NEW RESEARCH - AMALGAM MERCURY CAUSES HARM!

Breakthrough research has just been published in a highly respected medical scientific journal, the renowned FASEB Journal [see abstract in Science section of this issue,]. The study was led by Dr. Diana Eccheverria, Dr. H. Vasken Aposhian, and Dr. James S. Woods. To evaluate the effect of recent exposure to mercury and of mercury body burden on central nervous system function, a battery of neurobehavioral tests was administered to dental personnel. Urine mercury levels were measured before and after the administration of the mercury chelating agent DMPS. The urine mercury levels prior to DMPS chelation were very low, in the 0.4 mcg/liter range found in the general population with mercury amalgam dental fillings.

The authors described four aspects of mercury intoxication found in the scientific literature: 1) psychosomatic symptoms (salivation, insomnia, and loss of appetite); 2) alterations in affect or emotional liability (mood swings, irritability, fatigue, loss of interest, withdrawal, and sweating and blushing); 3) motor effects (in the arms, progressing to uncoordination, imbalance, and cerebella ataxia and tremor); and 4) insidious loss of mental capacity (progressively affecting memory, logical reasoning, or intelligence). Although these effects had previously been associated with higher levels of urine mercury, this study demonstrated the effects in the study group which had very low levels of urine mercury.

The study could not determine a toxic threshold, even at the low urine mercury levels. The authors stated: “Questions remain unanswered concerning the lower threshold of Hg⁰ exposure for behavioral effects, as we found no indication of a lower boundary in any or our subjective or objective results.”; and “Persistent symptoms that appear over a year were selectively associated with Hg body burden; this finding suggests that symptoms may remain undetected in evaluations that rely solely on prechelation urinary measurements of Hg⁰ exposure.”

In conclusion, the authors stated: “Concern for very low level Hg⁰ toxicity is supported by our observations of associations at HgU levels well below the proposed biological standard of 25 mcg/l and below urinary levels that would be expected at the OSHA permissible exposure limit of 50 mcg Hg⁰/m³ in air. The low Hg⁰ exposures between 0 and 4 mcg/l were partially attributable to the number of Hg amalgam fillings in the dental group.” They also stated: “It is clear from the present study that comparing associations with pre- and post-chelation urinary Hg levels revealed patterns of previously unobserved effects. These would not have been identified if they had been evaluated in relation to the traditional pre-chelation urinary Hg levels alone.”

The two main arguments used in defense of the use of mercury dental fillings are 1) dentists are as healthy as is the general population, so patients with amalgam fillings could not be suffering harm; and 2) urine mercury levels in patients with amalgam fillings are well below levels found in humans known to be harmed by mercury exposure. This study clearly destroys both of those arguments.
LAB TESTING FOR PERIODONTAL AND ENDODONTIC INFECTION!

[This article is courtesy of Dr. Boyd Haley and Dr. Curt Pendergrass; Affinity Labelling Technologies, Inc.]

REvised and IMPROVED GCF-TOXICITY REPORT: To date we have analyzed well over 1,500 gingival crevicular fluid (GCF) samples and have made some observations which have allowed us to improve our GCF toxicity test. ALT’s toxicity test now includes four markers of toxicity instead of one. These four markers of toxicity provide clear evidence of bacterial infection at the site of GCF collection and include the following.

* Inhibition of purified enzymes by bacterial toxins present in the GCF.
* Presence of human inflammatory proteins in the GCF.
* Presence of microbial proteins in the GCF.

In future ALT tests, determination of an increased level of toxicity in a GCF sample resulting from bacterial infection will be based on these four markers of toxicity. We now know that the presence of microbial and inflammatory proteins (since they are less mobile than the toxins) are very useful for determining the focal site of infection. For a more thorough discussion of these four markers of GCF toxicity along with the scientific basis and rational for their inclusion in the ALT toxicity assay please refer to Appendix II.

CONTROL GCF SAMPLES: In the future we request that all control samples be taken from the buccal region of an incisor or bicuspid. The rational for this is as follows. Our GCF test usually shows maximum toxicity and presence of bacterial proteins in the GCF of avital teeth. However, some GCF toxicity assays have given results that indicate that some of the control sites also contained significant anaerobic bacteria and toxicity (especially in the molar regions). This surprised us since we did not expect to see such toxicity or bacterial proteins associated with the GCF of healthy looking teeth and gingiva. We therefore collected and analyzed the data from the control GCF samples and did a thorough review of the periodontal literature. We also consulted with some research periodontists concerning the results found with the control GCF samples. What we learned is that virtually all GCF will harbor a resident population of subgingival anaerobic bacteria if a single infected site exists within the mouth. These bacteria also produce volatile toxins (e.g. hydrogen sulfide) which spread rapidly throughout the mouth and produce a measurable level of toxicity detectable by the ALT assay. This is especially true of the large molars toward the back of the mouth where air exchange is less, allowing volatile toxins to build up and anaerobic bacteria to flourish. Good dental hygiene prevents these bacteria from colonizing and setting up severe conditions, but they are there and constantly being reseeded by the major infected site, usually an avital tooth. The buccal region of incisors and bicuspid are more exposed to air which decreases growth of anaerobic bacteria and should represent the best area to get a "control GCF sample". For further discussion please read Appendix I.

BIOCALEX STUDY: There appears to be a lack of a consensus method for treating or reducing the level of toxicity in infected teeth once a problem has been identified. The choices appear to be to do, or redo, the root canal or extract the tooth. The debate rages in this area. The field of treating periodontal disease and endodontic infections is a very active area in dental research and it would be great if root canal treatments could last longer and be less likely to spread infection. However, as best we can determine from talking with many dentists, there is no consensus “best protocol” for treating infected teeth. ALT has been involved in testing the toxicity of extracted teeth with root canals. Some of these teeth were Biocalex filled root canal teeth which, as a group, showed a dramatic reduction in toxicity compared to that observed with gutta percha filled teeth. These results lead us to believe that replacing gutta percha filled root canal teeth with Biocalex may serve to limit bacterial infection and allow the patient to keep their teeth with less danger of infection. Biocalex is a calcium oxide based material which forms calcium hydroxide and calcium carbonate upon contact with water and carbon dioxide, respectively, in the root of an endodontically treated tooth. It thus has the possibility to maintain the canal in an alkaline environment (basic pH) which hinders bacterial growth and may possibly fill the dentinal tubules and limit the spread of bacteria back into the canal. Calcium hydroxide based disinfectants are widely used in endodontics to kill bacteria in infected roots before filling. We have recently tested the GCF from a root canal tooth filled with gutta percha both before and after replacement of the gutta percha with Biocalex. Two weeks after replacement with the Biocalex there was almost a 50% reduction in the level of toxicity measured in the GCF surrounding this tooth. We will have to retest the GCF of this tooth over a period of months to determine if this reduction in toxicity persists. However preliminary, these results with Biocalex do appear promising. What remains to be determined is whether or not the body’s immune system can clean up the external infection in the periodontal ligament and surrounding bone tissues.

As a result of the above observations, ALT is now beginning a research study into the effects of replacement of gutta percha filled root canal teeth with Biocalex. Please note that we have absolutely no financial involvement in Biocalex and would be happy to test...
other materials in a similar way. We are looking for volunteers to participate in this Biocalex Study. All GCF samples taken as part of this Biocalex Study will be analyzed by the company FREE of charge. We would like to test at least 50 such root canal teeth before filling with Biocalex and at 1 and 6 months post treatment. We feel this is an adequate period of time to determine whether or not the reduction in toxicity is lasting. If you or any of your colleagues or patients would like to participate in this study, simply notify us and we will make the necessary arrangements. When submitting these samples for analysis, please indicate on the Sample Requisition Form that the GCF is being analyzed as part of the Biocalex Study. At the conclusion of this study, the company would like to make a formal presentation of our results to all of our ALT customers. We are considering hosting a scientific workshop on analyzing the ALT toxicity reports where we can present and discuss the results of the Biocalex Study. In addition, we plan to invite experts in the fields of Root Canal Therapy, Periodontal Disease, Cavitations, Nutritional Biochemistry, Mercury Neurotoxicity, Oral Microbiology, and Prosthetic Replacement. This would be held either at our headquarters in Lexington, KY or in Cincinnati, OH. Those interested in attending such a meeting please contact: Affinity Labeling Technologies, Inc., A-215 ASTeCC Building, Lexington, KY 40506-0286; T: (606) 257-2300, ext. 291; F: (606) 252-9029.

Appendix I. Control GCF SamplesLT, Inc.

Initially, we were naive in thinking that healthy looking gums meant a non-toxic GCF. Recent studies have shown that the presence of subgingival bacteria results in a low-grade inflammatory reaction at gingival sites without any outward clinical signs of infection. Consulting periodontists have told us, that when a patient has one or more sites of extreme focal infection in the mouth there is a rapid, widespread dissemination of bacteria and bacterial toxins throughout the oral cavity. In such circumstances, there is virtually NO GCF in that person’s mouth which would not be expected to show significant levels of toxicity, even in the absence of outward clinical signs of infection. In other words, even GCF samples taken from a healthy control tooth in a person with a badly infected tooth or periodontal pocket may register a fairly high level of toxicity in the ALT assay. This stands to reason since many bacterial toxins (i.e. hydrogen sulfide and methyl mercaptan) are volatile and easily diffuse from one location to another within the oral cavity. In addition, the bacteria themselves readily spread from the site of focal infection to other sites in the mouth. The ability of oral bacteria and their toxins to spread from a site of focal infection, not only in the mouth, but also to other sites in the body is well documented in the scientific and medical literature. Any oral bacteria which can migrate throughout the whole body and cause a variety of systemic diseases, as numerous studies have shown, has the ability to spread from the site of a focal infection to other teeth in the mouth, even healthy ones. Therefore, we are asking that all ALT clients adhere to the following criteria when taking control GCF samples:

In the future, we ask that you take control GCF samples ONLY from the buccal aspect of incisors and bicusps, preferably the upper ones, since these are usually the least likely to be periodontally involved. As before, try to take the control as far away physically from the infected tooth or teeth as possible. As always, make sure the control tooth is free of any outward clinical signs of periodontal disease and gingivitis. Also, make sure the tooth is vital, nonendodontically treated and free of amalgam restorations, caps or crowns. In addition, always take the GCF samples BEFORE performing ANY type of dental procedures. We ask that you tell your patients to refrain from using any type of toothpaste, mouthwash or under the gum irritant for at least 1 hour before the GCF samples are taken. We have found that these contain compounds which can interfere with the ALT toxicity test. We feel that by following these simple guidelines, the control GCF sample will more accurately reflect the general level of background toxicity present in each individual’s mouth and still provide us with a measure of all the external, environmental factors to which each and every tooth in the mouth has been exposed. As always, ONE control GCF sample will be analyzed from each patient free of charge.

Appendix II. Four Markers of Toxicity Used in the ALT, Inc. GCF Test:

After having collected and analyzed the data from over 1,500 GCF toxicity tests, we have now broadened our original toxicity test to include four markers of toxicity. We have found, and the scientific literature supports, that these four markers provide clear evidence of bacterial infection at the site of GCF collection. These four markers of toxicity are listed below along with the scientific basis and rationale for their inclusion in the ALT toxicity assay. Determination of an increased level of toxicity in a GCF sample resulting from bacterial infection is based on the following four markers of toxicity:

Inhibition of purified enzymes by bacterial toxins present in GCF.

Anaerobic bacteria produce large amounts of metabolic waste products, many of which are toxic to a host of mammalian enzymes, including those used in the ALT toxicity assay. These toxins include volatile sulfhydryl compounds like hydrogen sulfide and methyl mercaptan, ammonia, nonvolatile organic acids such as 2-ketopropionic acid, 2-ketobutyric acid, phenylacetic acid and formic acid just to name a few. These compounds are all
toxic to mammalian enzymes if present in large enough quantities (“the dose makes the poison”). In a normal, vital tooth in a person with good oral hygiene, diet, immune function, etc. the number of these bacteria is kept to a minimum. However, if the subgingival bacteria are allowed to grow and multiply unchecked over time the patient will develop gingivitis, and/or periodontal disease at that site. Also, in an avital, endodontically treated tooth in which the blood supply and innervation have been removed, there is a slow, insidious rise in the level of bacterial involvement over time. Numerous studies have shown that bacteria can and do migrate from the gingival sulcus into the dentinal tubules of both vital and avital teeth, especially if the gingival sulcus is periodontally involved. Bacteria in these infected teeth are largely free from systemic or locally administered bactericidal agents. In fact, some research in periodontal disease has shown that bacteria residing in the dentinal tubules serve as a chronic source of re-infection of the gingival sulcus. These infected teeth can thus act as a “pathogenic reservoirs” from which bacteria and bacterial toxins are slowly released both locally into the sulcus and oral cavity and into the blood stream where they deposit in the tissues and organs of the body. The accumulation of these oral bacteria in extraoral sites has been linked to a whole litany of systemic diseases including infective endocarditis, atherosclerosis, ischemic heart disease, and stroke. The presence of these bacterial toxins in the GCF sample results in inhibition of the activity of the purified enzymes used in the ALT toxicity assay. The level of inhibition and hence the level of toxins can be accurately quantified in this assay.

Presence of human inflammatory proteins in the GCF.

Anytime the body is subjected to a wound or infection there is a stereotypical response involving activation of the immune system at the site of damage. This process involves the release of serum proteins such as albumin, antibodies, compliment proteins, clotting factors, etc. and recruitment of immune cells such as phagocytic white blood cells (PMNs). The purpose of this response is to kill invading bacteria, remove damaged tissues, and promote wound healing at the site of inflammation. These serum components are normally confined to the circulation and their concentration in GCF is usually quite low. In fact, studies have shown that the GCF in a noninflamed sulcus is very similar to the interstitial fluid that bathes the body’s tissues. Unfortunately, nearly all teeth, even those that are nonendodontically treated and show no outward signs of periodontal disease (Control teeth for ALT testing purposes) still harbor a substantial population of subgingival bacteria. As a result, there is very nearly always a low-level chronic inflammation in the gingival sulcus even in the absence of outward clinical signs. This is especially true of the large molars at the back of the mouth. The presence of these inflammatory proteins can be easily detected and quantified in the ALT toxicity assay.

Presence of bacterial proteins in the GCF.

As bacteria grow, multiply and eventually die they are continually releasing their proteins which, over time, accumulate at the site of infection. In the case of periodontal disease or infection of avital teeth, these bacterial proteins are present in the GCF in large quantities. Two of the most prevalent and widely studied bacterial proteins are bacterial alkaline phosphatase and bacterial acid phosphatase. Bacteria use these two proteins to metabolize phosphorylated substrate molecules which they need for growth. Several studies have shown that the level of alkaline phosphatase in GCF is positively correlated with the severity of periodontal disease. Also many studies have shown that the same bacteria which cause periodontal disease are also the same bacteria which cause endodontic infections. One confounding factor that must be considered here is that many human cells also contain both alkaline and acid phosphatases. This is especially true of phagocytic white blood cells (PMNs) which are one of the first immune cells to arrive at a site of inflammation. The presence of these cells and others undoubtedly contribute to the total phosphatase (both bacterial and human) being measured in the ALT GCF toxicity assay.

Presence of bacterial proteases in the GCF.

As with the afore mentioned phosphatases, bacteria also contain an abundant array of proteases which they use to degrade food particles and even host proteins at the site of infection. Many of these proteases, such as collagenase and elastase, are fairly specific as to which proteins they degrade. Others, such as trypsin and chymotrypsin, are fairly nonselective and will degrade almost any protein. When purified enzymes, like those used in the ALT toxicity assay, are added to a GCF sample containing these types of proteases they are literally “chewed up” into smaller pieces. The loss of these purified proteins and the appearance of their proteolytic fragments can be easily detected in the ALT assay. Numerous studies have shown that bacterial proteases can be detected in the GCF. In fact, the Perioscan test developed by Oral B for detection of periodontal disease relies on the presence of a protease found in three species of bacteria. Unfortunately, studies have shown that over 150 different species of bacteria can be found living below the gum line and many of these have been linked to both periodontal disease and endodontic infections.

We feel the results from this 4 part GCF toxicity assay will provide you and your patients with a wealth of information on the status of the tooth and/or gingival sulcus in question. We hope that once these sites of active bacterial infection have been identified, this information can then be used by you, the medically trained
professionals, to recommend a course of treatment to your patients. In addition, we feel this 4-part toxicity GCF assay will give you the ability to measure the lessening of the degree of infection over the course of the treatment. Lastly, and most importantly, we feel that routine use of this test will allow the patient to retain their teeth without the worry or the consequences resulting from the spread of bacteria or their toxins from the mouth to other sites in the body.

When we first developed the ALT GCF toxicity test, we were relying solely on the level of inhibition of our 5 purified enzymes resulting from the presence of bacterial toxins, to determine the level of toxicity present in the GCF. Early on it became apparent to us that the level of bacterial and human inflammatory proteins which we were able to detect in our toxicity assay often provided us with as much information about the status of a tooth or periodontal pocket as did the level of enzyme inhibition. Two of the best-characterized GCF markers of bacterial infection are acid and alkaline phosphatase. Unfortunately for us, two of our test enzymes (pyruvate kinase and creatine kinase) are the same size as these two phosphatases. Since a portion of our analysis relies on our ability to separate proteins on the basis of their size, we could not discern the level of alkaline and acid phosphatase already present in the GCF sample because of the purified pyruvate kinase and creatine kinase we were adding to the GCF sample. Since we feel the ability to quantify the presence of these two phosphatases is so important, we have decided to remove these two purified enzymes from our test mixture. Now, instead of using 5 enzymes in our GCF toxicity test, we will use three, phosphorylase a, phosphoglycerate kinase, and adenylyl kinase, to indicate the presence of bacterial toxins in the GCF. For the toxicity testing of teeth of cavitation materials, ALT will continue using all 5 of the purified enzymes. For these samples, we use only the filtered extract from the third 1 ml wash of the sample for toxicity testing so the confounding presence of these bacterial proteins is not a problem. As always, we at ALT, Inc. appreciate your business and we look forward to serving your toxicity testing needs in the future.

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DENTAL BOARD NEWS

[Provided by attorney Charles Brown of Swankin & Turner law firm in Washington, D.C., representing activities of “Consumers For Dental Choice.”]

Because 18 state dental boards had not responded to last year’s petition (Alabama, Arkansas, District of Columbia, Hawaii, Illinois, Maine, Maryland, Massachusetts, Mississippi, Missouri, Nebraska, New Hampshire, North Dakota, Rhode Island, South Dakota, Tennessee, Vermont, and West Virginia), we wrote them again on 2 July. Because we had written Governors and Attorneys General and many of them replied, I could note that point when writing a number of the 18 Boards. I am pleased to report that five of them responded in less than a month’s time.

Massachusetts and Missouri gave excellent responses. Massachusetts’ Executive Director stated in a letter to me dated 13 July 1998: “There are no regulations in place that would prohibit a licensee from discussing the pros and cons of particular restorative materials with a patient.” Obviously, Massachusetts does not subscribe to the ADA line!

Missouri’s Executive Director stated, via letter to me dated 9 July 1998: “There does not appear to be any statutory limitations on a dentist who wishes to practice mercury-free dentistry. That decision rests with each individual dentist. It is the belief of the Missouri Dental Board that consumers have the right to make the most informed decisions about their dental health. Obviously, the dental provider will be an important source for the consumer making informed dental choices.”

Hawaii and Rhode Island gave less specific but, nonetheless, quite acceptable and helpful responses. Hawaii’s Executive Director wrote on 14 July that “Hawaii does not regulate what materials a dentist may use in the dentist’s professional judgement, assuming there is no fraud, misrepresentations or false promises.” Rhode Island’s Administrator stated on 7 July that “There are no statutes or regulations which regulate what materials a dentist may use in his/her professional judgement.”

In contrast, New Hampshire’s board president on 8 July chose to give me a lecture about how good amalgams are. Of course, besides being wrong as a matter of emerging science, he missed the point about our request for a level playing field.

So we have added four more states endorsing a level playing field for mercury-free dentistry. More successes for our side! Also, on 13 July Carol Ward, DAMS vice president, and I, plus others, appeared before the Pennsylvania board to testify for a level playing field, to inform them of examples of mercury poisoning, and to complement them on their existing regulation.

BIO-PROBE COMMENT: The attorneys for Swankin & Turner, Charles Brown and James Turner, should be commended for their tremendous efforts in helping to protect the rights of mercury-free dentists and of patients. Their have been recent events indicating the extent of their success. In the past six or so months, at least seven mercury-free dentists have had complaints dismissed by the dental boards in their states (all of the complaints were filed by dentists, not by patients). The message is getting through to board members in most states, although a few states are still recalcitrant. All biological dentists, and all patients, should help in protecting their rights by financially supporting these efforts! Without financial support, these initiatives cannot
be continued. [Send your tax-free donations to Consumers For Dental Choice, c/o Swankin & Turner, 1424 16th Street, N.W., Washington, D.C., 20036. T: 202-462-8800.]

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SCIENCE

Neurobehavioral Effects from Exposure to Dental Amalgam Hg: New Distinctions Between Recent Exposure and Hg Body Burden.


ABSTRACT: Potential toxicity from exposure to mercury vapor (Hg\(^0\)) from dental amalgam fillings is the subject of current public health debate in many countries. We evaluated potential central nervous system (CNS) toxicity associated with handling Hg-containing amalgam materials among dental personnel with very low levels of Hg\(^0\) exposure (i.e., urinary Hg mcg/l), applying a neurobehavioral test battery to evaluate CNS functions in relation to both recent exposure and Hg body burden.

New distinctions between subtle preclinical effects on symptoms, mood, motor function, and cognition were found associated with Hg body burden as compared with those associated with recent exposure. The pattern of results, comparable to finding previously reported among subjects with urinary Hg 50 mcg/l, prevents convincing new evidence of adverse behavioral effects associated with low Hg\(^0\) exposures within the range of that received by the general population.

Mobilization of Mercury and Arsenic in Humans by Sodium 2,3-Dimercapto-propane Sulfonate (DMPS).

Aposhian, HV.


ABSTRACT: Sodium 2,3-dimercapto-1-propane sulfonate (DMPS, Dimaval) is a water soluble chelating agent that can be given by mouth or systemically and has been used to treat metal intoxication since the 1960s in the former Soviet Union and since 1978 in Germany. To better approximate the body burdens of Hg and As in humans, DMPS-Hg and DMPS-As challenge tests have been developed. The tests involve collecting an overnight urine, administering 300 mg DMPS at zero time, collecting the urine from 0 to 6 hr, and determining the urinary Hg before and after DMPS is given.

The challenge test, when applied to normal college student volunteers with and without amalgam restorations in their mouths, indicated that two-thirds of the Hg excreted in the urine after DMPS administration originated in their dental amalgams. In addition, there was a positive linear correlation between the amalgam score (a measure of amalgam surface) and urinary Hg after the challenge test. When the DMPS-Hg challenge test was used to study dental personnel occupationally exposed to Hg, the urinary excretion of Hg was 88, 49, and 35 times greater after DMPS administration than before administration in 10 dental technicians, 5 dentists, and 13 non-dental personnel, respectively.

DMPS also was used to measure the body burden of humans with a history of drinking water containing 600 microg As/liter. DMPS administration resulted in a tripling of the monomethylarsonic acid percentage and a halving of the dimethylarsinic acid percentage as related to total urinary As. Because South American animals studied were deficient in arsenite methyltransferase, a hypothesis is presented that arsenite and arsenite methyltransferase may have had a role in the evolution of some South American animals.

BIO-PROBE COMMENT: The current finding that two-thirds of the mercury in the bodies of humans with amalgam fillings comes from the fillings confirms 1992 findings. There can be no doubt that amalgam dental fillings contribute significantly to mercury body burden.

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Increased blood mercury levels in patients with Alzheimer's disease.

Hock C; Drasch G; Golombowski S., et al.


ABSTRACT: Alzheimer's disease (AD) is a common neurodegenerative disorder that leads to dementia and death. In addition to several genetic parameters, various environmental factors may influence the risk of getting AD. In order to test whether blood levels of the heavy metal mercury are increased in AD, we measured blood mercury concentrations in AD patients (n = 33), and compared them to age-matched control patients with major depression (MD) (n = 45), as well as to an additional control group of patients with various non-psychiatric disorders (n = 65). Blood mercury levels were more than two-fold higher in AD patients as compared to both control groups (p = 0.0005, and p = 0.0000, respectively). In early onset AD patients (n = 13), blood mercury levels were almost three-fold higher as compared to controls (p = 0.0002 and p = 0.0000, respectively). These increases were unrelated to the patients' dental status. Linear regression analysis of blood mercury concentrations and CSF levels of amyloid beta-peptide (A beta) revealed a significant correlation of these measures in AD patients (n = 15, r = 0.7440, p = 0.0015, Pearson type of correlation). These results demonstrate elevated blood levels of mercury in AD, and they suggest that this increase of mercury levels is associated with high CSF levels of A beta, whereas tau levels were unrelated. Possible explanations of increased blood mercury levels in AD include yet unidentified environmental sources or
release from brain tissue with the advance in neuronal death.

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**Activation of the Immune System and Systemic Immune-Complex Deposits in Brown Norway Rats With Dental Amalgam Restorations.**

Hultman, P; Lindh, U; Horsted-Bindslev, P.


**ABSTRACT:** Dental amalgam restorations are a significant source of mercury exposure in the human population, but their potential to cause systemic health effects is highly disputed. We examined effects on the immune system by giving genetically mercury susceptible Brown Norway (BN) rats and mercury resistant Lewis (LE) rats silver amalgam restorations in 4 molars of the upper jaw, causing a body burden similar to that described in human amalgam bearers (from 250 to 375 mg amalgam/kg body weight).

BN rats with amalgam restorations, compared with control rats given composite resinous restorations, developed a rapid activation of the immune system, with a maximum 12-fold increase of the plasma IgE concentration after 3 wks (p < 0.001; Mann-Whitney's test). LE rats receiving amalgam restorations showed no significant increase of plasma IgE (p > 0.05). After 12 wks, BN rats with amalgam restorations showed significantly increased (p < 0.05) titers of immune complex (IC) deposits in the renal glomeruli and in the vessel walls of internal organs. These rats also showed a significant (p < 0.05), from six- to 130-fold, increase in tissue mercury concentration in the concentration order kidney > spleen > cerebrum occipital lobe > cerebellum > liver > thymus, and the tissue silver concentration was significantly (p < 0.05) increased from three- to 11-fold. Amalgam implanted BN rats showed a significant ((p < 0.05) increase in copper concentration in the kidney and spleen, and in kidney selenium concentration.

We conclude that dental amalgam restorations release substantial amounts of their elements, which accumulate in the organs and which, in genetically susceptible rats, give rise to activation of the immune system and systemic IC deposits.

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**Shrinkage of Motor Axons Following Systemic Exposure to Inorganic Mercury.**

Pamphlett, R; Png, FY.


**ABSTRACT:** Systemically administered inorganic mercury localizes to motor neurons, but it is not known if mercury injures these neurons. We therefore looked for signs of damage to the motor and sensory neurons of mice that had been exposed to inorganic mercury. Young mice were injected intraperitoneally with either 1 or 2 microg/g of mercuric chloride and perfused 1 or 30 weeks later. The cellular distribution of mercury in the spinal cord was examined with silver nitrate autometallography. The numbers and sizes of myelinated axons in the L5 anterior and posterior roots were quantitated using an image analysis program.

Mercury was found throughout the cytoplasm of motor neuron cell bodies after 1 week and in paranuclear aggregations after 30 weeks. Thirty weeks after exposure to either 1 or 2 microg/g of mercury, fewer large myelinated axons were seen in mercury injected groups than in controls, though total numbers of myelinated axons did not differ between groups. A slight increase in numbers of small axons was seen in the posterior roots of mice exposed to 1 microg/g of mercury.

In conclusion, inorganic mercury remains within mouse neurons for prolonged periods and causes a reduction in the size of myelinated axons in the anterior root and to a lesser extent the posterior spinal root. Inorganic mercury within motor neurons therefore appears to behave as a slowly acting neurotoxin that shrinks motor neurons.

**BIO-PROBE COMMENT:** It is well established scientifically that mercury vapor, being lipid soluble, is more neurotoxic than is inorganic mercury. The effect found in this study, therefore, might be expected to be greater if the exposure is from mercury vapor.

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**Clinical Applications of N-Acetylcysteine.**

Kelly, GS.


**ABSTRACT:** N-acetylcysteine (NAC), the acetylated variant of the amino acid L-cysteine, is an excellent source of sulfhydryl (SH) groups, and is converted in the body into metabolites capable of stimulating glutathione (GSH) synthesis, promoting detoxification, and acting directly as free radical scavengers.

Administration of NAC has historically been as a mucolytic agent in a variety of respiratory illnesses; however, it appears to also have beneficial effects in conditions characterized by decreased GSH or oxidative stress, such as HIV infection, cancer, heart disease, and cigarette smoking. An 18 dose oral course of NAC is currently the mainstay of treatment for acetaminophen induced hepatotoxicity. N-acetylcysteine also appears to have some clinical usefulness as a chelating agent in the treatment of acute heavy metal poisoning, both as an agent capable of protecting the liver and kidney from damage and as an intervention to enhance elimination of the metals.

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**pH Measurement of Root Canal Sealers.**

Huang, TH; Kao, CT.

ABSTRACT: The purpose of this study was to compare the surface pH level of three different type sealers after mixing at various time intervals in vitro. The cements were mixed according to the manufacturer's instructions. They were incubated to set in 100% humidity at 37 degrees C for 1 hr, 24 h, 5 days, 8 days, 2 wk, 3 wk, 4 wk, 5 wk, and 7 wk. pH was calculated by a Twin pH meter.

The pH levels of the three sealers were different at various time intervals (p < 0.0001). The resin based cement had an acid pH level (pH < 7.0). The calcium hydroxide based cement showed a higher alkalinity pH level (pH > 7.0). The zinc oxide-eugenol based cement a similar pH level to the calcium hydroxide cement at the end of the measurement. We postulated that, in endodontic therapy when the most healing is needed, the alkaline based sealer is the choice.

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Interaction of Calcium Hydroxide With Zinc Oxide-Eugenol Type Sealers: A Potential Clinical Problem.
Margelos, J; Eliades, G; Verdelis, C; Palaghias, G.

ABSTRACT: When a ZnOE type sealer was placed in root canals treated previously with calcium hydroxide dressing, an accelerated sealer setting rate occasionally occurred. This clinical observation led to the present experimental design aiming to investigate the effect of calcium hydroxide on a ZnOE cement and ZnOE type sealers and to preliminarily assess the removal efficiency of a calcium hydroxide preparation from root canal systems. Micro-MIR FTIR spectroscopy was used to quantify the effect of calcium hydroxide on the setting reactions of a ZnOE cement and two ZnOE type sealers. The removal efficiency of calcium hydroxide from root canal systems was evaluated after treatment with NaOCl; NaOCl and filing; and NaOCl plus EDTA and filing.

Calcium hydroxide preferentially interacted with eugenol inhibiting the ZnO-eugenol chelate formation. The Ca(OH)2-eugenol interaction was rapid, and kinetically dependent, leading to residual eugenol in the set product. The set ZnOE cement and the ZnOE type sealers in contact with calcium hydroxide were brittle in consistency and granular in structure. Although none of the treatments tested completely removed calcium hydroxide from root canals, treatment with EDTA significantly reduced the extent of residual calcium hydroxide.

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FORUM
INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

The IAOMT has initiated the first formal Accreditation Program for biocompatible dentistry. This program is designed to be the "gateway to the future of dentistry", hopefully leading to eventual board certification. It will also satisfy the increasing demands of the public for "qualified" or "specially trained" biocompatible dentists. The program is comprehensive, including six required Core Curriculum Courses with a written examination, elective courses at IAOMT meetings, interview of two case presentations, and submission of a Standard of Care on a material, procedure or product. IAOMT members interested in enrolling in this program may do so through the office in Orlando, P.O. Box 608531, Orlando, FL 32860-5831, 407-298-2450.

The rapid growth of the IAOMT has provided the resources for addressing two additional areas. Committees have now been formed to develop procedures and policies for a biocompatible approach to periodontal therapy, chaired by Dr. Thomas Baldwin, and cavitations, chaired by Dr. Nicholas Meyer. As soon as possible, committees to address mercury detoxification and root canal therapy will also be formed.

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IAOMT MID-YEAR MEETING

DATE: Friday-Saturday, 19-20 March 1999.
SITE: Las Vegas, Nevada.

HOTEL: Riviera Hotel, 2901 Las Vegas Boulevard, South; Las Vegas, NV. 89109. T: (702) 734-5110; F: (702) 794-9410. IAOMT rate: $95.00/night, single/double (plus 9% tax); $20.00 each additional.

MEETING REGISTRATION: IAOMT, P.O. Box 608531, Orlando, FL. 32860-8531. T: (407) 298-2450; F: (407) 298-3075.

PROGRAM: Speakers to be announced. Friday morning dedicated to IAOMT presentation on biocompatible periodontal therapy.

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IAOMT 1999 ANNUAL MEETING

DATE: Friday-Saturday, 8-9 October 1999.
SITE: Atlanta, Georgia.

HOTEL: The Marque of Atlanta, Perimeter Center.

MEETING HOST: Dr. Ronald Dressler.

PROGRAM: To be announced.

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, DDS, P.O. Box 608531, Orlando, FL 32860-8531. Phone 407-298-2450, Fax 407-298-3075.