FLUORIDE
A STORM BREWING!
Michael F. Ziff, D.D.S., and Sam Ziff
A member of the New Jersey General Assembly (State Legislature) has recently called upon FDA Commissioner Dr. David Kessler to remove fluoride drops and tablets from the market immediately. Assemblyman John V. Kelly has discovered that these products have never been examined for safety or effectiveness, nor have they ever been approved for use by the FDA.

It all began in November of 1992, after the publication of a study by the New Jersey Department of Health that showed an increase of a certain bone cancer (osteosarcoma) in young males living in communities with fluoridated water supplies compared to those in non-fluoridated areas.

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The study had confirmed results from a larger national study, as well as several animal studies.

The alarmed Assemblyman began an investigation of the subject. He first requested studies demonstrating the safety and effectiveness of fluoride supplements (drops and tablets) from the American Academy of Pediatric Dentistry. They informed him that they had no such studies and referred him to the National Institute of Dental Research (NIDR), the dental branch of the National Institutes of Health (NIH). The NIDR also denied possession of any studies on the subject, claiming that it was the domain of the United States Food and Drug Administration (FDA).

Meanwhile, Assemblyman Kelly became aware of the controversy within the U.S. Environmental Protection Agency (EPA) over the fluoridation of water supplies. The many studies linking fluoride to a number of health problems were being suppressed. These included cancer, hip fractures and other bone fractures. Dr. William Marcus, the senior science advisor in the EPA’s water division had been fired for whistle blowing about the dangers of fluoride. A court trial had fully exonerated Dr. Marcus and ordered the EPA to reinstate him with back pay and damages. To this date the EPA has delayed compliance with the court order.

Since 1938 Federal Law has required that prescription drug manufacturers submit new drug applications with evidence of safety and effectiveness to the FDA for their approval. Assemblyman Kelly requested these studies from the FDA. He was shocked when the FDA informed him that they had no drug applications and no studies demonstrating either the safety or
effectiveness of fluoride drops or tablets. According to Frank Fazzari, the head of the FDA's Prescription Drug Compliance Branch, these supplements are "unapproved new drugs", even though they have been prescribed to millions of children for years.

Assemblyman Kelly, on 3 June 1993, formally requested that Commissioner Kessler comply with United States Law by removing fluoride drops and tablets from the market. He also urged the President of the American Academy of Pediatric Dentistry to immediately inform all members that fluoride supplements are unapproved new drugs for which the FDA has no evidence of safety or effectiveness.

IS FLUORIDE SAFE?

Fluoride is toxic, of that there is no doubt. It is used as an insecticide and rodent poison! One might argue that everything is poisonous if the dose is high enough, so "the dose makes the poison." While this may be true, there are certainly tremendous variances in the "toxic thresholds" of poisons, that dose which causes significant pathology. Who can argue that ethyl alcohol (the form in alcoholic beverages) is as toxic as methyl (wood) alcohol; or that Salmonella toxin is as poisonous as Botulism toxin?

Published research studies have shown that fluoride has a harmful effect on the immune system at exposure levels as low as 0.5 ppm, well below the level added to water supplies. It is also documented that children have died from fluoride exposure from treatments administered in dental offices and that fluoridated toothpaste tubes contain enough fluoride to kill small children.

Further, it has been scientifically demonstrated that fluoride causes brittle bones, leading to easy fracture, as well as a harmful effect on the thyroid gland and a contribution to the hardening of arteries. Finally, fluoride is clearly carcinogenic, a factor that by itself precludes its approval for general use by the public.

All of this is very well documented by Dr. John Yiamouyiannis in his recently updated book "Fluoride, the Aging Factor". Bio-Probe highly recommends this book and considers it "must reading" for individuals interested in the subject, and most certainly for all dentists.

IS FLUORIDE EFFECTIVE IN PREVENTING TOOTH DECAY?

In view of the compelling scientific evidence condemning the use of fluoride, one must question why its use continues. The obvious argument is that its use has resulted in a significant reduction of the incidence of dental caries (decay).

Dental journals are replete with studies and articles suggesting the effectiveness of fluoridated water in the prevention of dental decay. These reports have firmly convinced the vast bulk of the dental profession. On the other hand, other studies provide evidence that fluoridated water does not lead to the reduction of tooth decay.

So who is right? The answer to that question requires careful consideration. First, it must be acknowledged that the incidence of dental decay is decreasing, of that there is no doubt. The debate concerns the reasons for the decrease.

Studies on the effectiveness of fluoride are "epidemiologic" studies, which compare groups of individuals with or without the factor being investigated. One must be extremely careful in considering epidemiologic studies; remember the old adage "figures do not lie, but liars figure". There are a great many factors that can affect the results of epidemiologic studies; these are called "confounding variables."

There can be more than one possible factor influencing a disease, such as dental decay. For example, alterations in customary dietary habits and increased awareness of oral hygiene and dental care are definite possibilities. The wonders of modern communications, especially television advertising, have had a clear impact on life in the developed world.

To evaluate this factor, one must investigate the rates of dental decay in developed nations that have banned or forbidden the addition of fluoride to water supplies. There are eight such nations in western Europe, all of which exhibit reductions in dental decay comparable to those in the United States and other countries allowing fluoridation of water supplies. This evidence suggests that factors other than water fluoridation are responsible for reductions in tooth decay.
Then what about studies comparing fluoridated and non-fluoridated communities in the United States, many of which suggest that the decay rates are lower in the fluoridated areas? Once again, one must carefully evaluate "confounding variables". These studies compare decay rates at specific ages, usually in children. It has been well established that fluoride delays eruption of teeth! Consequently, seven-year-old children, for example, will have fewer adult teeth erupted into the mouth in fluoridated communities as opposed to those in non-fluoridated areas. Naturally, the longer a tooth is exposed to decay producing factors, the greater the incidence of decay. This confounding variable is not considered in the published studies favorable to fluoride.

Another possible confounding variable is investigator bias. Most investigations begin with a purpose in mind. It is very difficult to not look for information favorable to that purpose and to disregard information that is not, especially when continued research funding hangs in the balance. Rationalizing the selection of data in research findings is actually quite easy.

THE BOTTOM LINE!

Let us assume for the moment that fluoride is effective in the reduction of tooth decay, even though that assumption is very questionable. The price paid for that benefit must be considered. If the price consists of increased susceptibility to cancer, suppression of immune defenses, increased brittleness of bones, adverse effect on the thyroid gland, and possible increased susceptibility to cardiovascular disease - then the price is too high! That conclusion is inarguable!

Since fluoride drops and tablets have never been approved for use by the FDA and since the FDA possesses no scientific evidence demonstrating the safety or effectiveness of these products, FDA Commissioner Kessler has no choice but to obey the Law and withdraw these products from the market. Bio-Probe agrees with Assemblyman Kelly wholeheartedly.

As for the supplementation of fluoride to water supplies, this matter falls in the domain of the Environmental Protection Agency. It is clear from existing evidence, that politics has reigned supreme over science on this matter with the EPA. Should this not be corrected by the EPA itself, it is equally clear that a Congressional Investigation of the issue is necessary. Bio-Probe urges its readers to inform their individual United States Senators and Representatives of the conflict, in order that the public health be responsibly served.

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ABSTRACTS

Foo, SC; Ngim, CH; Salleh, I; Jeyaratnam, J; Boey, KW.
Neurobehavioral Effects in Occupational Chemical Exposure.
ABSTRACT:
Neurobehavioral Effects in 30 female workers (aged 18-41, mean 25.6) exposed to an average of 341 mg/m³ (SD 100) toluene for an average of 5.7 years (SD 3.3) compared with 30 matched controls (aged 18-48, mean 25.1), 24 male workers (aged 18-32, mean 24.7) exposed to 268 mg/m³ (SD 185) toluene equivalent of mixed solvent (82% toluene, 12.3% ethyl acetate, and 5.5% methyl ethyl ketone) for 2.3 years (SD 3.0) compared with 24 matched controls (aged 17-31, mean 24.2), and 94 dentists (aged 24-49, mean 31.7) exposed to 0.017 mg/m³ (SD 0.009) of elemental mercury for 7.4 years (SD 5.3) compared with 54 referents (aged 23-50, mean 33.6) were studied. The Z score (made up of Digit Span, Symbols Digits, and Grooved Peg Board) for the workers exposed to toluene was 0.79, for workers exposed to mixed solvents was 0.38, and for the dentists exposed to mercury was 0.42. The Z score for each group of exposed subjects was statistically poorer than that for its controls. Neurobehavioral performance was statistically related to exposure intensity for the toluene-exposed workers and to years of exposure or dose (exposure intensity x years of exposure) for mixed solvent- and mercury-exposed subjects. The type of chemical species and pattern of exposure appear to influence whether the adverse effects will be cumulative.

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ABSTRACT:
A computer-administered neurobehavioral evaluation system in a Chinese language version (NES-C) and a mood inventory of the profile of mood states (POMS) were applied to assess the psychological effects of low-level exposure to mercury vapor in a group of 88 workers (19 males and 69 females, with mean age of 34.2 years) exposed to mercury vapor (average duration of exposure 10.4 years). The well-matched group of 97 nonexposed workers was treated as the control. The intensity of current mercury vapor was relatively mild as reflected by the average level of mercury in the air of the workplace (0.033 mg/m³) and in urine (0.025 mg/liter). The results indicated that the profile of mood states posed was moving to the negative side in Hg-exposed group and most of the NES-C performances, in particular, the mental arithmetic, two-digit search, switching attention, visual choice reaction time, and finger tapping, were also significantly affected compared with those obtained from controls (P 0.05-0.01). The present study and the previous study on the validation of the system suggest that the NES-C we developed is valid for the neurotoxicity screening among the working population exposed to neurotoxic agents.

BIO-PROBE COMMENT: For years, dental authorities have claimed that dental personnel are not adversely affected by working with mercury. These two studies add further evidence to the number of studies that contradict that claim. It should be noted that dental personnel were included and that the mercury vapor exposure levels were below standards set by government agencies and levels frequently documented in dental offices. These studies found adverse neurologic and psychological effects at the low exposure levels, opening the door for further studies on the neurobehavioral status of dental personnel.

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Kubicka-Muranyi, M; Behmer, O; Uhrberg, M; Klonowski, H; Bister, J; Gleichmann, E.

Murine Systemic Autoimmune Disease Induced by Mercuric Chloride (HgCl₂): Hg-Specific Helper T-Cells React to Antigen Stored in Macrophages. Int J Immunopharmacol. 1993, Feb. 15(2):151-61.

ABSTRACT:
The adoptive transfer popliteal lymph node assay (PLNA) was used to demonstrate Hg-specific T-cell responses of mice that were continuously treated with HgCl₂ by a regimen known to induce a systemic autoimmune disease in H-2s (murine histocompatibility complex, haplotype s) mice, but not H-2d mice. We found that spleen cells of B10.S and A.SW donors (both H-2s) responded anamnestically to HgCl₂ by inducing a significant increase in cellularity in the draining PLN of the recipient: In contrast, spleen cells of HgCl₂-treated DBA/2 (H-2d) donors failed to induce an increase in PLN cellularity, and spleen cells of B10.D2/n (H-2d) donors induced no changes or even diminished PLN cellularity upon re-encounter with HgCl₂.

Kinetic studies showed that spleen cells of B10.S donors were stimulatory from day 3 until day 14 of donor HgCl₂ treatment and, when purified splenic T-cells were tested, still on day 28, the last point in time tested. The Hg-specific T-cells prepared from HgCl₂-treated B10.S mice not only induced an increased cellularity, but also B-cell activation to antibody secretion in the draining PLN of the recipient.

Moreover, the Hg-specific donor T-cells transferred could specifically be restimulated by killed peritoneal cells obtained from the same donors or from syngeneic donors previously treated with HgCl₂. Interestingly, when killed peritoneal cells were injected as antigen the amount of Hg required for T-cell restimulation was only 1/40 of that required when free HgCl₂ was used.

Taken together, these results show that an HgCl₂ treatment schedule designed to induce systemic autoimmune disease primes Hg-specific T-helper (Th) cells and generates immunogenic material in peritoneal cells to which the T-cells react. The possible contribution to the pathogenesis of HgCl₂-induced auto-immune disease of these Hg-specific T-cells and the autoreactive T-cells reported in the literature is discussed.
BIO-PROBE COMMENT: It has been well documented that inorganic mercury can induce auto-immune reaction. This important study now demonstrates the ability of this action of mercury to compound with other antigens, some of which may be induced by the mercury itself. The significance of auto-immunity in the development of numerous disease syndromes of previously unknown etiology is becoming increasingly recognized by medical science.

Zalups, RK; Knutson, KL; Schnellmann, RG.
In Vitro Analysis of the Accumulation and Toxicity of Inorganic Mercury in Segments of the Proximal Tubule Isolated from the Rabbit Kidney.

ABSTRACT:
Cellular accumulation and toxicity of inorganic mercury were studied in suspensions (1 mg protein/ml buffer) of proximal tubular segments isolated from the kidneys of rabbits. Mercuric chloride containing trace amounts of radiolabeled inorganic mercury (203Hg2+) was added to the buffer to produce a concentration of inorganic mercury ranging from 0.1 to 10 microM.

Significant release of lactate dehydrogenase (LDH) and significant decreases in oxygen consumption (QO2), which were used as indices of cellular injury, were detected only when the tubules were in the presence of 10 microM inorganic mercury. At this concentration of inorganic mercury, cellular release of LDH increased and QO2 decreased significantly between the 1st and 4th hour of exposure, by which time most of the proximal tubular cells were necrotic. Maximal cellular content of inorganic mercury was attained within the first 5 minutes of exposure, during which time nearly 70% of the inorganic mercury in the bath was removed.

Accumulation of mercury was more gradual when the tubules were exposed to 0.1 microM inorganic mercury. Addition of 40 microM glutathione, cysteine, or bovine serum albumin to the bath provided the segments of the proximal tubule with complete protection from the toxic effects of 10 microM inorganic mercury. The rate of uptake of inorganic mercury was also significantly decreased. By the end of 4 hours of exposure only about 30% of the content of mercury in the bath was abstracted.

These findings indicate that isolated segments of proximal tubules take up inorganic mercury very rapidly and subsequently become intoxicated. They also show that when compounds containing free sulfhydryl groups are in the presence of inorganic mercury in the bath, the rate of uptake of inorganic mercury is significantly decreased and the tubules are provided protection from the toxic effects of the inorganic mercury.

BIO-PROBE COMMENT: It has been well documented that inorganic mercury accumulates in the kidneys. Animal experiments conducted at the University of Calgary have demonstrated that mercury specifically from dental amalgam fillings does the same. This study not only clearly demonstrates a rapid pathologic effect of inorganic mercury on kidney cells, but also shows the protective effect of sulfhydryl compounds, such as glutathione.

Lachapelle, M; Guerin, F; Marion, M; Fournier, M; Denizeau, F.
Mercuric Chloride Affects Protein Secretion in Rat Primary Hepatocyte Cultures: A Biochemical Ultrastructural, and Gold Immunocytochemical Study.

ABSTRACT:
The toxicity of mercury on hepatocytes was studied at the ultrastructural, biochemical, and immunocytochemical levels. Albumin metabolism was examined because it is a representative liver-specific function. A novel cytochemical method using the protein A-gold technique for the in situ localization of albumin in hepatocyte cultures was applied. Primary rat hepatocyte cultures were exposed to increasing HgCl2 concentrations. Cytotoxicity was assessed by measuring the release of lactic dehydrogenase from the cells.

At the highest exposure concentration tested (50 microM), Hg was found to be significantly cytotoxic in contrast to what occurred at 5.0 and 0.5 microM. The level of albumin secreted, as measured by ELISA, was decreased by approximately 38% at 5.0 microM HgCl2 and was found not to be different from that of controls at lower concentrations.
The ultrastructural analysis showed that hepatocytes treated with 5.0 microM HgCl₂ undergo drastic morphological changes such as a decreased number of ribosomes associated with the rough endoplasmic reticulum, and the disappearance of the latter organelle, proliferation of the smooth endoplasmic reticulum, and dilation of both the Golgi apparatus and the biliary canaliculus-like structures.

Immunocytochemical detection of albumin-immunoreactive sites using protein A-gold labeling further revealed that these were less abundant in hepatocytes treated with 5.0 microM HgCl₂ (-64%) as compared to control preparations.

These results suggest that one of the effects of mercury on hepatocytes is to affect liver-specific functions such as albumin production, possibly through interference with ribosomal function. This study also demonstrates for the first time the applicability of the high-resolution protein A-gold technique for toxicological investigations on hepatocytes in vitro.

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Rawson, AJ; Patton, GW; Hofmann, S; Pietra, GG; Johns, L.
Liver Abnormalities Associated with Chronic Mercury Accumulation in Stranded Atlantic Bottlenose Dolphins.

ABSTRACT:
Eighteen stranded Atlantic bottlenose dolphins (Tursiops truncatus) examined postmortem were sampled for histologic study. All cases were examined for ferric ion and lipofuscin. Ages were determined from tooth growth layers. Electron microscopic (EM) examination and X-ray spectroscopy (EDAX) were performed. Chemical analysis for mercury was conducted on 12 of the animals by atomic absorption spectrophotometry.

Nine animals were found to have excessive lipofuscin in both liver and kidney. Four of these nine animals also exhibited active liver disease (fat globules, central necrosis, lymphocytic infiltrates) whereas, of the animals without the excessive pigment, only one animal had an active liver lesion. EM and EDAX showed electron-dense amorphous material presumably within lysosomes to be Hg with no deposits on mitochondrial or nuclear membranes noted. Age relationship to portal pigment deposition was positive.

Liver mercury concentrations ranged from 0.01 to 443 micrograms/g of wet weight with all animals having liver pigment yielding values of or above 61 micrograms/g, whereas all animals lacking pigment had values of or below 50 micrograms/g.

The evidence suggests that the excessive pigment accumulation is related to toxic effects of Hg and presents as increased active liver disease.

BIO-PROBE COMMENT: It is well recognized that mercury is a metabolic poison and that the highest accumulations of inorganic mercury occur in the kidneys, followed by the liver. These studies demonstrate metabolic pathology caused by inorganic mercury in liver tissue that will not be recognized by signs and symptoms analysis or cursory clinical examination.

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Tai, Y; DeLong, R; Goodkind, RJ; Douglas, WH.
Leaching of Nickel, Chromium, and Beryllium Ions from Base Metal Alloy in an Artificial oral Environment.

ABSTRACT:
The use of base metal alloys in dentistry has gained wide popularity in recent years. However, claims of their safety have not been universally accepted.

An artificial oral environment capable of reproducing three-dimensional force-movement cycles of human mastication was used to determine whether nickel, chromium, and beryllium ions were leached from base metal alloy. Twelve pairs of crowns were articulated in the following combinations: metal versus metal, metal versus enamel, metal versus porcelain, and metal versus metal without chewing as a control. In a simulated 1-year period of mastication, the results showed that nickel and beryllium metals were released both by dissolution and occlusal wear.

These findings suggest that if these conditions occur in the oral cavity, the stability of base-metal alloys is subject to question. Further studies are needed to determine whether the leaching reported has long-term consequences for patients receiving base metal restorations.
BIO-PROBE COMMENT: The authors point out that nickel, chromium, and beryllium are classified as carcinogenic, hazardous, and priority toxic pollutants. They state that "the attrition of metal caused by occlusion over a simulated 1-year period of mastication significantly increases the amount of metal ingested" and "both dissolution and occlusal wear were significant factors". The daily beryllium exposure (approx. 8 micrograms/day/crown) was over 200 times the level that the EPA found could increase the lifetime cancer risk to 1:100,000.

The authors also cite other studies published in respected dental journals that question the safety of the use of these materials in dentistry, especially nickel and beryllium. The dental profession can no longer ignore the increasing evidence contradicting the use of these materials.

Yess, NJ.
U.S. Food and Drug Administration survey of methyl mercury in canned tuna.
J AOAC Int. 76(1):36-38, Jan-Feb, 1993.

ABSTRACT:
Methyl mercury was determined by the U.S. Food and Drug Administration (FDA) in 220 samples of canned tuna collected in 1991. Samples were chosen to represent different styles, colors, and packs as available. Emphasis was placed on water-packed tuna, small can size, and the highest-volume brand names. The average methyl mercury (expressed as Hg) found for the 220 samples was 0.17 ppm; the range was 0.10-0.75 ppm. Statistically, a significantly higher level of methyl mercury was found in solid white and chunk white tuna than was found in chunk light and chunk tuna. Methyl mercury level was not related to can size. None of the 220 samples had methyl mercury levels that exceeded the 1 ppm FDA action level.

BIO-PROBE COMMENT: This study should finally put to rest the misleading statements continuously made by the ADA supporting their "fishy mercury" position. For example in their Special Report, When your patients ask about mercury in amalgam, published in JADA, Vol 20:395-398, April 1990 they state: "... A major source of mercury exposure is from the fish in the diet. Swordfish and tuna, and fish taken from mercury-polluted water, contain significant concentrations of mercury. Research shows that you may be exposed to more mercury from fish than from dental amalgams." Conversely, the World Health Organization (WHO) scientific committee that developed the 1991 WHO "Environmental Health Criteria 118 Inorganic Mercury" using the same data the ADA used to make their statements concluded that dental amalgam contributes 3.8-21 micrograms per day of elemental mercury vapor while fish only contributed an average of 2.4 micrograms per day of methylmercury.

Another major consideration missing from the information presented in the above FDA analysis and omitted from any of the ADA statements, is the selenium content of tuna and other fish in which mercury content is being evaluated. In a review of the subject, the National Academy of Sciences in a major paper published in 1978 titled "An Assessment of Mercury in the Environment" drew some very interesting conclusions. For example "The most consistent beneficial influence of selenium has been a reduction of the lethal and neurotoxic effects of methylmercury compounds." or "... Nishigaki et al. (1974) analyzed 279 samples of 24 species of marine fish and found that the fish with higher levels of methylmercury generally also contained even higher levels of selenium (0.5 to 1.0 ug.g). or "Recent studies of human populations that consume large quantities of tuna have revealed no definitive sign of mercury poisoning, although some individuals had elevated mercury levels in blood and hair."

It is obvious that, scientifically at least, there is something radically wrong and inaccurate with the ADA statements on fish Hg contribution to body burden and amalgam Hg contribution to body burden. The statements appear to be designed to lull the "uninformed" dentists and American people into believing you don't have to worry about Hg from amalgams because you get more in your diet from fish. That position ranks with fish that has been sitting in the sun for three days. "It stinks."

SUPPORT THE FOUNDATION FOR TOXIC FREE DENTISTRY LEGAL FUND!
SEND A TAX-FREE CONTRIBUTION TO FTFD, P.O. BOX 608010, ORLANDO, FL 32860-8010
Boffetta P; Merler E; Vainio H.
Carcinogenicity of Mercury and Mercury Compounds.

ABSTRACT:
Mercury and mercury compounds are widely used in modern society, but only sparse data are available on their carcinogenicity. Methylmercury chloride causes kidney tumors in male mice. Mercury chloride has shown some carcinogenic activity in male rats, but the evidence for female rats and male mice is equivocal. Other mercury compounds and metallic mercury have not been tested adequately in experimental animals. Epidemiologic data are available for chloralkali workers, dentists and dental nurses, and nuclear weapons workers, three groups occupationally exposed to low levels of mercury and its compounds, but those highly exposed in the past, such as miners, or populations which have suffered massive environmental exposure have not been adequately studied. However, the sparse epidemiologic data point toward the possibility of a risk of lung, kidney, and central nervous system tumors. Better data are needed on the carcinogenicity of mercury and mercury compounds in humans and experimental animals.

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ANNOUNCEMENT

The Holistic Book Project, Inc. is compiling an international guide listing qualified alternative health care physicians and professional practitioners. Listings are alphabetical by practice and geographic location and include "Biological Dentistry". The resource directory is being published as a companion to "Alternative Medicine: The Definitive Guide", an 800 page encyclopedia of alternative medicine. To be listed (no charge), send your name, address, phone number, professional initials, and specialty (-ies) to the offices of Future Medicine Publishers:

Melinda Bonk
Attn: Resource Guide
716 Mount Curve Blvd.
St. Paul, MN. 55116
Phone/FAX: (612) 690-9626

The Ninth Annual Science Symposium of the International Academy of Oral Medicine and Toxicology (IAOMT) will be held in Chicago, Illinois on 1-3 October 1993.
Date:
• Scientific Session: Friday-Saturday, 1-2 October 1993.
• Reception: Thursday evening, 30 September.
• Annual Meeting: Sunday morning, 3 October.
Site: Hyatt Regency, Oak Brook, Illinois. Room rate - $78.00/night (specify IAOMT).

Program:
» Murray J. Viny, M.D.: University of Calgary, Faculty of Medicine. Current research on mercury amalgam.
» Boyd Haley, Ph.D.: Univ. of Kentucky Alzheimer's Disease research.
» Richard Gordon Foulkes, M.D.: Previous Minister of Health for British Columbia, Canada - On fluoride.
» Peter Duesberg, M.D.: University of California retrovirologist - On HIV.
» James Masi, Ph.D.: Corrosion physicist - On dental amalgam corrosion.
» Others (to be announced).

Chair: Dr. Marcia A. Basciano. 2932 Finley Rd. Downers Grove, IL. 60515.

16th National Dental Seminar in Homeopathy
Date: 22-24 October 1993.
Site: Oak Brook Hills Hotel and Conference Center. 3500 Midwest Road, Oak Brook, IL. 60522. Room rate - $95.00/night. (800) 445-3315.

Contact: National Dental Seminar. P.O. Box 123. Marengo, IL. 60152.

IAOMT REGIONAL MEETINGS 1994
For planning purposes the following dates and places have been established.
» Tucson, Arizona, last week-end in January
» Pittsburgh, Pennsylvania May 7-8, 1994. Meeting will be held at the Sheraton Station Square Hotel. (412)-621-2000.