOE. NIOSH will request certification from DOE that record searches requested by NIOSH have been completed.

(i) As necessary, NIOSH will characterize the internal and external exposure environments for parameters known to influence the dose. For internal exposures, examples of these parameters include the mode of intake, the composition of the source term (i.e., the radionuclide type and quantity), the particle size distribution and the absorption type. When it is not possible to characterize these parameters, NIOSH may use default values, when they can be established reasonably, fairly, and based on relevant science. For external exposures, the radiation type (gamma, x-ray, neutron, beta, or other charged particle) and radiation energy spectrum will be evaluated. When possible, the effect of non-uniformity and geometry of the radiation exposure will be assessed.

(j) For individual monitoring records that are incomplete, doses may be imputed using techniques discussed in § 82.16. Once the resulting data set has been evaluated and validated, an occupational exposure matrix will be constructed, using the general hierarchical approach discussed in § 82.2. This matrix will contain the estimated annual equivalent dose(s) to the relevant organ(s) or tissue(s), for the period from the initial date of potential exposure at a covered facility until the date the cancer was diagnosed. The equivalent dose(s) will be calculated using the current, standard radiation weighting factors from the International Commission on Radiological Protection (ICRP, Publication 60),[1] indicated in Table 1.

TABLE 1.—RADIATION WEIGHTING FACTORS

| Radiation type and energy range | Radiation weighting factor, $w_R$ |
|---|---|
| Photons, all energies | 1 |
| Electrons and muons, all energies | 1 |
| Neutrons, energy <10 keV | 5 |
| 10 keV to 100 keV | 10 |
| >100 keV to 2 MeV | 20 |
| >2 MeV to 20 MeV | 10 |
| >20 MeV | 5 |
| Protons, other than recoil protons, energy >2 MeV | 5 |
| Alpha particles, fission fragments and heavy nuclei | 20 |

(k)(1) At any point during steps in paragraphs (f)–(j) of this section of dose reconstruction, NIOSH may determine that sufficient research and analysis has been conducted to complete the dose reconstruction. Research and analysis will be determined sufficient if one of the following three conditions is met:

(i) From acquired experience, it is evident the estimated cumulative dose is sufficient to qualify the claimant for compensation (i.e., the dose produces a probability of causation of 50% or greater);

(ii) Dose is determined using worst-case assumptions related to radiation exposure and intake, to substitute for further research and analyses; or,

(iii) Research and analysis indicated under steps in paragraphs (f)–(j) of this section have been completed.

(2) Worst-case assumptions will be employed under condition in paragraph (k)(1)(ii) of this section to limit further research and analysis only for claims for which it is evident that further research and analysis will be extremely unlikely to produce a compensable level of radiation dose (a dose producing a probability of causation of 50% or greater), because even using worst-case assumptions it cannot be determined that the employee may have incurred a compensable level of radiation dose. For all claims in which worst-case assumptions are employed under condition in paragraph (k)(1)(ii) of this section, the reasoning that resulted in the determination to limit further research and analysis will be clearly described in the draft of the dose reconstruction results reported to the claimant under § 82.25 and in the dose reconstruction results reported to the claimant under § 82.26.

(l) After providing the claimant with a copy of a draft of the dose reconstruction report to be provided to DOL, NIOSH will conduct a closing interview with the claimant to review the dose reconstruction results and the basis upon which the results were calculated. This will be the final opportunity during the dose reconstruction process for the claimant to provide additional relevant information that may affect the dose reconstruction.

(m) Subject to any additional information provided by the claimant under § 82.10(l), the claimant is required to return form OCAS–1 to NIOSH, certifying that the claimant has completed providing information and that the record for dose reconstruction should be closed. Upon receipt of the form and completion of any changes in the dose reconstruction resulting from new information provided under § 82.10(l), NIOSH will forward a final dose reconstruction report to DOL and to the claimant.

(n) NIOSH will not forward the dose reconstruction report to DOL for adjudication without receipt of form OCAS–1 signed by the claimant or a representative of the claimant authorized pursuant to 20 CFR 30.600. If the claimant or the authorized representative of the claimant fails to sign and return form OCAS–1 within 60 days, after notifying the claimant or the authorized representative, NIOSH may administratively close the dose reconstruction and notify DOL of this action. Upon receiving this notification by NIOSH, DOL may administratively close the claim.

(o) Once actions under § 82.10(m) are completed, the record for dose reconstruction shall be closed unless reopened at the request of DOL under 20 CFR part 30.

[1] International Commission on Radiological Protection (ICRP) 60: "1990 Recommendations of the International Commission on Radiological Protection." Ann. ICRP 21(1–3): 6.

**§ 82.11 For which claims under EEOICPA will NIOSH conduct a dose reconstruction?**

NIOSH will conduct a dose reconstruction for each claim determined by DOL to be a claim for a covered employee with cancer under DOL regulations at 20 CFR 30.210(b), subject to the limitation and exception noted in § 82.12. Claims for covered employees who are members of the Special Exposure Cohort seeking compensation for a specified cancer, as determined by DOL under 20 CFR 30.210(a), do not require and will not receive a dose reconstruction under this rule.

**§ 82.12 Will it be possible to conduct dose reconstructions for all claims?**

It is uncertain whether adequate information of the types outlined under § 82.14 will be available to complete a dose reconstruction for every claim eligible under § 82.11.

(a) NIOSH will notify in writing any claimants for whom a dose reconstruction cannot be completed once that determination is made, as well as in the closing interview provided for under § 82.10(l).

(b) Notification will describe the basis for finding a dose reconstruction cannot be completed, including the following:

(1) A summary of the information obtained from DOE and other sources; and,

(2) A summary of necessary information found to be unavailable from DOE and other sources.

(c) NIOSH will notify DOL when it is unable to complete a dose reconstruction for the claimant. This will result in DOL producing a recommended decision to deny the claim, since DOL cannot determine probability of causation without a dose estimate produced by NIOSH under this rule.

(d) A claimant for whom a dose reconstruction cannot be completed, as indicated under this section, may have recourse to seek compensation under provisions of the Special Exposure Cohort (see 20 CFR part 30). Pursuant to section 3626 of EEOICPA, the Secretary of HHS is authorized to add additional classes of employees to the Special Exposure Cohort.

**§ 82.13 What sources of information may be used for dose reconstructions?**

NIOSH will use the following sources of information for dose reconstructions, as necessary:

(a) DOE and its contractors, including Atomic Weapons Employers and the former worker medical screening program;

(b) NIOSH and other records from health research on DOE worker populations;

(c) Interviews and records provided by claimants;

(d) Co-workers of covered employees, or other witnesses with information relevant to the covered employee's exposure, that the claimant identified during the initial interview with NIOSH;

(e) Labor union records from unions representing employees at covered facilities of DOE or AWEs; and,

(f) Any other relevant information.

**§ 82.14 What types of information could be used in dose reconstructions?**

NIOSH will obtain the types of information described in this section for dose reconstructions, as necessary and available:

(a) *Subject and employment information*, including:

(1) Gender;

(2) Date of birth; and,

(3) DOE and/or AWE employment history, including: job title held by year, and work location(s): Including site name(s), building number(s), technical area(s), and duration of relevant employment or tasks.

(b) *Worker monitoring data*, including:

(1) External dosimetry data, including external dosimeter readings (film badge, TLD, neutron dosimeters); and,

(2) Pocket ionization chamber data.

(c) *Internal dosimetry data*, including:

(1) Urinalysis results;

(2) Fecal sample results;

(3) In Vivo measurement results;

(4) Incident investigation reports;

(5) Breath radon and/or thoron results;

(6) Nasal smear results; and,

(7) External contamination measurements.

(d) *Monitoring program data*, including:

(1) Analytical methods used for bioassay analyses;

(2) Performance characteristics of dosimeters for different radiation types;

(3) Historical detection limits for bioassay samples and dosimeter badges;

(4) Bioassay sample and dosimeter collection/exchange frequencies; and,

(5) Documentation of record keeping practices used to record data and/or administratively assign dose

(e) *Workplace monitoring data*, including:

(1) Surface contamination surveys;

(2) General area air sampling results;

(3) Breathing zone air sampling results;

(4) Radon and/or thoron monitoring results;

(5) Area radiation survey measurements (beta, gamma and neutron); and,

(6) Fixed location dosimeter results (beta, gamma and neutron).

(f) *Workplace characterization data*, including:

(1) Information on the external exposure environment, including: Radiation type (gamma, x-ray, neutron, beta, other charged particle); radiation energy spectrum; uniformity of exposure (whole body vs partial body exposure); irradiation geometry; and work-required medical screening x rays.

(2) [Reserved]

(g) *Information characterizing internal exposures*, including:

(1) Radionuclide(s) and associated chemical forms;

(2) Results of particle size distribution studies; and,

(3) Respiratory protection practices.

(h) *Process descriptions for each work location*, including:

(1) General description of the process;

(2) Characterization of the source term (i.e., the radionuclide and its quantity);

(3) Extent of encapsulation;

(4) Methods of containment;

(5) Other information to assess potential for airborne dispersion.

**§ 82.15 How will NIOSH evaluate the completeness and adequacy of individual monitoring data?**

(a) NIOSH will evaluate the completeness of an individual's monitoring data provided by DOE through one or more possible measures including, but not limited to:

(1) Comparisons with information provided by claimants, co-workers, and other witnesses;

(2) Comparisons with available information on area monitoring, production processes, and radiologic protection programs;

(3) Comparisons with information documented in the records of unions representing covered employees;

(4) Comparisons with data available on co-workers; and

(5) Reviews of DOE contractor record systems.

(b) NIOSH will evaluate the instruments and procedures used to collect individual monitoring data to determine whether they adequately characterized the radiation environments in which the covered employee worked, (adequately for the purpose of dose reconstruction,) based on present-day scientific understanding. For external dosimeter measurements, this includes an evaluation of the dosimeter response to the radiation types (gamma, x-ray, neutron, beta, or other charged particle) and the

associated energy spectrum. For internal exposure, the methods used to analyze bioassay samples will be reviewed to determine their ability to detect the radionuclides present in the work environment. An analysis of the monitoring or exchange frequencies for the monitoring programs will also be conducted to determine the potential for undetected dose.

**§ 82.16 How will NIOSH add to monitoring data to remedy limitations of individual monitoring and missed dose?**

(a) For external dosimeter results that are incomplete due to historical record keeping practices, NIOSH will use commonly practiced techniques, such as those described in the NIOSH Research Issues Workshop,[2] to estimate the missing component of dose and to add this to the total dose estimate. For monitoring periods where external dosimetry data are missing from the records, NIOSH will estimate a claimant's dose based on interpolation, using available monitoring results from other time periods close to the period in question, or based on monitoring data on other workers engaged in similar tasks.

(b) NIOSH will review historical bioassay sample detection limits and monitoring frequencies to determine, when possible, the minimum detectable dose for routine internal dose monitoring programs. This "missed dose" will establish the upper limit of internal dose that a worker could have received for periods when bioassay sample analysis results were below the detection limit. Using ICRP biokinetic models, NIOSH will estimate the internal dose and include an associated uncertainty distribution.

**§ 82.17 What types of information could be used to supplement or substitute for individual monitoring data?**

Three types of information could be used:

(a) Monitoring data from co-workers, if NIOSH determines they had a common relationship to the radiation environment; or,

(b) A quantitative characterization of the radiation environment in which the covered employee worked, based on an analysis of historical workplace monitoring information such as area dosimeter readings, general area radiation survey results, air sampling data; or,

(c) A quantitative characterization of the radiation environment in which the employee worked, based on analysis of data describing processes involving radioactive materials, the source materials, occupational tasks and locations, and radiation safety practices.

**§ 82.18 How will NIOSH calculate internal dose to the primary cancer site(s)?**

(a) The calculation of dose from ingested, inhaled or absorbed radioactivity involves the determination of the types and quantities of radionuclides that entered the body. NIOSH will use the results of all available bioassay monitoring information as appropriate, based on assessment of the technical characteristics of the monitoring program. If bioassay monitoring data are unavailable, the dose reconstruction will rely on the results of air sampling measurements.

(b) NIOSH will calculate the dose to the organ or tissue of concern using metabolic models published by ICRP. Using data available to NIOSH, the models will be based on exposure conditions representative of the work environment. When NIOSH cannot establish exposure conditions with sufficient specificity, the dose calculation will assume exposure conditions that maximize the dose to the organ under consideration.

(c) Internal doses will be calculated for each year of exposure from the date of initial exposure to the date of cancer diagnosis.

**§ 82.19 How will NIOSH address uncertainty about dose levels?**

The estimate of each annual dose will be characterized with a probability distribution that accounts for the uncertainty of the estimate. This information will be used by DOL in the calculation of probability of causation, under HHS guidelines for calculating probability of causation estimates at 42 CFR part 81. In this way, claimants will receive the benefit of the doubt in cases in which the actual dose may have exceeded the best estimate calculated by NIOSH.

## Subpart D—Reporting and Review of Dose Reconstruction Results

**§ 82.25 When will NIOSH report dose reconstruction results, and to whom?**

NIOSH will report dose reconstruction results to DOL and to the claimant, as provided for under § 82.10. Draft results will be reported to the claimant upon tentative completion of the dose reconstruction. Final results will be reported to the claimant and DOL after NIOSH receives certification from the claimant that the claimant has completed providing information to NIOSH for the dose reconstruction (Form OCAS–1).

**§ 82.26 How will NIOSH report dose reconstruction results?**

(a) NIOSH will provide dose reconstruction results to the claimant and DOL in a report: "NIOSH Report of Dose Reconstruction under EEOICPA." The report itself will not provide information on probability of causation, which DOL must calculate to determine a recommended decision on the claim.

(b) The report will include the following information, as relevant:

(1) Annual dose estimates (or a fraction thereof) related to covered employment for each year from the date of initial radiation exposure at a covered facility to the date of cancer diagnosis;

(2) Separate dose estimates for acute and chronic exposures, different types of ionizing radiation, and internal and external doses, providing dose information for the organ or tissue relevant to the primary cancer site(s) established in the claim;

(3) Uncertainty distributions associated with each dose estimated, as necessary;

(4) Explanation of each type of dose estimate included in terms of its relevance for estimating probability of causation;

(5) Identification of any information provided by the claimant relevant to dose estimation that NIOSH decided to omit from the basis for dose reconstruction, justification for the decision, and if possible, a quantitative estimate of the effect of the omission on the dose reconstruction results; and

(6) A summary and explanation of information and methods applied to produce the dose reconstruction estimates, including any factual findings and the evidence upon which those findings are based.

(c) As provided under § 82.10(l), NIOSH staff will conduct a closing interview with claimants to explain the dose reconstruction report.

**§ 82.27 How can claimants obtain reviews of their dose reconstruction results by NIOSH?**

Claimants can seek reviews of their dose reconstruction through the processes established by DOL under 20 CFR part 30. DOL will request NIOSH to review dose reconstructions under the following conditions, as provided under 20 CFR 30.318:

(a) DOL may determine that factual findings of the dose reconstruction do

[2] NIOSH (1995). NIOSH research issues workshop: Epidemiologic use of nondetectable values in radiation exposure measurements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 224647 (NTIS–PB 95189601).

not appear to be supported by substantial evidence; or,

(b) Although the methodology established by HHS under this Part is binding on DOL, DOL may determine that arguments concerning the application of this methodology should be considered by NIOSH.

**§ 82.28 Who can review NIOSH dose reconstruction files on individual claimants?**

(a) Claimants and DOL will be provided individual dose reconstruction files, upon request. Claimants should note, however, that a complete summary of the data and methods used in a dose reconstruction will be included in the "NIOSH Report of Dose Reconstruction under EEOICPA".

(b) Researchers and the public will be provided limited access to NIOSH dose reconstruction files, subject to provisions and restrictions of the Privacy Act for the protection of confidential information on individuals. Researchers will not receive names of claimants or covered employees associated with dose reconstructions.

Dated: September 21, 2001.

**Tommy G. Thompson,**

*Secretary, Department of Health and Human Services.*

[FR Doc. 01-24879 Filed 10-4-01; 8:45 am]

BILLING CODE 4160-17-U

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## FEDERAL COMMUNICATIONS COMMISSION

**47 CFR Part 73**

**[MM Docket No. 01-235; FCC 01-262]**

**RIN 4207**

## Cross-Ownership of Broadcast Stations and Newspapers

**AGENCY:** Federal Communications Commission.

**ACTION:** Proposed rule.

**SUMMARY:** This document initiates a proceeding to consider whether to eliminate, modify, or retain the Commission's newspaper/broadcast cross-ownership rule and/or related waiver policies. The takes this action in part because it committed to do so in its first biennial review of its broadcast ownership rules. The intended effect is the harmonization of the Commission's competition and diversity goals with the current realities of the local media marketplace.

**DATES:** Comments are due December 3, 2001; reply comments are due January 7, 2002.

**ADDRESSES:** Federal Communications Commission, 445 12th Street, SW., Washington, DC 20554.

**FOR FURTHER INFORMATION CONTACT:** Eric J. Bash, (202) 418-2130 or ebash@fcc.gov.

**SUPPLEMENTARY INFORMATION:** This is a synopsis of the *Notice of Proposed Rule Making* ("*NPRM*") in MM Docket No. 01-235, FCC 01-262, adopted September, 13, 2001, and released September 20, 2000. The complete text of this *NPRM* is available for inspection and copying during normal business hours in the FCC Reference Center, Room CY-A257, 445 12th Street, SW., Washington, DC and may also be purchased from the Commission's copy contractor, Qualex International, Portals II, 445 12th Street SW, Room CY-B-402, Washington, DC 20554, telephone (202) 863-2893, facsimile (202) 863-2898, or via email *qualexint@aol.com*. The *NPRM* is also available on the Internet at the Commission's website: *http://www.fcc.gov*.

### Introduction

1. In this proceeding, the Commission seeks comment on whether and to what extent it should revise the newspaper/broadcast cross-ownership rule, which prohibits common ownership of a broadcast station and a newspaper in the same geographic area. The rule rests on the "twin goals" of diversity of viewpoints and economic competition. The Commission adopted the rule in 1975. The local multimedia marketplace in which broadcast stations and newspapers operate has changed significantly since that time. This proceeding seeks comment on the relevance of these changes to the newspaper/broadcast cross-ownership rule.

### Background

2. The newspaper/broadcast cross-ownership rule prohibit common ownership of a full-service broadcast station and a daily newspaper when the broadcast station's service contour (2mV/m contour for AM, 1 mV/m contour for FM, Grade A for TV) fully encompasses the newspaper's city of publication. When adopting the rule in 1975, the Commission not only prohibited future newspaper/broadcast combinations, but also required existing combinations in highly concentrated markets to divest holdings to come into compliance within five years. The Commission grandfathered combinations in other markets, so long as the parties to the combination remained the same. The Commission, however, contemplated waiving the rule, for existing or future combinations, if: (1) A combination could not sell a station; (2) a combination could not sell a station except at an artificially depressed price; (3) separate ownership and operation of a newspaper and a station could not be supported in a locality; or (4) for whatever reason, the purposes of the rule would be disserved. The Supreme Court has reviewed the rule and the Commission's related waiver policies, and upheld them in their entirety. The Commission has granted only four permanent waivers in the twenty-six years since it adopted the rule.

3. In February 1996, the Telecommunications Act of 1996 also became law. Section 202(h) of the 1996 Act instructs the Commission to review each of its ownership rules biennially, to determine whether the rule is "necessary in the public interest as a result of competition" and repeal or modify any rule it finds is no longer in the public interest. As required by section 202(h) of the 1996 Act, the Commission examined the newspaper/broadcast cross-ownership policies in its first biennial review on broadcast ownership rules. The Commission concluded that the newspaper/broadcast cross-ownership rule continues to serve the public interest because it furthers diversity, and therefore should be retained. However, the Commission also noted that the rule might not be necessary to achieve its intended public interest benefits under certain circumstances. Thus, the Commission committed to undertaking a rulemaking proceeding to tailor the rule accordingly.

### Discussion

4. Since the Commission adopted the newspaper/broadcast cross-ownership rule over twenty-five years ago, the local media marketplace has changed dramatically. In this proceeding, we seek to examine our newspaper/broadcast cross-ownership policies in the context of these changes in the local media marketplace, taking into account section 202(h) of the Telecommunications Act of 1996, and our diversity and competition goals.

5. *Current Status of the Media Marketplace.* The number of local media outlets has grown substantially since 1975. A significant portion of this growth has occurred within the broadcast industry itself. A total of 7,785 radio stations were on the air as of January 1, 1975; as of June 30, 2001, the Commission had licensed 12,932 radio stations. A total of 952 TV stations were on the air on January 1, 1975; as of June 30, 2001, the Commission had

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May 12, 2000 10-6003

Mr. James Love
Attorney at Law
111 West 5th
Suite 640
Tulsa, OK 74103
USA

EXHIBIT
3

Derivation of Mercury Exposure for Dr. David Barnes

Dear Mr. Love:

You will find attached our determinations of Dr. Barnes' mercury exposure as a result of his practice of dentistry. Sources of that exposure included: the leaking amalgam capsules and resulting mercury 'spray' and contamination throughout the dental operatories caused by the trituration of those capsules; the removal of old amalgam fillings from his dental patients; and the placement of new amalgam fillings in his dental patients. Pathways of exposure included inhalation of mercury vapour and amalgam particulate matter, and dermal (skin) absorption of the vapour.

The methods employed to quantify these exposures are consistent with standard methods developed and prescribed for quantitative exposure assessment by a variety of agencies and organizations including the U.S. Environmental Protection Agency and the Canadian federal Department of Health (Health Canada), as well as numerous state, provincial and international agencies.

Should you have any questions or require clarification, please contact the undersigned.

Yours truly,

O'CONNOR ASSOCIATES ENVIRONMENTAL INC.

G. Mark Richardson, Ph.D.
Senior Risk Assessment Specialist

*SUMMARY*

Mercury exposures were determined for Dr. David Barnes, who experienced mercury exposure. Exposures were determined for amalgam removals, amalgam placements, and from room air levels arising from leaking amalgam capsules and the resulting office contamination. Exposures can be summarized as follows:

| | | |
|---|---|---|
| Amalgam placement | vapour | 64 µg/day |
| | particulate | 595 µg/day |
| Amalgam removal | vapour | 62 µg/day |
| | particulate | 7100 µg/day |
| Room air contamination | vapour | 198 - 958 µg/day |
| Total | | 8019 - 8779 µg/day |

## 1.0 INTRODUCTION

This report summarizes the determination of mercury exposure for Dr. David Barnes of Shelbyville, Tennessee. Dr. Barnes' occupation as a dentist resulted in exposure to mercury. Dr. Barnes' average daily exposure to mercury was determined for routine dental procedures (amalgam placements and removals) undertaken between 1989 and 1998 when he reported working 10 to 11 hours per day, 5 days per week.

## 2.0 SOURCES AND PATHWAYS OF EXPOSURE

Mercury exposure in Dr. David Barnes would have arisen from three primary sources:

- General office air contamination due to the leaking amalgam capsules, the spray of mercury droplets caused by trituration (mixing) of those leaking capsules, and the subsequent dispersion of mercury throughout the dental operatories in which he worked;
- The removal of old amalgam fillings, undertaken to facilitate the placement of new amalgams and crowns; and
- The placement of new amalgams.

Dr. Barnes' exposure to mercury would have occurred via three pathways: inhalation of vapours, inhalation of amalgam particulate matter containing mercury, and dermal absorption of mercury vapour. The air of dental offices is routinely contaminated with mercury leading to elevated exposure in dental personnel (Martin et. al., 1995). Mercury leakage from defective capsules and from amalgam triturators has been a significant contributor to mercury exposure experienced by dental personnel (Hooper, 1980; Warfvinge, 1995). The widespread mercury contamination throughout Dr. Barnes' dental operatories would have emitted mercury vapour on an on-going basis until a thorough decontamination was completed. Despite two attempts at mercury decontamination, mercury contamination throughout Dr. Barnes' dental operatories is still significant (Gobbell Hays Partners, Inc., 1999; State of Tennessee, 1999), being approximately double the average level of surface contamination reported in the literature (Schneider, 1974). Therefore, it is apparent that the dispersion of liquid mercury through the office was considerable.

Besides being inhaled, mercury vapour is also absorbed through the skin (US ATSDR, 1999; Hursh et al., 1989). For a given air mercury concentration, dermal absorption contributes an additional 2.6% to the mercury intake via inhalation.

The removal of old amalgam fillings and the placement of new ones results in the generation of high concentrations of mercury vapour in the dentist's breathing zone (Richards and Warren, 1985), and large concentrations of respirable amalgam particulate matter in the dentist's breathing zone (Nimmo et al., 1990). Amalgam particulate is approximately 50% mercury by weight (Berry et al., 1994). The inhalation of this particulate matter and vapour will have contributed significantly to Dr. Barnes' mercury burden. The mercury inhaled as amalgam particulate matter is systemically absorbed within about 3 days (Cutright et al., 1973).

## 3.0 MERCURY EXPOSURES

### 3.1 Exposure due to Amalgam Placements

For amalgam placement procedures, average daily mercury exposure is the result of inhalation of mercury vapour and inhalation of amalgam particulate matter, the latter being 50% mercury by weight.

#### 3.1.1 Vapour inhalation dose during amalgam placement

$$Vapour\ inhalation\ dose = C_{air} * IR * D * AR * N$$

where, $C_{air}$ = concentration in air during procedure ($\mu g/m^3$)
IR = inhalation rate ($m^3/hr$)
D = average duration of each procedure (hours)

AR = absorption rate for mercury in the lungs (unitless)
N = number of amalgam placements performed per working day (placements/day)

Data provided by Dr. Barnes indicated that, during the years of 1996 and 1998, he performed a total of 1295 and 779 amalgam procedures, respectively (average = 1037 procedures). Dr. Barnes also indicated during our telephone interview of April 20, 2000, that he has placed at least 1000 amalgam fillings each year from the beginning his practice in 1983 until the end of 1997. In 1998 he began a reduced work schedule of 4 days per week. For purposes of calculations performed here, I have assumed an annual average of only 1000 fillings placed.

Dr. Barnes also indicated during our telephone interview that he worked 288 days per year in 1997. For purposes of this report, I have assumed that he worked an average of 220 days per year for the majority of his career. Based on a average of 1000 amalgams placed per year and his working 220 days per year, this would indicate that an average of 5 amalgams were placed per working day.

Haikel et al.(1990) and Powell et al. (1994) have demonstrated, qualitatively, that insertion of an amalgam filling produces the same average breathing zone mercury level as does amalgam removal. Richards and Warren (1985), using continuous monitoring, reported that the average mercury vapour concentration in the breathing zone of the dentist during an amalgam removal procedure was 100 $\mu g/m^3$.

Trituration, condensation, insertion and carving of a newly placed amalgam requires approximately 13 minutes (0.22 hours) (Powell et al., 1994).

Average inhalation rate for an adult male is 17.5 $m^3$/24 hours (Allan and Richardson, 1998), or 0.73 $m^3$/hour. The absorption rate for mercury vapour from the lungs is 80% (WHO, 1991; US ATSDR, 1999).

Therefore,

$C_{air}$ = 100 $\mu g/m^3$ (Richards and Warren 1985; wet grinding with aspiration)
IR = 0.73 $m^3$/hour (Allan and Richardson, 1998)
D = 13 minutes = 0.22 hours (Powell et al., 1994)
AR = 80% or 0.8 (US ATSDR, 1999)
N = 5 fillings placed per day

As a result, Dr. Barnes' average daily vapour inhalation dose due to placement of new amalgam fillings was:

Vapour inhalation dose = 100 $\mu g/m^3$ * 0.73 $m^3$/hour * 0.22 hour * 0.8 * 5 = 64 µg per day

### 3.1.2 Particle inhalation dose during amalgam placement

$$Particulate\ inhalation\ dose = PM * P(Hg) * N * R$$

where, PM = mass of amalgam particulate inhaled during an amalgam removal (µg/filling)
P(Hg) = the weight percent of Hg in amalgam
N = number of amalgam placements per day (fillings/day)
R = relative proportion of amalgam particulate inhaled during placement relative to during removal (unitless)

The average mass of fully respirable amalgam particulate inhaled by a dentist during a removal procedure is 3550 µg (Nimmo et al., 1990; with water spray). The procedures of grinding the newly-set amalgam to contour surfaces for proper occlusion with opposing teeth, as well as final polishing, also release amalgam particulate matter. Contouring and polishing is anticipated to remove 0.1 mm (4 thousandths of an inch) of the new amalgam. This is 6.7% of an average depth of an amalgam, which is typically 1.5 mm in depth into the tooth (Nimmo et al., 1990). Therefore, it can be conservatively assumed that 6.7% of the new amalgam placed in the prepared cavity would be ground and polished off to achieve occlusion and finishing of the filling, and would result in amalgam particulate exposure equal to 6.7% of that for the removal of an old amalgam filling.

Therefore,

PM = 3550 µg/filling
P(Hg) = the weight percent of Hg in amalgam = 50% or 0.5 (Berry et al., 1994)
N = number of amalgam placements per day = 5 fillings/day
R = 6.7% or 0.067

As a result, Dr. Barnes' average daily particulate inhalation dose due to placement of new amalgam fillings was:

Particulate inhalation dose = 3550 µg/filling * 0.5 * 5 fillings/day * 0.067 = 595 µg/day

## 3.2 Exposure Due to Amalgam Removals

For amalgam removal procedures, average daily mercury exposure is the result of inhalation of mercury vapour and inhalation of amalgam particulate matter, the latter being 50% mercury by weight.

### 3.2.1 Vapour inhalation dose during amalgam removal

$$Vapour\ inhalation\ dose = C_{air} * IR * D * AR * N$$

where, $C_{air}$ = concentration in air during procedure (µg/m³)
IR = inhalation rate (m³/hr)
D = average duration of each procedure (hours)
AR = absorption rate for mercury in the lungs (unitless)
N = number of amalgam removals performed per working day (placements/day)

Dr. Barnes indicated during our telephone interview of April 20, 2000, that the removal of pre-existing amalgams was required in at least 70% of placement procedures. Therefore, for 1000 amalgams placed per year, 700 amalgam removals were undertaken.

Data provided by Dr. Barnes also indicated that, during 1996 and 1998, he installed 375 crowns in 1996 and 440 crowns in 1998 (average = 408 crowns). Dr. Barnes also indicated that 90% of procedures involving the placement of crowns first required the removal of an existing old amalgam. Assuming an annual average of 400 crown procedures, then 360 additional amalgam removals were undertaken per year. Total removals undertaken was, therefore, 1060 per year. Based on 220 working days per year, this amounts to 5 removals per day.

As outlined previously, average inhalation rate for an adult male is 0.73 m³/hour (Allan and Richardson, 1998) and the respiratory absorption rate for mercury vapour is 80%, or 0.8 (US ATSDR, 1999; WHO, 1991). It has been assumed herein that the average duration of an amalgam removal procedure was 10 minutes (0.17 hours).

Richards and Warren (1985), using continuous monitoring, demonstrated that the average mercury vapour concentration in the breathing zone of the dentist during an amalgam removal procedure was 100 µg/m³. The data of Richards and Warren (1985) were applied herein for determination of Dr. Barnes' mercury vapour exposure during removal procedures.

Therefore,

$C_{air}$ = 100 µg/m³ (Richards and Warren 1985; wet grinding with aspiration)
IR = 0.73 m³/hour (Allan and Richardson, 1998)
D = 10 minutes = 0.17 hours
AR = 80% or 0.8 (US ATSDR, 1999)
N = 5 fillings removed per day

As a result, Dr. Barnes' average daily vapour inhalation dose due to the removal of old amalgam fillings was:

Vapour inhalation dose = 100 µg/m³ * 0.73 m³/hour * 0.17 hour * 0.8 * 5 = 62 µg per day

### 3.2.2 Particle inhalation dose during amalgam removal

*Particulate inhalation dose = PM * P(Hg) * N * AR*

where, PM = mass of amalgam particulate inhaled during an amalgam removal (µg/filling)
P(Hg) = the weight percent of Hg in amalgam
N = number of amalgam removals per day (fillings/day)
AR = absorption rate for mercury in the lungs (unitless)

The average mass of fully respirable amalgam particulate inhaled by a dentist during a removal procedure is 3550 µg (Nimmo et al., 1990; with water spray). Other parameters are described above.

Therefore,

PM = 3550 µg/filling removed
P(Hg) = the weight percent of Hg in amalgam = 50% or 0.5 (Berry et al., 1994)
N = number of amalgam removals per day = 5 filling removals/day
AR = 80% or 0.8 (US ATSDR, 1999)

As a result, Dr. Barnes' average daily particulate inhalation dose due to removal of old amalgam fillings was:

Particulate inhalation dose = 3550 µg/filling * 0.5 * 5 fillings/day * 0.8 = 7100 µg/day

## 3.3 Exposure Due to Contaminated Dental Office Air

Exposure to mercury vapour also resulted from the high levels of mercury that would have been in the dental operatory air due to the spray and leakage of mercury from the trituration of leaking amalgam capsules, which resulted in widespread mercury contamination throughout the dental operatories (Gobbell Hays Partners, Inc., 1999; State of Tennessee, 1999). Operatory contamination was twice that measured in a published survey of dental offices, despite the fact that Dr. Barnes' operatories had been subjected to two separate cleanup efforts prior to measurements of office contamination being made.

Mercury leakage from amalgam capsules and contamination of amalgamators (triturators) have been the subjects of numerous published articles. Serious mercury exposure in dental personnel (Warfvinge, 1995) and subsequent poisoning (Hooper, 1980) have been attributed to this problem. Recommended remedial steps have included replacement of the defective equipment, office decontamination, and recommendations to discontinue use of slip-fit style (specifically Kerr products) amalgam capsules (Brooks Air Force Base, 2000). Office cleanup and decontamination results in a 3 to 5 fold decrease in general office air contamination compared to pre-cleanup levels (Warfvinge, 1995).

Dr. Barnes' mercury exposure due to this room air contamination would result from both inhalation of mercury vapour as well as dermal absorption (US ATSDR, 1999; Hursh et al., 1989).

### 3.3.1 Vapour inhalation exposure

$$Vapour\ inhalation\ dose = C_{air} * IR * D * AR$$

where, $C_{air}$ = concentration in air ($\mu g/m^3$)
IR = inhalation rate ($m^3/hr$)
D = average duration in contaminated environment (hours)
AR = absorption rate for mercury in the lungs (unitless)

No measurements of mercury levels in the air of Dr. Barnes' office were conducted prior to cleanup and decontamination efforts. However, the air concentration can be estimated from the general surface contamination measured in his operatories after cleanup, and factoring in the likely reduction of that contamination due to the cleanup efforts. Room air concentration can be derived according to:

$$C_{air} = Q * SA_{Hg} / V_{air}$$

Where,

$C_{air}$ = concentration of mercury in indoor air ($\mu g/g$)
$Q$ = Mercury evaporation rate ($\mu g/cm^2 \bullet hour$)
$SA_{Hg}$ = surface area of mercury in dental operatories ($cm^2$)
$V_{air}$ = volume of air exchanged in the room per hour ($m^3/hour$)

The evaporation rate for mercury from droplets of the liquid metal is 7 $\mu g/cm^2$/hour at 20°C (Henke et al., 1993). Mercury evaporation increases as temperature increases. Dr. Barnes' operatories were maintained at 20°C from November through February. However, from March through October, ambient temperature in the operatories increased to about 30°C, due to southerly exposure, large picture windows and an inadequately sized air conditioning system (Dr. D. Barnes, personal communication, April 20, 2000). The increased mercury evaporation rate at 30°C is equal to the

Also, dermal absorption would have contributed an additional dose equivalent to 2.6% of the inhaled dose, which would range from 5 µg/day to 24 µg/day.

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## 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 $\alpha$ and $\beta$ subunit. In order for the two to join, the $\beta$-subunit must bind a GTP molecule. The researchers found that the $\beta$-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, $36 \times 10^{-9}$ 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.

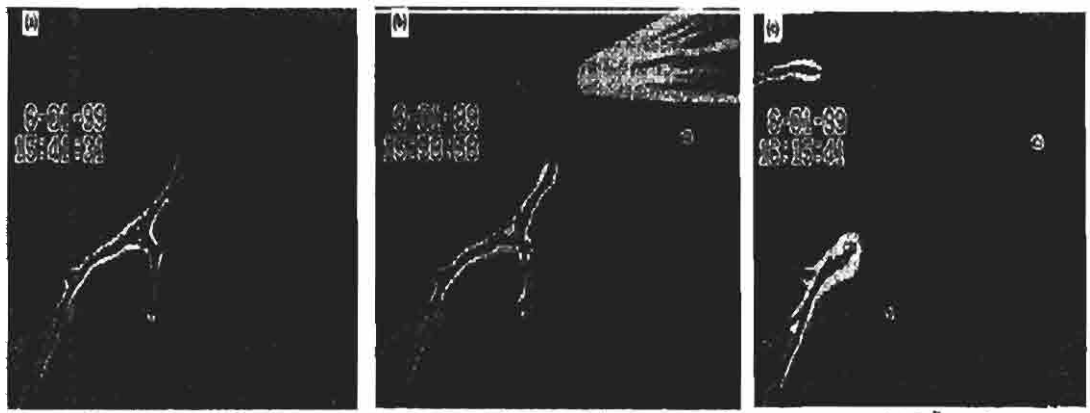

Figure 7 – Retrograde degeneration of neurite growth cone in the presence of $10^{-7}$ molar mercury chloride. Note the triangle reference mark. (From Leong, et. al. 2000)

## 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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## PUBLIC COMMENT

## EXHIBIT 2

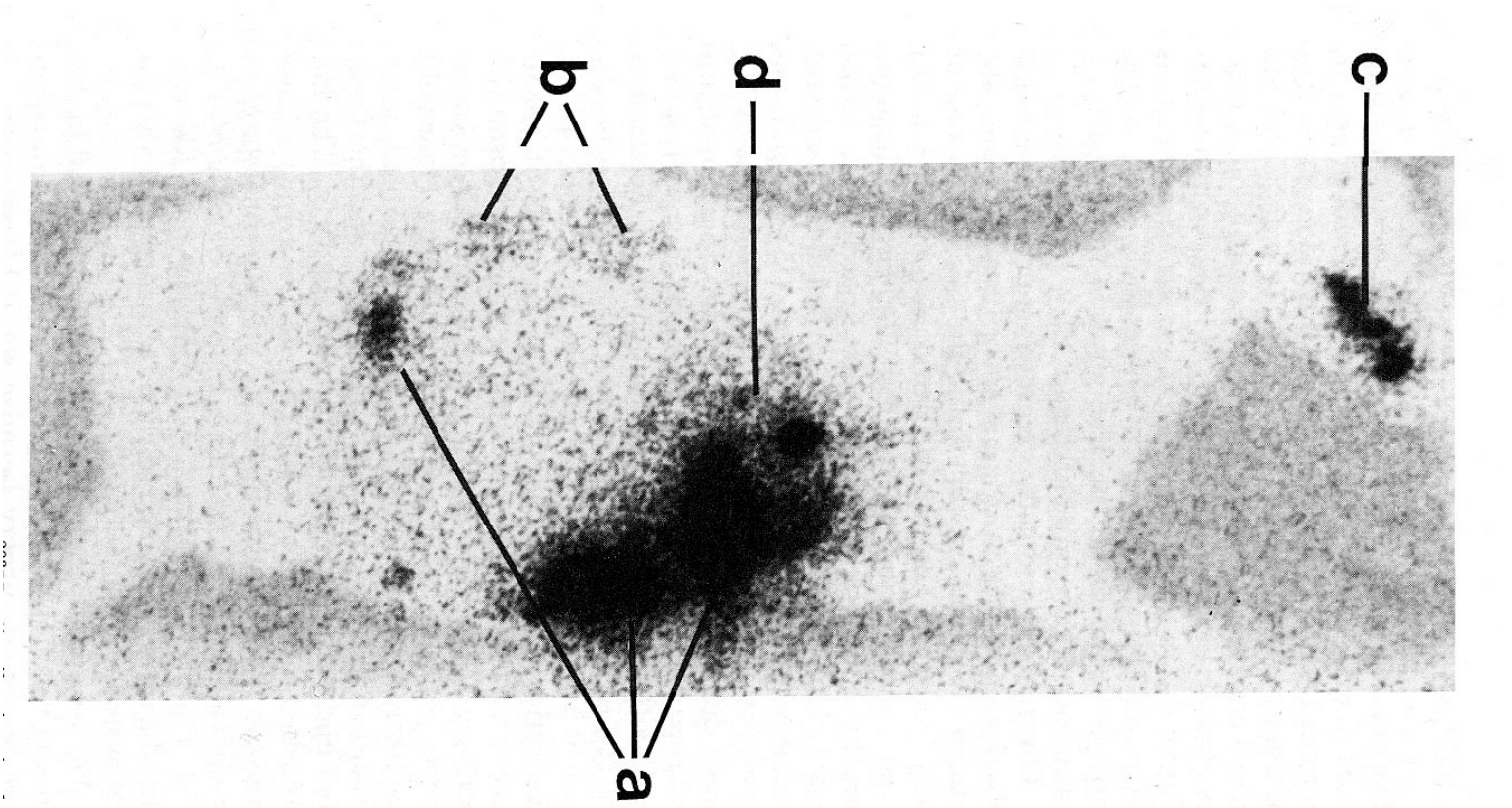


Figure 4 – Full body scan of a sheep 29 days after placement of 12 occlusal amalgams labeled with 203Hg. The fillings were removed prior to the scan. (a) digestive tract. (b) kidneys. (c) gums and alveolar bone. (d) liver, partially obscured by the digestive tract. (From Hahn, et. al., 1989)

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## PUBLIC COMMENT

## EXHIBIT 3

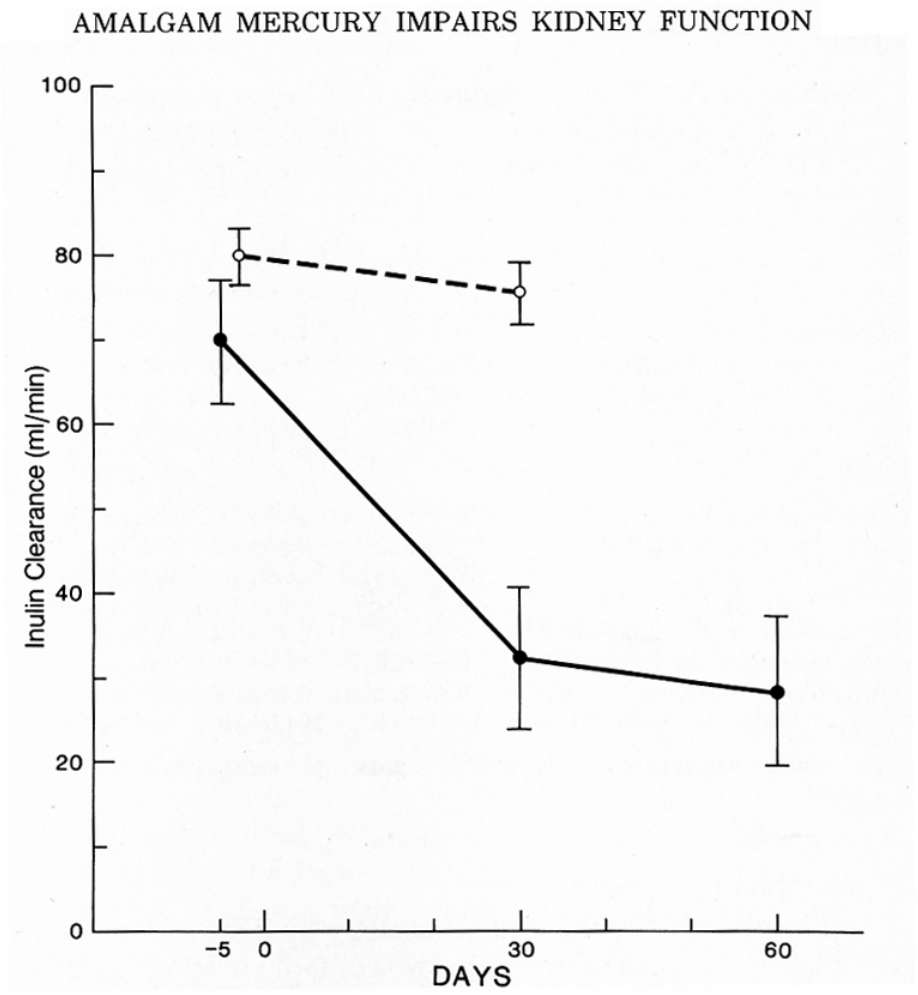


Figure 6 – Plasma inulin clearance (+/- SEM) of six sheep with twelve occlusal amalgam fillings (solid line) and two controls with glass ionomer fillings (dashed line). (from Boyd, et. al., 1991)

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 4

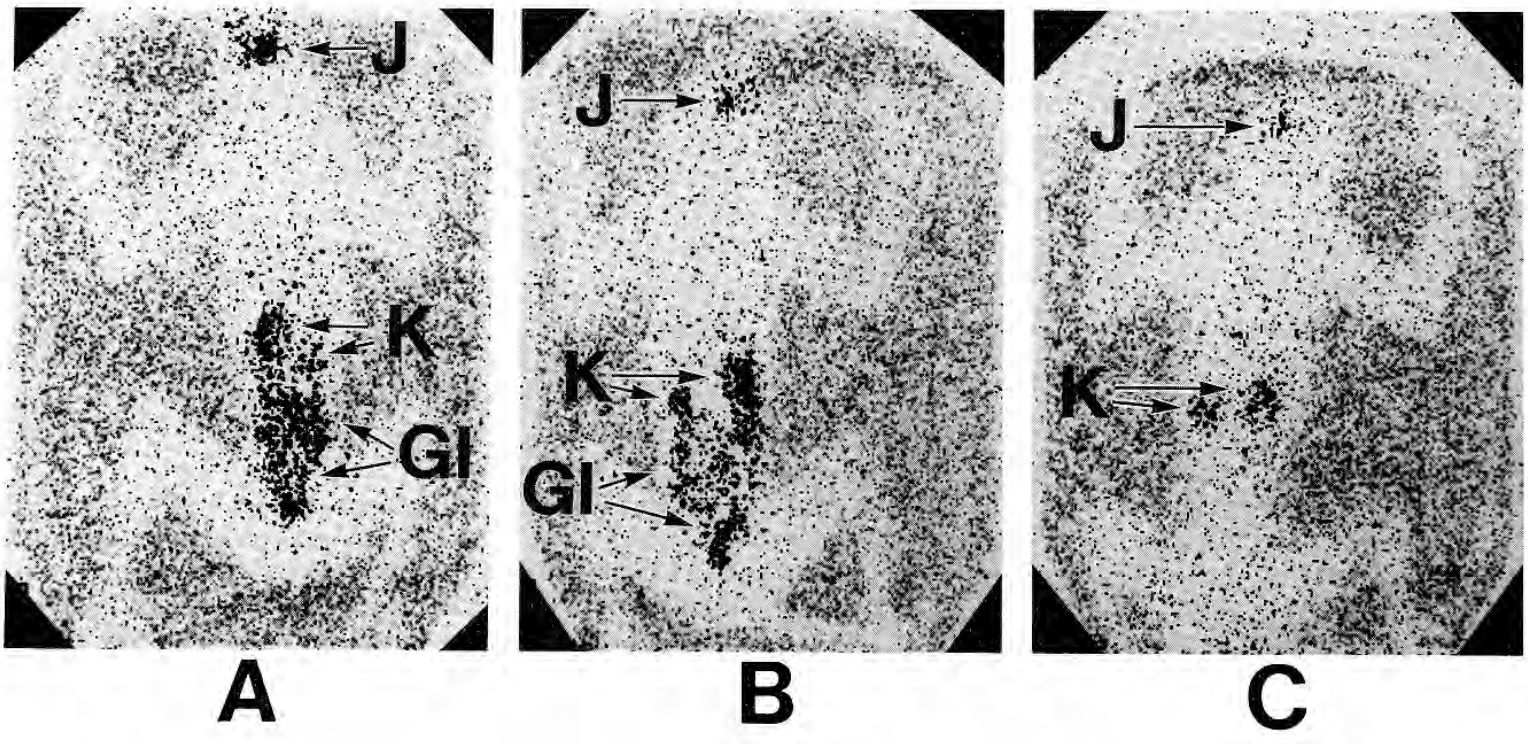


Figure 5 – Full body scan of a monkey 28 days after the placement of 16 occlusal fillings, labeled with 203Hg, showing radioactivity in the jaws, kidneys and GI tract. (A) ventral view. (B) dorsal view. (C) dorsal view with the GI tract removed, clearly showing radioactive mercury accumulation in the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

INTERNATIONAL ACADEMY OF
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PUBLIC COMMENT

EXHIBIT 5

## REFERENCES DOCUMENTING SYMPTOMS TO MERCURY EXPOSURE

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# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 6

### Questions for Committee Consideration

1) Based on the peer-reviewed scientific literature, the draft FDA White Paper, and any other information, including the information you heard in the public session, please discuss the following topics, including any issues of quality, experimental design, or other attributes of specific studies that may affect the weight that should be given to conclusions drawn from them:

a) Please discuss the direct evidence, if any exists, supporting or refuting the occurrence of adverse health effects from mercury vapor released from dental amalgam devices.

b) Please discuss tile indirect evidence (e.g., extrapolation from higher dose studies, animal studies), if any exists, supporting or refuting a link between dental amalgam devices and adverse neurological effects at the absorbed doses received from these devices.

c) Please discuss the indirect evidence (e.g., extrapolation from higher dose studies, animal studies), if any exists, supporting or refuting a link between dental amalgam devices and adverse non-neurological effects at the ab orbed doses received from these devices

d) Please discuss the indirect evidence (e.g., extrapolation from higher dose studies, animal studies) , if any exists, supporting or refuting a link between dental amalgam devices and adverse effects specific to vulnerable populations such as children and pregnant women at the absorbed doses received from these devices.

2) Does the draft FDA White Paper objectively and clearly present the current state of knowledge about the exposure and health effects related to dental amalgam?

3) Given the amount and quality of the information available for the draft FDA White Paper, are the conclusions reasonable?

INTERNATIONAL ACADEMY OF
ORAL MEDICINE AND TOXICOLOGY

PUBLIC COMMENT

EXHIBIT 7

**Exhibit 7**

# 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 and provide relevant documentation from the peer reviewed scientific literature.

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. In an elegant series of experiments he documented that his own breath contained 10 micrograms of mercury per cubic meter of air [12]. His conclusion was, "It will then likely be found that the thoughtless introduction of amalgam as a filling material for teeth was a severe sin against humanity." 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 spectrophotometer and the Jerome mercury vapor detector [3]. Today 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 [4]. 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 the IAOMT website under articles (www.iaomt.org), 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? It is a simple mixture like alcohol and water. 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 with the other metals. Figure 1 is a photomicrograph of a polished metallurgic sample of amalgam which has been pressed on by a round micro-probe [5]. Where the probe touched the surface, droplets of free liquid mercury appear on the surface because they were squeezed into view. This process does not require heating the sample, as some have objected; it was repeated down to the temperature of liquid nitrogen [6].
[See Masi Exhibit 14]

The clearest, most gut wrenching way to comprehend that amalgam releases free elemental mercury vapor 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, "Smoking Teeth," is available for download or viewing 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 25 years. A pdf version with scientific references and still photos is also available download. [Exhibit 9 ] The Smoking Tooth.

This graphically dramatic process was hinted at by the fact that old amalgams contain significantly less mercury than new ones [7] [8]. It was quantified in the human mouth by Svare, et. al., Gay et. al., Vimy and Lorscheider, and others [9] [10] [11] [12] [13]. 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[14] and Berglund et al. [15], 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.[16] reported absorbed doses of as much as 27 μg per day. Skare and Engqvist,[17] 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 [18], [Exhibit 13] 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[19] presented a chart [Exhibit 12] 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 [20] 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 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 [Exhibit 2] was the result [21].

The graphic results are dramatic. [Exhibit 2] 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 [22].

| 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 $Hg^{203}$ 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 [23]. 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.[24] 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. [Exhibit 4]

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 [25] [26] [27] [28] [29]. Blood levels of mercury correspond to amalgam exposure [30] [31] [32]. Subjects with amalgam excrete higher amounts of mercury in the feces [33] [34]. Mercury in urine, blood, and feces declines after amalgam removal [35] [36] [37].

Aposhian et al.,[38] 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 [39] [40].

**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" [41].

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 [42] [43] [44] [45] [46] [47] [48] (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 [49]. 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, [50] 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 $Hg^{203}$, 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,[51] 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 $Hg^{2+}$ [52]. Drasch, et al.[53] 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 [54] [55].

## 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.[56] [57]

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 Exhibit 12]

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: [58]

- 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. [59] By using antibody – antigen flocculation tests on blood serum, the number is over 90%.[60] Djerassi and Berova [61] 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. [62] 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. [63]

Mercury exposure is know to induce autoimmune reactions in susceptible animals,[64] [65] [66] and one investigation shows the same for amalgam. Hultman et. al.[67] 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. [68] 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 Exhibit 3] 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.[69] Molin, et. al.[70] 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.

## 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? [71]

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. [72]

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.[73] 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.[74] [75]

The urinary mercury levels of 4272 dentists were measured at random at dental conventions by Naleway,[76] 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. [77] Many other studies point to a negative effect of mercury vapor exposure on reproductive outcomes. [78 79 80 81]

Depression and mood alteration is a known feature of chronic mercury toxicity.[82] 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. [83 84 85 86] Other labs found elevated mercury in the blood and cerebrospinal fluid of AD patients. [87 88]

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.[89] 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! [90]

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. [91 92 93]

The mercury story is making its way in the laboratory, if not yet in the press. Recently, Olivieri, et. al.[94] 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,[95] 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.[96 97] 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.[98]

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. [99] 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. Exhibit 15 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.

## 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. [100]

| 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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INTERNATIONAL ACADEMY OF
ORAL MEDICINE AND TOXICOLOGY

PUBLIC COMMENT

EXHIBIT 8

# NORTH CAROLINA STATE BOARD OF DENTAL EXAMINERS



July 9, 2007

98-587-6822

Dr. W. Carl McMillan, DMD
2024 Renaissance Park Place
Cary NC 27513

Request for Declaratory Ruling

Dear Dr. McMillan

On May 10, 2007, the N.C. State Board of Dental Examiners received your request for a declaratory ruling regarding certain statements that you desire to include in advertisements and in statements to patients and prospective patients. Pursuant to 21 NCAC 16N .0402, the Board has enclosed its response to your request.

Thank you.

Very truly yours,

Carolin Bakewell

Carolin Bakewell
Board Counsel

Enclosure
cc: Dr. Joseph S. Burnham, President
Bobby D. White, Chief Operations Officer

| | |
|---|---|
| STATE OF NORTH CAROLINA | BEFORE THE NORTH CAROLINA STATE BOARD OF DENTAL EXAMINERS |
| In re: W. Carl McMillan, DMD Petitioner | DECLARATORY RULING |

## PROCEDURAL HISTORY

On or about May 10, 2007, W. Carl McMillan, DMD (Petitioner), submitted a Request for Declaratory Ruling (Petition) to the North Carolina State Board of Dental Examiners (Board) pursuant to 21 NCAC 16N .0402. The Petitioner asked the Board to determine whether he may make certain statements in advertisements and/or communications with patients and prospective patients without risking discipline by the Board for making false or misleading communications in violation of 21 NCAC16P .0101. The Petitioner indicated that he did not wish to appear at an oral hearing before the Board. The Petitioner's current request follows a similar petition submitted by the Petitioner on Dec. 8, 2006, to which the Board responded on February 5, 2007. The Board's February 5, 2007 Declaratory Ruling is incorporated herein by reference.

## PETITIONER'S CURRENT INQUIRY

The specific statements about which Petitioner inquires fall into two categories. First, Petitioner presents a series of statements that he wishes to make to patients and prospective patients. Second, he inquires about a set of nearly identical statements that he proposes to disseminate as newspaper advertisements.

## A. STATEMENTS TO PATIENTS AND PROSPECTIVE PATIENTS

The specific statements that the Petitioner desires to make to patients and prospective patients are as follows:

1. My dental practice is "mercury-free."

2. People should eliminate their exposure to mercury.

3. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that people should eliminate their exposure to mercury.

4. Silver fillings contain mercury that may leak into your body and cause health problems.

5. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that silver fillings contain mercury that may leak into your body and cause health problems.

6. Silver fillings contain mercury that may leak into your body.

7. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that silver fillings contain mercury that may leak into your body.

8. Mercury can cause health problems.

9. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that mercury can cause health problems.

10. Mercury fillings are harmful.

11. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that mercury fillings are harmful.

12. Even non-allergic persons should have their mercury fillings replaced for the purpose of removing a toxic substance from the body.

13. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that even non-allergic persons should have their mercury fillings replaced for the purpose of removing a toxic substance from the body.

14. Amalgam fillings represent the largest source of mercury exposure in the general population.

15. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that amalgam fillings represent the largest source of mercury exposure in the general population.

16. Ingestion of mercury derived from dental amalgam can cause neurotoxic effects.

17. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that ingestion of mercury derived from dental amalgam can cause neurotoxic effects.

18. Ingestion of mercury derived from dental amalgam can cause nephrotoxic effects.

19. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that ingestion of mercury derived from dental amalgam can cause nephrotoxic effects.

20. The inhalation of mercury derived from dental amalgam can cause bronchiolitus, pneumonitis and/or pulmonary edema.

21. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that the inhalation of mercury derived from dental amalgam can cause bronchiolitus, pneumonitis and/or pulmonary edema.

22. Ingestion and/or inhalation of mercury derived from dental amalgam can aggravate kidney disorders.

23. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that ingestion and/or inhalation of mercury derived from dental amalgam can aggravate kidney disorders.

24. Ingestion and/or inhalation of mercury derived from dental amalgam can cause nervous irritability, weakness, tremors, gingivitis, erethism and/or greying of the lens of the eye.

25. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that ingestion and/or inhalation of mercury derived from dental amalgam can cause nervous irritability, weakness, tremors, gingivitis, erethism and/or greying of the lens of the eye.

26. Ingestion and/or inhalation of mercury derived from dental amalgam by pregnant women can be harmful to their unborn children.

27. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that ingestion and/or inhalation of mercury derived from dental amalgam by pregnant women can be harmful to their unborn children.

28. Dental amalgams should not be placed in pregnant women or woman [sic] of child-bearing age.

29. In my honest opinion, the greater weight of peer-reviewed scientific research demonstrates that dental amalgams should not be placed in pregnant women or woman [sic] of child-bearing age.

30. My dental practice is "mercury-free." People should eliminate their exposure to mercury. Silver fillings contain mercury that may leak into your body. Mercury can cause health problems. Mercury fillings are harmful. Amalgam fillings represent the largest source of mercury exposure in the general population. Ingestion of mercury derived from dental amalgam can cause neurotoxic and nephrotoxic effects. The inhalation of mercury derived from dental amalgam can cause bronchiolitus, pneumonitis and/or pulmonary edema. Ingestion and/or inhalation of mercury derived from dental amalgam can aggravate kidney disorders. Ingestion and/or inhalation of mercury derived from dental amalgam can cause nervous irritability, weakness, tremors, gingivitis, erethism and/or greying of the lens of the eye.

B. STATEMENTS IN NEWSPAPER ADVERTISEMENTS

Petitioner also asks whether he may disseminate certain statements in newspaper advertisements. The text of the proposed advertisements matches the statements Petitioner wishes to make to patients and prospective patients, except that each newspaper ad would also disclose the Petitioner's name and dental degree. For example, as to Petitioner's second inquiry, the advertisement would read: "People should eliminate their exposure to mercury. – W. Carl McMillan, D.M.D."

In his Request for a Declaratory Ruling, Petitioner represents that each of the 30 foregoing statements constitutes the complete communication or advertisement. Petitioner also explicitly represents that the advertisement or communication would not be part of a larger or different ad or communication and that no other information or disclaimer would be included. In issuing the requested declaratory rulings, the Board

Second, to comply with the standard of care, dental professionals must ensure that their patients give informed consent to any plan of treatment. In the case of fillings, dentists must educate their patients about the available filling materials, relative costs, risks and benefits of each treatment option and the preferred mode of treatment in light of each patient's particular needs and circumstances. In the case of a patient who is considering replacing amalgam fillings with another material, the dentist should inform the patient that living tooth structure will have to be removed to extract and replace the filling. Likewise, the patient should be warned that the procedure could cause the tooth to fracture or could kill the nerve of the tooth, necessitating a root canal.

Since the statements proposed by Petitioner do not contain any such discussion, they do not comply with the standard of care. Petitioner might be subject to discipline for neglect and incompetence in violation of G.S. §§90-41(a)(12) and (14) if he proceeds as proposed.

Third, the statements improperly portray the alleged dangers of dental amalgam as established fact. Leading health organizations such as the Federal Drug Administration (FDA), World Health Organization (WHO), and American Dental Association (ADA) have rejected similar assertions as unsupported by peer-reviewed, controlled research. To avoid misleading patients and potential patients, Petitioner must disclose that the FDA, WHO and ADA have approved dental amalgam for use in non-allergic individuals. If Petitioner makes statements 4, 6, 10, 16, 18, 20, 22, 24, 26, 28 or 30 to patients or prospective patients without the disclosure, he could be subject to discipline for making a misleading statement in violation of 21 NCAC 16P .0101.

2. Personal Opinions Regarding Studies of Amalgam

Proposed statements numbers 5, 7, 11, 13, 15, 17, 19, 21, 23, 25, 27 and 29 also discuss the alleged harmful effects of amalgam or "mercury fillings." These statements are identical to proposed statements number 4, 6, 10, 16, 18, 20, 22, 24, 26, and 28 except that they are couched as Petitioner's personal opinion regarding what the "greater weight of peer-reviewed scientific research demonstrates" regarding amalgam.

Petitioner owes a duty to ensure that his patients and prospective patients are fully informed before they make treatment decisions. Petitioner must therefore disclose that the ADA, FDA and WHO have approved dental amalgam as safe for use in non-allergic individuals. If Petitioner makes statements 5, 7, 11, 13, 15, 17, 19, 21, 23, 25, 27 or 29 to patients or prospective patients without the disclosure, he could be subject to discipline for failing to comply with the standard of care in violation of G.S. §§90-41(a)(12) and (14).

3. Statements Regarding Mercury

Proposed statements 3 and 9 discuss Petitioner's opinions regarding what the "greater weight of peer-reviewed scientific research demonstrates" about mercury. Proposed statement number 8 states that mercury can cause health problems. Petitioner has represented that these statements about mercury would not be made in the context of any other discussion, including any reference to amalgam fillings or the practice of dentistry. The difference between proposed statements 3, 8 and 9 and the proposed opinion statements about amalgam fillings is crucial. Because statements 3, 8 and 9 do not deal with the use of mercury in dentistry, the statements are unlikely to impact treatment decisions of Petitioner's patients and prospective patients. It follows that

Petitioner may give his opinion about matters outside the scope of dentistry without further disclaimers or explanations. The Board cautions Petitioner, however, that its opinion regarding proposed statements 3, 8 and 9 are limited to the questions as drafted and is based on the representations contained in Petitioner's request. For example, the inclusion of proposed statement number 8 in the context of other statements about amalgam or the practice of dentistry, such as set out in proposed statement number 30, is not covered by this ruling.

4. Miscellaneous Statements to Patients and Prospective Patients

**Inquiry No. 1. My dental practice is "mercury-free."**

Petitioner may make this statement to patients and potential patients if Petitioner's practice is in fact entirely free of mercury. If, however, the Petitioner continues to use equipment that contains mercury, such as thermometers and fluorescent lights, or removes amalgam fillings, the statement would be false and/or misleading and would violate 21 NCAC16P.0101.

**Inquiry No. 2. People should eliminate their exposure to mercury.**

This statement implies that it is possible to completely eliminate all exposure to mercury. Total elimination of mercury exposure is not possible, as mercury is present in the environment, some fish and shellfish, and in man-made objects, such as thermometers, fluorescent lights and batteries. Moreover, there is a substantial risk that ordinary, prudent patients and prospective patients will confuse dental amalgam with other forms of mercury in the environment. As drafted, this statement is misleading and violates 21 NCAC 16P .0101.

**Inquiry No. 12. Even non-allergic persons should have their mercury fillings replaced for the purpose of removing a toxic substance from the body.**

The suggestion that all individuals should have amalgam fillings replaced to remove a toxic substance from the body is problematic for three reasons. First, it is not proper for a dentist to suggest treatment without first evaluating the patient and his or her medical history. There is nothing in Petitioner's statement to suggest that this has been done. As drafted, the proposed statement violates the standard of care and G.S. §§90-41(a)(12) and (14). See discussion in Section A(1), above.

Second, the reference to amalgam fillings as "toxic substances" is not supported by reliable peer-reviewed scientific studies. The great bulk of credible studies indicate that amalgam fillings are safe for non-allergic individuals. Unless Petitioner advises patients and prospective patients that the FDA, ADA and WHO have approved amalgam fillings for non-allergic patients, the statement as drafted is misleading and therefore violates 21 NCAC 16P .0101.

Third, it is not true that amalgam fillings should be removed from all patients, regardless of their health, age, finances, and aesthetic concerns. Setting aside the risks associated with removing otherwise serviceable fillings and the dubious "benefit" of removing dental amalgam from non-allergic individuals, some patients will be unable to afford to replace all of their amalgam fillings, while others will be too old or ill to tolerate the procedure. Petitioner's blanket suggestion that all individuals would benefit from the removal of all amalgam fillings is thus false and/or misleading. Petitioner's statement as drafted violates 21 NCAC 16P. 0101.

**Inquiry No. 14. Amalgam fillings represent the largest source of mercury exposure in the general population.**

This statement is false. As drafted, it violates 21 NCAC 16P .0101.

B. PROPOSED NEWSPAPER ADS

The Petitioner has also inquired whether he may properly disseminate certain statements in newspaper advertisements. The statements are identical to those he proposes to make to patients and prospective patients, with the addition of his name and dental degree. The proposed advertisements fall into three categories: statements about the alleged harmful effects of amalgam fillings, statements couched as Petitioner's opinion regarding scientific studies about amalgam fillings or mercury and several miscellaneous proposed statements. The Board will address each category of proposed advertisement in turn.

1. Assertions Regarding Alleged Harmful Effects of Amalgam

Proposed statements numbers 4, 6, 10, 16, 18, 20, 22, 24, 26, 28 and 30 make various assertions about the alleged harmful effects of amalgam and "mercury" fillings. The statements are phrased as statements of fact, rather than Petitioner's personal opinion and they are clearly attributed to Petitioner in his capacity as a licensed dentist. The public is likely to place considerable reliance upon these statements, owing to Petitioner's specialized training and experience as a dentist.

A substantial risk exists that an ordinary, prudent reader of such advertisements will not know that similar statements have been rejected by leading government, health and dental organizations such as the FDA, ADA and WHO as unsupported by objective, peer-reviewed scientific studies. Even if aware of such information, few ordinary,

prudent readers will be equipped to find and understand the often highly technical scientific literature on the subject of dental amalgams. Consequently, there is a substantial risk that the statements could or would mislead members of the public into believing that the assertions are accepted by the great majority of dentists and government health care agencies, which is not accurate. Unless the ads also disclose that the ADA, FDA and WHO have approved dental amalgam for use in non-allergic individuals, the statements as drafted are misleading and therefore violate 21 NCAC 16P .0101.

2. Personal Opinions Regarding Studies of Amalgam

Proposed statements numbers 5, 7, 11, 13, 15, 17, 19, 21, 23, 25, 27 and 29 also discuss the alleged harmful effects of amalgam fillings or mercury. These statements correspond to proposed statements number 4, 6, 10, 16, 18, 20, 22, 24, 26, and 28 except that they are couched as Petitioner's personal opinion regarding what the "greater weight of peer-reviewed scientific research demonstrates" regarding amalgam or mercury.

As presented, Petitioner merely proposes to state to the public at large his opinion regarding what certain studies do or do not prove. He owes no particular duty of care to individuals who may read the newspaper ads, as they are not patients or prospective patients. Dentists in this state are not subject to discipline merely for expressing their personal opinions in a newspaper advertisement. Consequently, Petitioner may disseminate proposed statements numbers 5, 7, 11, 13, 15, 17, 19, 21, 23, 25, 27 and 29, as drafted, without risking discipline by the Board for violating 21 NCAC 16P .0101, provided that he also includes in the advertisement that he is a general dentist as required by 21 NCAC 16P. 0102. Petitioner should be mindful, however, that there is no First

Amendment right to practice incompetent dentistry and that whatever treatment he undertakes must comport with the accepted standard of care in this state. Moreover, the Board strongly disagrees with the opinions expressed by Petitioner and its ruling herein, which is narrowly confined to the precise language posed, should not be construed as support for the propositions set out in 5, 7, 11, 13, 15, 17, 19, 21, 23, 25, 27 and 29.

3. Statements Regarding Mercury

Proposed statements 3 and 9 discuss Petitioner's opinions regarding what the "greater weight of peer-reviewed scientific research demonstrates" about mercury. Proposed statement number 8 states that mercury can cause health problems. As drafted, these statements may be made in the newspaper ads proposed by Petitioner without further disclaimer, for the reasons set out in Section A(3).

4. Miscellaneous Statements in Newspaper Ads

**Inquiry No. 1. My dental practice is "mercury-free."**

See discussion in Section A(4).

**Inquiry No. 2. People should eliminate their exposure to mercury.**

See discussion in Section A(4).

**Inquiry No. 12. Even non-allergic persons should have their mercury fillings replaced for the purpose of removing a toxic substance from the body.**

The suggestion that amalgam fillings are "toxic substances" that should be removed from the body in every case is not supported by objective, peer-reviewed scientific studies. Moreover, it is not true that removal of dental amalgam is indicated for every individual, regardless of his or her age, finances and overall health. As drafted,

therefore, this statement is false and/or misleading and may not be disseminated in a newspaper ad. See discussion in Section A(4).

**Inquiry No. 14. Amalgam fillings represent the largest source of mercury exposure in the general population.**

This statement is false. See discussion in Section A(4).

This the 9th day of July, 2007.

Joseph S. Burnham, D.D.S.

Dr. Joseph Burnham, President
The N.C. State Board of Dental Examiners

has relied upon the representations in Petitioner's request. Any change in these representations or other conditions not included in Petitioner's request might alter the Board's responses. The Board will address each of Petitioner's inquiries in turn.

## DECLARATORY RULING

### A. STATEMENTS TO PATIENTS AND PROSPECTIVE PATIENTS

#### 1. Statements Concerning Alleged Dangers of Amalgam Fillings

Petitioner may not properly make proposed statements numbers 4, 6, 10, 16, 18, 20, 22, 24, 26, 28 or 30 to patients or prospective patients, for several reasons. First, as drafted, the statements appear to be intended as advice to patients or prospective patients, yet there is no indication that the dentist has gathered any information about the patient's health history, aesthetic concerns, finances or treatment goals before making the statements. Dentists must collect this background information to enable them to make appropriate treatment recommendations. For example, by reviewing the health history the dentist will learn whether the patient has conditions that might impact treatment decisions. A patient with a history of certain heart problems, for instance, may require pre-medication before a procedure.

Awareness of a patient's finances and aesthetic concerns may also impact treatment. A well-to-do patient may opt for gold fillings, whereas patients with limited resources may select less expensive amalgam fillings. Until a dentist is fully informed about each patient's needs and desires, the dentist cannot make an informed recommendation about the proper treatment for each patient. Failing to collect relevant data could subject the dentist to discipline for negligence or incompetence in violation of N.C. Gen. Stat. §§90-41(a)(12) and (14).

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 9

25 - year old mercury / silver amalgam filling with visible elemental mercury vapor after stimulation with warm water ( from the Smoking Teeth Video )

INTERNATIONAL ACADEMY OF
ORAL MEDICINE AND TOXICOLOGY

PUBLIC COMMENT

EXHIBIT 10

O'CONNOR ASSOCIATES ENVIRONMENTAL INC.

639 5TH AVENUE S.W., SUITE 1000, CALGARY, ALBERTA T2P 0M9 TELEPHONE: (403) 294-4200 FAX: (403) 294-4240 WATTS: (800) 661-8141

Ottawa office: 14 Clarendon Ave., Ottawa, ON CANADA K1Y 0P2
Phone/FAX: (613) 729-8536 E-Mail: gmrich@sonetis.com

April 17, 1998 10-5127

Mr. James Love
Attorney at Law
4363 East 70th Street
Tulsa, OK 74139-4605

Dear Mr. Love:

Re: Estimated Mercury Exposure Resulting from the Placement of a New Amalgam in Dr. Dixie Cranmer-McReynolds, D.C.

The purpose of this letter is to complete the estimates of exposure to mercury experienced by Dr. Cranmer-McReynolds, as a result of her dental treatment by Dr. Mindrup. Through an oversight on my part, my letter of April 2, 1998 did not include the additional mercury exposure resulting from the placement of the new amalgam filling.

The dose of mercury systemically absorbed by Dr. McReynolds as a result of the placement of a new amalgam filling, following the removal of an old filling, is conservatively estimated to be 93 µg. This brings the total estimated exposure from all sources (amalgam removal, amalgam placement, waste mercury and waste amalgam in the dixie cup) and pathways (inhalation of vapours, inhalation of particulate matter, ingestion of particulate matter) to 1,807 µg.

The calculations, assumptions and reasoning employed to estimate Dr. McReynold's mercury exposure due to the placement of the new amalgam are set out below. The filling was described by Dr. Mindrup as being a "very large" 3-surface amalgam filling located in the upper left first molar of Dr. McReynolds. Therefore, it was assumed that the filling was of general 'average' size, thereby ensuring that exposure was not over-estimated.

For the placement procedure, the following equation was constructed:

Total dose = vapour inhalation dose + particle inhalation dose + particle ingestion dose (1)

Vapour inhalation dose = $C_{air}$ * IR * D * AR (2)

.../2

where, $C_{air}$ = concentration in air during procedure ($\mu g/m^3$)
IR = inhalation rate ($m^3/hr$)
D = duration of procedure (hours)
AR = absorption rate for mercury in the lungs (%)

Haikel et al.(1990) and Powell et al. (1994) have demonstrated, qualitatively, that insertion of an amalgam filling produces the same average breathing zone mercury level as does amalgam removal. Therefore, the data of Richards and Warren (1985) equally apply to insertion as to removal. Unfortunately, Hailel et al. (1990) and Powell et al. (1994) do not employ continuous monitoring for their studies. Instead, they use spot monitoring, which is unreliable for a quantitative evaluation of inhalation exposure for short-term procedures such as amalgam removal and placement. Richards and Warren (1985) use continuous monitoring.

Tituration, condensation, insertion and carving of the newly placed amalgam (to achieve occlusal contouring) requires approximately 13 minutes (Powell et al., 1994).

Inhalation rate and absorption rate for mercury in the lungs will be the same as that described in my letter to you dated April 2, 1998.

Therefore,

$C_{air}$ = 100 $\mu g/m^3$ (Richards and Warren 1985; wet grinding with aspiration)
IR = 15 $m^3$/24 hours (average for a female aged 20 to 59 years)
= 0.63 $m^3$/hour (Allan and Richardson, 1998)
D = 13 minutes = 0.22 hours (Powell et al., (1994)
AR = 80% or 0.8 (U.S. ATSDR, 1994)

As a result, the inhalation dose due to placement of the new amalgam was:

Inhalation dose = 100 $\mu g/m^3$ * 0.63 $m^3$/hour * 0.22 hour * 0.8 = 11.1 $\mu g$

Particle inhalation dose

The procedures of grinding the newly-set amalgam to contour the occlusal surface for proper occlusion with opposing teeth, as well as final polishing, will release amalgam particulate matter. It is reasonable to assume that these procedures will release particulate amalgam proportional to that of a removal, recognizing that much less than 100% of the new amalgam is ground or polished off to achieve proper occlusion and finishing of the filling. Perhaps 0.1 mm (4 thousandths of an inch)

.../3

of the surface will be removed to achieve occlusion and polishing. This is 6.6% of an average amalgam which is 1.5 mm in depth into the tooth (Nimmo et al., 1990). Therefore, it can be conservatively assumed that 5% of the amalgam originally placed in the prepared cavity would be ground and polished off to achieve occlusion and finishing of the filling. During an amalgam removal, obviously 100% of the amalgam is ground out and removed.

Therefore, the particulate inhalation exposure arising from the placement of a new amalgam is conservatively estimated at 5% of that arising from removal:

Particulate inhalation dose = Particulate inhalation dose during removal * 5%
= 1600 µg * 5%
= 80 µg

Particle ingestion dose

The procedures of grinding the newly-set amalgam to contour the occlusal surface for proper occlusion with opposing teeth, as well as final polishing, will release amalgam particulate matter into the oral cavity which will be ingested. It is reasonable to assume that these procedures will release particulate amalgam proportional to that of a removal, but recognizing that much less than 100% of the new amalgam is ground or polished off to achieve proper occlusion and finishing of the filling. Conservatively, 5% of the amalgam originally placed in the prepared cavity would be ground and polished off to achieve occlusion and finishing of the filling.

Therefore, the particulate ingestion exposure arising from the placement of a new amalgam is conservatively estimated at 5% of that arising from removal:

Particulate ingestion dose = Particulate ingestion dose during removal * 5%
= 31.2 µg * 5%
= 1.6 µg

Therefore, the total dose of mercury received by Dr. McReynolds during the placement of the new amalgam was 11.1 µg + 80 µg + 1.6 µg = 92.7 µg

Considering the calculations above and my calculations contained in my letter to you dated April 2, 1998, the total absorbed dose experienced by Dr. McReynolds is conservatively estimated to be 1,807 µg (1,644 µg due to amalgam removal procedure, 93 µg due to amalgam placement, and 70 µg due to poor hygiene practices leading to air contamination). This likely under-estimates the true exposure received by Dr. McReynolds for the following reasons:

.../4

- The amalgam filling was assumed to be of average size, approximately 750 mg of amalgam. The amalgam filling removed from Ms. McReynolds' was described as "very large". Dr. J. Osborne, on behalf of the defendants in this case, estimated the size of Dr. McReynold's filling to be 990 mg of amalgam. Therefore, it is likely that it exceeded the size assumed by me for purposes of this exposure assessment.

- The concentration in the air of the patient's breathing zone during the drilling, removal and placement of the amalgam filling was most likely greater than the assumed 100 $\mu g/m^3$. This assumption was derived from experimental work presented by Richards and Warren (1985). In their work, the background concentration of mercury in the dental operatory was less than 50 $\mu g/m^3$. Dr. Mindrup's dental operatory maintained open containers of liquid mercury and also had amalgam scrap and other mercury-contaminated waste and debris in unsealed trash containers. These will have contaminated the air quality of the dental operatory in excess of 50 $\mu g/m^3$, as calculated above. Therefore, the combination of operatory air contamination and the increased contamination resulting from the removal of the amalgam filling likely combined to exceed the assumed 100 $\mu g$ $Hg/m^3$.

- The inhalation rate used here was based on the average rate over 24 hours, which includes low inhalation rates common during sleep; inhalation rates are higher when a person is anxious, nervous and active (as might be expected when attending a dental appointment).

- The assumed surface area of the dixie cup holding waste mercury was for the bottom of the dixie cup. Dixie cups are tapered. A dixie cup of approximately 2.5 to 3 inches tall has a bottom radius of 2 cm and a top radius of 2.75 cm. Using the bottom diameter to estimate mercury vaporization will underestimate the actual air concentration.

- Dermal absorption has not been considered. Mercury vapour is readily absorbed through the skin at a rate of 240 $\mu g$ $Hg/m^2$ skin surface area per minute of exposure and per mg $Hg/m^3$ in air. Dermal absorption is estimated to increase the total dose from contaminated air by approximately 3% over that by inhalation alone (Hursh et al., 1989).

- The estimate of exposure due to ingestion of mercury as a result of the amalgam removal and placement only considered the mercury evident in faeces on day 2 following the removal procedure. Not all the mercury is passed in just 2 days (Bjorkman et al., 1997). Also, the oral mucosa act as a reservoir for mercury and ingestion of saliva presents a continuing source of mercury from the amalgam removal procedure even up to 60 days after the removal (Bjorkman et al., 1997).

Therefore, the conservative assumptions employed would combine to under-estimate, rather than over-estimate, the mercury exposure experienced by D. McReynolds.

.../5

Should you have any questions or require clarification, please contact me at your convenience.

Yours truly,

G. Mark Richardson, Ph.D.
Senior Risk Assessment Specialist

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April 2, 1998 10-5127

Mr. James Love
Attorney at Law
4363 East 70th Street
Tulsa, OK 74139-4605

Dear Mr. Love:

Re: Estimated Mercury Exposure Experienced by Dr. Dixie Cranmer-McReynolds, D.C.

As per your request, I have now completed my estimate of mercury exposure experienced by your client as a result of the removal and subsequent replacement of an amalgam filling. This estimated exposure is based on the information provided by you in your letter of March 25, 1998, as well as information contained in the depositions of Dr. Jerome Mindrup and Ms. Kim Lockhart, Dr. Mindrup's assistant.

The total dose of mercury systemically absorbed by Dr. McReynolds as a result of her dental treatment by Dr. Mindrup is conservatively estimated to be 1,714 μg. The total mercury exposure experienced by Dr. McReynolds was a combination of the direct exposure to mercury vapour and particulate matter resulting from the removal of an old amalgam filling and subsequent placement of a new amalgam filling, and due to the respiratory absorption of mercury vapours present in the dental operatory due to poor occupational and industrial hygiene practices respecting the storage and disposal of waste liquid mercury.

The calculations, assumptions and reasoning employed to estimate Ms. McReynold's mercury exposure are set out below. The filling was described by Dr. Mindrup as being a "very large" 3-surface amalgam filling located in the upper left first molar of Dr. D. McReynolds. Therefore, it was assumed that the filling was of general 'average' size, thereby ensuring that exposure was not over-estimated.

For the removal procedure, the following equations were constructed:

PLAINTIFF'S EXHIBIT A-21

.../2

Total dose = vapour inhalation dose + particle inhalation dose + particle ingestion dose (1)

Vapour inhalation dose = $C_{air}$ * IR * D * AR (2)

where, $C_{air}$ = concentration in air during procedure
= 100 µg/m³ (Richards and Warren 1985; wet grinding with aspiration)
IR = inhalation rate = 15 m³/24 hours (average for a female aged 20 to 59 years)
= 0.63 m³/hour (Allan and Richardson, in press)
D = duration of procedure = 0.25 hours (duration of McReynolds' drilling procedure)
AR = absorption rate for mercury in the lungs = 80% or 0.8 (U.S. ATSDR, 1994)

Therefore, inhalation dose = 100 µg/m³ * 0.63 m³/hour * 0.25 hour * 0.8 = 12.6 µg

Particle inhalation dose = PM * PHg * N * AR (3)

where, PM = particle mass deposited in lungs per average sized amalgam removed
= 4,000 µg (Nimmo et al. 1990; wet grinding)
P(Hg) = the weight percent of Hg in amalgam = 50% or 0.5 (Berry et al., 1994)
N = number of amalgam fillings = 1
AR = absorption rate for mercury in the lungs = 80% or 0.8 (U.S.ATSDR, 1994)

Therefore, particle inhalation dose = 4,000 µg * 0.5 * 1 * 0.8 = 1,600 µg

$$\text{Particle ingestion dose} = \sum_{i=1}^{n} ((C_{feces\text{-}i} * Mass_{feces\text{-}i} * AF * AR)/(1\text{-}AR)) \quad (4)$$

where, $C_{feces\text{-}i}$ = the concentration of Hg in feces (µg/g) on day i
= 17 µg/g feces (median value on day 2 following removal (Bjorkman et al. 1997)
$Mass_{feces\text{-}i}$ = the average mass of feces emitted on day i
= 150 g (Thomas, 1989)
AF = adjustment factor for 1 filling in McReynolds and 9.5 in Bjorkman et al. study
= 1/9.5 = 0.11
AR = absorption rate = 10% or 0.1 (U.S.ATSDR, 1994)

For only the peak day (day 2) particle ingestion dose = (17 µg/g*150 g*0.11*0.1)/ 0.9 = 31.2 µg

.../3

Therefore, Total dose = vapour inhalation dose + particle inhalation dose + ingestion dose
= 12.6 µg + 1,600 µg + 31.2 µg = 1,644 µg

Note that this accounts for ingestion exposure based only on Hg in feces on day 2 following removal of fillings; elevated levels of mercury were observed in feces up to 60 days (Bjorkman et al. 1997). The mercury present on day 2 will not be due to excretion of inhaled mercury as the whole body half-life for excretion of inhaled mercury is 2 months (Hursh et al. 1976).

Exposure to mercury vapour also resulted from the high levels of mercury that would have been in the office and operatory air due to the open containers (dixie cups) used to store waste liquid mercury. Mercury is extremely volatile; it is more volatile and evaporates more readily than water.

To estimate the exposure from the air in the operatory, unrelated to the removal procedure, it is first necessary to determine the evaporation rate of mercury. A 3 $cm^3$ (3 mL) volume of mercury will saturate the air in a room in 7 days (Chang, 1980). The radius of a sphere of mercury of 3 $cm^3$ volume is 0.895 cm. The surface area of a sphere of mercury of 3 $cm^3$ volume is 10.1 $cm^2$. The saturation concentration of mercury in air is 20 $mg/m^3$ (Shoemaker et al., 1996). For a room of 30 $m^3$ volume (approximate size if Dr. Mindrup's dental operatory (Lockhart deposition)) , the total mass of mercury in the room air at saturation is: 20 mg $Hg/m^3$ * 30 $m^3$ = 600 mg Hg. 600 mg of mercury would evaporate from 10.1 $cm^2$ surface area in 168 hours (7 days). Therefore, the evaporation rate for mercury is 0.35 mg $Hg/cm^2$/hour.

The concentration of mercury vapour in the operatory air due to the mercury present in the open dixie cups can be calculated as:

$$C_{air} = (ER_{Hg} * SA_{cup} * 24\ hr) / TV_{air} \qquad (5)$$

where, $C_{air}$ = concentration of Hg in air
$ER_{Hg}$ = evaporation rate of mercury = 0.35 $mg/cm^2$/hr
$SA_{cup}$ = surface area of dixie cup = 12.6 $cm^2$ (assumed radius of dixie cup is 2 cm)
$TV_{air}$ = total volume of air in office in 24 hours
= volume of room * air exchanges/hour * 24 hours
= 30 $m^3$ * 2 /hour * 24 hours
= 1,440 $m^3$

Therefore, $C_{air}$ = (0.35 mg $Hg/cm^2$/hr * 12.6 $cm^2$ * 24 hr) / 1,440 $m^3$ = 0.074 $mg/m^3$ = 74 $\mu g/m^3$

.../4

To estimate the exposure from the air, unrelated to the removal procedure:

Vapour inhalation dose = $C_{air}$ * IR * D * AR (6)

where, $C_{air}$ = concentration in operatory air = 74 µg Hg/m³
IR = inhalation rate = 15 m³/24 hours (average for a female aged 20 to 59 years)
= 0.63 m³/hour (Allan and Richardson, in press)
D = duration of time spent in the dental operatory = 1.5 hours (statement from Dr. McReynolds)
AR = absorption rate for mercury in the lungs = 80% or 0.8 (U.S. ATSDR, 1994)

Therefore, absorbed inhalation dose = 74 µg/m³ * 0.63 m³/hour * 1.5 hour * 0.8 = 69.9 µg

Considering all sources and routes of exposure, the total absorbed dose experienced by Dr. McReynolds is estimated to be 1,714 µg (1,644 µg due to amalgam removal procedure and 70 µg due to poor hygiene practices leading to air contamination). The estimated exposure set forth above likely under-estimates the true exposure received by Dr. McReynolds for the following reasons:

- The amalgam filling was assumed to be of average size. The amalgam filling removed from Ms. McReynolds' was described as "very large". Therefore, it is likely that it exceeded the size assumed for purposes of this exposure assessment.

- The concentration in the air of the patient's breathing zone during the drilling and removal of the amalgam filling was most likely greater than the assumed 100 µg/m³. This assumption was derived from experimental work presented by Richards and Warren (1985). In their work, the background concentration of mercury in the dental operatory was less than 50 µg/m³. Dr. Mindrup's dental operatory maintained open containers of liquid mercury and also had amalgam scrap and other mercury-contaminated waste and debris in unsealed trash containers. These will have contaminated the air quality of the dental operatory in excess of 50 µg/m³, as calculated above. Therefore, the combination of operatory air contamination and the increased contamination resulting from the removal of the amalgam filling likely combined to exceed the assumed 100 µg Hg/m³.

- The inhalation rate used here was based on the average rate over 24 hours, which includes low inhalation rates common during sleep; inhalation rates are higher when a person is anxious, nervous and active (as might be expected when attending a dental appointment).

- The assumed surface area of the dixie cup holding waste mercury was for the bottom of the dixie cup. Dixie cups are tapered. A dixie cup of approximately 2.5 to 3 inches tall has a bottom radius of 2 cm and a top radius of 2.75 cm.

- Dermal absorption has not been considered. Mercury vapour is readily absorbed through the skin at a rate of 240 $\mu g\ Hg/m^2$ skin surface area per minute of exposure and per mg $Hg/m^3$ in air. Dermal absorption is estimated to increase the total dose from contaminated air by approximately 3% over that by inhalation alone (Hursh et al., 1989).

- The estimate of exposure due to ingestion of mercury as a result of the amalgam removal only considered the mercury evident in feces on day 2 following the removal procedure. However, not all the mercury is passed in just 2 days (Bjorkman et al., 1997). Also, the oral mucosa act as a reservoir for mercury and ingestion of saliva presents a continuing source of mercury from the amalgam removal procedure even up to 60 days after the removal (Bjorkman et al., 1997).

Therefore, the conservative assumptions employed would combine to under-estimate, rather than over-estimate, the mercury exposure experienced by D. McReynolds.

Should you have any questions or require clarification, please contact me at your convenience.

Yours truly,

G. Mark Richardson, Ph.D.
Senior Risk Assessment Specialist

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INTERNATIONAL ACADEMY OF
ORAL MEDICINE AND TOXICOLOGY

PUBLIC COMMENT

EXHIBIT 11

# EFFECTS OF AMALGAM REMOVAL ON HEALTH

25 studies comprising 5821 patients


## Mats Hanson

Tandvårdsskadeförbundet, Tf;
Swedish Association of Dental Mercury Patients, 2004


### Introduction

The Swedish Association of Dental Mercury Patients (Tf) started the more systematic investigation of symptoms related to amalgam and the effects replacing the alloy with less toxic materials (Tf-bladet, 1986). Since then a considerable number of studies of varying quality have been presented, most of them by dentists. Most of them give a very consistent result: there is hardly any medical treatment which gives so positive results on so many health problems as amalgam removal.

Amalgam is a mixture of mercury and other metals (usually silver, tin, copper and sometimes zinc) and all metals are released by the fillings and are absorbed by the body. In addition, many persons are exposed to metals from other types of restorations, e.g. gold, platinum, palladium in pharmacologically relevant doses. "Amalgam poisoning" is therefore not quite equivalent with mercury poisoning. Many metals have similar biological effects and although mercury is the most poisonous of the dental metals, each one might have specific effects. However, treatment of most poisonings follow the same rule: elimination of exposure and addition of antidotes, nowdays often in the form of antioxidants.

### Comments on the various studies

No study can pass without objections. Criticism can be raised against selection of patients (self-selected), frequency of replies to questionnaires, variations in symptoms reporting, follow-up time, uncertainty about materials replaced (amalgam, gold alloys, gold + amalgam, metal-bound porcelain etc.), replacement materials, precautions during amalgam removal, rate of removal, dental cheating (amalgam remaining under plastics).

Some studies have overlapping patient material and especially the Swedish studies have more or less comprised members of the Swedish patient organisation, however, during a period of 16 years with a considerable turnover of members. Some studies have reported the state of health for all patients collectively in various phases of exchange, ongoing or at varying times after amalgam removal. Other studies have divided the patients into groups with completely removed amalgam, partly removed and controls without any exchange at all.

There is rarely any information on new treatments initiated in connection with amalgam removal. Especially have many patients started with antioxidant therapy as a new form of treatment, whereas conventional medical treatments do not appear to have had any remarkable effects and are not different from treatments which have been ineffective for years.

Even if some information is lacking in each study, the various investigations from different parts of the world complement each other and information lacking in one study is provided by other studies, however from other patients.

**Sex, age.**
Most of the patients are women, something which influences how they are treated in the health care system (negative influence). The mean percentage of women in the total of all studies is 71 %, most of them within the age range 40-60 years.

**Patient selection**
The reason these patients visit the dentist is to have amalgam removed. A minority of patients remove amalgam to reduce the risk of future problems, for the unpleasant feeling of having a load of poison in the mouth, for aesthetic or environmental reasons. One study has monitored health changes also in the group of persons that considered themselves healthy before amalgam removal (Eriksson et al, 2000). Often the removal of amalgam is a final, unpleasant and expensive measure after many years of ill health where conventional medical therapies have not improved the situation. In at least one study (Klock et al, 1989) patients with "other causes"

(according to the dentists judgement) for their problems have been excluded beforehand. One study (Hugosson, 1986) excluded nearly all the patients for this reason, but these, symptom descriptions and treatments are so poorly described that the study had to be excluded from the curent meta-analysis.

Several studies that have tried to relate symtoms to levels of Hg in blood and urine. These have been excluded from this analysis since none has provided any correlations. This is completely consistent with the experience from the acrodynia epidemic and more recent reports. There is no relation between symptom severity and levels of Hg in blood or urine. Individual sensitivity is more important.

**Replies to questionnaires**
The result might be affected if patients with imroved healt answer questionnaires more frequently than persons where amalgam removal has been without effect. Some studies have, by using telephone interviews and reminders, tried to estimate such effects. In the study by the Swedish patients organization, Tf, from 1986, the percentage of replies was low (519 replies on a questionnaire sent out in the patient organization bulletin, the membership then about 3-4000). Mörnstad et al sent a questionnaire to members of Tf in Västerbotten County 1990 and obtained 62 % replies. The result was the same as in 1986, which indicates that no systematic skewed reporting had taken place, even when the frequency of replies was low.

Other studies with the same degree of health improvement have reached a higher response rate. Lindforss et al, 1994 had a 72 % response, Östlin, 1991 80 %, Strömberg and Langworth, 1998 78 %, including a follow-up of non-respondents by telephone interviews. The pattern of replies and results from those answering by telephone did not differ from those sending in written reports. Klock et al, 1989 reported 80 % replies; in a follow-up study 1992 67 %. Some persons did not want to reply since they disliked the conclusion from the first study that the health improvements were caused by a placebo effect. Lindh et al, 2002, 65 %. A follow-up of those not answering did not show any change. I a study by Siblerud, 1990, 86 of 300 patients replied (29 %).

Thus, we have not the impression that persons who answer these questionnaires constitute a group which is enriched with patients who have improved after amalgam removal and that the reported results might give a too optimistic view. On the contrary, persons who have recovered leave the organization after some years, more so than persons with remaining problems or recovering who want information on possible treatments that can enhance the process.

**Reporting symptoms**
The questions regarding symptoms have varied. In the 1986 Hanson study we asked for the 10 worst symptoms. In other studies a list of symptoms has been presented to the patients, symptoms the investigator considered belonging to the amalgam syndrome. The list can be very long or only comprise a few symptoms. Questions on both symptom frequency and intensity occur. Frequency is of less interest since you can live with minor problems but if they get intense the patients will try to get help from the health care system.

The study by Hanson, 1986, has influenced later studies regarding which symptoms are connected with amalgam. The study also lists symptoms of inorganic mercury exposure from a number of studies, not connected with amalgam. All amalgam symptoms can be found in the latter. The questionnaire used in Hanson, 1986, had no clues or directives on what to expect. At that time the general knowledge of amalgam poisoning was less than today. Irrespective of the type of questionnaire some symptoms are constantly reported.

The type of questioning has some bearing on the issue of which symptoms appear first. From the studies where the patients indicated their problems in a list it appears that psychic/cognitive effects are the first to appear and that after longer and more intense exposure tiredness, pains etc dominate.

**Symptoms**
Most studies report many and varied symptoms. Some of these appear constantly. A compilation of symptoms from all studies according to frequency shows the following: (only the most common ones included): Fatigue, anxiety/depression, muscle pains, headache, concentration problems, joint problems, metal

taste, mouth symptoms, vertigo/dizziness, gastrointestinal problems, memory disturbances, problems with sight, irritability, sleep disturbances, heart problems, skin problems, allergies, problems with hearing, numbness, infection prone.

**Changes in health**
Health changes after amalgam removal (much better or better) in the studies which give possibility for percentual calculations: 94; 78; 88; 76; 80; 80; 40; 70; 68; 88; 79; 68; 31; 71; 70; 79; 60; 89; 88; 63; 72; Mean 73 % See diagram for each study.

**Incomplete removal.**
In some studies patients who have not or only partly removed amalgam been documented separately. Lindforss et al, 1994, obtained much better or better in 88 % of the cases when all amalgam fillings had been removed, compared to 54 % with partial removal and 39 % when no fillings had been exchanged.
Östlin, 1991, 91 % after complete exchange and 46 % after partial exchange; Olsson & Lindh 75-80 % after total and 38 % after partial or no exchange. Strömberg & Langworth 80 % and 61 % respectively.

In Bjerner & Hjelm, 1991, only 1/3 had removed all amalgam. Total exchange increased joint- and muscle pains, general weakness, concentration problems, memory disturbances, and dry mouth. Metal taste, tremor, headache were reduced. The same data a year earlier, presented to the Health and Welfare Board (SoS) "heavy metal group", gave results, which were generally the reverse. At that time (1-year follow-up) 142 patients had been evaluated and 1/4 removed all amalgam fillings, later, when the 1-year follow-up had been completed, 207. At the 2-year follow-up 98 patients were contacted. If these were patients which had participated in the earlier follow-up is not stated. The number of patients described had thus increased with 65 and 1/3 had exchanged all amalgam fillings. Tf has the information that at the visit to the referral dentist and doctor no information on amalgam beneath gold crowns and bridges was given. Neither was any information presented on possible materials, which could be used instead of amalgam. In addition the responsible dentist and doctor gave several lectures to the county dentists with the message that you cannot get ill from amalgam.

The Bjerner &. Hjelm report deviates markedly from the results of other studies.

Klock et al, 1989, reported 70 % improvement in those with complete removal and 17 % in those with partial removal or other treatments. The follow-up showed 68 and 44 % improvement respectively.
Lichtenberg, 1996, reports that nearly 1/3 (39 of 118) patients had remaining gold or porcelain after visible amalgam had been removed. Under such restorations amalgam might remain. The result was 79 % healthy or better when visible amalgam had been removed. The explanation is certainly that patients hesitate to remove expensive constructions when also the replacements can be expected to be expensive. The same conditions should be applicable to most studies. Eriksson et al, 2000, report that 30-40 % of the patients, before starting exchange, had other metals in their mouths. After amalgam removal and dental work 10-15 % of the patients had remaining other metals.

**What and how much has been removed?**
Amalgam is certainly the most common material which is removed. However, nobody has systematically examined how carefully the alloy has been removed. Our experience during 20 years has shown that dentists often cheat; all amalgam is not removed but only part of it and the rest is covered with composite plastics. The patients will often first become better but gradually the symptoms recur. Amalgam is regularly present beneath gold-crowns and -bridges and gold-amalgam contacts are often the worst causes of ill health. There are also brass screw posts with a thin and porous gold plating, often directly cemented into amalgam fillings. Such constructions often result in periapical inflammations.

Metal fused to porcelain contains, in addition to the noble metals, undeclared additions of easily oxidized metals, added to obtain a god fusion between the components. Constructions of metal fused to porcelain give as many health disturbances as amalgam. Endodontically treated teeth contain, in addition to root screws of various metals (sometimes even pieces of paper clips!), not uncommonly broken-off root files, endodontic filling materials containing everything

possible poisonous. It is not just the infamous N2, which is a hazard to health.

A thorough sanitation should, logically, include removal of everything which is suspect and as innocuous materials as possible be used instead. Few dentists work without neglecting some of these principles.

**Reactions during removal**

Hanson, 1986 reports 113 short acute patient reactions (from 519 patients) during the period of amalgam removal (Spontaneous patient reports, not included in questionnaire). Strömberg & Langworth, 1998, reported that 1-2 fillings were removed every month with the use of high volume vacuum and cofferdam whenever possible. Despite this, reactions following amalgam removal were common. Out of the 26 patients who replied to this question, 46 % reported increased health problems after each amalgam removal session, 33 % sometimes and 21 % never. A majority of those who experienced problems (93 %) the symptoms appeared within 6 days, 7 % after a week or more. Most patients (59 %) noticed an intensification of symptoms already present, but 41 % had quite new symptoms. The intensification usually lasted for 2-6 days or more.

Bjerner & Hjelm, 1991, reported that 75 % had no unpleasant effects in addition to those which are normally connected with reparative dentistry, 13 % experienced symptoms the same day, 8 % 2-3 days later and 2 % >3 days. Mörnstad et al, 1994, however, reported 79 % negative health effects in connection with amalgam removal, usually after 2 days (0-20) and remained in the mean 4 weeks (0 days to several months). 10 % of the group reported no reaction at all.

Eriksson et al, 2000, write that 39 % of all patients in the study felt worse or much worse after drilling out amalgam. Persons who considered themselves sick from amalgam felt worse to 56 %. Persons who removed amalgam for other reasons reacted much less frequently. Hovmand, 1987, writes that some patients had unpleasant reactions in connection with amalgam removal but that this group later showed the most pronounced health improvements. The temporary reactions were typically acute attacks of already existing symptoms.

**Follow-up**

The idea that health improvements after amalgam removal should be caused by a placebo effect (if such even exists) has been proposed by e.g. Klock et al. Also "placebo-believers" consider such effects short-lasting. A number of the studies have evaluated the patients a long time after amalgam removal.

Olsson & Lindh, 1997, followed the patients in different groups up to 10 years. Changes appearing in the 0-3 year group did not differ from those in the 3-5 group and 3-10 year group (different patients).

Strömberg & Langworth, 1998: 14 % of the patients reported improvements starting within 1-2 months after removal of amalgam had been finished, 46 % successive improvement starting within 6 months. For 40 % the improvement came after more than 6 months, not indicating a placebo effect.

Klock et al evaluated 1/2 - 1 year after the time of examination. Continuing evaluation 42 months later. The results were persistent. The group with no removal or other treatment had also improved but not to the same degree (68 % am. removal and 44 no removal). Lichtenberg, 1993, evaluation 1 year after amalgam removal was finished and Lichtenberg, 1996, 1-4 years.

Redhe evaluated 3 years 11 months after removal. I the study by Eriksson, Falk, Liukkonen, 2000, the patients reported 0-15 years after removal. 11 % were then still in the process of removal. Of those under treatment 20 % had experienced improvement; of those who had finished the treatment 1 year or more ago 70 %. Engel evaluated a mean 16 months after treatment (0-116), Hovmand: mostly 6-12 months and Zamm mostly 4-12 months.

**Period of ill health**

The results from studies reporting duration of illness regularly indicate several years. In Hanson, 1986, those who reported better/much better health had been ill for a mean of 13 years; patients who got worse 23 years. Other studies report a mean of 10,3 years; >6 years, 14-15 years, mostly 5-20 years; >2 years and about 35 % 4 up to more than 10 years, 20 % 1-4 years.

## DAMS/ Foundation for Toxic-free Dentistry, USA

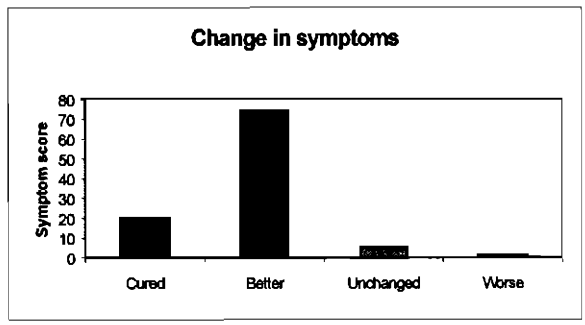
## Patient Adverse Reaction Report, 1993

## 762 patients

534 women, mean age 45 y.
228 men, mean age 46 y.

Common symptoms: Depression, allergies, exhaustion, lack of energy, headache, loss of memory, metal taste, gastrointestinal problems, vertigo/dizziness, irritability, sleep disturbances, joint problems, difficult concentrate, muscle weakness and pains, nervousness, numbness, hearing problems, vision problems, skin problems, multiple sclerosis.

Published by DAMS, sept. 1993




## Hanson, 1986

## 519 patients

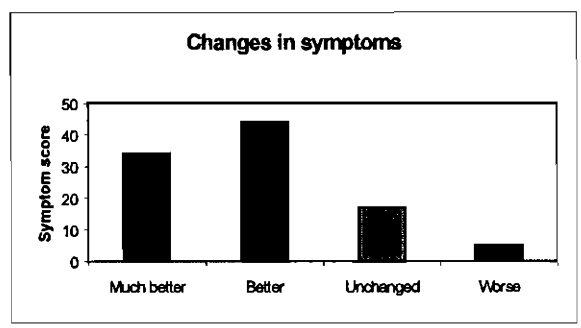
**Estimate of health status after amalgam removal. Changes in 62 symtoms, reported 3319 times in 519 patients**

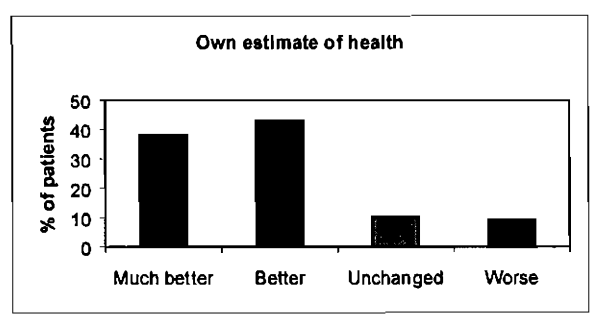
The 10 most troublesome symptoms were reported. It is vey little difference between a health score based on individual symptoms and a general estimate of health.

379 women , 136 men. Not reported 4.
Mean age women 51 (12-78) y.
Mean age men 47 (18-79) y.

Most troublesome symptoms: Joint and muscle pains, tiredness, vertigo, headache, gastrointestinal problems, problems with vision, mouth problems, heart trouble, loss of memory, problems with breathing/asthma, ear problems, depression, concentration difficulties.

Hanson, M. Tf-bladet 1, 1986 (Bulletin of the Association of Swedish Dental Mercury Patients)



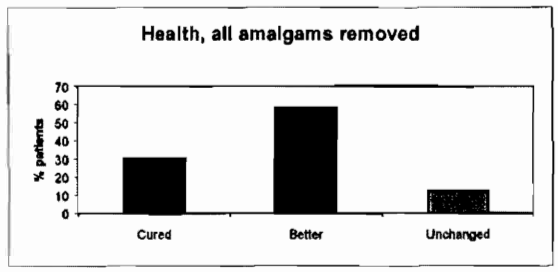
## Lindforss et al, 1994

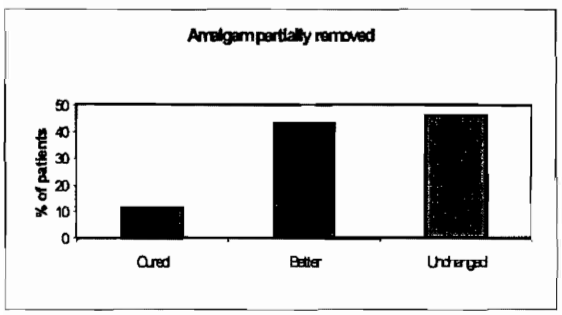
## 503 patients

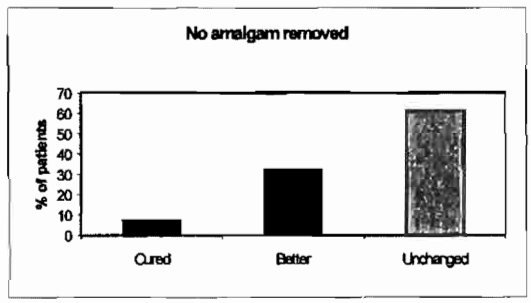
72 % women, 28 % men, the majority 40-60 y. Several symptoms increased in frequency but reduced in intensity.

Common symptoms: Pains, tiredness, anxiety, vision disturbance, disrupted sleep, metal taste, dry mouth, oral smarting pains.

Lindforss, H, Marqvardsen, O, Olsson, S, Henningsson, M. Effekter på hälsan efter avlägsnandet av amalgamfyllningar (Effects on health after removal of amalgam fillings) Tandläkartidn. 86(4), 1994, 205-211.

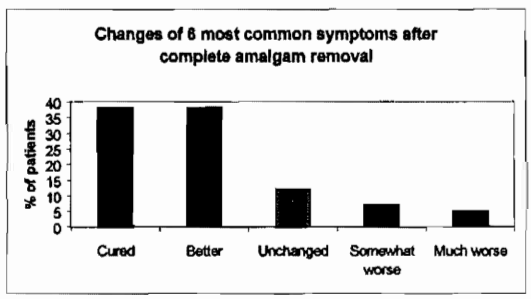


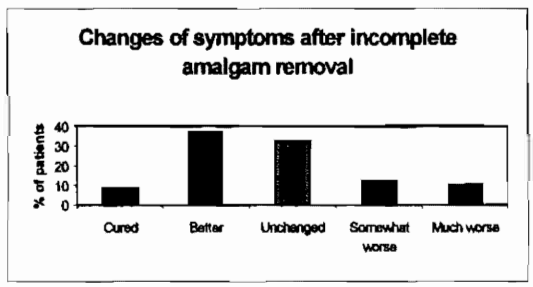




## Försäkringskassan (Stockholm health insurance), Stockholm, L. Östlin, 1991
## 308 patients

Diagram based on changes in symptoms for the 6 most common symptoms. Other symptoms showed the same tendency to varying degrees.
74 % women, 26 % men, mostly 40-59 år.
73 % had exchanged all amalgam fillings, the rest in various stages of removal.




Common symptoms: Blisters and ulcers in the mouth, smarting/metal taste, tiredness, muscle pains, headache, tooth- &. jaw pains, joint pains, problems concentrating, vertigo, gastrointestinal problems, shoulder pains, anxiety /restlessness/ depression, skin affections, vision disturbances, heart problems, and tinnitus.




Östlin, L. Amalgamutbyte - en väg mot bättre hälsa? (Amalgam removal - a road to better health?) Försäkringskassan, Stockholms län, 1991

## Olsson & Lindh, 1997
## 253 patients in 4 groups

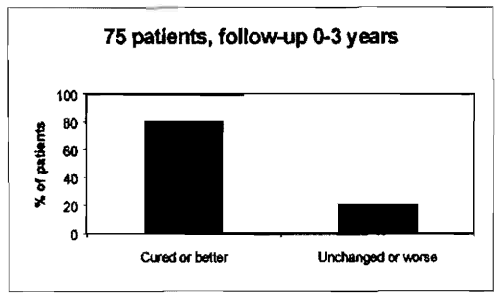
Based on % changes of 13 symptoms

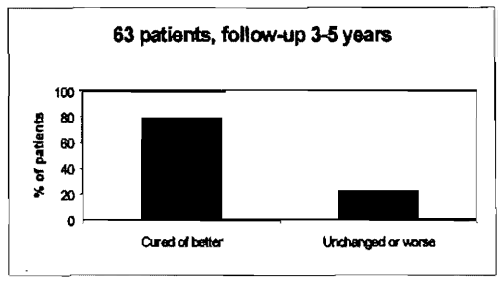
Group IV comprises patients who have started amalgam removal but not completed (59 %) and patients who never started (41 %)

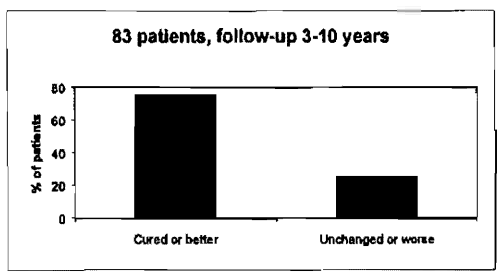
Most frequent symptoms: Pronounced tiredness, oral mucosal smarting & metal taste, difficulty concentrating, headache, muscle pains, anxiety, restlessness and depression, joint pains, shoulder pains, gastrointestinal disturbances, tooth & jaw pains, blisters & ulcers in oral mucosa, heart problems, skin eruptions, vertigo, infection prone, and visual disturbances.

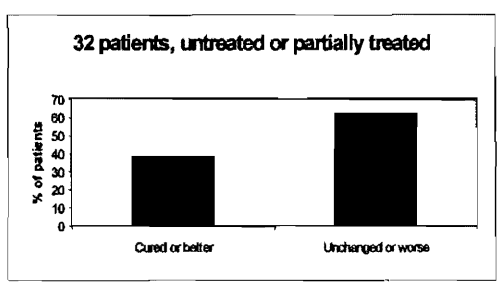
Oral smarting and metal taste improved in 87 % of patients who had removed amalgam; in 48 % of patients who had only partially or not at all removed their amalgam fillings.

Olsson, G & Lindh, U. Veränderung des allgemeinen Gesundheitszustand nach Amalgamentfernung. (Changes in general health after amalgam removal) GZM, Ganzheitl. Zahnmed. 2(1), 1997, 22-28.



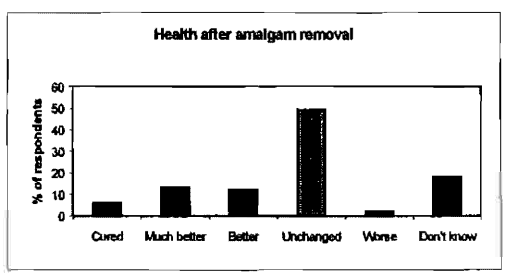




## SIFO, telephone questionnaire, 1993
## 100 patients (opinion poll institute)

SIFO: Allmänhetens inställning till och besvär av tandfyllningar med amalgam., (Attitudes and problems from amalgam dental fillings in the general population) Södelind, M. SIFO Research AB, 1993. Comment: There is no information on completeness of amalgam removal or how well the respondents understood the question (M.H.)



## Strömberg & Langworth, 1998
## 233 patients

No significant difference between 0-1 y. and >6 y. follow-up.
31 % men
69 % women
36 out of 280 were initially convinced that their problems were amalgam-related.
The development of symptoms up to start of amalgam removal was a successive increase in 78 % , in 18 % unchanged and in 4 % abating.
The duration of symptoms a mean of 10,3 years.

Common symptoms: headache, tiredness, vertigo, difficult concentrating, oral smart, changes of taste, recurrent respiratory infections, and gastrointestinal problems.
Strömberg, R, Langworth, S. Förbättras hälsan efter borttagning av amalgam? (Does health improve after removal of amalgam?) Tandläkartidn. 90(9), 1998, 23-29.

## 207 patients
## Bjerner & Hjelm, dec. 1991
## 207 patients

72 % women, 28 % men, mean age 48 y.

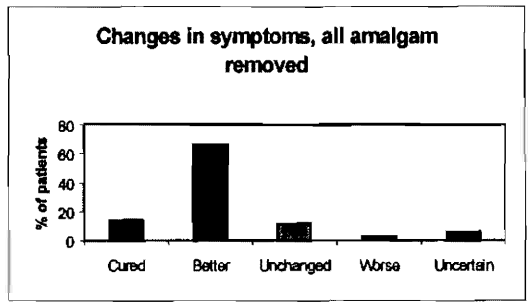
Follow-up 1 y. after first visit.
36,1 % had exchanged all amalgam (75 ), 27,4 % none, 19,2 % a few, 17,3 % many..

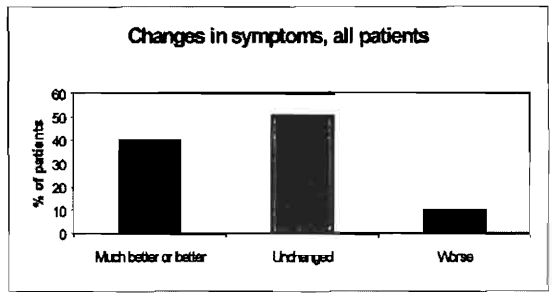
Common symptoms: Headache, metal taste, vertigo, difficulty concentrating, joint and muscle pains, numbness, general weakness, parenthesis, sweating, loss of sleep.

Most symptoms have lasted >6 years

Metal taste and headache had diminished after amalgam removal.
Joint and muscle pain, general weakness, difficulty concentrating, memory impairment and dry mouth had increased, based on 74 of 207 pat. who had removed amalgam

When the same data, but with fewer patients followed-up, 37 of 146 who had removed amalgam, were presented for the Health and Welfare "Heavy Metal Group" one year earlier (November 1990), joint and muscle pains, general weakness, problems concentrating, shyness,



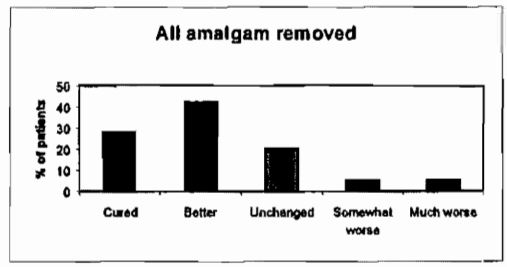

memory impairment and metal taste had diminished and salivation and gingivitis increased after amalgam removal.

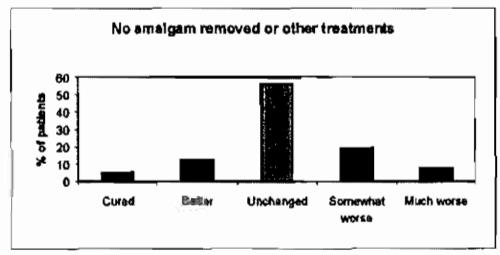
LEK-studien, Landstinget Dalarna, Bjerner, B & Hjelm, H. dec. 1991
LEK-studien Dalarna. Sammanställning inför hearing med SoS:s "tungmetallgrupp", 90-11-21

## Klock, Blomgren, Ripa, Andrup, 1989
## 198 patients

Based on 6 most common symptoms: tiredness, smarting and metal taste, difficulty concentrating, muscle pains, anxiety/restlessness/depression, and headache. Evaluation 1/2-3 y. after examination by referral dentist.
Other symptoms showed the same improvement after amalgam removal.
The authors are of the opinion that the patient's symptoms do not belong to those which should occur at light mercury poisoning (hand tremor, loss of appetite, loss of weight according to the authors) and that it must be a placebo effect since all, widely varying symptoms, were reduced after a single form of therapy.

Klock, B, Blomgren, J, Ripa, U, Andrup B. Effekt av amalgamavlägsnande på patienter som misstänker att de lider eller har lidit av amalgamförgiftning. (Effect of amalgam removal in patients who suspect amalgam poisoning) Tandläkartidningen 81, 1989, 1297-1302.

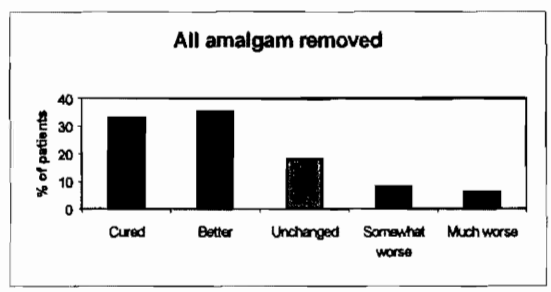


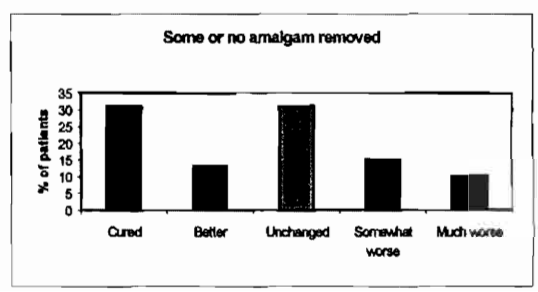




## Klock & Ripa, 1992, follow-up
## 166 patients

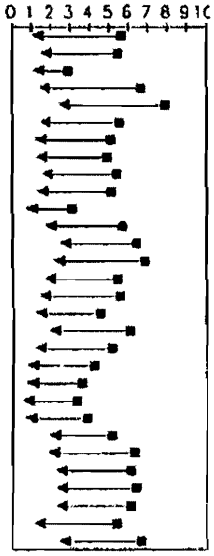
Klock, B, Ripa, U. Effekt av amalgamavlägsnande på patienter som undersökts av hänvisningstandläkare. (Effect of amalgam removal in patients, examined by referral dentists) Tandläkartidningen 84(17), 1992, 988-994.

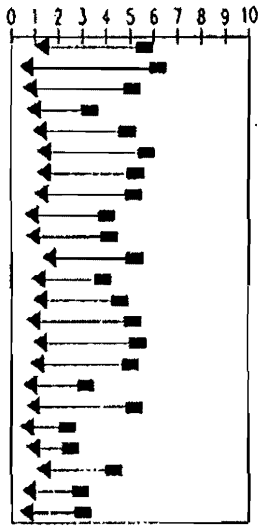
All amalgam removed, n = 102
Some amalgam removed: n = 43
No amalgam removed or other treatments: N = 14
"Other treatments" not specified.



**Mörnstad, Teivens, Wänman, 1994**
**132 patients**




Questionnaire to members of patient organization (Tf) in Västerbotten County. 159 had completely removed amalgam and out of these 132 had adequately replied.

Common symptoms (most severe symptoms): muscle pains, joint pains, general tiredness, headache, back pain, shoulder pain, vertigo, gastric pain, heart trouble, tinnitus, chest pains, temperomandibular pains, problems concentration, sleep disturbances mm.

Amalgam removal had most effect on general tiredness (7,9 -->2,8), headache (6,6 -->1,8), vertigo (5,5 --> 1,3), difficulties concentrating (6,7 -->2,5) and feeling frozen (6,4 -->2,2). Depending on symptom between 14 and 34 out of 132 reported that they had completely got rid of the symptoms. Total resolution of symptoms was most often reported for vertigo (34), eye troubles (31), headache (27) and sleep disturbances (27). One person reported that all earlier symptoms had completely disappeared after amalgam removal.

Most common mouth symptoms were metal taste, oral smarting, jaw pain and easily exhausted jaw muscles. Most effect of amalgam removal was found for metal taste (5,7 -->0,9), jaw pain (5,3 -->1,3), sinusitis (4,9 -->1,0), diffuse facial pain (5,0 -->1,3) and oral smarting 5,3 -->1,6). 79 % had reactions directly in connection with amalgam removal, most often after 2 days (0-20) and remaining a mean of 4 weeks.

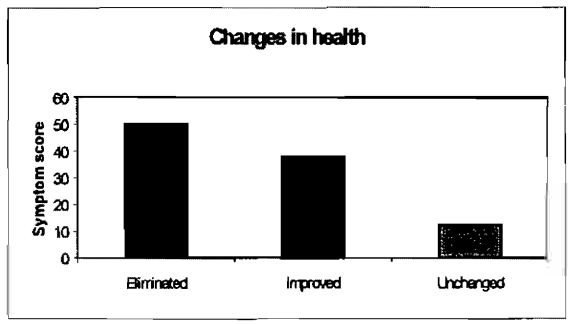
46 % considered that they had been badly treated in the health care system, 12 % well treated, and the rest neither well nor badly.
Most of the patients were living in the Skellefteå community, a mining area where Rönnskärsverken is situated, earlier the largest point source of mercury release in Sweden.

Mörnstad H, Teivens A, Wänman A. Sjukdomsbild och attityder till amalgam. En enkätstudie bland medlemmar i Tandvårdsskadeförbundet. (Health status and attitudes to amalgam. A questionnaire to members of the Dental Patient Organization) Tandläkartidningen 86, 1994, 196-204.

## Lichtenberg, 1993
## 120 patients

120 patients, 97 women, 23 men, mean age 48 years (25-72)

Adequate protection during amalgam removal.
Replacement material composite plastics.
Evaluation 1 y. after completed amalgam removal.
% change in symptoms in patients who before amalgam removal had a specific symptom.
Variation 75-100 %.



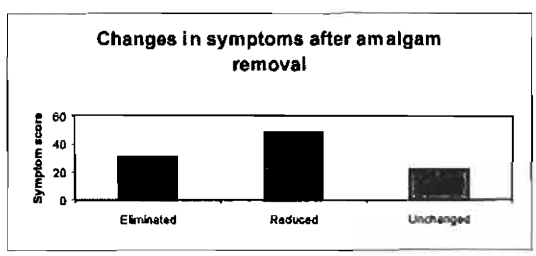

100% elimination for tender teeth (reported by 62 pat.), metal taste (69 pat.), lack of appetite (12 pat.).
Continued evaluation shows a transfer from group improvement to group elimination of symptoms.

Common symptoms: Joint pains, leg cramps, exhaustion, easily tired muscles, headache, metal taste, bleeding gums, tender teeth, irritability, difficulty concentrating, vertigo.

Lichtenberg H. Eliminering of symtoms by removal of dental amalgam from mercury poisoned patients, as compared with a control group of average patients. J. Orthomol Med. 8, 1993, 145-148

## Lichtenberg, 1996
## 110 patients

79 of the patients had no metals remaining after treatment, 39 had remaining gold or porcelain beneath which there might have been amalgam remaining.
Follow-up 1-4 years




Common symptoms: Tiredness, difficulty concentrating, bad memory, irritability, muscle weakness, metal taste, headache, joint pains, throat pains, bloating, allergies, bad appetite.

Metal taste was present in 72 patients before treatment, after am. removal eliminated in 59 and reduced in 12.

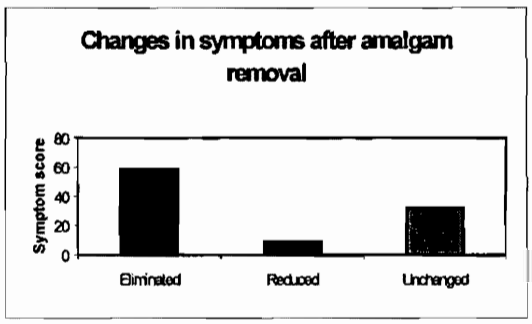
Lichtenberg, H. Symptoms before and after proper amalgam removal in relation to serum-globulin reaction to metals. J Orthomol Medicine11(4), 1996, 195-204.

## Redhe, 1991
## 100 patients

Follow-up 3 y. 11 mo.
71 women, 35 men (drop-out 6 persons before completion of study)

Common symptoms: Headache, tender teeth/tooth ache, strained jaws, tiredness, vertigo, joint and muscle problems, anxiety/restlessness/stress feeling, metal taste, recurrent infections.

Sjuk av amalgam. (Sick from amalgam) Redhe, O. R-Dental, Falun, 1991



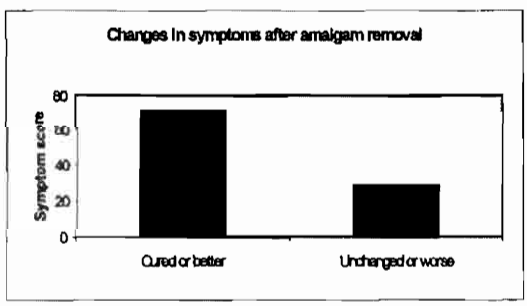

## Eriksson, 1996
## 97 patients

64 women
33 men
Follow-up > 3 years

Common symptoms: General tiredness, feelings of stress and tension, back/neck pains, joint pains and problems, metal taste, stiffness of joints and muscles, vertigo, forgetfulness, headache/migraine, muscle pains.

Metal taste remained in 2 of 62 patients

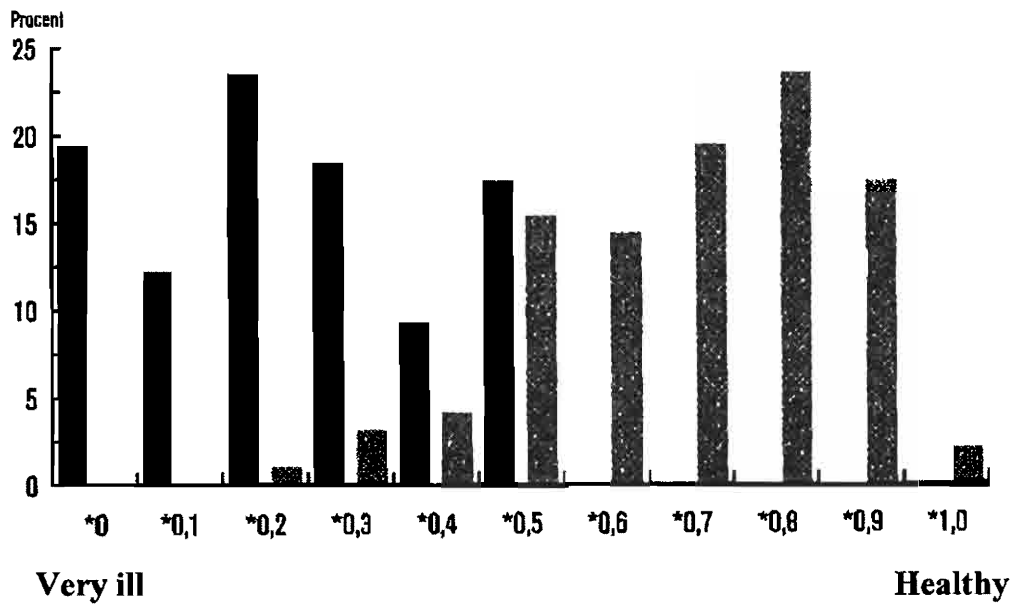
G. Eriksson, Torsås. Amalgamsanering - kliniska resultat efter tre år. (Amalgam removal - clinical results after three years) IAOMT, 1996



**Eriksson, Falk, Liukkonen, 2000**
**98 patients**

Changes in health after amalgam removal

Black: Category scaling before amalgam removal
Shaded: Category scaling after amalgam removal



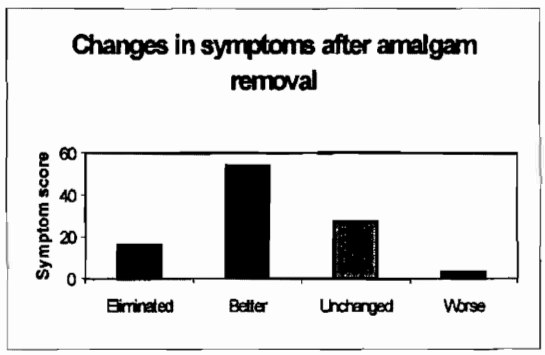

98 patients. Questionnaire to patients to dentists, which by Tf had been judged to remove amalgam in an adequate way, using replacement materials, which most patients, tolerate.

Most common symptoms: Joint and muscle pains, memory disturbances, headache, difficulty concentrating, irritability, sensitivity to light/sound, pains/sensitivity in teeth, sleep disturbances, paralysing tiredness, anxiety, skin problems, allergies.

Eriksson, L. Amalgam, hälsa och pengar. (Amalgam, health and money) Falktext, Växjö 2000.

## Siblerud, 1990
## 86 patients

The total number of symptoms 1815 in a questionnaire where the patients indicated their symptoms from a list and reported degree of change.
80 % reported feeling better. The degree of health improvement 59 %
91 % were satisfied with the decision to remove amalgam and 88 % that they should do it again with the result of the removal as a decision basis.




32 patients reported metal taste. Disappeared or reduced in 30, 1 unchanged, 1 worse.

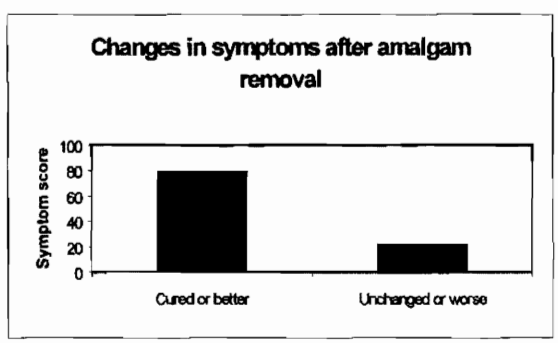
Most common symptoms: Depression, irritability, forgetfulness/memory loss, difficulty concentrating, metal taste, vision disturbances, easily exhausted, fatigue, tired in the morning, gastrointestinal problems, hypoglycaemia, anger easily, depression, irritability, often restless, nervousness, low self esteem, sensitive to foods, sensitive to light (eyes), cold hands and feet, vertigo/dizziness, sleep disturbances.

Siblerud, RL. Health effects after dental amalgam removal. J. Orthomol. Med. 5, 1990, 95-106.

## Godfrey & Campbell, 1994
## 80 patients

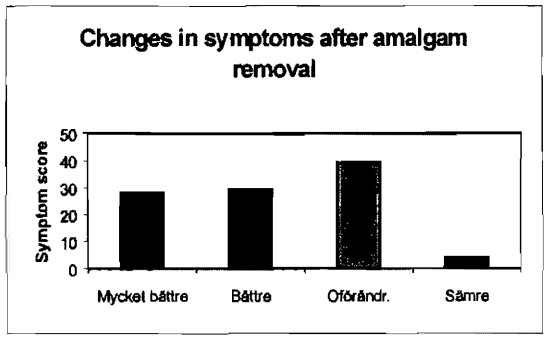
Women 48
Men 32
Mean age 43 y. (19-71)

Most common symptoms or symptom groups: Tiredness, neurological, psychological, gastrointestinal problems. Bad memory, metal taste, headache, cold extremities, tinnitus. Metal taste was present in 59 of 80 patients. After amalgam removal none.




Godfrey, M. & Campbell, N. Confirmation of mercury retention and toxicity using 2,3-dimercapto-1-propane-sulphonic acid sodium salt (DMPS). J. Adv. Med. 7(1), 1994, 19-30.

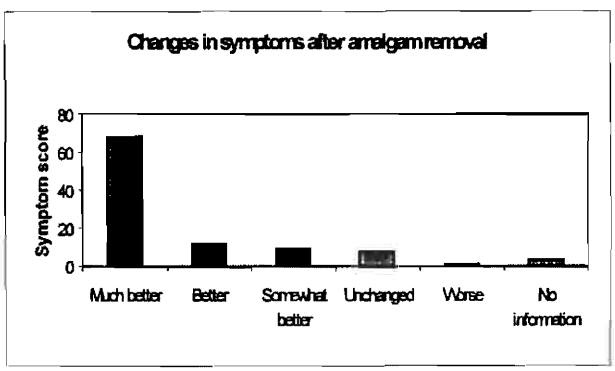
## Larose, 1992
## 80 patients




Evaluation 3 months after finished amalgam removal.
Common symptom: Tiredness (much better or better in 32 of 40), problems concentration (21/27), headache (12/20), metal taste (20/22), high blood pressure (1/33), tachycardia (7/30), MS (5/10), muscle and joint pains (9/17), depression (14/18), loss of memory (9/18), nervousness/anxiety (12/17).

P. Larose. The effect of amalgam removal on 37 health symptoms in humans. Updated 1992 from study reported in Dental Health & Facts 2(1) 1989. Foundation for Toxic-free Dentistry/Bio-Probe, Orlando

## Engel, 1998
## 75 patients




Women 52
Men 23
Mean age 48,4 y. (19-73)
No. am-fillings, mean 10,8 (3-19)
Follow-up, mean 16 mo. (0-116)
Evaluation: Patients own estimate of health before and after amalgam removal.
Clean-up and cofferdam used during drilling.

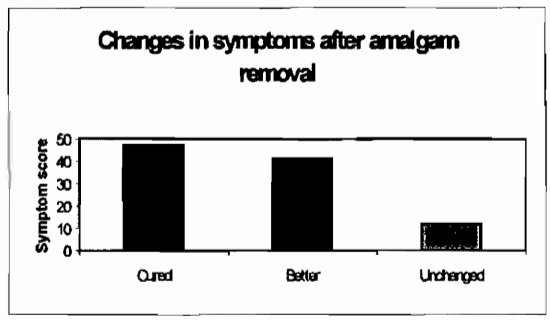
Common symptoms:
Pains in joints, back, neck, shoulder, arms
Headache, migraine
Vertigo
Vision disturbances
Paresthesias
Allergies
Gastrointestinal problems
Tiredness
Psychic problems

Engel, P. Beobachtung über die Gesundheit vor und nach Amalgamentfernung. Schw. Monatschr. Zahnmed. 108, 1998, 811-813.

## Hovmand, 1987
## 59 patients

59 patients. All fillings removed in 43, partly in 16.
Evaluation according to % improvement for patients with a certain symptom.
Common symptoms: Headache/migraine, metal taste (100 improvement.), tiredness, back/joints, gastrointestinal, depression, and eczema.

Hovmand, O. Oral galvanisme - erfaringer fra praxis. Tandlaegebladet 91, 1987, 473-476.



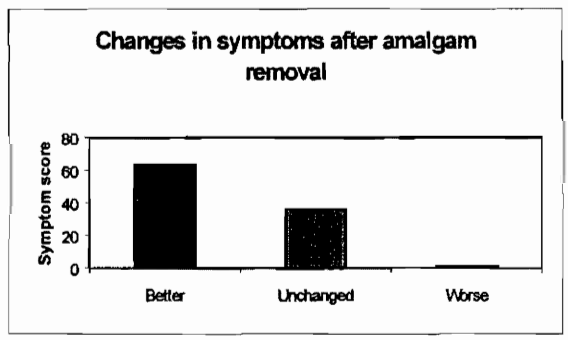

## 22 patients

## Zamm, 1990

22 patients reported a total of 584 symptoms
Evaluation according to changes in symptom severity for patients with certain symptoms.
Follow-up, mean 7,3 mo. (1,5-15)

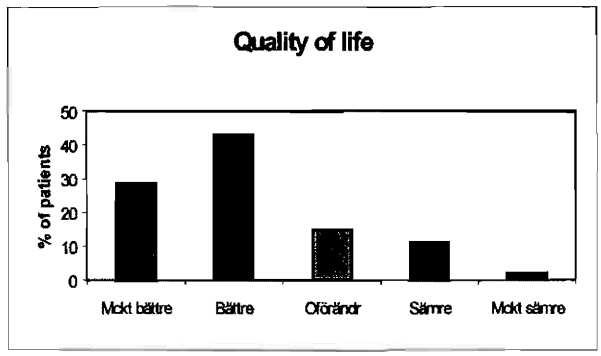
Common symptoms: headache, vertigo, irritability, anxiety, difficulty concentrating, difficult to think, mental confusion, "groggy", reading difficulties, bad memory, depression, dermatitis, sinusitis, achycardia, myalgia, exhaustion, weakness.

Zamm, AV. Removal of dental mercury: often an effective treatment for the very sensitive patient. J Orthomol. Med. 5, 1990, 138-142.



## Lindh et al, 2002
## 463 patients

Changes in health after amalgam removal according to fig. 1 in the report (Much better 4-6 on scale, better 1-3, worse -1 till -3, much worse -4 till -6.)

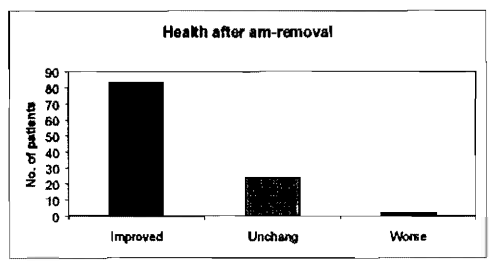



Dropout investigation did not demonstrate any difference between those answering and not answering questionnaire.

Common symptoms: Tiredness, depression, muscle pains, easily exhausted, difficulty concentrating, muscle problems in whole body, gastrointestinal problems, mouth symptoms, sleeping disturbances, unpleasant feelings in hands and feet, memory disturbance, vertigo/unsteady, headache, difficulty thinking, joint problems.

Lindh, U, Hudecek R, Danersund A, Eriksson S, Lindvall A. Removal of dental amalgam and other metal alloys supported by antioxidant therapy alleviates symptoms and improves quality of life in patients with amalgam-associated ill health. Neuroendocrinol Lett 5/6, 2002, 459-482

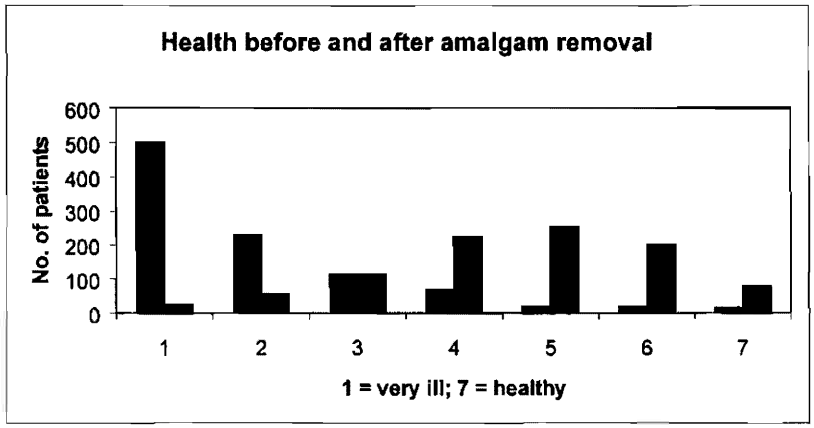
## Stejskal et al, 1999
## 98 patients




The majority of patients (77 %) fulfilled the criteria for CFS, the remaining ones lacked one of the major criteria or several of the minor ones.

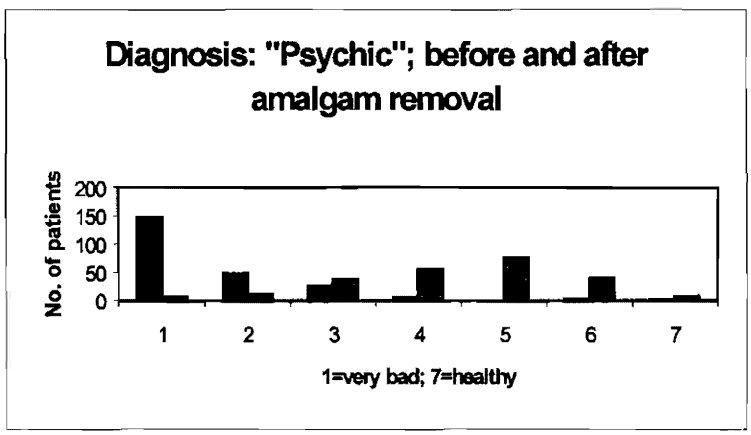
Stejskal VDM, Danersund A, Lindvall A, Hudecek R, Nordman V, Yaqob A, Mayer W, Bieger W, Lindh U. Neuroendocrinol Lett. 20, 1999, 289-98.

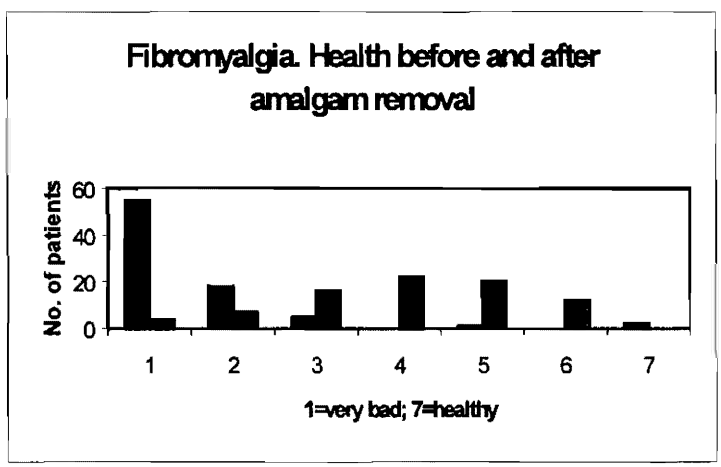
**Dental Materials and Health,**
**Swedish Dept. of Health, 2003, SOU**
**2003:53, Appendix 10.**

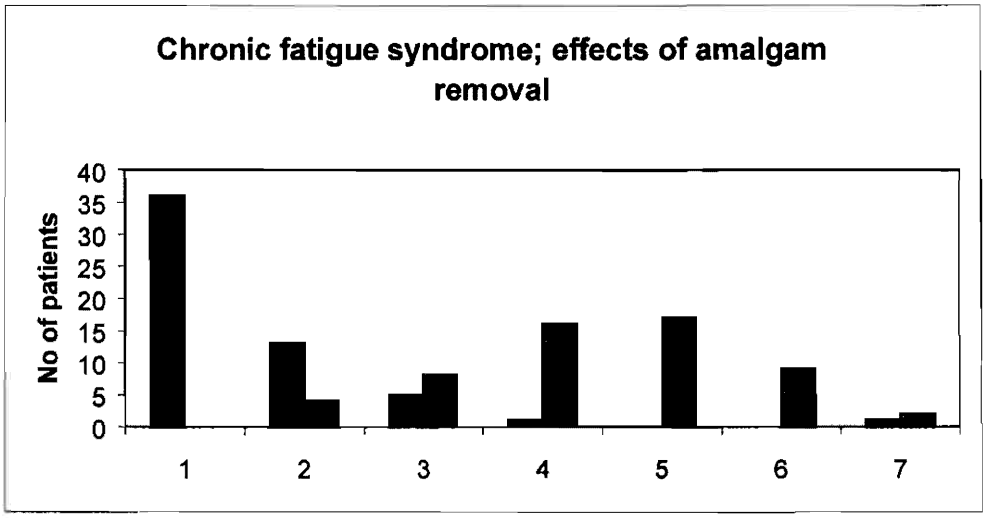


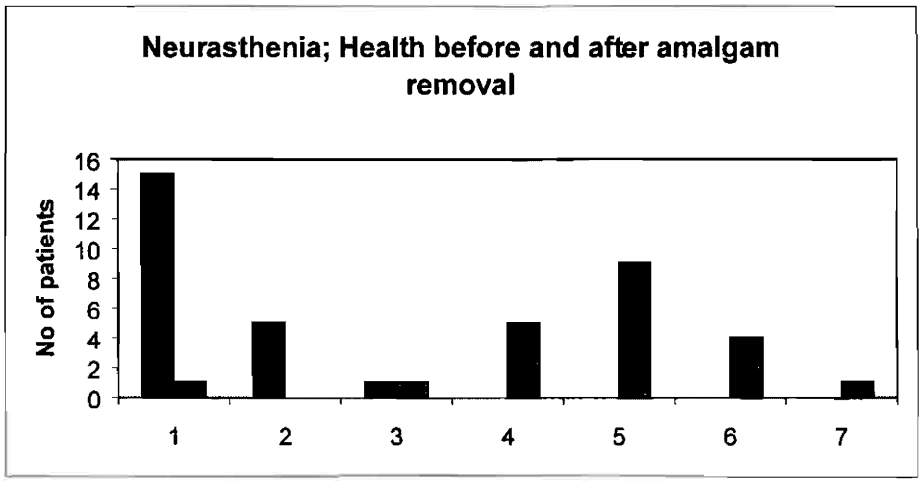

**Before amalgam removal**

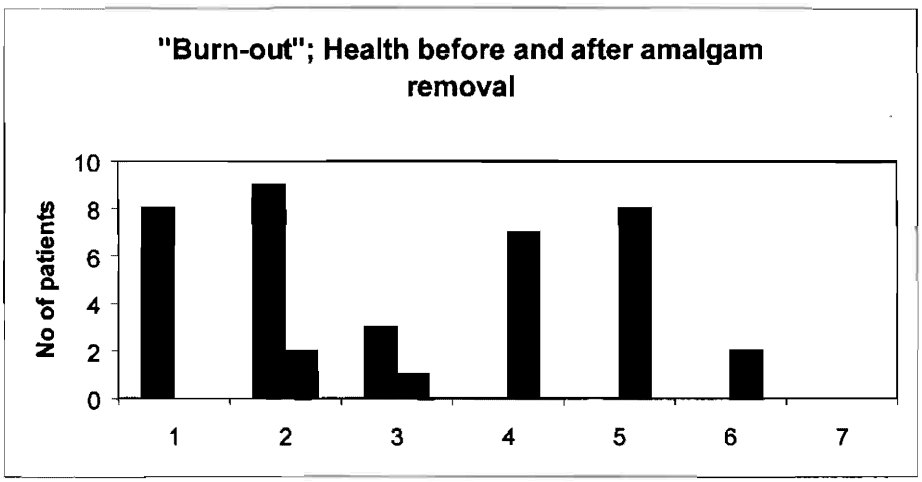
**After amalgam removal**

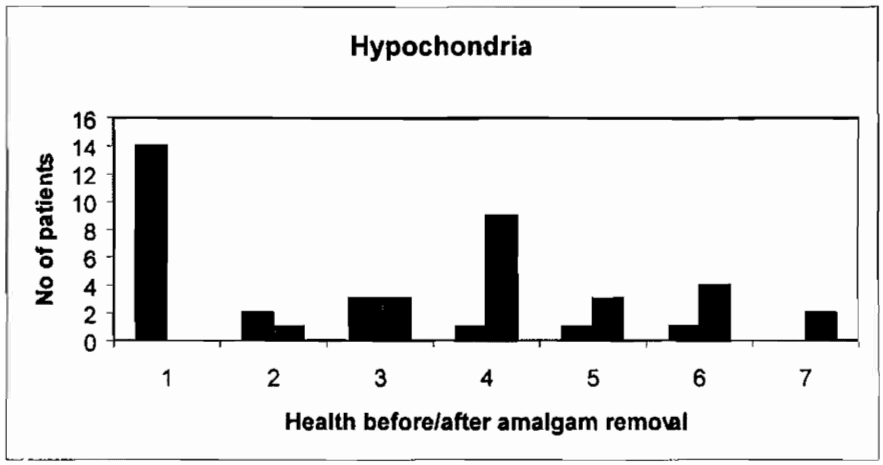


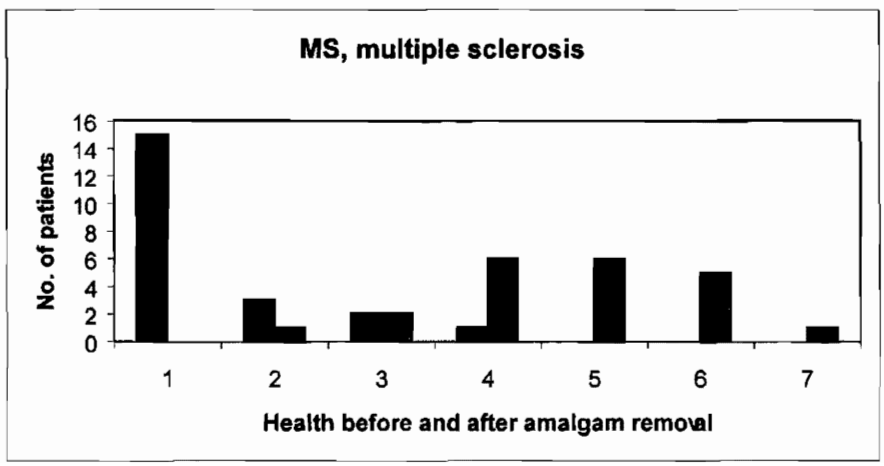




Chronic fatigue syndrome; effects of amalgam removal
No of patients
40
35
30
25
20
15
10
5
0
1
2
3
4
5
6
7


Neurasthenia; Health before and after amalgam removal
No of patients
16
14
12
10
8
6
4
2
0
1
2
3
4
5
6
7


"Burn-out"; Health before and after amalgam removal
No of patients
10
8
6
4
2
0
1
2
3
4
5
6
7


Hypochondria
No of patients
16
14
12
10
8
6
4
2
0
1
2
3
4
5
6
7
Health before/after amalgam removal


MS, multiple sclerosis
No. of patients
16
14
12
10
8
6
4
2
0
1
2
3
4
5
6
7
Health before and after amalgam removal

INTERNATIONAL ACADEMY OF
ORAL MEDICINE AND TOXICOLOGY

PUBLIC COMMENT

EXHIBIT 12

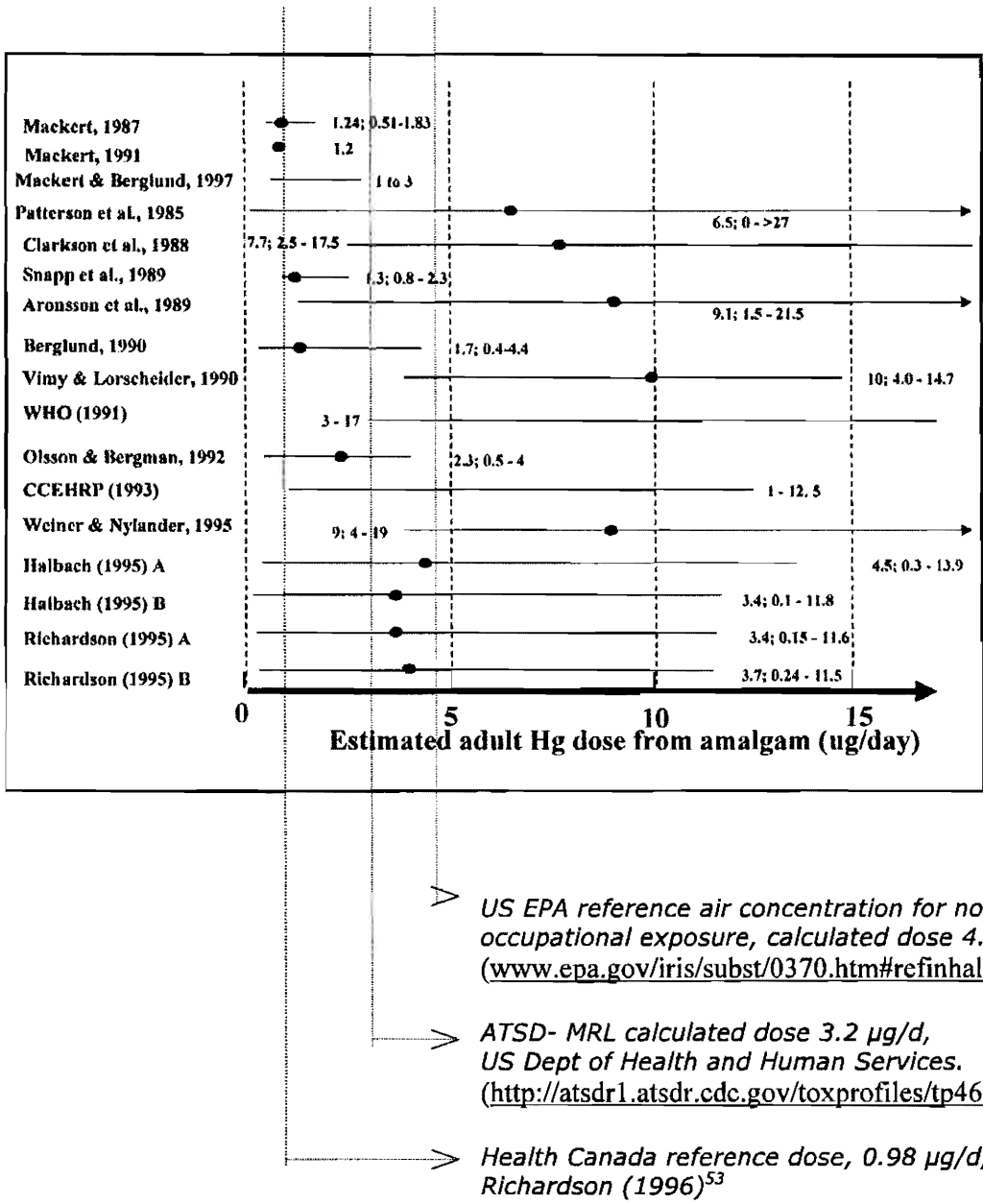
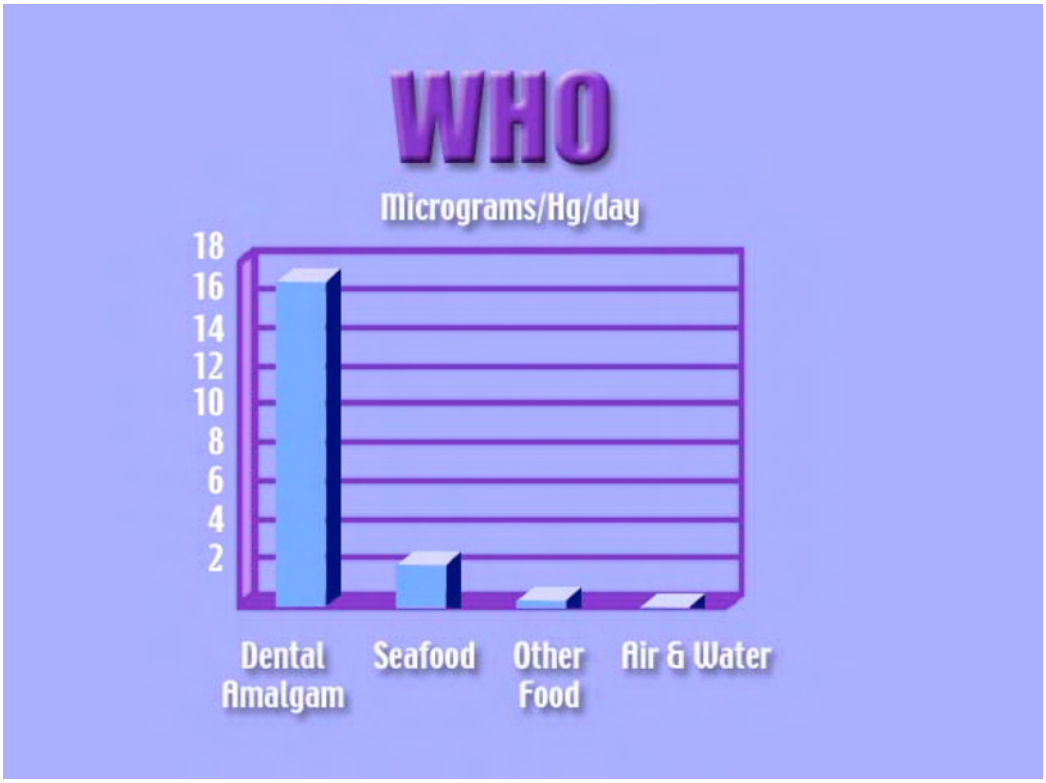



Figure 3 – Summary of seventeen literature citations estimating average mercury exposure in adults from amalgam fillings. The intersecting red lines show current allowable limits for non-occupational exposure to inorganic mercury from three different government agencies. The green dot in each horizontal 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)

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 13




| Medium | Intake (retention) (µg)a | | Reference |
|---|---|---|---|
| | Mercury vapour | Inorganic mercury compounds | |
| Atmosphere | 0.04–0.2 (0.03–0.16)b | 0c | IPCS, 1991 |
| Food: Fish | 0 | 0.6d (0.06) | IPCS, 1991 |
| Food: Non-fish | 0 | 3.6 (0.36) | IPCS, 1991 |
| Drinking-water | 0 | 0.05 (0.005) | IPCS, 1991 |
| Dental amalgam | 1.2–27 (1–21.6) | 0 | ATSDR, 1999 |
| Total | 1.2–27 (1–22) | 4.3 (0.43) | |

a Figures in parentheses are the amounts retained that were estimated from the pharmacokinetic parameters; i.e., 80% of inhaled vapour and 10% of inorganic mercury are retained.

b Assumes an air concentration of 2–10 ng/m3 and a daily respiratory volume of 20 m3.

c For the purposes of comparison, it is assumed that the atmospheric concentrations of species of mercury other than mercury vapour are negligible.

d It is assumed that 20% of the total mercury in edible fish tissues is in the form of inorganic mercury compounds. It should be noted that fish intake may vary considerably between individuals and across populations. Certain communities whose major source of protein is fish may exceed this estimated inorganic mercury intake by an order of magnitude or more.

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 14

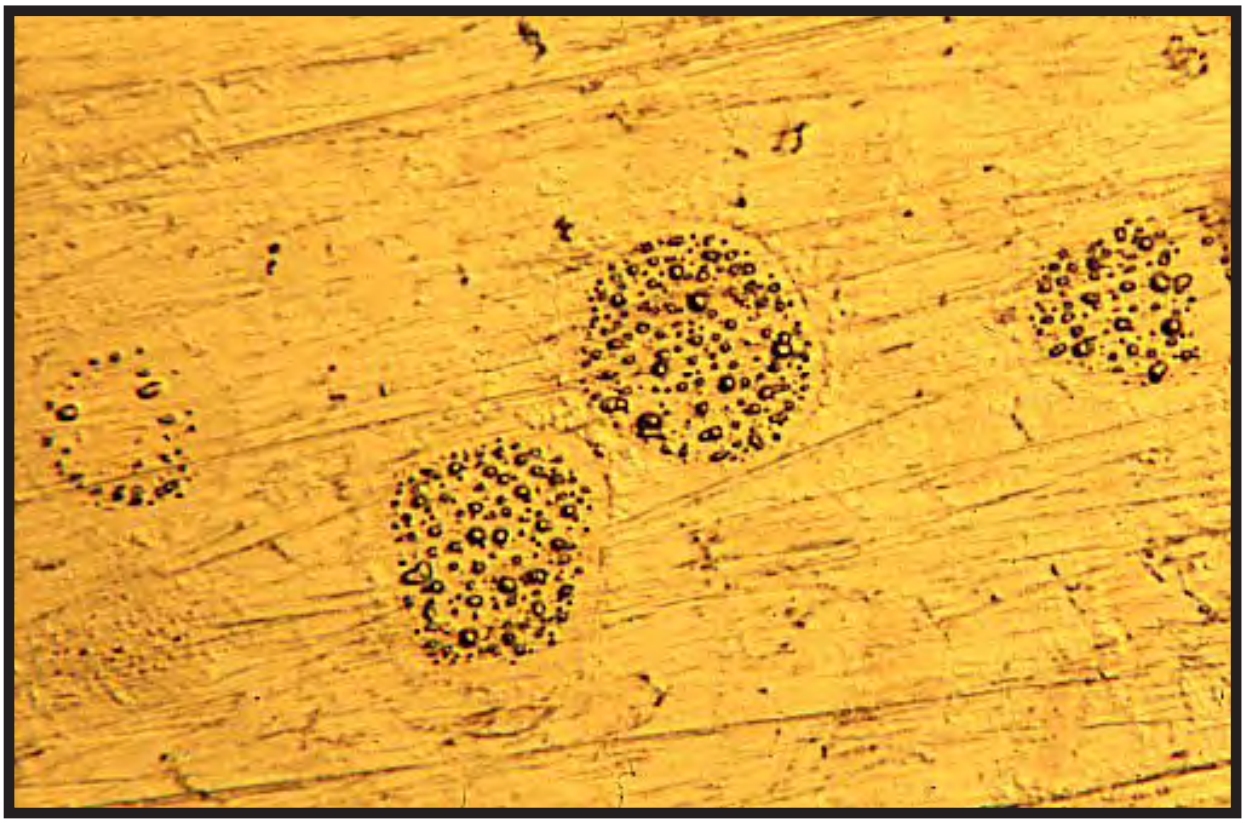


Figure 1 – Microscopic beads of liquid mercury expressed from the surface of amalgam metallurgical sample, following pressure from a microprobe. (from Masi, 1994)

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 15

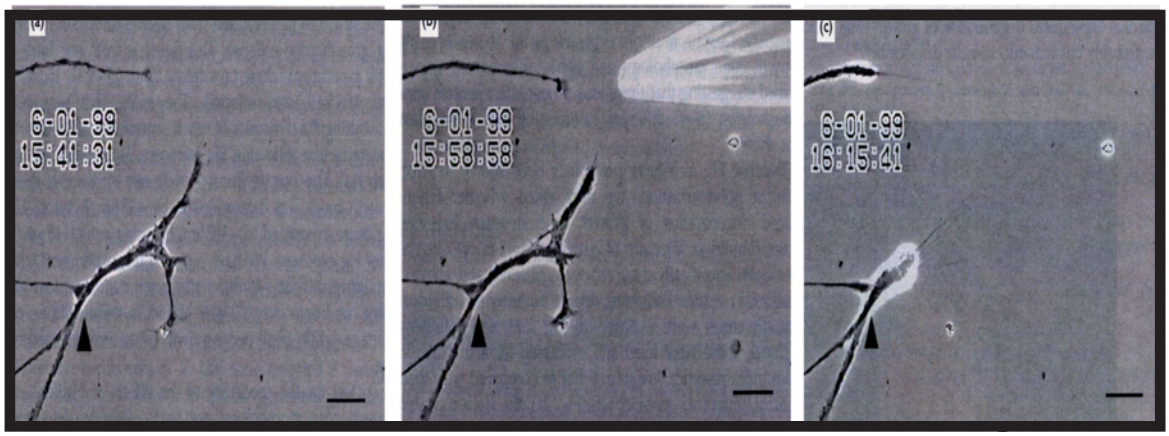


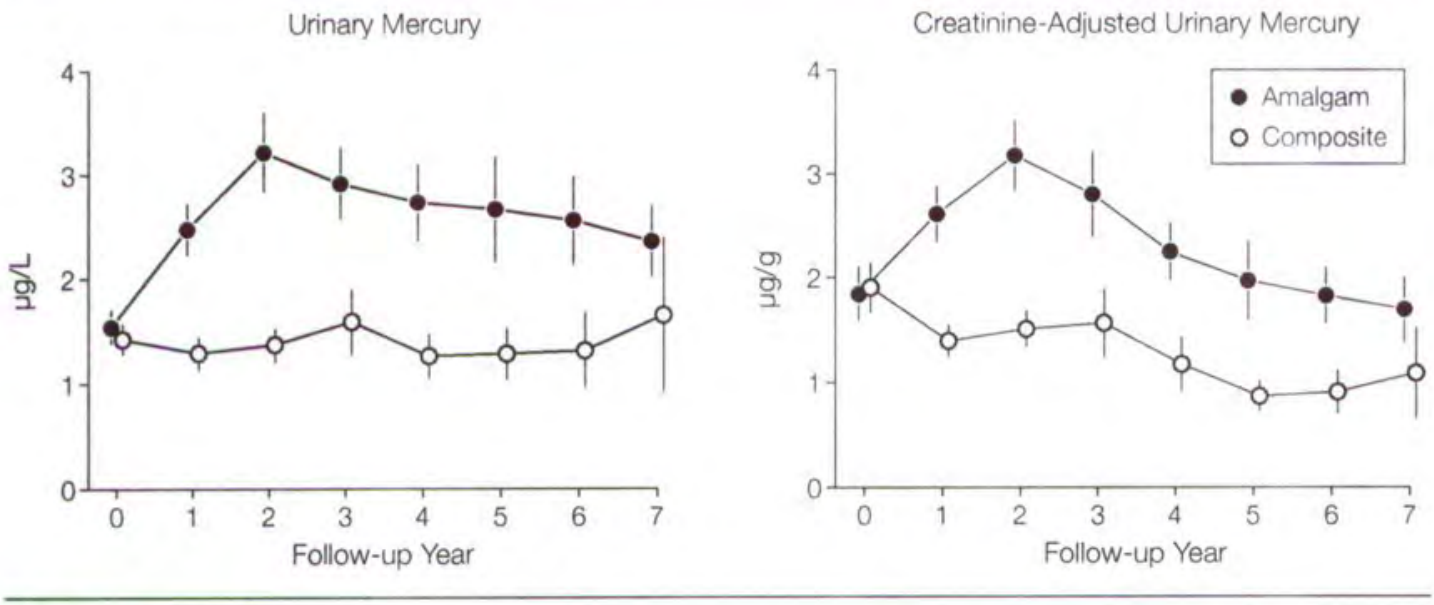

Figure 7 – Retrograde degeneration of neurite growth cone in the presence of 10-7 molar mercury chloride. Note the triangle reference mark. (From Leong, et. al. 2000) ( http://www.youtube.com/watch?v=VImCpWzXJ_w )

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 16

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




Error bars indicate 95% confidence intervals.

**Casa Pia childrens amalgam trial JAMA 4/2006 shows decline in urine mercury while exposure increased.**

# INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

## PUBLIC COMMENT

## EXHIBIT 17

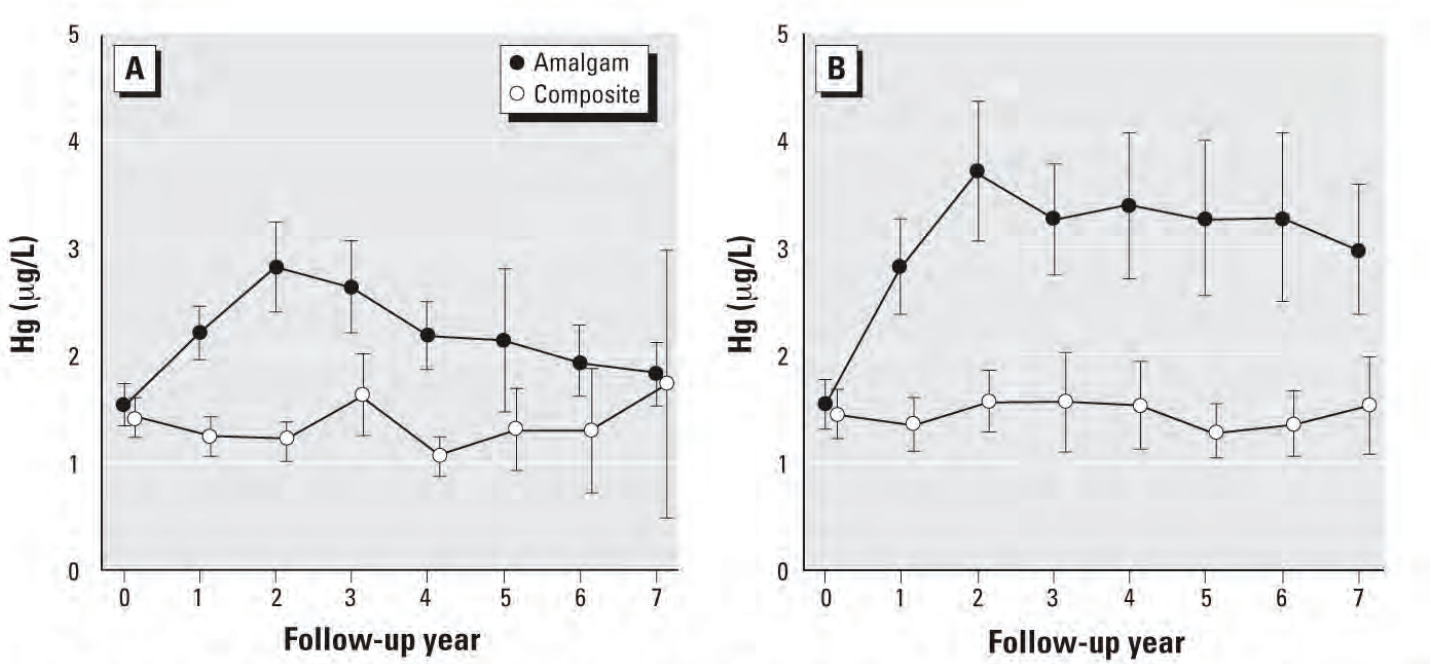



**Figure 3.** Mean urinary mercury concentrations for the amalgam group and composite group separately for male (*A*) and female (*B*) participants. Error bars show 95% confidence intervals for the group means. Differences between males and females in the amalgam group were statistically significant ($p < 0.05$) at all follow-up years except follow-up year 3. The sex comparisons were not altered significantly by adjustment for creatinine (results not shown).

8/12/08
Dear Dr. Kennedy,

Thank you for your interest in our study. The points we would make in response to your question are: (1) placement of amalgams increases urinary Hg content, and (2) the urinary Hg does not continue to rise, but instead falls off over time after placement of the fillings stops, i.e., there is negative correlation between urinary Hg and time since placement toward the end of the study.

Yours truly,

James S. Woods, Ph.D., Professor <jwoods@u.washington.edu>