August 10, 1995
Mr. Sam Siff
Editor and Publisher
Bio-Probe, Inc.
P.O. Box 608010
Orlando, Florida 32860-0010

Dear Mr. Siff:

This reply to your letter of July 20, 1995 concerning the article that appeared in the July 1995 issue of the Bio-Probe Newsletter, is intended with the intention which is the subject of that article.

As a preliminary matter, I should note that it is common in the publishing business to solicit consent on articles before they are published, not after. However, the following information is provided in the hope it will be of interest to your readers.

The July 1995 issue of the Bio-Probe Newsletter reported on a lawsuit pending in Santa Clara, California that was brought by a patient who claimed he was injured as a result of mercury-containing dental amalgam. The ADA was one of defendants named in the lawsuit. The Superior Court for the County of Santa Clara dismissed the ADA from the lawsuit in January 1993 on the grounds that because of public policy considerations the ADA had no legal duty to the plaintiff in this case.

The ADA is a voluntary association whose object is to encourage the improvement of the health of the public and to promote the art and science of dentistry. One way the Association carries out this object is by disseminating scientific information to the profession on a wide variety of dentally-related topics including dental amalgam. The ADA, the position, based on currently available scientific evidence, that dental amalgam is a safe and effective restorative material, except in the case of the rare patient who is allergic to one of its components. The ADA’s position is consistent with the views of U.S. Public Health Service and its agencies, including the Food and Drug Administration and National Institute of Dental Research.

Others have different views. They are free to communicate their views to the profession in any way of means. For example, the same issue of “Bio-Probe Newsletter” that discusses the Santa Clara lawsuit contains numerous articles critical of the use of dental amalgam and alerts the reader to several seminars and symposia devoted to the issue.

The concept of free speech is key to the court’s decision in the Santa Clara case. In its opinion, the court stated:

To subject Defendant Association to liability would be, in this Court’s opinion, contrary to public policy which the Court perceives to be the promotion rather than suppression of the free flow of scientific information directed to the practicing membership of the professional community to which the Defendants’ publication are directed, i.e., professional dentists. Otherwise stated, to impose a duty/liability in this case would effectively suppress the publication of scientific literature, controversial or not, not only with respect to the members of this professional association but likely with respect to the publications of other professions such as physicians, attorneys, pharmacists and accountants.

It is a fundamental tenet of free speech that truth is most likely to be discovered in the free marketplace of competing ideas, rather than in courts of law. The ADA makes no apology for defending the free exchange of scientific information on the safety of dental amalgam. I hope this information is helpful. All future media inquiries should be directed to the ADA's Office of Media Relations at (312) 440-3865.

Sincerely,

[Signature]

[Name], F.D.S.
Executive Director

[Date]

[Address]

CC: Dr. Daniel M. Meyer, associate executive director, Division of Science
Mr. Peter M. Selkes, associate executive director, Division of Legal Affairs
Mr. Chris Martin, manager, Department of Media Relations

ADA RESPONDS TO BIO-PROBE!

In the July 1995 issue (Vol. 11, No. 4), the Bio-Probe Newsletter featured the news that the American Dental Association (ADA) declared the following in a Court of Law: “The ADA owes no legal duty of care to protect the public from allegedly dangerous products used by dentists, etc.” After discussion with numerous members of the ADA, Bio-Probe was unable to find any members that were aware of that court declaration. We had even received reports (undocumented) that officers in the ADA believed that our report was false. In
view of the absence of awareness of the ADA action, Bio-Probe formally requested a response from the ADA, for publication in the September Newsletter issue. A letter, dated 10 August 1995, was received from John S. Zapp, D.D.S., the Executive Director of the ADA. That letter, in its entirety appears above.

BIO-PROBE COMMENT: Statements contained in the letter from Dr. Zapp, along with those of the ADA in the Court and from the Court itself, raise some interesting, if not critical issues:

1. Dr. Zapp confirmed the ADA position stated in Court and acknowledged that he was aware of the court case (Para. 1), but did not address why the membership of ADA had not been informed of the ADA declaration in court, which was the specific concern of Bio-Probe. This omission demands concern over the motive of ADA for not revealing its vitally important court declaration to its membership, as well as the dental profession generally.

2. While pointing out that "the ADA had no legal duty to the plaintiff in this case" (Para. 3), Dr. Zapp emphasized the ADA's continuing support for the safety and effectiveness of dental amalgam (Para. 4). This indicates that while the ADA encourages dentists to use dental amalgam, legal liability belongs only to the dentist, manufacturers and distributors if the ADA is wrong about its safety. It would seem, therefore, that "this case" should more accurately be stated as "any case."

3. Dr. Zapp states that the ADA position on the safety of dental amalgam is consistent with the U. S. Public Health Service (USPHS) and its agencies. This is clearly inaccurate. In 1994, the U. S. Public Health Service, through its Agency for Toxic Substances and Disease Registry (ATSDR), published its "Toxicological Profile for Mercury" update. New Minimal Risk Levels (MRL’s) for chronic and acute general population exposures to mercury vapor were established and calculated to be 0.28 and 0.40 micrograms of mercury per day, respectively. The ATSDR document on page 125 states "both MRL’s are below estimated exposure levels from dental amalgam." This clearly establishes that the United States Public Health Service considers mercury exposure from dental amalgam to present a risk to patients and renders all previous positions of USPHS and its agencies obsolete and invalid.

4. Dr. Zapp defines the objectives of the ADA as "to encourage the improvement of the health of the public and to promote the art and science of dentistry." (Para. 4) These two goals could very well be conflicting in regard to its policies on dental amalgam. In 1984, the ADA formally admitted that patients with amalgam fillings are chronically exposed to amalgam-derived mercury. Now, the USPHS declares that exposure to be well above its MRL, clearly establishing that the use of amalgam does not improve the health of the public.

On the other hand, the ADA, in its efforts to promote the art and science of dentistry, has clearly been instrumental, if not paramount, in encouraging and promoting the use of dental amalgam. The influence of the ADA in establishing the use of dental amalgam as a "standard of care" for the dental profession is well documented and undeniable. The ADA has published its opinion that dental amalgam is both safe and effective numerous times. In 1987 the ADA altered its "Principles of Ethics and Code of Professional Conduct" to declare the advocacy of the removal of dental amalgam as a means of eliminating the daily exposure to mercury to be unethical, and published encouragement that dentists who do so be disciplined by Dental Boards. This, coupled with and the fact that its membership includes approximately 74% of the dental profession clearly establishes its influence on the use of dental amalgam as the "standard of care" for the dental profession.

5. The statement that "it is common in the publishing business to solicit comment on articles before they are published, not after" came as quite a surprise. The ADA has never requested pre-publication comment from Bio-Probe on any of the numerous articles on dental amalgam it has published in JADA or the ADA News. Is Dr. Zapp establishing a new policy for ADA that will be open to all interested parties?

6. Finally, we reserve the most important topic for last! The comments of Dr. Zapp, and those of the Court itself, clearly establish support for "freedom of speech" on the amalgam controversy. Dr. Zapp even declares that "the ADA makes no apology for defending the free exchange of scientific information on the safety of dental amalgam." In point of fact, this contradicts the 1987 addition on dental amalgam to the ADA "Principles of Ethics and Code of Professional Conduct." The Boards of Dentistry in every state should be made aware of this current ADA position, along with emphasis that the right of freedom of speech is not constitutionally limited to the American Dental Association. This letter, and court case, provides strong new support for the practice of mercury-free dentistry in case of challenges by Boards of Dentistry. In the event that a "scientific information" restriction be utilized, remember the formal position of the ADA on dental amalgam published in JADA in 1990, whereby the ADA states that the "strongest support" for the safety of dental amalgam is that so many have been placed for over 150 years. That position could hardly be characterized as scientific; it is certainly anecdotal to the extreme! 

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CLINICAL RESEARCH ASSOCIATES (CRA) - NEW POSITIONS!

In its most recent two newsletter issues, the widely known and respected Clinical Research Associates featured lead-article attention to the use of dental amalgam and metal crown and bridge alloys.

The "Silver Amalgam Update-1995" [19(8):1, Aug 1995] stated: "Amalgam has been controversial since its introduction, & 1995 is no exception. Current activity shows: (1) Amalgam is, or will be banned in some countries because of its alleged toxicity & allergenic potential; (2) Amalgam debris & mercury are environmental hazard concerns in some countries; (3) Preliminary results of recent CRA survey of practitioners shows 8% of respondents think amalgam should not be used & 79% still consider it useful; (4) Increasing number of patients deny use of amalgam when informed of other restorative alternatives; & (5) Esthetics continues to stimulate people toward tooth colored restorations." CRA concluded: "Increasing numbers of patients have legitimate or supposed reasons to avoid silver amalgam, & today various alternatives are available for their treatment needs. Legally, patients need to be made aware of alternatives."

The subject in the July 1995 issue [19(7):1], entitled "Metal Phobia & Metal Contraindications in Fixed Prosthodontics-Update 1995", discussed non-metal alternatives and concluded: "Increasing numbers of patients have legitimate or supposed reasons to avoid metal alloys for crowns & fixed prostheses."

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EUGENOL IN ENDODONTIC THERAPY

[Bio-Probe is grateful to Darick Nordstrom, D.D.S. for providing the following article.]

In dental school, we were taught to be wary of eugenol-containing endodontic sealers. Only in pedo endos did we use such a sealer (sans gutta percha). As the various calcium sealers reached market, we began investigating their actual effects in hope of finding a more-ideal sealer than AH-26 or eugenol-based. Along the way, we faced up to very realistic concerns about the viability of ANY endodontic sealer.

For most practitioners, there isn't even a hint given in the commonly-read journals that there is a general concern among scientists for the cytotoxicity of eugenol in sealers. In fact, most noticed are the leakage and obturation studies (more about those later). And to add confusion, there are studies that accurately show the pulp tolerating IRM bases. What is the truth about eugenol sealers?

EUGENOL IS INFLAMMATORY. "A Histological Comparison of Periapical and Neural Responses to Two Endodon-
NOTES: Vasculature in the sinus area is poor, with resulting tendency towards inflammation and fibrous proliferation, rather than osteogenesis. Eugenol sealers should NEVER be used in roots that closely approximate the sinus! It may be impossible to reverse such inflammation, even with the use of proven calcifying sealers.

Teeth, especially maxillary posteriors, should be regularly checked for vitality. Endodontia using non-inflammatory techniques and materials (Biocalex) should be performed as soon as tests confirm irreversible pulpitis, thus minimizing the potential for chronicity and various occult effects. Extraction should also be considered for teeth with questionable prognosis in this light.

REFERENCES

BIO-PROBE COMMENT: The information provided by Dr. Nordstrom on the desirability of calcium oxide over other endodontic materials is bolstered by the 1995 study by Granchi, et al [See BPNL 11(4):7, Jul 1995].

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SCIENCE

Halbach, S.
Combined Estimation of Mercury Species Released From Amalgam.

ABSTRACT: Amalgam fillings constitute, after food, the main source of exposure to mercury for the general population. Banning amalgam would incur huge costs for additional dental treatment. An evaluation of potential health risks must be based on the mercury dose released from fillings. In this study, dose is estimated by a new procedure of mercury speciation which elutes the released elemental and inorganic mercury with solvents of differing polarity. We tested the procedure by incubating spherical amalgam pellets in a mixture of light paraffin oil and saline (0.9% NaCl).

Release of mercury into paraffin and saline was linear in relation to both amalgam surface area and exposure time. Measurements with this method were then extended to a group of 21 amalgam-bearing volunteers. The absorbed dose averaged 4.8 micrograms/day compared with 3.7 measured conventionally in intra-oral air from the same persons. With both methods, the dose was significantly correlated to the number of amalgam-covered tooth surfaces. This dose, combined with the nearly equal mercury uptake from food, is far below the acceptable daily intake limit.

BIO-PROBE COMMENT: "Acceptable daily intake limit" TO WHOM?? The United States Public Service's Minimal Risk Level (MRL) for chronic exposure to mercury vapor for the general population is 0.28 micrograms Hg/day intake! This is the legal standard in the United States for non-occupational exposure. Halbach's findings are 17 TIMES higher than the U.S. MRL Standard! This may be acceptable to the author, and apparently to the dental journal that published the study, but it certainly isn't acceptable to the United States Government. Furthermore, the statement in the first sentence that amalgam fillings constitute, after food, the main source of exposure to mercury for the general population is erroneous. In 1991, the World Health Organization in their publication "Environmental Health Criteria 118 - Inorganic Mercury" concluded that the estimated daily intake and retention of "elemental mercury vapor" from dental amalgams exceeds intake and retention from all other sources.

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The following study was published in a medical journal, rather than a dental journal, and provides an interesting contrast of the differing perspectives of the dental and medical communities on this subject.

Barregard, L; Sallsten, G; Jarvholm, B.
People With High Mercury Uptake From Their Own Dental Amalgam Fillings.

OBJECTIVES: To describe people with high mercury (Hg) uptake from their amalgam fillings, and to estimate the possible fraction of the occupationally unexposed Swedish population with high excretion of urinary Hg.

METHODS: Three case reports are presented. The distribution of excretion of urinary Hg in the general population was examined in pooled data from several sources.

RESULTS: The three cases excreted 23-60 micrograms of Hg/day (25-54 micrograms/g creatinine), indicating daily uptake of Hg as high as 100 micrograms. Blood Hg was 12-23 micrograms/l, which is five to ten times the average in the general population. No other sources of exposure were found, and removal of the amalgam fillings resulted in normal Hg concentrations. Chewing gum and bruxism were the probable reasons for the increased Hg uptake. Extrapolations from data on urinary Hg in the general population indicate that the number of people with urinary excretion of >or =50 micrograms/g creatinine could in fact be larger than the number of workers with equivalent exposure from occupational sources.

CONCLUSION: Although the average daily Hg uptake from dental amalgam fillings is low, there is a considerable variation between people; certain people have a high mercury uptake from their amalgam fillings.

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Warfinge, K; Hansson, H; Hultman, P.
Systemic Autoimmunity Due to Mercury Vapor Exposure in Genetically Susceptible Mice: Dose-Response Studies.

ABSTRACT: Six groups of genetically mercury-susceptible female SJL/J (H-2) mice were exposed to mercury vapor at a concentration of 0.3-1.0 mg Hg/m3 air for 0.5-19 hr/day 5 days a week for 10 weeks. The absorbed doses were calculated to be between 75 and 2365 mcg Hg/week/kg body wt (mcg Hg/week/kg). The correlation between the dose and the concentration of Hg in kidney, spleen, and thymus was highly significant (p; Spearman's rank correlation test).

The lowest observed adverse effect level (LOAEL) for serum IgG antinucleolar antibodies (ANoA) was 170 mcg Hg/week/kg, corresponding to a renal mercury concentration of 4.0 +0.76 mcg Hg/g wet wt. The correlation between the absorbed dose and the ANoA titer was highly significant (p; Spearman's rank correlation test), and all mice were ANoA-positive at a dose of 480 mcg Hg/week/kg. High-titer ANoA targeted the nucleolar 34-kDa protein fibrillarin. The LOAEL for B-cell stimulation, measured as an increase in serum IgG2a and IgG1 concentrations, was 360 mcg Hg/week/kg, but the increase was fivefold higher and also included IgE at a dose of 690 and 2365 mcg Hg/week/kg.

The serum Ig concentrations peaked after 2-4 weeks and then slowly declined but, except for IgE, remained significantly increased during the entire exposure time. Glomerular, mesangial IgG immune complex (IC) deposits, accompanied by systemic vessel wall IC deposits, were first detected at a dose of 480 mcg Hg/week/kg. The mesangium also showed increased titers of IgM IC deposits and complement factor C3c. The correlation between the absorbed dose, and the individual titer of IgG, IgM, and C3c, was highly significant (p; Spearman's rank correlation test).

In conclusion, mercury vapor efficiently induced an autoimmune syndrome in genetically susceptible mice, and the LOAEL for the adverse effects varied in the order ANoA-cell stimulation deposits. Comparing the body burden of mercury in mice at the LOAEL for autoantibodies with the body burden in populations of occupationally exposed humans suggests that the safety margin may be narrow for genetically susceptible individuals.

**BIO-PROBE COMMENT:** The authors stated: "We suggest that genetically susceptible individuals, especially in occupational settings, may be at risk for developing adverse immunological reactions due to mercury vapor exposure. Using the data from rodents on mercury vapor exposure, and applying safety factors for interspecies variations and transformation of LOAEL values to NOAEL values, the allowable daily intake for an ordinary adult can be calculated to 11-17 mcg Hg/day. This is very similar to an average mercury dose resulting from inhalation of mercury vapor in persons with dental amalgam fillings."

This information and position, of course, completely contradicts the position of organized dentistry! The following study adds further evidence that so-called "safe" exposures to mercury may not be so safe.

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Perlingeiro, RC; Queiroz, ML.

ABSTRACT: The chemotactic and nitroblue tetrazolium reducing activities of neutrophils from 48 mercury-exposed workers were examined and compared with those of non-exposed, age- and sex-matched individuals. At the time of testing, the exposed population had a mean (± s.d.) urinary mercury concentration of 24.0 ± 20.1 micrograms g-l creatinine and in 44 of these workers urinary mercury levels were below the accepted threshold level (TLV) of 50 micrograms g-l creatinine.

The two neutrophil functions were significantly reduced in the mercury-exposed workers compared with controls. In 28
of these workers, chemotaxis was re-evaluated 6 months later. During the intervening 6 months, the level of hygiene was improved throughout the plant and urinary mercury concentrations were determined monthly in each worker. Despite a significant reduction in urinary mercury concentrations, neutrophil migration did not return to within the normal range.

These results suggest that "safe" level mercury exposure may lead to impairment of neutrophil function.


**ABSTRACT:** Mercury is a recognized environmental toxin. Several organ systems are targeted by this substance and impairment of immune function is known to result from exposure to mercury. Using the patch clamp technique in the whole cell configuration on resting human B lymphocytes we have identified an outward potassium current and studied the effects of mercury on this current.

We present data that demonstrate: (i) the absence of inward currents; (ii) a time and voltage dependent outward current with a threshold of -40 mV and reversal potential near EK⁺; (iii) blocking of this current by TEA (tetraethylammonium chloride) in a dose dependent manner; (iv) a slow time course for recovery from inactivation of this outwardly rectifying K⁺ current and, (v) the diminution and final block of this potassium current by mercury.

These data supplement the findings from our laboratories that demonstrate inhibitory effects on B cell activation by mercury. We propose that the movement of potassium ions across the B cell membrane, an event presumed to be one of the first signals in the mitogenic process, is a target of mercury toxicity.

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**SUMMARY:** Female SJL/N mice were exposed to mercury vapour 5 days/week for 10 weeks, at a mercury concentration of approximately 0.5 mg/m³, 19 h/day; 1 mg/m³, 3 h/day; 0.3 mg/m³, 6 h/day or 1 mg/m³, 1.5 h/day. The total mercury concentrations in the brain were 6.4, 6.3, 1.6 and 0.64 mcg/g tissue, respectively. The mercury distribution in the brains was examined. Mercury was found in almost the whole brain in the two groups with the highest exposure. In the third group, mercury was primarily found in the neocortical layer V, the white matter, thalamus, and the brain-stem. In the fourth group, the white matter and the brain-stem were the targets for mercury accumulation. Similarities and differences between rats and mice in the distribution pattern are discussed.

**DISCUSSION:** Mercury treated, genetically susceptible mice of the H-2a haplotype exhibit a general activation of the immune system with splenic cell hyperplasia, a strong B-cell activation, an increased number of immunoglobulin-secreting cells and hyperimmunoglobulaemia. How the immune disease is linked to the mercury distribution in the brain is not known. However, the present study demonstrates the distribution of mercury within the brain of a mercury sensitive mouse strain.

**BIO-PROBE COMMENT:** Data from Table 1, page 30, reveals that the brain mercury concentration of Group A (0.5 mg Hg/m³, 19 h/day) was virtually identical to that of Group B (1 mg Hg/m³, 3 h/day) and that of Group C (0.3 mg Hg/m³, 6 h/day) was 250% higher than of Group D (1 mg Hg/m³, 1.5 h/day). This information confirms that long-term, lower dosage exposure to mercury vapor is just as dangerous, if not more so, than short-term higher exposures.

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**ABSTRACT:** The effects of mercury vapour on the production of nerve growth factor during development have been examined. Pregnant rats were exposed to two different concentrations of mercury vapour during either embryonic days E6-E11 (early) or E13-E18 (late) in pregnancy, increasing the postnatal concentration of mercury in the brain from 1 ng/g tissue to 4 ng/g tissue (low-dose group) or 11 ng/g (high-dose group).

The effect of this exposure in offspring was determined by looking at the nerve growth factor (NGF) concentration at postnatal days 21 and 60 and comparing these levels to age-matched controls from sham-treated mothers. Changes in the expression of mRNA encoding NGF, the low- and high-affinity receptors for NGF (g75 and p140 trk, respectively) and choline acetyltransferase (ChAT) were also determined.

When rats were exposed to high levels of mercury vapour during early embryonic development there was a significant (62%) increase in hippocampal NGF levels at P21 accompanied by a 50% decrease of NGF in the basal forebrain. The expression of NGF mRNA was found to be unaltered in the dentate gyrus. The expression of p75 mRNA was significantly decreased to 39% of control levels in the diagonal band of Broca (DB) and to approximately 50% in the medial septal nucleus (MS) whereas no alterations in the level of trk mRNA expression were detectable in the basal forebrain. ChAT
mRNA was slightly decreased in the DB and MS, significantly in the striatum.

These findings suggest that low levels of prenatal mercury vapour exposure can alter the levels of NGF and its receptors, indicating neuronal damage and disturbed trophic regulations during development.

**BIO-PROBE COMMENT:** This study is the latest of many showing neurologic damage from prenatal exposure to mercury, even at low levels of exposure and brain tissue accumulation (parts per billion). There are now three animal studies and one human autopsy study clearly demonstrating the transfer of dental amalgam mercury from the fillings of pregnant females into the fetal brain tissue. This is certainly sufficient documentation to call for an elimination of amalgam mercury exposure to unborn babies!

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Kim, C-Y; Watanabe, C; Satoh, H.
Effects of Buthionine Sulfoximine (BSO) on Mercury Distribution after Hg⁰ Exposure.
Toxicology. 98:67-72. 1995.

**ABSTRACT:** Effects of buthionine sulfoximine (BSO), a glutathione (GSH) depletor, on mercury (Hg) distribution were studied after mercury vapor (Hg⁰) exposure (3.2 mg/m³) in mice. BSO (1.6g/kg, i.p.) significantly decreased the GSH levels in the liver, kidney and lung. Pretreatment with BSO increased Hg concentrations in the lung, liver and plasma, but decreased Hg concentration in the kidney. These findings indicate that GSH functions as a determinant in the changes of Hg distribution after Hg⁰ exposure.

Possible causes of the alterations in Hg distribution are discussed, including the anticipated enhancement in the Hg⁰ oxidation process in the tissues with decreased GSH levels as well as the transportation of Hg²⁺ derived from oxidation of Hg⁰. Concentrations of thiobarbituric acid reactive substances (TBARS) were determined as an indicator of oxidative damage, but the increase in TBARS was not detected in any tissue examined.

**BIO-PROBE COMMENT:** The significance of these findings was discussed, but not yet fully determined. It is clear, however, that glutathione levels are a factor determining the influence of mercury vapor exposure on individuals.

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Goodwin, CA; Wheeler-Jones, CPD; Namirian, S; Bokkala, S; Kakkar, VV; Authi, KS; Scully, MF.
Increased Expression of Procoagulant Activity on the Surface of Human Platelets Exposed to Heavy-Metal Compounds.

**ABSTRACT:** One of the essential roles for platelets in haemostasis is in the potentiation of blood clotting due to the contribution of anionic phospholipid from the surface of the cells, as an essential cofactor to the proteolytic reactions of coagulation (platelet procoagulant activity). Only a limited number of agonists are known to initiate platelet procoagulant activity.

In this study the rate of thrombin formation on the platelet surface was observed to increase in a dose-dependent manner upon treatment of washed platelets with heavy-metal compounds. Unlike the immediate increase observed upon treatment of platelets with calcium ionophore, A23187, the change due to these agents was progressive, approaching a maximum after 10 min. The maximum-fold acceleration of the rate of thrombin formation compared with control platelets was calculated for HgCl₂ (56-fold), AgNO₃ (42-fold), phenymercury acetate (24-fold) and thimerosal (14-fold).

The increase in procoagulant activity due to HgCl₂ coincided with a large increase in intracellular calcium and phosphorylation of 22 and 45 kDa proteins. It is considered that the mechanism responsible for the increase in procoagulant activity is exposure of anionic phospholipids. This was detected by a 2-fold increase in the binding of 125I-annexin V upon addition of HgCl₂, compared with resting platelets (3-fold on treatment of platelets with calcium ionophore).

In contrast to the generation of activity by A23187 and other known agonists of this reaction, heavy-metal compounds appeared to cause little or no release of microparticles from the platelet surface. Since HgCl₂ did not cause aggregation of platelets or significant release of serotonin, these findings may give further support to the need for exposure and ligation of glycoprotein IIb:IIIa for vesiculization to occur. Treatment of platelets with heavy metals may constitute a new approach to studying the early changes in the cell membrane which lead to increased expression of anionic phospholipid.

**BIO-PROBE COMMENT:** The authors stated: "In the present paper we demonstrate that heavy-metal compounds are potent agonists for platelet procoagulant activity. This information provides food for thought regarding a possible influence of chronic exposure to mercury on various cardiovascular pathological conditions."

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Kerosuo H; Moe G: Kleven E.
In vitro release of nickel and chromium from different types of simulated orthodontic appliances.

**ABSTRACT:** Five identical samples, each consisting of a fixed appliance, a headgear and a quad-helix for one-half of a dental arch, were immersed in 0.9% sodium chloride for 2 hours, 24 hours and 7 days. A control appliance was subjected to dynamic test conditions in a specially built "oral simulator"
under similar test conditions. A significant release of nickel was detected from the quad-helix during the first two hours in static conditions, whereas during the following two periods significantly less nickel was released from the quad-helix than from the other appliances. The fixed appliance with simulated function showed a significantly higher cumulative release of nickel than the similar appliance in static conditions, 44.2 micrograms (SD 22.8) and 17.1 micrograms (SD 3.4). The total amounts of chromium released from the fixed appliance were significantly lower than those of nickel. No difference in the release of chromium was seen between the static and dynamic. The results indicate certain differences in the amount and pattern of nickel release from different stainless steel orthodontic appliances in vitro. The release rate of nickel from dynamically loaded fixed appliances was found to be accelerated compared with that released under static conditions. Caution should be exercised when applying the results to the in vivo situation.

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Bruce GJ; Hall WB.
Nickel hypersensitivity-related periodontitis.

**ABSTRACT:** As the cost of providing dental care escalates, there has been a substantial increase in the substitution of nonprecious alloys for gold and precious metals found in porcelain-fused-to-metal crowns. These alloys are frequently 69% to 81% nickel. Nickel hypersensitivity is quite common in the general population and periodontal responses have been associated with nickel-containing crowns in nickel-sensitive individuals. In this article, the authors report the case of a nickel-sensitive patient who demonstrated loss of alveolar bone after the placement of crowns with a high nickel content. Non precious alloy crowns can be well tolerated in most cases, however, a history of metal sensitivity should be evaluated.

**BIO-PROBE COMMENT:** The above two articles emphasize the need for the dental profession and the individual dentist to focus more attention on the patient's medical history in regards to potential hypersensitivity to all metals used in dentistry. When in doubt, refer to an Allergist to evaluate or have a materials reactivity test performed. This type of test is done by several laboratories around the country. One that members of the IAOMT have used frequently is Clifford Consulting & Research (719) 550-0008.

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**FORUM**

**IAOMT - 1996 SPRING SESSION**

**DATE:** 22-24 March 1996.
**SITE:** Reno, Nevada.

**HOTEL:** Reno Hilton. 2500 East 2nd Street, Reno, NV 89595. Room rate = $89.00/night + tax, double occupancy. IAOMT Code=ORALT. (800) 648-5080.

**CHAIR:** Duane E. Christian, DMD. 810 N. Nevada St., Carson City, NV 89701. (702) 882-4122

**PROGRAM:**
- Murray J. Vimy, DMD: Scientific Update on Dental Amalgam.
- H. Vasken Apostian, Ph.D.: The DMPS Research Study on IAOMT and other Dentists.
- Diana Echeverria, Ph.D.: Neurobehavioral Effects of Mercury on Dental Personnel.
- John Lee, MD: Fluoride.

**Plus Workshops:**  

**18th NATIONAL DENTAL SEMINAR IN HOMEOPATHY**

**DATE:** 13-15 October 1995.

**HOTEL:** Oak Brook Hills Hotel and Conference Center, 3500 Midwest Rd., Oak Brook, IL 60552. (800) 445-3315. Rate: $99.00/nite (single or double; includes daily continental breakfast and early evening Hors d'oeuvres).

**PROGRAM:** Craig A. Zunka, D.D.S.; Robert R. Canida, D.D.S.; Harris M. Kimbrough, D.D.S.

**REGISTRATION:** National Dental Seminar, P.O. Box 123, Marne, IL 60152. Basic Course = $395; Advanced Course = $375; Spouses/Auxiliaries = $125 (Reduced to $375/$350/$100 if paid by 9/1/95).

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**FREE RADICAL THERAPY**

**DATE:** 27-28 October 1995.

WHERE: Cheyenne Mountain Conference Resort, Colorado Springs, Colorado.

**PROGRAM:** H.L. "Sam" Queen, M.A., C.N.S., C.C.N.
- A Mouthful of Evidence™ and
- Rebuilding Your Patients' Health Through Free Racial Therapy™.

**REGISTRATION:** Queen and Company Health Communications, Inc. (719) 598-4968 - Fax (719) 548-1785. Call or Fax for a complete information package.