AMALGAM WARNING FROM BRITAIN!

On 29 April 1998, the Department of Health of the Government of Great Britain issued a warning against the use of mercury amalgam dental fillings in pregnant women. The warning, in the form of an "advisory", was issued through the Committee on Toxicology of Chemicals in Food, Consumer Products and the environment (COT) and was presented as a "precautionary" measure. The COT maintained its previous position that, as yet, there is insufficient scientific evidence to determine the presence or absence of risk from the prenatal exposure to mercury. Elevated exposures to amalgam mercury during placement and removal of the fillings were emphasized.

The government announcement received widespread national attention throughout the written and electronic media, and has raised an uproar with the public. Although the announcement was couched in terms of perceived lack of risk, many citizens view the situation as "where there is smoke, there is bound to be fire." Similar advisories had previously been issued by the governments of Germany, Sweden, Canada, and possibly Norway, Denmark, Finland and Austria.

The British Dental Association (BDA) agreed to honor the government advisory, but maintained its previous position that patient exposure to amalgam mercury is harmless. The United Kingdom Chapter of the International Academy of Oral Medicine and Toxicology (IAOMT) applauded the government action and advised of the need to follow proper protocols for the removal of mercury amalgam fillings.

WILL THE REAL "DFU" PLEASE STAND UP!

In the last issue [BPNL, 14(2), March 1998], we reported that Densply/Caulk (D/C) had placed side effects, warnings, precautions and contraindications for its Dispersalloy and other amalgam products in the "Directions For Use (DFU)" on their Internet site. Subsequently, we learned that Ivoclar had issued even more stringent warnings and contraindications in the DFUs for their amalgam products, although these were not placed on the Internet.

As might be expected, the hue and cry resulting from this was monumental! Densply/Caulk quickly removed the DFUs from its Internet site and has announced that the DFUs would be "modified" to conform with the "current" position that amalgam products are harmless to patients. The explanation for the original posting was that it was in response to Proposition 65 in California and European Union
(EU) requirements for the CE labeling, for marketing specifically in Germany. A letter from Dentsply, United Kingdom stated: “Regulations in the State of California, USA and Germany only, stipulate that these regulations must be adhered to. This however, does not apply to the UK where BDA [Ed note: British Dental Association] assurances are currently fully supported by DENTSPLY.”

Bio-Probe has learned that Dispersalloy sold in California does contain the DFU insert containing the warnings and contraindications, whereas that sold in some other states do not. The message, apparently, is that if Dispersalloy is placed in California, it could have adverse effects and requires warning the patient. If, however, it is placed in other states there could not be any adverse effects and therefore warning patients is not required. The same, apparently, would apply to the other European Union countries other than Germany.

While the logic of this dichotomy defies explanation, the legal question should provide fertile grounds for investigation by attorneys. As an example, if a manufacturer issues warnings, precautions and contraindications for its product in one locale, does the potential for liability extend to any locale where the product is sold? If so, does the duty to inform require that the patients be warned in all locales, or only where the warning is required? In the event of liability, who bears the responsibility in the locale requiring warning, and in other locales [More on this issue in the next article.]

Of further interest is the posting of MSDSs [Material Safety Data Sheets] on the amalgam products. These clearly depict adverse effects from exposure to mercury, including chronic exposure. Dentists are responsible for the information found in MSDSs; they must make the information available to patients and staff members. There is no defense for not doing so. This presents dentists using amalgam with another medico-legal dilemma. It also provides a strong defense to mercury-free dentists for informing patients of potential adverse effects from exposure to amalgam mercury. State dental boards should take note of this when addressing the amalgam controversy.

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DENTISTS LEFT HANGING?

AMALGAM MANUFACTURERS: The recent action by amalgam manufacturers to issue warnings, adverse effects, precautions and contraindications to the use of their products (in certain locales) has provided the final step in focusing the potential liability on the dentist placing amalgams. The only conceivable reason for amalgam manufacturers to take this action is to protect themselves from legal liability. Indeed, the package insert from Kerr for its Tytin Whitecap amalgam contains the following statement: “Kerr’s technical advice, whether verbal or in writing, is designed to assist dentists in using Kerr’s products. Such advice does not expand Kerr’s limited warranty or relieve the dentist of testing Kerr’s products to determine their suitability for the intended uses and procedures. The dentist assumes all risk and liability for damages arising out of the improper use of Kerr’s product.”; and: “In no event shall Kerr be liable for any indirect, incidental, or consequential damages.” [Our emphasis.] This message should be perfectly clear to practicing dentists.

THE AMERICAN DENTAL ASSOCIATION (ADA): For years, the ADA has insulated itself from responsibility for the use of mercury fillings. Years ago, the ADA contracted with the American National Standards Institute (ANSI), a private company, to develop evaluation of dental materials. The initial ADA/ANSI Document (#41) for the “Recommended Standard Practices for Biological Evaluation of Dental Materials” listed “dental amalgam” and required a number of tests to ensure its biocompatibility. However, the ADA never certified dental amalgam itself, only “amalgam alloy” as ANSI/ADA Specification #1 and “dental mercury” as ANSI/ADA Specification #6. Neither of these two specifications have any biological evaluation listed. This ADA policy was developed through its Council on Dental Materials, Instruments and Equipment (CDMIE), the director of which was John W. Stanford, Ph.D. Later, in response to a letter of inquiry, Dr. Stanford stated: “There appears to be confusion regarding both the role of the Council and the scope of ANSI/ADA Specification No. 1 for Alloy for Dental Amalgam. The Specification in not for dental amalgam. It is only for the alloy for dental amalgam. The amalgam does not form until the dentist mixes the alloy with mercury. Therefore, dental amalgam per se cannot be certified. We cannot certify a reaction product made by the dentist.” [Letter: John W. Stanford, Ph.D., Secretary, Council on Dental Materials, Instruments and Equipment, ADA, 22 May 1986.] This policy on dental amalgam is most fascinating, as the ADA certifies other “reaction products” mixed in the dental office, such as impression materials and cements.

In 1992, the ADA made its position on potential liability for dental amalgam perfectly clear and, in
doing so, left amalgam-using dentists on their own. The ADA had been named as one of the defendants in a lawsuit against amalgam. Attorneys for the ADA pleaded in Court: "The ADA owes no legal duty of care to protect the public from allegedly dangerous products used by dentists. The ADA did not manufacture, design, supply or install the mercury-containing amalgams." [In The Superior Court of the State of California, In and For the County of Santa Clara, Case No. 718228, 22 October 1992.]

THE U.S. FOOD AND DRUG ADMINISTRATION (FDA): In 1975, the Congress and President of the United States directed the FDA to evaluate and classify all medical devices intended for human use. The FDA Commissioner appointed panels for various specialties, including dentistry. The Chairman of the Dental Devices Panel was John W. Stanford, Ph.D. [FR 40(97):21848, 19 May 1975.] Although FDA regulations required biological evaluation of products based on valid scientific evidence, there were no physiologists or toxicologists on the Dental Device Panel. It consisted of four dentists, a consumer liaison, an industry liaison, a dental lab technician, a dental school materials instructor, and Dr. Stanford.

Not surprisingly, the FDA accepted “Dental Mercury” and “Amalgam Alloy” as safe and effective dental devices. The proposed rules in 1980 had both placed in Class II, requiring evidence of performance standards to guarantee safety and effectiveness. [FR 45(251):85979-86034, 30 Dec 1980] The Final Rule in 1987 switched “Dental Mercury” to Class I, which only requires acceptable manufacturing procedures. [FR 52(155):30062-30108, 12 Aug 1987]

The well known 1993 USPHS CCEHRP document on the safety of dental amalgam verifies that the FDA still has not accepted and classified mixed dental amalgam [Summary and page VI-1]. A formal letter from the FDA further clarifies their position on mixed dental amalgam: “No manufacturer produces mixed dental amalgams. The mixed dental amalgam is prepared by dental clinicians. FDA does regulate manufacturers of dental mercury and amalgam alloys, but the only control FDA has over the ultimate, mixed amalgam is through the labeling for dental mercury and amalgam alloys.” [Letter: Lillian Yin, Ph.D., Director, Division of Ob-Gyn, ENT, and Dental Devices, FDA, 2 April 1991.]

THE U.S. PUBLIC HEALTH SERVICE (PHS): The foundation for support for the safety of dental amalgam is the now famous 1993 PHS document “Dental Amalgam: A Scientific Review and Recommended Public Health Service Strategy for Research, Education and Regulation.” The use of this document for the purpose of “proving” the safety of dental amalgam is a misrepresentation and nothing short of irresponsible.

In 1997, a three judge Court of Appeals in Colorado reversed a District Court ruling against a mercury-free dentist and cited the 1993 PHS document: “The report determined, rather, that the department: could not conclude with certainty whether or not the mercury in amalgam might pose a public health risk”; and “the possibility that these materials could pose health risks cannot be ruled out.” These cites are found on page 3 and page III-29 of the PHS document. Moreover, Dr. James O. Mason, Assistant Secretary for Health and head of the Public Health Service, stated in his introductory letter to the 1993 PHS document: “Because the possibility of adverse health effects resulting from the use of dental amalgam cannot be fully discounted based on available scientific evidence, I am requesting the National Institutes of Health, the Centers for Disease Control and Prevention, and the Food and Drug Administration to undertake an expanded and targeted program of research, professional and consumer education and product regulation.” Even the preface to the document states: “This report is not intended to serve as the authoritative source on dental amalgam safety, but rather as a planning tool to assist policy makers in deciding on appropriate risk management actions.” Clearly, it is a misrepresentation to claim that this document “proves” the safety of dental amalgam, a tactic that is the basis for the defense of the material.

OTHER COUNTRIES: Although no country has yet banned the use of dental amalgam, several have announced intentions to do so. Other countries have issued recommendations that amalgam should not be used in various situations. These countries include Austria, Canada, Germany, Sweden, and possibly Denmark, Finland and Norway (not verified on these three). Obviously, the trend is to not confirm the absolute safety of amalgam. There must be a sound reason for the positions of these governments.

SCIENCE: Much is made of the “science” on dental amalgam. A few studies concluding the safety of amalgam have been published. These studies, almost without exception, have been conducted by dentists and published in dental journals. The validity of these must be questioned, since state dental boards discipline dentists for “practicing be-
yond the scope of dentistry” if they address medical issues. You cannot have it both ways; either dentists are qualified in toxicology or they are not!

Mercury toxicology is a medical issue, and must be addressed by scientists qualified in the discipline investigated. Studies addressing immunology should be published in immunology journals, those on neurology in neurology journals, and so forth. The studies addressing the amalgam mercury issue that have been conducted by qualified scientists have been published in appropriate medical journals and have almost invariably concluded or suggested a health risk from amalgam mercury. These studies have been noted by the countries listed above!

Recently, a spate of “Consensus Committees” on amalgam safety have been formed. These committees are always dominated by dentist members, ignore the science published in medical journals, and have concluded that amalgam is harmless. It really does not matter how many of these committees are formed; they will not change the validly published scientific studies!

**SUMMARY:** Where does all of this leave the practicing dentist medico-legally? Without doubt, the ADA has attempted to insulate itself from liability over the use of dental amalgam. For over forty years, the FDA has steadfastly refused to accept and classify mixed dental amalgam. Both the ADA and the FDA have placed in writing that mixed dental amalgam is the responsibility of the practicing dentist. Amalgam manufacturers have begun issuing warnings of potential adverse effects, limitations and contraindications to use. Governments other than the United States have issued recommendations against use in various situations. If liability exists for the use of dental amalgam, who is left? The practicing dentist who places the material, especially if no warnings are issued to patients!

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**AMALGAM LAWSUIT IN CANADA!**

On 30 March 1998, a class action lawsuit was filed in the Ontario Court General Division under the Class Proceedings Act, 1992. The defendants are: Johnson & Johnson, Inc.; Dentsply Canada Ltd.; Federal Government of Canada, specifically Health Canada; and every Provincial Dental Regulatory body.

The foundation of the lawsuit is one of informed consent, plaintiffs claiming: Not informed of the risks associated with mercury amalgam fillings; placement of mercury amalgam fillings would not have been accepted if informed of potential risks; the Federal Government was negligent in allowing the manufacturers of mercury and dental amalgam to provide their product to Canadians without prescribed regulations and/or pre-market testing or review; the manufacturers of mercury and dental amalgam have suppressed the information about the potential health and environmental risks both from the dentists and the Canadian public. Plaintiffs claim that they should be reimbursed for the cost of removal and replacement of the mercury amalgam fillings.

The class action lawsuit was announced through press conferences in major cities across Canada, and numerous media articles were generated. In the first week, thousands of Canadians inquired about participation in the lawsuit. Interested Canadians may contact “Canadians for Mercury Relief”, 36 Toronto St., Suite 850, Toronto, ON M5C 2C5, Canada; (416) 410-6314, or email: communications@talkinternational.com.

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**SCIENCE**

**AMALGAM AND ANTIBIOTIC RESISTANCE!**

[Excerpts from “Filling and Drilling Breeds Superbugs”; The Sunday Telegraph, United Kingdom, 24 September 1997, page 17, by Robert Matthews.]

“The fashion among dentists for ‘drilling and filling’ with mercury amalgam in the seventies may have spawned dangerous superbugs immune to antibiotics, say leading microbiologists.” The scientists are studying samples from thousands of patients to determine the extent of the problem, and state that the use of mercury fillings may have to be discontinued if their results confirm earlier results in animals.

The superbugs being investigated include streptococci (responsible for bacterial pneumonia and meningitis), some types of mycobacteria (which cause TB), and staphylococci (a cause of lethal septic shock). The numbers of these superbugs is rising as they pass on their antibiotic resistance to others. The previous animal research demonstrated that mercury derived from amalgam fillings caused both mercury- and antibiotic-resistance, because of the proximity of the gene factors.

The research team is lead by Professor Rohn Rowbury of University College, London, who stated: “If there is an association between the use of mercury amalgams and antibiotic resistance, then the implications are enormous.”
Prenatal Exposure to Methyl Mercury During Late Gestation Affects Cerebral Opiateergic System in Rat Offspring.
Zanoli, P; Truzzi, C; Veneri, C; Brandoli, C; Baraldi, M.
ABSTRACT: Pregnant female rats were orally administered a single dose (8 mg/kg) of methyl mercury chloride (MMC) on day 15 of gestation. The binding characteristics of opioid receptors were studied in the brain of developing rats at different stages of age.

An increased density of opioid receptors was found in whole brain of MMC exposed rats at 21 days (delta receptors) and 60 days (mu and delta receptors) of age, in comparison to matched controls. An enhanced response to morphine administration was detected in MMC exposed rat offspring at day 60 of postnatal life, which, however, was not apparently due to an impaired liver metabolism or renal excretion. Hence, it is reasonable to surmise a possible correlation between receptor up-regulation and increased response to pharmacological challenge.

These data seem to indicate that neurochemical alterations produced in the rat developing organism by prenatal exposure to methyl mercury involves the opiateergic system which undergoes a supersensitivity phenomenon. This effect, which is not detectable in the first postnatal period, shows a delayed onset, being detectable only at the adult stage. These findings seem to indicate that pre- and postnatal methyl mercury exposure induces latent neurochemical and behavioral alterations which could last even after the clearance of the metal from the brain.

BIO-PROBE COMMENT: Astounding evidence that exposure to mercury, especially prenatally, may be a factor in later addiction to drugs!

Metallothionein Induction in Fetal Rat Brain and Neonatal Primary Astrocyte Cultures by In Utero Exposure to Elemental Mercury Vapor.
Aschner, M; Lorscheider, FL; Cowan, KS; Conklin, DR; Viny, MJ; Lash, LH.
ABSTRACT: Brain metallothionein (MT) protein and mRNA levels were determined in the fetal rat following in utero (gestational days 7-12) exposure to elemental mercury vapor (Hg0: 300 microg Hg/m3; 4/h/day). Total RNA was probed on Northern blots with [alpha-32PdCTP-labeled synthetic cDNA probes specific for rat MT isoform mRNAs. The probes for MT-I and MT-II mRNA hybridized to a single band of approximately 550 and 450 nucleotides, respectively.

Expression of whole brain MT-I mRNA in full term fetal rats (day 21) was significantly increased (P) by in utero exposure to Hg0 compared to nonexposed controls. This corresponded to a 14-fold increase (P) in fetal brain Hg concentration after in utero Hg0 exposure. In addition, astrocytes from both control and in utero Hg0 exposed fetuses were isolated, and neonatal primary astrocyte cultures were established and maintained in vitro for up to 3 weeks without additional experimental intervention.

Astrocyte monolayers derived from in utero Hg0 exposed fetuses consistently expressed increased abundance of MT-I mRNA transcripts after 1, 2, and 3 weeks in culture (P, P, and P, respectively) compared with controls. The abundance of astrocyte MT-II mRNA was unchanged at 1 and 2 weeks in culture, but was significantly increased at 3 weeks in cultures derived from brains of Hg0 exposed fetuses (P). Consistent with the increase in MT mRNA, an increase in astrocytic levels of MT proteins was noted by Western blot analysis and MT immunoreactivity.

These studies suggest that in utero exposure to Hg0 induces brain MT gene expression, and that MT mRNAs and their respective proteins are useful quantitative biochemical markers of intrauterine exposure to Hg0, a potentially cytotoxic challenge to astrocytes in the developing brain. It is concluded that induction of MT by fetal/Neonatal astrocytes represents an attempt by these glial cells to protect against Hg cytotoxicity in maintaining cerebral homeostasis.

BIO-PROBE COMMENT: This study presents solid evidence that pre- and neonatal exposures to mercury vapor, in amounts that could be derived from amalgam dental fillings, results in conditions in brain tissue reflective of potential harm.

Mercury in Biological Fluids after Amalgam Removal
Sandorh Englund, G; Elinder, C-G; Langworth, S; Schutz, A; Edstrond, J.
ABSTRACT: Dental amalgam is the major source of inorganic mercury (Hg) exposure in the general population. The objective of the present study was to obtain data on changes in Hg levels in blood, plasma, and urine following removal of all
amalgam fillings during one dental session in 12 healthy subjects. The mean number of amalgam surfaces was 18 (range, 13 to 34). Frequent blood sampling and 24 hour urine collections were performed up to 115 days after amalgam removal, and in eight subjects additional samples of plasma and urine were collected up to three years after amalgam removal.

A transient increase of Hg concentrations in blood and plasma was observed within 48 hours after amalgam removal. In plasma, the peak concentrations significantly exceeded the pre-removal plasma Hg by, on average, 32% (1.3 nmol/L; range, 0.1 to 4.2). No increase in the urinary Hg excretion rate was apparent after amalgam removal. An exponential decline of Hg was seen in all media. Sixty days after the amalgam removal, the Hg levels in blood, plasma, and urine had declined to approximately 60% of the pre-removal levels. In seven subjects, who were followed for up three years, the half lives of Hg in plasma and urine were calculated. In plasma, a bi-exponential model was applied, and the half life was estimated at median 88 days (range, 21 to 121). The kinetics of Hg in urine (nmol/24 hrs) fit a mono-exponential model with a median half life of 46 days (range, 35 to 67).

It is concluded that the process of removing amalgam fillings can have a considerable impact on Hg levels in biological fluids. After removal, there was a considerable decline in the Hg levels of blood, plasma, and urine, which slowly approached those of subjects without any history of amalgam fillings.

**BIO-PROBE COMMENT:** Published in a major dental journal! Does this provide documentation for the benefit of removing amalgams? In this study, amalgam removal was accomplished in one setting, using high speed water spray cutting and vacuum evacuation, without rubber dam. The median duration of the dental procedure was 45 minutes (range, 20 to 75). It is notable that, following amalgam removal, plasma mercury levels increased by 32%, but urine mercury levels did not increase.

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Entry of Low Doses of Mercury Vapor Into the Nervous System.

Pamphlett, R; Coote, P.


**ABSTRACT:** Inorganic mercury remains within neurons indefinitely and has been implicated in some human neurodegenerative diseases. We were interested in finding the lowest dose of mercury vapor that resulted in mercury deposition in neurons. Female BALB/c mice were exposed to 25 micrograms mercury/m3 for 2-20 hr or 500 micrograms mercury/m3 for 5-240 min. To see if mouse neurons were more susceptible to mercury vapor than male neurons, male and female BALB/c mice were exposed to 50 micrograms mercury/m3 for 4-24 hr. Mice were perfused with formalin 1-30 weeks after exposure and paraffin sections of brain, spinal cord and kidney were stained for mercury with silver nitrate autometallography.

On light microscopy, spinal motor neurons contained mercury granules after 12 hr exposure to 25 micrograms mercury/m3 or after 30 min exposure to 500 micrograms mercury/m3. Mercury remained in motor neurons 30 weeks after exposure. In female mice, mercury was seen in motor neurons at half the exposure times of male mice. In conclusion, low doses of mercury vapor, well within WHO guidelines for safe human occupational exposure, enter and remain within motor neurons of mice.

**BIO-PROBE COMMENT:** "Lou Gehrig's Disease (Amyotrophic Lateral Sclerosis, ALS)" is a disease of motor neurons. The doses in this study were well within the range that can be encountered from amalgam dental fillings. The authors' comment about the WHO occupational guideline is also notable.

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Metal Ion-Induced Toxic Histamine Release from Human Basophils and Mast Cells.

Schedle, A; Samorapoonpichit, P; Fureder, W; Rausch-Fan, XH; Franz, A; Sperr, WR; Sperr, W; Slavicek, R; Simak, S; Klepetko, W; Ellenger, A; Ghannadan, M; Baghestanian, M; Valen, P.


**ABSTRACT:** Recent data suggest that distinct metal ions can be released from dental alloys or other biomaterials, and may cause toxic effects on various cells. In this study, the effects of 14 metal ions on histamine release from human blood basophils (n= 4), isolated tissue mast cells (lung n= 8, uterus n=2, skin n= 1, gingiva n= 1), the basophil cell line KU-812, and the mast cell line HMC-1 were analyzed.

Of the 14 metal ions, Ag+ (0.33 mM) and Hg2+ (0.33 mM) were found to induce release of histamine in blood basophils, KU-812, mast cells, and HMC-1. The effects of Ag+ and Hg2+ were dose dependent and were observed with 60 min if incubation. In primary mast cells and basophils, Au3+ (0.33 mM) also induced histamine release, whereas no effects of Au3+ on HMC-1 or KU-812 cells were seen. The other metal ions showed no effects on primary or immortal cells within 60 min. However,
Pt4+ (0.33 mM) induced histamine liberation in HMC-1 and lung mast cells after 12 h. The Ag+ and Hg2+ induced rapid release of histamine from HMC-1 was associated with ultrastructural signs of necrosis, but not apoptosis. In contrast, prolonged exposure to Pt4+ (0.33 mM, 14 h) induced apoptotic cell death in HMC-1 cells, as assessed by electron microscopy and DNA analysis.

Together, certain metal ions induce distinct cytotoxic effects in mast cells and basophils. Whereas Ag+, Hg2+, and Au3+ cause direct toxicity, Pt4+ causes cell death through induction of apoptosis. Whether such effects contribute to local adverse reactions to metal-containing biomaterials in vivo remains to be determined.

**BIO-PROBE COMMENT:** The authors of this study are at the School of Dentistry, University of Vienna, Austria. Perhaps the time has come to cease the use of all metals in the oral cavity, including gold. Hopefully, the dental profession in North America will soon begin paying attention to what is occurring in Europe.

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Mercury Compounds and The Immune System: A Review.
Moszczyński, P.

**ABSTRACT:** This article reviews the literature data concerning the immunologic monitoring of animals and cell cultures exposed to mercury compounds. Mercury is present in nature as metallic mercury, mono- and bivalent inorganic compounds, and organic alkyl, aryl and alkoxy-alkyl compounds. Methyl mercury is most important in terms of environmental exposure while metallic mercury is the most common form to which workers are exposed.

The database on immune function disturbances in humans induced by mercury compounds is limited. Immunotoxicity assessment in animals, mainly in rodents, with subsequent extrapolation to man, is the basis of human risk assessment. The strength of in vitro immunotoxicity testing lies in studies aimed at unraveling mechanisms of immunotoxicity. These experimental investigations show clearly that mercury compounds can have immunomodulating activity. Mercuric chloride and methyl mercury inhibit most of animal and human lymphocyte functions including proliferation, expression of cell activation markers on cell surface and cytokine production. These cells exhibit a greater sensitivity to the immunotoxic effects of methyl mercury than to mercuric chloride. Repeated administration of mercuric chloride to rats, mice and rabbits can induce autoimmune response and a membranous nephropathy. In contrast, Lewis rats injected with mercuric chloride do not develop autoimmunity but exhibit immunosuppression. The immunosuppressive effects associated with exposure to chemical substances are often accompanied by increased susceptibility to challenge with infections agents or tumor cells.

Only few reports are available on animal studies of increased mortality connected with exposure to mercury compounds and challenge with infectious agents. It is difficult to establish a relationship between the observed immunomodulatory properties of mercury compounds and their possible carcinogenicity. In fact, the epidemiological studies performed so far failed to bring any conclusive evidence of carcinogenicity of mercury in animal experiments. The induction of renal tumors in male rodents by methyl mercury was observed only.

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Mercury Sensitization Induced by Environmental Exposure
Mori T, Hirai T, Tomiyama T. Iida K., et al.
Nippon Eiseigaku Zasshi. 52(4):661-666, Jan 1988

**ABSTRACT:** We investigated mercury sensitization in relation to urinary and hair mercury concentrations. Patch tests were performed on 215 medical students and these tests demonstrated that 28 students were mercury-sensitized (13.0%). Life-styles were studied by questionnaire in 26 of the mercury sensitized students and 46 of the non-sensitized subjects. Urinary mercury concentrations were measure in 25 sensitized and 46 non-sensitized and hair mercury concentrations were measured in 19 sensitized and 22 non-sensitized subjects. The eating of fish was not significantly associated with mercury sensitization (one-tailed t-test). The number of teeth treated with metals in the sensitized group was significantly higher than in the control group (6.8 +/- 4.3 in sensitized vs. 4.8 +/- 4.1 in non-sensitized, one-tailed t-test, p <0.05). The usage of mercurochrome was not significantly associated with mercury sensitization (chi-squared test). Urinary mercury concentrations were not significantly higher in sensitized subjects. Hair mercury concentrations were significantly higher in sensitized subjects (1.98 +/- 0.91 micrograms/g in sensitized vs. 1.23 +/- 0.53 in non-sensitized, one-tailed t-test p <0.05). These results suggest that mercury sensitization is associated with increased hair mercury concentrations but not with urinary mercury concentrations. In this study it is confirmed that
dental amalgam for treating teeth may be an important factor relating to mercury sensitization.  

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FORUM

Queen and Company.

“The Six Fundamental Defects That Underlie All Oral and Medical Diseases.”

DATE: 4-7 June 1998.
SITE: Colorado Springs, Colorado.

HOTEL/ROOM RESERVATIONS: Summer Suites (800) 747-8483. Suites @ $129/night (up to 4 per suite; by 10 May).

MEETING REGISTRATION: Queen and Company Health Communications, Inc., P.O. Box 49308, Colorado Springs, CO 80949-9308. T: (719) 598-4968; F: (719) 548-1785; email: bev@queenhealth.com. Before 15 April= $695.00; after 15 April= $750.00.

PROGRAM: 2 1/2 day seminar for medical doctors, dentists, chiropractors and other health professionals.

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American Academy of Head, Neck and Facial Pain (AAHNFP)


DATE: 30-31 July, 1 August 1989.
SITE: Chicago, Illinois.

HOTEL/ROOM RESERVATIONS: The Westin Hotel, N. Michigan Ave. & Delaware St., Chicago, IL. (800) 228-3000; (312) 943-7200. AAHNFP rate (by 25 June): $149.00/night (S/D).

MEETING REGISTRATION: AAHNFP, 520 West Pipeline Rd., Hurst, TX 76053. Tuition varies with attendance at presentations, participation in fellowship, etc.

PROGRAM: Presentations for dentists, assistants; workshops, forums, special events.

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IAOMT 1998 ANNUAL MEETING

DATE: 10-12 September 1998.
SITE: Colorado Springs, Colorado.

HOTEL/ROOM RESERVATIONS: Antlers Doubltree Hotel, 4 South Cascade, Colorado Springs, CO 80903. T: (719) 473-5600; F: (719) 389-0259. Rate: $115.00, single or double.

MEETING REGISTRATION: Dr. Michael F. Ziff, Executive Director, P.O. Box 608531, Orlando, FL 32860-8531; T: (407) 298-2450; F: (407) 298-5075. Lunches, Banquet and one auxiliary included in meeting registration: Members= $450.00; non-members= $550.00.

PROGRAM:

☐ W. J. Clifford, MS: “Progress in Reactivity and Sensitivity Testing.”


☐ Mats Hanson, PhD: “European Developments in the Dental Amalgam Issue.”

☐ Walter J. Loesche, DMD, PhD: The Possible Role of Dental Disease in Heart Disease.”

☐ J. C. Pendergrass, PhD: “Gingival Crevicular Fluid: Components and Analysis.”

☐ H. L. “Sam Queen”, DSc, CCN: “Periodontal Disease: An Emerging Risk Factor For Arterial Disease and Other Chronic Conditions.”

☐ Murray J. Viny, DMD: “Scientific Review of Dental Amalgam Biocompatibility.”

IAOMT ACCREDITATION: Core Curriculum Courses, Written and Oral Examinations: Thursday, 10 September. Registration: $300.00 (Includes lunch, courses, exams.)

SPECIAL EVENTS: Welcome No-Host Reception: Thursday, 10 September, 7:30pm. Annual Membership Meeting: Friday, 11 September, 5:00pm; Annual Banquet: Saturday evening, 12 September, 7:00pm.

If you are a mercury-free dentist or are contemplating going mercury-free, you need to join the IAOMT. The IAOMT has helped fund or has been the catalyst for much of the current scientific research demonstrating that dental amalgam is not the benign dental material that 150 years of use and the ADA would like you to believe. Furthermore, the IAOMT is doing something about Standards of Care and Protocols that protect you, your staff and the patient. For membership information contact Dr. Michael F. Ziff, D.D.S. FIAOMT, P.O. Box 608531, Orlando, FL 32860-8531. Tel: 407-298-2450 and Fax: 407-298-3075.