ORIGINAL ARTICLE

THE TAURINE/MERCURY/GLYCINE CONNECTION

EPILEPSY - HEART DISEASE - CHOLESTEROL - GALL STONES

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"Taurine, one of the lesser-known amino acids, plays several important roles in the body and is essential to newborns of many species. Along with methionine, cystine and cysteine, it is a sulfur amino acid." "Glycine and taurine have virtually identical action on neurons during experimental conditions...Glycine, taurine and GABA are the major inhibitory neurotransmitters of the human brain." The quotes are from the outstanding book on amino acids written by Eric R. Braverman, M.D. with Carl C. Pfeiffer, M.D., Ph.D. published by Keats Publishing, Inc. New Canaan, CT. The book is entitled "The Healing Nutrients Within - Facts, Findings and New Research on Amino Acids."(1) Every health care provider should read this book. It is written in an easy to read style and contains a wealth of clinical applications in the use of amino acids to treat a myriad of health problems. The book is available from Bio-Probe at the cover price of $27.95 plus $2.00 postage.

Our interest in the sulfur amino acids is the ability of the toxic element mercury to seriously affect the biochemical pathways in the body. In Chapter 11 of Infertility and Birth Defects we explore, in some detail, how mercury affects the transsulfuration pathways and other biochemical pathways not under the direct influence of the sulfur amino acids.(2)

When we think of sulfur amino acids, cysteine and methionine come immediately to mind because so much more has been written about them. Taurine was not considered an essential nutrient and thus did not receive much attention. However, the research that has been done in recent years proves that it plays a major role in electrically excitable tissues, being intimately involved in normal functioning of the brain, heart, gallbladder, eyes and vascular system. Taurine's involvement is in its ability to stabilize electrically the cell membranes by helping the passage of sodium and potassium into and out

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HIGH LEVELS OF MERCURY FOUND IN THE BRAINS OF ALZHEIMER'S VICTIMS.

See page 8
of cells. There is also data showing the same kind of involvement with calcium and magnesium.

We found this of particular interest because of the ability of mercury to influence intracellular levels of all these electrolytes. For example, Shier and Du Bourdieu found that mercury can mimic calcium, competing for the same cellular binding site as calcium and through this mechanism induce cell death.(3) At about the same time, Miyamoto found that at motor nerve terminals, mercury caused irreversible depolarization leading to an irreversible block of transmitter release. This study indicated that the neurotoxic action of mercury was at an intracellular site, possibly involving sulphydryl groups, and that entry was gained through both sodium and calcium channels(4)

In a recent in vitro study, using Glycera red blood cells, mercuric chloride rapidly inhibited taurine influx 95% within one minute of exposure. The results suggested that the sulphydryl groups associated with transport protein are readily accessible to the mercury and that the sodium independent component of taurine transport is susceptible to inhibition. Furthermore, inhibition of transport also occurred without sodium in the medium suggesting that the mercury also acts on the sulphydryl fraction of the carrier protein.(5)

Braverman and Pfeiffer state that in the developing brain, taurine is the most plentiful amino acid and that in the adult brain it is the second most plentiful. Further, within the brain taurine appears to be concentrated in the olfactory bulb (taste and smell), the hippocampus (memory center), and pineal gland thought to be involved in the body's responses to light and dark.(1) In a recent radiographic study with rats, taurine was found in the cortex of the kidney, the liver, pituitary, thymus, adrenals, eyes, nasal mucous membranes, salivary glands, heart and the mucous membranes lining the digestive tract.(6)

It is fascinating to note that in autopsy studies on humans, mercury has also been present in most of these same locations, which again only demonstrates the great affinity of mercury for sulphydryls.

A recent study found that there are approximately two million children who suffer from epilepsy. The authors of the 450 page study concluded that little is certain about the prenatal and perinatal causes of epilepsy.(7) A neurotransmitter is released when a neuron or nerve cell is excited and helps to carry the nerve impulse across the synapse to the next nerve cell, which it inhibits or excites. An important role of taurine is to stabilize electrical activity and help maintain homeostasis, especially in the brain. In addressing the possible relationship of taurine to epilepsy, Braverman and Pfeiffer (1) bring out the following points:

1. One theory of the cause of epilepsy relates to abnormal amounts of glutamic acid in the brain. Taurine works by normalizing the level of glutamic acid and research with mice has shown that a lack of protein or taurine during the first two weeks of life permanently affects the level of glutamic acid in the brain.

2. Taurine increases the rate of action of glutamate decarboxylase, an enzyme which breaks down glutamic acid. Taurine’s anticonvulsant action must be due to its ability to lower brain glutamic acid levels.

3. Vitamin B6 is necessary for the synthesis of taurine. Taurine is closely associated with or bound to zinc and manganese. Stress can deplete the body of zinc and B6 which may also lead to lowered levels of taurine. Certain types of seizure disorders are worsened by stress.

4. The total evidence suggests that brain taurine deficiencies can be corrected by oral taurine and this fact has been demonstrated clinically.

5. Newborns fed formula often develop higher levels of bilirubin (a red bile pigment that can cause jaundice). The main danger of high bilirubin levels at birth is their potential for causing brain damage. When the infant is breastfed or sufficient levels of taurine are added to infant formula, jaundice is rare.

Glycine is also a factor that must be considered when discussing neurological functions within the brain. "In the treatment of epilepsy, data about glycine seem to be similar to those on taurine, with increased levels of glycine in the brain, particularly at the epileptic focus. The brain naturally accumulates more glycine at
the seizure site to protect itself."(1) "Werman and associates (1968) assembled neurochemical and electrophysiological evidence that strongly supports a role for glycine as the inhibitory transmitter between spinal interneurons and motoneurons. Glycine is the most abundant amino acid with inhibitory activity found in the ventral-quadrant gray matter of the spinal cord.(8) "Areas containing the highest concentration of glycine are the thalamus, amygdala, substantia nigra, putamen and globus pallidus, regions that are particularly involved in Parkinson's disease. Glycine is thought to be involved in behaviors related to convulsions and retinal function. It may increase acetylcholine neurotransmission in the memory center of the brain called the hippocampus."(1)

What Drs. Braverman and Pfeiffer have laid out above can become a working hypothesis for mercury vapor from dental fillings to be considered a primary etiological feature in epilepsy. I say working hypothesis because there are clinical case histories reflecting the amelioration or complete cessation of seizures when dental amalgams have been replaced with non-metallic materials. Lets look at some of the facts that would be involved.

**MERCURY:**

1. There is scientific documentation that mercury vapor crosses the placental barrier and is taken up by the fetus.(9)

2. There is evidence that the fetal brain levels of mercury are 30% greater than maternal blood levels of mercury.(10)

3. Pre-term and term infants have very little, if any, capacity to synthesize the taurine necessary for their normal development. In newborn rats and probably also in humans, taurine is supplied almost completely by mother’s milk during the first few days of life.(1)

4. Breast milk contains mercury, reflective of maternal mercury body burden. In a study using rats, the brains of the nursing pups contained a higher concentration of mercury than the mother's brain after only 5 days.(11) Mothers with large numbers of amalgam fillings would be getting a much larger intake of mercury than those without amalgam fillings. This should also translate into higher mercury levels in the breast milk of amalgam bearers. For example, in a recent study, 57 prenatal patients, with no known exposure to mercury, had their blood mercury concentrations monitored from the initial prenatal clinic examination through delivery and postpartum hospitalization. Whole blood total mercury increased 46% by the end of the monitoring period. In their conclusions, the authors stated that patients with large numbers of dental fillings exhibited a tendency to higher maternal blood-mercury levels.(12) This would also mean that the potential for mercury to bind to taurine exists because of the great affinity mercury has for all sulfur molecules.

5. Mercury has the ability to reduce the availability of zinc and vitamin B6. Moreover, cysteine and vitamin B6 are needed in the manufacture of taurine in the body. As a result, mercury could also inhibit the amount of maternal taurine produced by binding with cysteine and reducing the available B6. This same sequence could also cause a similar effect in the developing breast fed child as soon as it was able to start producing its own supply of taurine.(2)

6. In a 1983 study Goodman et al. stated "The mechanisms of action of mercury and cadmium-induced fetotoxicity--whether directly on the differentiating embryonic tissue, indirectly through action on the maternal and placental tissues, or a combination of both--remain to be elucidated. A possible contributory factor in cadmium and mercury fetotoxicity may be an effect on the transmembrane transport of nutrients, such as amino acids, across the placenta to the fetus. An inhibition of nutrient transport may cause fetal death, congenital malformations, or growth retardation."(13) In a review of the neurotoxic effects of mercury published in 1977, Chang also points out the inhibition of amino acid transport into the brain after the administration of mercury.(14)
7. Recent major autopsy studies have demonstrated a positive correlation between numbers and surfaces of amalgams and brain concentrations of mercury.(15-16-17-18)

8. The February 1988 revision to the toxicology text book Biological Monitoring of Toxic Metals now contain a chapter, written by world renowned mercury toxicology experts, entitled The Prediction of Intake of Mercury Vapor From Amalgams, in which the major conclusion is "The release of mercury from dental amalgams makes the predominant contribution to human exposure to inorganic mercury including mercury vapor in the general population."(19)

GLYCINE:

1. In a 1985 study, Clarkson and Kench investigated the urinary amino acids of industrial workers exposed to mercury vapor and noted an increase in glycine excretion.(20)

2. Mercury will couple with glycine through the coordinating amino nitrogen.(21)

3. The conversion of methionine to cysteine may be under the influence of an adequate supply of glycine.(22) This could then affect the quantity of cysteine available for production of taurine.

We feel that the sets of scientific data outlined above present sufficient evidence to support the hypothesis that mercury vapor from amalgam dental fillings may be a totally overlooked etiological factor in epilepsy. Needed now is a very simple epidemiological study to determine that a correlation does exist. This could be easily done by determining the oral metal status of children with medically confirmed epilepsy. After a correlation has been established, a controlled study evaluating the therapeutic efficacy of replacing amalgams with non-metal materials, could be undertaken in a suitable sample of children. The therapeutic efficacy of nutritional augmentation also could be evaluated at the same time.

With minor modification, the above data is also applicable to the other major functions of taurine in the body. More importantly, it is apparent from the scientific literature that the role of taurine in the maintenance of a healthy cardiovascular system may be much greater significance than all other metabolic functions of taurine combined. The fact that almost one million people a year die from heart disease serves to emphasize both the significance of taurine and the possible negative results that mercury may have on its utilization and metabolic functions.

"Taurine is the most important and abundant free amino acid in the heart, surpassing the combined quantity of all the other. It modulates the activity of cyclic AMP, which activates important enzymes in heart muscle and contributes to the muscle's contractility. Taurine also plays a role in the metabolism of calcium in the heart and may affect the entry of calcium into the heart muscle cells where it is essential in the generation and transmission of nerve impulses. The taurine content of the heart is increased during chronic stress as part of the body's adaptive response. Following ischemia (low oxygen in the heat) or necrosis (heart attack), taurine levels drop--sometimes to as low as one-third of normal."(1) That quotation from Braverman and Pfeiffer sets the framework of understanding the variety of scientific data that follows:

1. There is a phenomenon termed the "calcium paradox" involving the irreversible loss of electrical mechanical activity that occurs when a heart is reperfused with Ca$$^{++}$$ after a short period of Ca$$^{+}$$-free perfusion. Calcium paradox recently investigated in the chick heart to determine whether oral pretreatment with taurine or taurine added directly to the perfusate had any effect upon calcium paradox-induced heart failure. In both protocols, taurine significantly reduced the mechanical dysfunction and in taurine pretreatment partially inhibited the excess accumulation of calcium in the myocardium that occurs upon calcium repletion. The protective influence of taurine was accompanied by a reduction of the gain of sodium content that occurs during calcium depletion, and a reduction of the late gain in calcium that occurs during calcium repletion. From this data the conclusion was drawn that taurine plays a role in regulation of calcium homeostasis and membrane stabilization.(23)
2. Renin stimulates an increased intake of water and salt and angiotensin II stimulates only the intake of water in a strain of spontaneously hypertensive rats. The effect of oral taurine on fluid intake was assessed. Previous administration of taurine antagonized the effects of renin and angiotensin II on fluid intake. When the spontaneously hypertensive rats received water containing 3% taurine from 32 to 105 days of age, development of hypertension was inhibited. Taurine was also able to markedly inhibit the salt intake induced by the administration of renin. (24)

3. In another study utilizing DOCA-salt rats that develop hypertension after being given deoxycorticosterone acetate (DOCA) and salt, the hypotensive actions of dietary taurine supplementation were evaluated. Supplementation with 1% taurine could reduce blood pressure after hypertension occurred. At 23 degrees centigrade, cardiac norepinephrine (NE) turnover was markedly accelerated in DOCA-salt rats when compared to normotensive rats, but the 1% taurine supplement restored it toward normal. The 1% taurine supplement was also able to normalize hypothalamic NE turnover. Stimulation of sympathetic nerve discharge by cold exposure after the administration of a NE antagonist, produced marked depletion of NE in most tissues. However, the 1% taurine supplement could normalize this. The authors felt the evidence presented suggested that the hypothalamic noradrenergic system might be involved in the hypotensive action of taurine in DOCA-salt rats. (25)

4. Thousands of feline pets die each year of dilated cardiomyopathy, a disease that can be reversed by supplementing the diet with taurine, an organic compound that cats are unable to synthesize. Studies at the School of Veterinary Medicine at the University of California, Davis, show that when taurine levels in the blood plasma rise, dilated cardiomyopathy symptoms lessen. The scientists say that "chronic taurine depletion in the cat may provide a new model for studying ventricular function, potential inotropic agents and the mechanisms by which taurine affects myocardial function." (26)

5. A recent double-blind study using taurine to treat congestive heart failure was conducted in Japan and produced some excellent results attesting to taurine’s efficacy. Twenty-four patients were treated with 4 grams of taurine given orally each day for four weeks. Nineteen of the twenty-four patients improved on the taurine regime. (27)

Braverman and Pfeiffer (1) also point out other significant aspects concerning taurine that have a direct bearing on the heart: 1) "Taurine content naturally increases in failing hearts, which is thought to be the body’s attempt at metabolic correction." 2) "Taurine also acts as a heart stimulant, like digitalis. Taurine may be safer than the conventional treatments, which do not nourish the heart muscle." 3) Taurine and magnesium are depleted in arrhythmia and some clinical evidence demonstrated that intravenous taurine prevented arrhythmias caused by digitalis. [Bio-Probe Comment: There is scientific evidence that mercury can inhibit magnesium function. With a specific effect on ATP which is actively involved in heart homeostasis.] 4) "Taurine also inhibited the drop in potassium levels inside heart cells which can cause electrical instability and thus arrhythmias." [Bio-Probe Comment: Mercury influences the normal sodium/potassium pump action and one of the documented symptoms of chronic exposure to mercury vapor is arrhythmias. There are also clinical case histories demonstrating the cessation of arrhythmias after amalgam replacement.] 5) "Some patients with mitral valve prolapse, a sometimes rapidly progressive form of congestive heart disease, have been found to have depressed levels of heart muscle taurine. This inborn error further underscores taurine’s importance in the heart and suggests that there may be some cases of the common diagnosis ‘mitral valve prolapse’ which might respond to taurine." 6) "Taurine also improves fat metabolism in the liver and seems to accelerate regression of atherosclerotic plaques inside arteries." (1)

Fats in your diet are broken down (emulsified) in the small intestine so that they can be absorbed through the intestinal wall. This is accomplished by acids contained in the bile excreted by the gall bladder. There are two primary bile acids needed to break down fats: taurocholic acid which cannot be produced without taurine; and glycocholic which cannot be produced without glycine. There are two aspects of bile acid
function that are of great significance in our discussion. The first relates to the normal body processes that serve to control cholesterol levels. Cholesterol is normally excreted in the bile and subsequently in the feces. A recent study demonstrated that taurine did stimulate the formation of taurocholate, which increases cholesterol excretion in the bile; and blood cholesterol levels increased in taurine-dependent rats when their diet contained less sulfur amino acids. Cystine or taurine added to the diet normalized the animals’ cholesterol levels.(22) In other studies, Katan et al. (1982) demonstrated the serum cholesterol lowering effect of dietary-supplemented glycine in rabbits and rats.(28) Sugiyama and his colleagues have observed that when methionine is added to the diet together with sufficient glycine, plasma cholesterol levels were markedly decreased.(22) The second relates to taurine and its possible role in the formation of gall stones. In a 1984 study the binding of calcium ions to micelles of glycine and taurine bile acid conjugates was studied. The findings of this study caused the author’s to speculate that the binding of Ca$^{2+}$ to bile salt micelles may act as one mechanism to lower Ca$^{2+}$ activity in bile and, thus, reduce its tendency to precipitate as insoluble calcium salts and further growth into gallstones.(29) In a 1985 study addressing this issue the author’s reported that taurine conjugation almost completely inhibits the binding of these bile acids to insoluble calcium phosphate. They further observed that because the glycine-conjugated dihydroxy bile acids are predominant in the rabbit, but not in the rat, their results suggested an explanation for the intriguing species-dependence of casein-induced hypercholesterolemia, which is high in the rabbit but absent in the rat.(30)

[Bio-Probe Comment: It is significant to note that research has demonstrated that dietary and environmental methylmercury and mercury is excreted via the bile bound to sulfur containing amino acids and/or glutathione, which contains both cysteine and glycine. Is it possible that amalgam bearers have higher cholesterol levels and a greater incidence of gall stones than non-amalgam bearers? Mercury related reduction of available cysteine/taurine and glycine with its accompanying rise in blood cholesterol and diminished binding of calcium ions could easily be one of the mechanisms at work. Additionally, we are all exposed to lead in the environment and in our diets and lead also has a great affinity for binding with sulfhydryls. The question that needs to be answered is: Are amalgam bearers who continually have an additional chronic exposure to ionic mercury and mercury vapor, over and above normal dietary and environmental intakes of mercury and lead, at greater risk of having higher LDL cholesterol levels and being more prone to gall stones, than non-amalgam bearers?]

It is obvious from the information presented that taurine plays a significant role in the maintenance of a healthy heart and that glycine and taurine are both involved in homeostasis of cholesterol levels. Just as obvious, is the fact that very little research has been done evaluating the effect of mercury on taurine or glycine function. However the limited research that has been done clearly indicates that mercury has a very negative affect on taurine function and a potentially negative effect on glycine. Consequently, in advancing our hypothesis, we have also used the other scientific literature that irrefutably demonstrates the ability of mercury to bind with any thiol (organic compound containing the -SH group) and most sulfur linkages.

A WORD OF CAUTION REGARDING SUPPLEMENTING WITH TAURINE. "AT THE BRAIN BIO CENTER, WE HAVE FOUND THAT TAURINE SUPPLEMENTS CAN CAUSE ACID STOMACH AND ULCERS IN HIGH-HISTAMINE PATIENTS AT DOSES OF 1000 MG PER DAY. IF THIS OCCURS, WE STOP OR CUT BACK THE DOSE OF TAURINE. THIS PROBLEM CAN PROBABLY BE AVOIDED BY TAKING TAURINE WITH FOOD, MILK OR MILK OF MAGNESIA. TAURINE SHOULD NEVER BE TAKEN WITH ASPIRIN."

(1)

In view of the wonderful work being done by Dr. Pfeiffer and Braverman at the Princeton Brain Bio Center we openly urge them to add the following additional parameter to the diagnostic evaluations of their patient population: Chart the numbers and surfaces of amalgam fillings to determine the correlation between body fluid amino acid levels, symptomatology and amalgam dental fillings.
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ABSTRACTS/REVIEWS


REVIEW: In an earlier study (Ehmann et al., 1986) the concentration of 16 trace elements was determined in brain specimens from individuals who had died of Alzheimer's disease (AD) and compared to brain samples from age matched neurologically normal control patients. This earlier study was investigating the hypothesis that trace-element toxicity had a possible etiological role in Alzheimer's disease because of previous studies implicating Aluminum as a possible neurotoxic factor. The 1986 study of bulk AD brain samples reported imbalances for eight elements: Bromine (Br), Chlorine (Cl), Cesium (Cs), Mercury (Hg), Nitrogen (N), Sodium (Na), Phosphorus (P) and Rubidium (Rb). The most persistent differences observed in AD samples compared to controls was the elevation of Br and Hg and the depletion of Rb. [Bio-Probe Note: In a recent study Tayarani et al. (1987) demonstrated "a substantial decrease in 86Rb uptake by brain microvessels in the presence of high concentrations of mercury compounds. The percent of uptake inhibition was almost identical irrespective of the form of mercury used. However,
Thimerosal was found to be less potent than either mercuric chloride or methylmercury chloride." Tayarani et al. The effect of mercurials on amino acid transport and rubidium uptake by isolated rat brain microvessels. NeuroToxicology 8(4):543-552, Winter 1987]

In this study, samples were taken from the hippocampus, amygdala and nucleus basalis of Meynert (nbM). The hippocampus and amygdala are involved in memory function of the brain, and the nbM is the primary cholinergic projection to the cerebral cortex. Previous research has demonstrated that all three regions in AD brains have a severe loss of neurons and the presence of neurofibrillary tangles, with the hippocampus showing the most severe and consistent pathological alterations of any brain region.

Results of the present study found significant increased levels of Hg in AD nbM and to a lesser degree in AD amygdala. In the discussion of their findings Thompson and his colleagues state "The AD-control difference for Hg in the nbM is, to our knowledge, the largest trace-element imbalance observed to date in the AD brain. The source of Hg in brain is not known although environmental pollution and dental amalgams have been widely suggested. Why Hg is elevated so unevenly among different AD brain regions is another interesting question. The diversity of the regions in which we find the highest Hg elevations would tend to argue against a single route of entry, such as the olfactory route." The authors go on to conclude that the persistent imbalances or trends observed for the monovalent cations of Na, K, Rb and Cs may indicate an electrolyte imbalance in the AD brain.

BIO-PROBE COMMENT: We consider the trace-element work being done at the University of Kentucky, Lexington to be in the forefront of etiological investigative studies of major neurological diseases. In addition to the work being done on AD there is also work in progress to determine the significance of Hg in the cerebral spinal fluid (CSF) and brain of Amyotrophic Lateral Sclerosis (ALS) victims. (ALS is commonly referred to as Lou Gehrig’s Disease.) What we consider so noteworthy about the Thompson et al. study is the mention of dental amalgam as a possible source of the mercury being found in the brains of AD victims. The work of Schiele, Friberg, Nylander and Eggleston establishing positive correlations between numbers and surfaces of amalgam fillings and brain mercury levels; Alhrot-Westerlund’s findings of high mercury levels in the CSF of Multiple Sclerosis (MS) patients; and the work of Vimy and Lorscheider demonstrating intra-oral mercury levels with its application to the four compartment distribution model, must be considered in future investigative studies of major neurological diseases.

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ABSTRACT: Pattern reversal, brain stem auditory and somatosensory evoked potentials (PREPs, BAEPs, SEPs) have been recorded on 13 patients occupationally exposed to inorganic lead compounds, in 9 patients occupationally or accidentally exposed to inorganic mercury compounds and in 26 chronic alcoholics. The results were compared to those of a normal control group. Peripheral conduction velocities were decreased in lead exposed workers and in alcoholics, but not modified in the mercury exposed patients. In the three exposed groups, an amplitude increase (PREPs and upper limb SEP cortical components), more important in the mercury group, an increase of central conduction time in the case of lower limb stimulation, could be interpreted as early signs of nervous cortical impairment.

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ABSTRACT: A variety of heavy metals are recognized as environmental pollutants, and although a significant body of literature exists on the acute toxicity of these metals in various tissues, little is known
about the effects of metals such as mercury on host defense. Therefore, the effect of mercuric chloride (HgCl₂) on human polymorphonuclear leukocytes (PMN) function in vitro was evaluated. Direct effects on PMN were evaluated and results indicated concentrations of HgCl₂ less than or equal to 10(-6) to 10(-7) M did not induce significant lactate dehydrogenase release or produce any ultrastructural alterations in the PMN respectively. However, in evaluating the effects of HgCl₂ on human PMN functions involved in host defense, the researchers found that HgCl₂ consistently suppressed human PMN adherence, polarization, chemotaxis, and erythrophagocytosis at concentrations between 10(-6) and 10(-17) M. Because of the established role of oxygen metabolites in host defense, the effects of HgCl₂ on chemiluminescence and H₂O₂ production were evaluated next. These studies demonstrated that low concentrations of HgCl₂ (i.e., 10(-9)-10(-15) M) significantly enhanced chemiluminescence, as well as stimulated H₂O₂ production by the PMN. The authors concluded that their studies clearly demonstrated the ability of extremely low levels of HgCl₂ not only to suppress various PMN functions involved in host defense but also to stimulate oxygen metabolism. In vivo, these HgCl₂ effects would not only compromise host defense but also promote tissue injury via the local production of oxygen metabolites.

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ABSTRACT: We investigated the effects of mercuric chloride on phagocytic capacity, formation of toxic oxygen species and release of lysosomal enzymes of human polymorphonuclear leukocytes (PMNL). Our results show that HgCl₂ may alter these microbicidal function of human PMNL without remarkable damage of cell viability. The phagocytic capacity was markedly depressed in a concentration-dependent manner. The formation of toxic oxygen species was also diminished by mercuric chloride when induced by phagocytosis. It was furthermore reduced when the PMNL were activated without phagocytosis by binding of IgG to Fc-receptors or by binding of phorbol myristate acetate to the membrane. In contrast, the release of lysosomal enzyme lysozyme was enhanced in the presence of mercuric chloride, but not the release of beta-glucoronidase. These effects may lead to impaired defense against infections and possible to inflammatory reactions in adjacent tissues induced by released lysosomal enzymes.

BIO-PROBE COMMENT: Two more studies demonstrating the potentially devastating effects on the immune defense systems that can be caused by exposure to mercury. Hopefully, the chapter on the predicted intake of mercury vapor from dental amalgams included in the February 1988 edition of "The Biological Monitoring of Toxic Metals" published by Plenum Press will begin to alert the medical profession to the necessity of considering mercury toxicity as a possible etiological factor when working with immunoincompetent patients.

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ABSTRACT: There are situations in which the exposure to more than one agent results in an enhanced risk for the exposed organism, that is in which the observed effect exceeds the effect expected from the addition of the individual effects. Our knowledge of such hazards is rather limited, in particular for those agents that occur in the environment of man. When early mouse embryos in vitro were exposed to ionizing radiation and mercuric chloride, the observed risk was higher than expected from the individual effects. This increase in risk was due to an interaction between mechanisms induced by ionizing radiation and mercury. To gain some more insight into the mode of interaction, the time requirements of mercury exposure were studied. The amount of interaction did not depend on mercury exposure before or during irradiation. However, to achieve an enhanced risk, exposure had to start as soon as possible after irradiation and had to last as long as possible. This time dependence suggests that if inhibition of DNA-repair is
involved in the mechanism of interaction at all, then there must be an additional late process that is also impaired by mercury.

BIO-PROBE COMMENT: Amalgam bearers are continuously exposed to additional mercury vapor from their dental fillings that is being absorbed and distributed throughout the body. Therefore, the immediate question that arises is the applicability of the findings of Müller and Streffer to the other routine radiation exposures (medical and dental) to which amalgam bearers are subjected? Are individuals with ten or more amalgam fillings at greater risk?

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ABSTRACT: This study assesses the early cavomarginal breakdown of the newer posterior composite resin restorations compared with that of amalgam restorations. A total of 432 posterior composite restorations and 73 amalgam restorations were examined: 121 composite restorations (28%) and 44 amalgam restorations (60%) clinically showed a marginal crevice at some point on the cavosurface margin of the restoration at 6-month, 1-year, and 2-year recalls. The largest single reason for poor marginal adaptation was marginal fracture. Up to 2 years, the marginal integrity of the studied posterior composites was superior to that of an amalgam alloy. It was determined that smaller cavities, greater bulk of resin at the margin (especially at functional cusp areas), and well-finished margins without overfilling seem to reduce the occurrence of marginal fracture on composite resin restorations.

BIO-PROBE COMMENT: NO COMMENT!

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FORUM

The following was sent to us by Tom Valentine in Iowa City, Iowa. Tom is an excellent reporter who writes for many publications and also does a program on Radio Free America. He has done an outstanding job in bringing the mercury issue to the attention of all his readers and listeners.

Machine Design, August 25, 1988, page 274:

"From Europe comes reports of doubts about the safety of amalgam dental fillings. Swedish, Austrian, and German doctors have raised questions about the danger posed by deterioration of the fillings, made of a mercury and silver alloy. The continuing decay process may release tiny particles of mercury. The Ministry of Health in Bonn has warned expectant mothers against use of the fillings."

Bio-Probe called West Germany and talked to personnel in Bonn and in Berlin who confirmed the information stating "It wasn’t a warning. Just discussion, and that the press release said that discussions were going on related to the safety of amalgam and that pregnant women should delay having any amalgam work done until after their pregnancy."

From the rumor department, still unconfirmed, we understand that also within West Germany it is considered "maltreatment" for a dentist to place gold in direct connection with amalgam. It appears that at least someone in the German dental establishment hierarchy is paying attention to what the dental materials text books have been saying for more than 30 years. That is more than can be said for the NIDR and ADA in this country.

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NATIONAL ENVIRONMENTAL CONFERENCE, GREENSBORO, NORTH CAROLINA, OCTOBER 8, 1988.

Elizabeth Ridenour of Greensboro, NC, was the person responsible for setting up the conference and bringing everybody together. Elizabeth is an ex-amalgam victim who is still trying to fully recover from
the devastating effect mercury had on her. It was an excellent program with presentations being made by Dr. John C. Munday Jr., Christian Broadcasting Network University; Paul Petersen of Hollywood (Donna Reed Show for 8 years); Lola Falana who is recovering from diagnosed Multiple Sclerosis which Miss Falana now believes may have been mercury poisoning from her amalgam fillings; Douglas N. Rader, Senior Scientist with the N.C. EDF; Steve Young, Greenpeace, Boston, MA; Allen Spalt, N.C. Agricultural Resources Center; Jim Pierce, Staff Scientist with the Environmental Action Foundation in Washington; Clinton Miller, Legislative Advocate for the National Health Federation and last but not least, Sam Ziff made a presentation on the scientific facts about the mercury/amalgam issue and Louise Herbeck DAMS told her first person story about her recovery from MS by amalgam replacement. There was excellent media coverage by two TV stations and the Greensboro newspapers. Associated Press had Lola Falana’s story on the wires for national coverage. As a direct result of this meeting and Elizabeth Ridenour’s efforts, there is a strong commitment from a N.C. Senator to introduce informed consent and freedom of choice legislation at the next session of the North Carolina legislature. Further activity took place in the week following the conference, Pat Preyer a prior MS patient who had recovered after amalgam replacement, contacted Elizabeth Ridenour and an excellent TV interview was filmed and shown on Channel 12 in Greensboro.

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Dr. Joyal Taylor, author of The Complete Guide to Mercury Poisoning From Dental Fillings just returned from a successful media tour in Anchorage Alaska that was put together by Bob Stephenson, a wildlife biologist for the Alaska Department of Fish and Game and Burton Miller, D.D.S. of Anchorage.[All of us in the anti-amalgam movement, owe a debt of gratitude to Bob Stephenson, an ex-amalgam victim himself, for his unselfish efforts in making people in Alaska aware of the issue.] Several senators and legislators attended a two hour public meeting on October 15, which featured a slide presentation by Dr. Taylor and a 20 minute video interview with Dr. David Eggleston.

In a television interview following the meeting, senator Patrick Rodey (D) of Anchorage said, "The information that was presented would cause any reasonable person to conclude that there is a difficulty here, that there is a problem worthy of study, and that extreme caution should be used with mercury filings...for those people who have difficulty, removal is perhaps the only alternative." Senator Rodey also stated that we would work toward legislation for informed consent laws and that he is having his amalgams removed. Representative Walt Furnace (R) a member of the house of representatives said he would also give support toward informed consent legislation. Representative Furnace had his amalgams removed last year. Doctors who are interested in sponsoring further tours for their area contact Dave Collins at 619-586-7623. For further information on the Alaskan reaction contact Dr. Burton Miller in Anchorage at 907-258-1390.

One important event that occurred subsequent, was an invitation for Dr. Miller to make a presentation of the scientific literature to members of the Anchorage Dental Society on October 27, 1988, which Dr. Miller states was well received.

BIO-PROBE COMMENT: WE THINK THE POTENTIAL STATE LEGISLATIVE ACTION THAT MIGHT ENSUE FROM THESE EFFORTS IS REAL PROGRESS IN OUR FIGHT FOR INFORMED CONSENT AND FREEDOM OF CHOICE! THERE IS CERTAINLY NO QUESTION THAT A MUCH LARGER SEGMENT OF THE U.S. POPULATION IS AWARE OF THE ISSUE BECAUSE OF THE TV AND MEDIA COVERAGE GIVEN TO LOLA FALANA WHO IS MOTIVATED TO HELP GET THE WORD ABOUT MERCURY/AMALGAM TOXICITY TO EVERY PERSON IN THE U.S. SUFFERING FROM MULTIPLE SCLEROSIS.

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