Dental Mercury: A Factor That Aggravates and Induces Xenobiotic Intolerance

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I know that most men, including those at ease with problems of the greatest complexity, can seldom accept even the simplest and most obvious truth, if it be such as would oblige them to admit the falsity of conclusions which they have delighted in explaining to colleagues, which they have proudly taught to others, and which they have woven thread by thread into the fabric of their lives.

— Leo Tolstoy

Abstract

Individuals who are xenobiotically sensitive to chemicals comprise a living indicator system that enables us to identify that group in the population that is also mercury sensitive.

There is a spectrum of xenobiotic intolerance in the general population that is a function of, among other things, the spectrum of efficiency of the cytochrome P-450 system that exists in the population due to a spectrum of genetic polymorphism.

Dental mercury inactivates those groups, whose function is protection of the cytochrome P-450 system. This inactivation and consequent loss of protection induces xenobiotic intolerance in individuals who are already compromised due to genetic polymorphism and who are the most susceptible individuals in the genetic population to further compromise. These compromised individuals will exhibit a variety of diagnostically confusing heterogenous symptoms.

On March 15, 1991, the Food and Drug Administration convened a hearing on the "Potential Toxicity of Dental Amalgam". I was one of the invited speakers. The following is based on the speech I delivered at that meeting.
My purpose in this presentation is to

1. Mercury from dental amalgam induces symptoms in a sensitive group of the population that has also been observed to be sensitive to xenobiotic substances. (Xenobiotic substances are substances which are foreign to the natural state of an organism. Examples of such foreign substances are petrochemical vapors, chlorinated hydrocarbons, sulfites, and metals which are not metabolically useful.)

2. This sensitive group serves as a marker that warns of the potential danger of dental mercury to the rest of the population who are also at risk but may not yet exhibit symptoms.

3. Dental mercury should be banned.

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I. Symptomatology

The following is a small sample of common symptoms that I have observed to improve when mercury fillings are removed: fatigue, headache, central nervous system dysfunction, inappropriate coldness, sugar intolerance, sugar cravings, gastrointestinal disturbances, myalgia, arthralgia, rhinitis, dermatitis, asthma, and genitourinary dysfunction.[1] These symptoms are so varied and seemingly disconnected that misdiagnosis or no diagnosis is more often the rule. These and many other symptoms can also be produced at will in these sensitive patients by exposure to xenobiotic substances.

These symptoms of xenobiotic intolerance often develop after dental mercury has been inserted into a patient's mouth and remit after the filling has been removed. The duration of time before the onset of symptoms subsequent to the insertion of dental mercury and the duration of time before the remission of symptoms after the mercury has been removed varies from individual to individual due to genetic polymorphism. The great variability of these two time-oriented factors, the

onset of symptoms and the remission of symptoms, makes for difficulty in identifying a cause-and-effect relationship and adds to the confusion in making a diagnosis. This clinically evident cause-and-effect relationship indicates that the protective mechanism against xenobiotic poisoning has been compromised by the presence of mercury in the tissues.

In the words of Gerstner and Huff, as quoted in Goodman and Gilman's classic textbook *The Pharmacological Basis of Therapeutics,* mercury poisoning is often "misdiagnosed for months and even years. The reasons for these tragic delays included the insidious onset of the affliction, vagueness of early clinical signs, and the medical profession's unfamiliarity with the disease".[2]

One of my clinically observed findings is that the mercury intolerant, xenobioti-cally intolerant group often exhibit sugar cravings and sugar intolerance. In some cases even the smallest amount of table sugar will produce a temporary "high" followed by one to two days of symptoms such as those mentioned above. These symptoms gradually improve to some extent over a one to two year period after complete removal of dental mercury. The following is one of the possible explanations for this observed phenomenon.

Thiamine is important in the decarboxylation process of cellular respiration.[3] [4] There is a critical step at the entrance into the aerobic oxidation cycle (Krebs cycle) from the anaerobic (Embden-Meyerhof) pathway. This step involves "coenzyme A". Coenzyme A contains a sulfhydryl group (-SH). These "-SH" groups are susceptible to being inactivated by mercury,[5] and, hence, unable to produce acetyl-coenzyme A. Some molecules will escape the poisoning, depending upon how much mercury is available, and only limited amounts of functional coenzyme A will be available.

Adding more thiamine will enhance an impaired area of the metabolic cycle and compensate for its inefficiency by pushing the reaction "to the right", as follows: The patient will more efficiently utilize whatever limited amounts of still unpoisoned coenzyme A are available by a greater amount of thiamine provided to the decar-boxylation process.

In regard to the interference of thiamine's action by poisons, there is a similarity between the toxic reactions of mercury and arsenic. Selenium is protective against both mercury and arsenic poisoning. Both mercury and arsenic interfere with thia-mine-dependent enzymes (and excess thiamine can be protective, to some extent, against poisoning from both metals). Arsenic poisoning can imitate thiamine deficiency disorders, as it interferes with the thiamine-dependent conversion of pyruvate to acetyl coenzyme A.[6]

These relationships also add weight (but no absolute proof) to the concept that thiamine could be taken in a predictive way to determine if mercury intoxication is present and benefit could ensue from the removal of amalgam dental fillings. Such benefit from taking thiamine is a frequent finding in my practice.

Clinical Cases of Dental Mercury Poisoning that Parallel Laboratory Investigation

1. The T-4 helper cells perform a major function in the immunological protective process that defends against invading pathogenic organisms and cancer cells. David Eggleston, D.D.S., at the University of Southern California School of Dentistry showed that when he removed the mercury amalgam fillings from four volunteers, the number of T-4 helper cells increased by 50%. He was then able to depress the level of T-4 helper cells by 50% by reinsert-ing mercury fillings. When he finally removed these last mercury fillings and replaced them with a non-mercury substitute, the T-4 helper cells recouped this 50% loss.[7]

The following two clinical case studies parallel Dr. Eggleston's laboratory work and show that dental mercury does suppress the immune response.

Case of Mr. J.S.

This is a 60-year-old man who for 30 years suffered from generalized eczema. He was able to obtain some relief and control by avoiding various allergenic foods. At age 57 he developed intractable staphylococcal furunculosis. Despite almost constant antibiotics for two years, the condition

persisted. Mr. J.S. was investigated by a board certified hematologist/oncologist who could find no reason for his illness. Subsequent questioning of Mr. J.S. revealed that his furunculosis began about three months after two mercury fillings were installed. Within one month after these fillings were removed, his condition started to abate. By three months he was entirely free of furuncles, and the antibiotics were stopped. He has been off antibiotics and has remained free from furuncles this last year-and-a-half. Over the last year his chronic eczema of 30 years' duration has practically disappeared, and he is now able to eat foods that were previously allergenic, if they are not eaten in excess.

Case of Ms. M.C.

This is a 22-year-old woman who at age 11 developed a variety of symptoms which later became so incapacitating that she had to leave college. Numerous medical investigations failed to reveal the reason for her illness. History revealed that her illness started at age 11, one year after she had her first mercury filling installed. When I first saw her four months ago, I recommended that her nine mercury fillings be removed.

The following notes are from a letter I received from Ms. M.C. when she was five weeks mercury-free: sleeping well - I wake up rested, fatigue gone, physically stronger, mental acuity recovered, food reactions less severe, pain on side no longer constant or extreme, memory restored, no more diarrhea, headaches rare and not severe, appetite improved, no longer constantly cold, co-ordination returned, immune system strengthened - bad cut healed quickly with no invection. (Ms. M.C. always developed an infection whenever her skin was broken. She described that even when she used prophylactic topical antibiotics, she still developed infections. This represents a manifestation of immune dysfunction. Eggleston is right!) Her complexion improved, menstrual problems were all but eradicated, her overall health improved, not only physically and mentally, but also emotionally, and her self-confidence was restored.

2. The rapidity with which mercury pervades the body is demonstrated in the following experiment. Fritz Lorscheider, Ph.D., of the University of Calgary School of Medicine, inserted ordinary mercury dental amalgam into the teeth of sheep. Radioactive mercury was used as a marker. Within 29 days, mercury could be found in every organ in the sheeps' bodies.[8]

A parallel case study that clinically confirms this experiment is that of Ms. S.B. This 22-year-old female was in good health until age 20. Within weeks of having a single mercury filling installed, she developed a progressive illness. Most of her symptoms were similar to those of Ms. M.C. (above). I advised her to have this single mercury filling removed. Within 17 days after removal, she reported that her symptoms started to abate. By three months her general health had improved, and she was able to eat most foods without difficulty, provided they were not eaten in excess. I reported to the Food & Drug Administration 30 similar cases out of the hundreds that I have seen. These were filed as "Adverse Reaction Reports". The following is a summary of these 30 cases:

- Four cases of interstitial cystitis responding to removal of dental mercury, one of whom was one week away from having her bladder excised as the only therapy her board-certified urologist could offer to relieve her 20 to 30 bloody urinations a day.
- One case of the recalcitrant sequelae of Lyme Disease in a patient who was fully treated and who only improved after her mercury fillings were removed.
- Five cases of patients who were exposed to excessive amounts of xenobiotic vapors and who subsequently developed immune dysfunction. They improved only after removal of their mercury fillings.
- Thirteen cases of patients who had various intolerances to exogenous substances such as inhalants (particles and vapors) and ingestants (foods and chemicals). These patients were also often intolerant to endogenous organisms, most prominently Candida albicans. These cases include a 62-year-old female (Ms. C.R.) who for about ten years suffered from Meniere's syndrome. She had been investigated by a small army of physicians, including two extensive investigations at the Lahey Clinic — all without benefit.

After her mercury fillings were removed, it took nine months for her to experience some benefit. She subsequently became practically symptom-free and is now able to tolerate previously provocative allergenic foods.

• One case of asthma and dyslexia who recovered after removal of her mercury fillings.

• One case of epilepsy initiating at age ten, at a time when his first mercury filling was installed. He improved only after his mercury fillings were removed at age 40.

• One case of two sisters who had multiple sensitivities and who improved only after their mercury fillings were removed. This suggests to me a genetic biologic predisposition for susceptibility to mercury poisoning.

II. Mercury, Selenium and Cadmium

Mercury is immunosuppressive and immunodisregulatory.[1 7 9 10 12 13 14 15] Because mercury also combines with and inactivates selenium it may be a contributory factor to the development of cancer.

Selenium is protective against cancer:[16 17 18 19] Schamberg has shown that the incidence of cancer is high in areas where the selenium content of the soil and water is low (and vice versa). The lowest cancer rate (94 cancer deaths per 100,000) was found in Rapid City, South Dakota, which had the highest soil and water levels of selenium in the United States. The highest cancer rate was found in Lima, Ohio, which had the lowest soil and water levels of selenium in the United States. Other U.S. cities fell in between these extremes in a similar pattern when the cancer rate was compared to the selenium levels. Blood selenium levels were used in these studies as an accurate measure of the actual ingestion of selenium resulting from these soil and water levels.[20]

Selenium binds mercury and is protective against mercury poisoning.[21 22 23 24 25 26] Is one of the mechanisms by which selenium protects against cancer the Inactivation of mercury?.

Selenium is a component in glutathione peroxidase. Selenium enhances xenobiotic tolerance by increasing the available glutathione.[27 28 29] Glutathione via glutathione peroxidase and glutathione reductase is involved in the protection against xenobiotic substances and free radicals. The primary function of glutathione peroxidase is the reduction of hydrogen peroxide in organic hydroperoxides via the oxidation of glutathione.[29 30] Glutathione peroxidase is also protective against lipid peroxides.[31]

Glutathione is also utilized by the cytochrome P450 enzyme system in the phase II reaction for detoxifying xenobiotic substances.

Selenium is protective against free radical damage and carcinogen-induced chromosomal breakage.[28 29 32 33 34] Free radicals are thought to be involved with the generation of neoplasms. The absorption of dental mercury will lead to a reduction in biologically available selenium because the selenium will bind to mercury,[22] thus reducing the potential benefits of protection against free radicals by selenium, since less selenium will be available.

Just as arsenic interferes with selenium activity,[21 24 26] so does mercury interfere with selenium activity. The result is diminished availability of selenium and loss of protection against free radical damage. This results in greater potential risk for the development of neoplasms.

Dental mercury may also be a contributory factor in the development of cardiovascular disease. Cadmium has been incriminated in the development of cardiovascular disease.[38] Hypertension in animals was able to be modulated at will by Schroeder by removing cadmium by chelation and then adding it back.[39]

Coronary artery disease is adversely affected by low blood and tissue selenium levels[40] and was benefited by increased intake of selenium.[41 42]

When selenium binds to cadmium both atoms become biologically inactivated.[21] The additional load of mercury from dental amalgam will bind additional amounts of selenium and render it biologically inactive; thus, less selenium will be available for Inactivation of cadmium. The result will be an increased biological availability of cadmium and a greater potential for cardiovascular disease.

Cadmium is encountered as an environmental contaminant. Cadmium is routinely leached out of copper water pipes.[43 44 45]

III. Xenobiotic Intolerance and the Cytochrome P-450 System

Evidence of Xenobiotic Intolerance in the General Population

A segment of the population is intolerant to xenobiotic substances. My findings reveal that this segment often develops intolerance or has existing intolerance aggravated by the presence of dental mercury. It appears that the cytochrome P450 enzyme system may be involved in this hypersensitivity via its "assigned" role as a protective mechanism against poisoning from xenobiotic substances.

The ability of a subset of the population to resist toxicity from exposure to xenobiotic chemicals is, among other things, proportional to the quantity and quality of cytochrome P450 present. There are individual differences in these two factors, and the effective protection in a population varies with the frequency distribution curve. The intensity of clinical symptoms of xenobiotic intolerance similarly varies with this curve and is a function of genetic polymorphism,[46 47 48 49 50 51] making for a wide variety of confusing symptoms.

Work at the Department of Molecular Carcinogenesis at the National Institutes of Health demonstrated that these extremes in xenobiotic intolerance in individuals exist, and that these extremes are a function of the effectiveness of cytochrome P450, which is a result of genetic differences.[52]

The Metabolism of Debrisoquine Serves as a Prototype for Other Cases of Xenobiotic Intolerance

The occurrence in the general population of a human autosomal recessive trait for P450dbl is about one in twelve.[51] The P450dbl enzyme presides over the hydroxylation of debrisoquine, an antihypertensive agent used in Europe and Canada but not in the United States (hence the "db" suffix). This enzyme also inactivates **a** variety of other xenobiotic molecules.

Individuals with this defective gene metabolize the debrisoquine at 1/10 to 1/200 of the rate found in the normal population. Thus they are much less able to deal with exposure to this and other xenobiotic substances. Through this prototype one can get a glimpse into the potential danger of dental mercury.

Dental mercury further compromises these individuals by adding to their metabolic burden by inactivation of the thio groups that protect their cytochrome P450 system. These genetically compromised individuals will be damaged by even so-called "small quantities" of mercury. It is tantamount to compounding a misdemeanor into a felony.

My clinical observations confirm this relationship: that there is a similar wide variety in the ability of individuals to resist poisoning from chemicals and dental mercury. Thirty years of observations have convinced me that these two groups, the chemically sensitive and the mercury sensitive, are in fact one group.

Some Specifics About the Cytochrome P450 System

This system contains a variety of enzymes, all with the common characteristic of containing an oxygen-binding heme group. All of these enzymes deal with oxidative (electron transfer) reactions. The mechanism by which cytochrome P450 system protects against xenobiotic substances involves a two-phase reaction: Phase I - Oxidation of the xenobiotic molecule; Phase II - The oxidized intermediate form of the xenobiotic molecule is hydrated or conjugated with glutathione or glucuronic acid or sulfate. The result is a water-soluble end product that can be excreted through the kidneys.

Laboratory Confirmation of the Clinical Observation that Mercury Aggravates and Induces Xenobiotic Intolerances

It is well known that there are protective -SH groups, i.e., protective thio groups which function to protect the cytochrome P-450 system.[53 54] The affinity of mercury for -SH groups is ten times greater than its affinity for oxygen and chlorine.[55] Mercury will bind firmly to and inactivate these critical -SH groups, which protect the cytochrome P-450 system.[53 54] It is by this process that the unique protective function of these -SH groups is lost due to mercury poisoning and the cytochrome P-450 enzyme system becomes compromised.

This laboratory evidence confirms my clinical observation that mercury is capable

of inducing xenobiotic intolerance. The following is from a personal communication to me from Harry V. Gelboin, Ph.D., Chief, Laboratory of Carcinogenesis, National Institutes of Health: "There are several references in the bibliography which indicate that mercury may interact with some P450s and also may interact with some of the second phase enzymes. Mercury is also a very good candidate for interaction with glutathione through the SH group and thereby reducing the concentration of this compound which has a very high detoxification role. In addition to these enzymes, a host of other metabolic enzymes would contain SH groups sensitive to mercury toxicity."

The Clinical Aspects of Xenobiotic Intolerance

There are certain universally accepted examples of subpopulations that are xenobiotically intolerant by virtue of a genetic inability to deal with foreign substances. Examples of these non-allergenic intolerances are sulfite intolerance, monosodium glutamate intolerance, and aspirin idiosyncrasy.

Do not confuse hypersensitivity with allergy. Allergy is an antigen-antibody reaction. Hypersensitivity includes not only antigen-antibody reactions but also non-antigen-antibody chemical reactions such as enzyme deficiency and enzyme inactivation.

The incidence of allergy to mercury is low, as proven by patch tests. This low incidence contrasts with the higher incidence of hypersensitivity to mercury, as suggested by the commonplace finding of xenobiotic intolerance. This relationship is depicted in the common clinical finding of intolerance among patients to petrochemical vapors, such as solvents, cleansing agents, gasoline fumes, kerosene heater fumes, the vapors of incompletely oxidized hydrocarbons from gas stoves used for cooking and automobile exhaust.

Sulfite Intolerance

Individuals collapse and even die on ingestion of sulfite-laden salads in restaurants. Guidelines and warnings have been issued for the use of sulfites in foods, thereby acknowledging that a subset of the population exists that is genetically different.

Monosodium Glutamate Intolerance and Aspirin Idiosyncrasy

Patrons of Chinese restaurants are alerted on menus that foods without MSG are available on request. Warnings are given to aspirin consumers on labels concerning their possible aspirin idiosyncracy. Yet no warnings are offered to purchasers of dental mercury amalgam.

The contents of mercury amalgam dental fillings have been kept secret from the consumer. The aforementioned warnings are all acknowledgements of society's responsibility to respect and warn this genetically different group. Dental patients are not informed that their so-called "silver" fillings are really 50% mercury — and that mercury is a health hazard. This is mislabeling — or non-labeling.

A juice vendor who sells a 30% orange juice solution as orange "juice" rather than orange drink would be liable for misrepresentation and fraud. Yet fraudu-lant dental mislabeling persists, with the canard that these amalgam fillings are called "silver" because they are silver-colored. Why not call them mercury fillings? Mercury is the same color.

To call them "amalgam" fillings is even more misleading. It is the equivalent of calling them a "soup" of metals without mentioning the ingredients of the soup. "Soup" means nothing to the average consumer.

The following are some clinical observations that the identical symptoms found in these two groups (the xenobiotically intolerant group and the mercury-intolerant group) are in fact the symptoms of a single group. The xenobiotically sensitive group routinely develops many symptoms from exposure to mercury following either a simple dental cleaning or the removal of a mercury filling. The mercury "normally' released during these procedures provides an indicator system for identifying this mercury-sensitive group.

Dental Cleaning and Amalgam Polishing

During the abrasive cleaning process mercury is released and then absorbed by the patient. I have used dental cleaning as

a diagnostic test for mercury intolerance by observing that significant symptoms are routinely produced in this group shortly after the cleaning and may last up to two weeks.

After the cleaning process symptoms are further produced by exposure to small amounts of mercury that are subsequently released when the mercury fillings are polished. This polishing removes a layer of previously reacted metal that was in a higher state of oxidation (i.e., it was less reactive and thus more stable). The polishing reveals the shiny unreacted, unoxidized and more reactive metal below. Following the cleaning process, this shiny, more reactive metal is additionally released by mastication over the next few weeks. The release of mercury provides symptoms throughout this period.

The Process of Removal of Mercury Fillings via Dental Drilling

A cloud of mercury vapor and particles is dispersed by the abrasion of the drill. Even with all the precautions taken by using multiple simultaneous suctions, it has been my experience that this group of xenobiotically intolerant patients routinely get markedly ill from exposure to mercury. The symptoms of mercury poisoning are identical to the symptoms developed from exposure to xenobiotic chemicals. I have seen these symptoms last from days to weeks after removal of a single mercury filling.

That these symptoms from cleaning and drilling are due to mercury poisoning is proven to my satisfaction by the fact that administration of selenium produces abrupt relief of symptoms within hours. The protective effect of selenium against mercury is the most likely reason for this beneficial effect.

Selenium and the "Tuna Obfuscation"

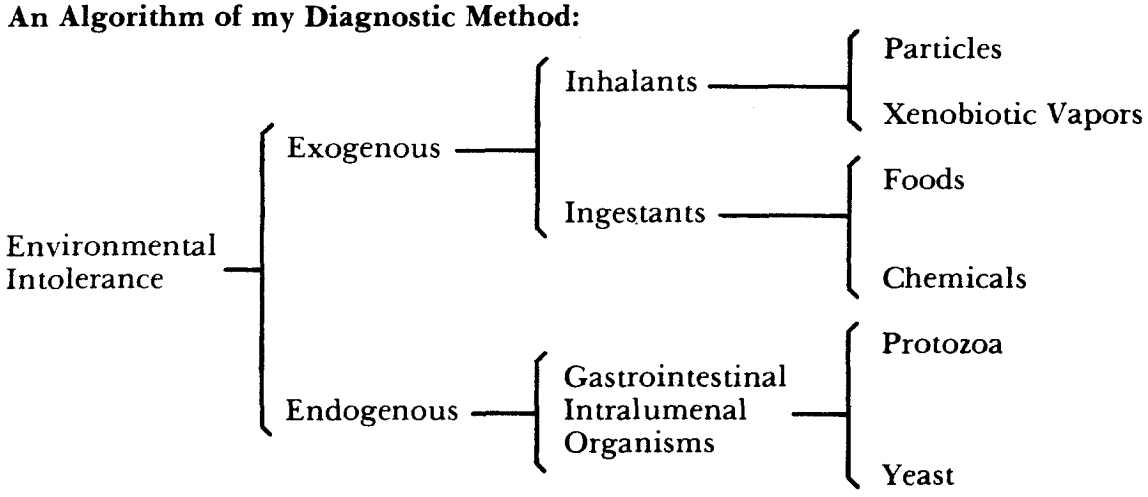
Some mercury advocates say that there is more mercury in tuna than from dental mercury exposure. They leave out the important fact that the tuna protects itself from poisoning by selectively absorbing two molecules of selenium for every molecule of mercury.[22] [26] Thus, the consumer of tuna ingests both the poison and the selenium antidote and is thereby protected, despite the presence of the mercury. No such protection via a selenium antidote is afforded by the dental mercury filling.

Diagnosis

The method I use to determine that a clinical-pathological relationship exists between dental mercury and xenobiotic sensitivity is as follows. I am a dermatologist. Dermatologists are uniquely able to observe the skin as a visible and objective indicator of disease. The skin shows us a relation between the presence and severity of an external eruption vis-a-vis the presence and severity of an internal disease.

The patient's skin condition and his internal medical symptoms get better and worse when I remove and then reintroduce a causative agent, such as an inhalant, a food, or a chemical. Thus, as a dermatologist, I am able to clinically identify the responsible substances causing the disease and then produce and verify a cure.

An Algorithm of my Diagnostic Method:



I divide the patient's world into exogenous and endogenous substances and then make a detailed, customized inventory, item by item, of these substances. A scheme of omission and reintroduction is used, using customized elimination diets as well as environmental controls.

The patient keeps a detailed record of ingestants and inhalants encountered and relates them to symptoms by time of day and geographical location. Preprinted, grid-like guidance forms are provided to the patient as well as printed instructions, books, and detailed verbal guidance. Patients are often studied for months and soon learn to do much of this detective work themselves.

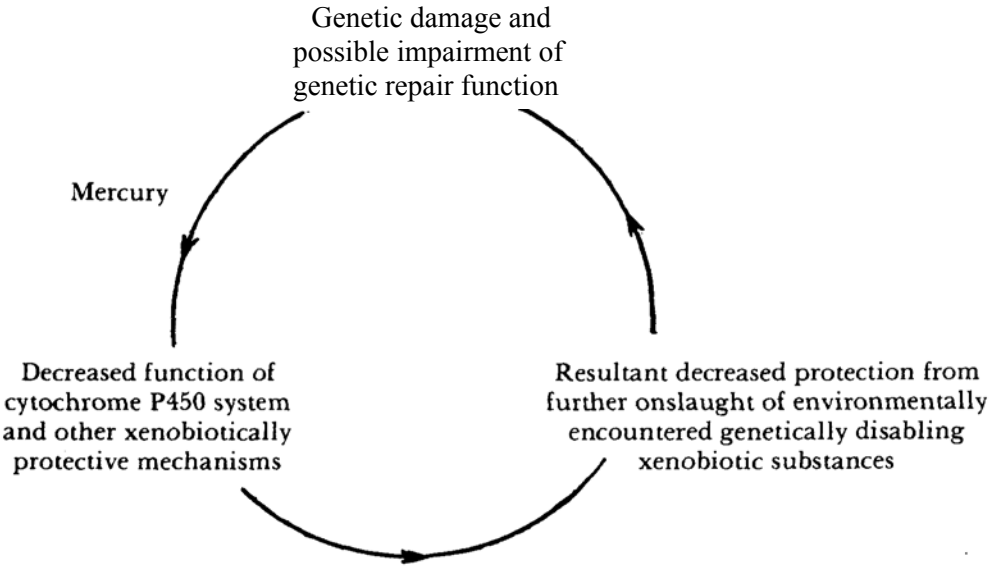
This detailed clinical method contrasts with the usual practice of dentistry.

Dentists are not in a position to make a decision as to whether dental mercury does or does not represent a danger to the patient, because they do not routinely follow the patient's medical health in non-dental parameters after they have inserted mercury into the patient's body.

It is evident that clinical observations have value, and one could not practice medicine without them. Medicine is as much an art as it is a science. The value of clinical observation is that it is a first step toward providing an index of suspicion. It is a starting point for the creation of a protocol that may lead to a statistically valid proof. A reasonable clinical index of suspicion now exists that incriminates dental mercury as a poison.

Not all xenobiotically intolerant individuals (including universal reactors) exhibit this intolerance at birth. The following diagram summarizes the previous discussion and illustrates a possible explanation of how a genetically disposed individual can go on to develop xenobiotic intolerance.

A Vicious Circle of Ever-Increasing Dysfunction



Do these genetically predisposed xenobiotically intolerant individuals leave the world further genetically compromised than when they arrived?

The Dental Mercury Controversy

What are the essential elements of this controversy? The points of agreement are:

1. Mercury is a poison.
2. Mercury is released from the amalgam fillings.
3. The amount of mercury released is only a very small amount.

So why the disagreement? Some believe that this small amount of mercury released from the mercury fillings is not clinically significant. Others believe that this small amount of mercury is clinically significant. The belief that a small amount of mercury is not clinically significant is the result of a major error in analysis. It assumes a binary function: either something is clinically

significant (to all) or is not clinically significant (to all). The error lies in assuming a yes or not answer.

The truth is that intolerance in a large population is not binary, but analog. It indicates "shades of gray" rather than "black or white". The degree of illness developed by an individual is a function of that part of the frequency distribution curve in which he finds himself by virtue of his genetic sensitivity.

To the most sensitive patient this so-called "only small" amount of mercury will be clinically significant. To the least sensitive patient this small amount of mercury may not produce clinical symptoms. The rest of the population will fall somewhere in between these two poles.

The magnitude of disease (MD) induced by mercury in a particular individual is directly proportional to the concentration of mercury (Hg) times the magnitude of his sensitivity (MS). Bear in mind that both of these measurements must be made: the concentration of mercury and the magnitude of his sensitivity:

$$[Hg] \bullet [MS] \propto MD$$

It is not scientific to dismiss an exposure to mercury as "only a little bit" (and therefore not important), if only one measurement (mercury) is made. It is mandatory to also measure the sensitivity of the patient so that two factors are available and a multiplication process can take place. No scientific conclusion can be reached under circumstances in which only one variable is measured.

In other words, you cannot predict what will happen to an individual patient just because you measure the little bit of mercury in his mouth. You need to know "who" he is — genetically.

The concentration of mercury can be measured easily enough. But how does one objectively measure the other variable, the sensitivity of a patient? How many more grams does a severe headache weigh than a mild headache? How many more centimeters does severe fatigue measure than mild fatigue? This is where clinical experience and observation are valuable tools.

We must acknowledge, by using clinical observation, that a sensitive subset of the population does exist; and, like the canaries used in the coal mines, they serve to warn the rest of us. We should apply common sense and remember the cardinal rule of medicine: Before you attempt to do good, first do no harm.

In summary, dental mercury is a dangerous substance. It is a 170-year-old anachronistic mixture of crude coin filings and mercury. It has been grandfathered in without scientific proof of safety and should be banned.

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Editor's Note

Medical Post, April 23, 1991, under heading, "Amalgam Safe For Most People, FDA Panel Agrees," carries the conclusion of a nine member expert panel of the U.S. FDA. They did not conclude mercury amalgams were unsafe, but admitted there were still unanswered questions about how much mercury may leach into the body, and that such questions have to be answered.

Perhaps they are beginning to listen to Dr. Zamm and other experts in mercury amalgam toxicology.

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