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DETOXIFICATION
Part II
Sam Ziff

Part I in Bio-Probe Newsletter vol 3, issue 1, Feb 1985 covered Cysteine, Glutathione and Vitamin E. In Part II we will address the nutrients Selenium and Zinc.

SELENIUM

In the late 1960s a pure selenium deficiency was produced in laboratory animals although the first salutary effect of selenium in the diet was reported in 1957. The 1957 data was from Schwartz and Poltz who demonstrated that selenium would prevent dietary liver necrosis in the rat.

Selenium closely resembles sulfur in its physical and chemical properties and the selenium concentration in the blood is 19-25 mcg/100 ml (U.S. population studies). It is found in highest concentrations in the kidney, heart, spleen and liver and to some degree in all other tissues except fat. Average dietary intakes in the U.S. are approximately 60-216 mcg/day and a provisional Recommended Dietary Allowance of 50 to 200 mcg/day for adults has been given.

It is paradoxical that although selenium can be toxic by itself it also prevents the toxicity of several other metals such as Ag⁺, Hg²⁺, Cd²⁺ and Pb²⁺ (Frost 1972). Toxicity of selenium may be caused by interference with sulfur metabolism which in turn would inhibit several enzymes (including succinic dehydrogenase, choline oxidase and proline oxidase). In excess it can cause abnormal development of bone and cartilage and also impair embryonic development.

Conversely, low-selenium intakes have been related epidemiologically to a higher incident of death from cancer of the digestive organs, lung, breast and lymph when compared to populations living in areas having a high-selenium content in the soil and forage crops. In one study comparing the incidence of cancer in 27 different countries the number of individuals getting cancer was significantly lower in those countries/populations with a high dietary intake of selenium from foods rich in selenium. Significantly lower levels of selenium have also been seen in patients with various types of cancer such as lymphocytic leukemia, breast, pulmonary carcinoma, gastrointestinal, colon, genitourinary carcinoma, skin cancer and Hodgkin's disease.

There is also epidemiological evidence suggesting that a lower cardiovascular mortality exists in high selenium areas compared with low selenium areas. Finland has done extensive population studies relating serum selenium levels to the incidence of myocardial infarction (MI). One study involving 8113 men and women indicated that a serum level of less than 35 mcg/l was associated with a six-to seven-fold increase in risk of death from heart disease and a two-fold increase in risk of MI. However, there are other studies presenting conflicting results which indicates that the association between serum selenium concentrations and the risk of cardiovascular disease remains uncertain.

Unfortunately, none of these studies looked for mercury as a possible confounding factor in the different results obtained. For example, it is well established by these studies that the cardio-
vascular mortality is higher in eastern Finland than in southwestern Finland. It was perplexing to the researchers though that the serum selenium levels were higher in the east than in the west. Dietary evaluations showed that the men in the east ingested more fish than the men in the west, 89 g/day vs. 24 g/day. Although fish has a fairly high selenium content, the mercury content is higher. An evaluation of serum mercury levels as well as separating the cohort into those with amalgams and those without might have provided some insights into some of the variances observed.

A significant factor in the utilization of selenium in any detox protocol relates to the concurrent use of ascorbic acid in the protocol. A great deal of confusion exists regarding the inhibition of selenium absorption by ascorbic acid. To start with I think it would be appropriate to outline the various forms of selenium: elemental selenium, natural dietary selenium, sodium selenite, sodium selenate, selenocystine, and selenomethionine. The inorganic forms are sodium selenite or sodium selenate and the organically-bound forms are selenocystine and selenomethionine. The organically-bound forms of Se are normally derived from wheat or yeast.

Recent studies have shown that ascorbic acid can act as a reducing agent, reducing inorganic selenite to elemental selenium, which is insoluble and biologically unavailable to the body (Ganter, 1979; Vokal-Borek, 1979; Shils and Levander, 1982). Such does not appear to be the case when the selenate form is used. A recent study by Mutanen and Mykkanen (1985) demonstrated that ascorbic acid did not have the same interaction with sodium selenate. Their study also showed that ascorbic acid supplementation increased the availability of natural selenium in the diets of subjects with low selenium intakes. Now that presents a problem because selenium in natural foods consists of a wide variety of inorganic and organic compounds which may react differently with the supplemental ascorbic acid. Mutanen and Mykkanen conclude their article: "Additional information is needed regarding different Se compounds in foods and the possible effects of ascorbic acid on these." This fact notwithstanding, their study confirmed earlier studies indicating that supplementation with sodium selenate in humans could alter the selenium status of those individuals with low dietary intakes of selenium.

Based on available data it would appear that sodium selenite should not be used in any protocol also utilizing vitamin C without stipulating that the selenium supplement be taken at least three hours away from supplemental vitamin C. Although at this point the same prohibition does not appear to be supported for the other forms of selenium, unless absolutely necessary, the prudent course might well be to insure selenium bio-availability by simply requiring a suitable time-separation between any selenium and vitamin C supplementation.

What is the relationship between selenium and mercury and does selenium reduce or change the toxicity of mercury? A tremendous amount of research on selenium has been done in the past few years. However, only one enzymatic function of selenium is presently known. Selenium is an essential constituent of glutathione peroxidase (GSH-Px) and metabolically possesses the ability to destroy hydrogen peroxide and organic hydroperoxides using reducing equivalents from glutathione. If we accept the fact that mercury is capable of generating peroxides as an oxidation by-product, the relationship to
selenium seems clear. Not so simple. The exact role of GSH-Px is certainly not clear at this point. For example, recent discoveries that a non-selenium dependent GSH-Px exists, that a selenium-dependent factor that prevents lipid peroxidation and is not GSH-Px also exists, and that catalase can also remove hydrogen peroxide all complicate the picture. There was even some confusion regarding applicability of animal studies to humans. The relationship in humans though was recently confirmed by findings of Anneren et al. (1985) that a significant positive correlation between GSH-Px activity and selenium level, both in plasma and in erythrocytes, did exist thus confirming the relationship between the selenium status and GSH-Px activity in man.

Autopsy studies done by Kosta, Byrne and Zelenko in 1975 revealed that contrary to accepted belief that the kidney was the prime accumulator of inorganic mercury the thyroid and pituitary had retained and accumulated more inorganic mercury than the kidney. These same authors went on to see if there was a correlation between mercury and selenium. Using other post-mortem samples of exposed humans and analyzing both elements they found an approximate 1:1 molar ratio for those organs which accumulate and retain mercury strongly i.e. thyroid, pituitary and kidney. In brain samples the same 1:1 molar ratio was observed in various sections of the brain. The 1:1 molar ratio suggested a direct Hg-Se linkage. The authors also observed that the coaccumulation of selenium was not the result of comparable exposures to both elements. Concluding that the effect can occur where mercury is present as the methyl (tuna) or the inorganic form (marine mammals and man), and whether selenium is relatively abundant in the diet or not and that the coaccumulation of selenium may be a natural or autoproductive effect.

Two recent papers Friberg et al 1986 and Ansari et al. 1985 bring additional light to the complex problem of mercury in the brain. Ansari and his colleagues found that "Inorganic peroxides, which are formed in the brain, can be reduced by GSH-Px, other peroxidase and catalase. Organic hydroperoxides can be reduced only by GSH-Px. These peroxides are an oxidation product of polyunsaturated fatty acids, lipids which are located primarily in the brain. Therefore, the brain may be more susceptible to peroxidative damage." The authors concluded that their work with fresh and autopsied human brain samples confirmed that the tissue contained significant quantities of GSH-Px activity and speculated that alterations in GSH-Px activity and tissues damage because of peroxide accumulation might be involved in the pathogenesis of senescence and some degenerative neurologic diseases. The study by Friberg and his associates revealed a direct correlation between the amount of inorganic mercury in the brain and the number and surfaces of amalgam fillings. The autopsy specimens were from accidental death victims.

I have written Dr. Friberg to see if would be possible to determine the brain selenium status of these same victims. Such additional research would seem to be appropriate when viewed in context with the work of Kumei and Sato (1981) who demonstrated that metallic corrosion products from dental amalgam caused lysis of the L cells utilized in the experiment. Simultaneous administration of sodium selenite counteracted the cytotoxicity of amalgam. The authors suggested that a mutual detoxication is possible between amalgam and sodium selenite.

Kling and Soares, 1978 postulated that since mercury tends to combine with selenium, part of the damage caused by mercury when
administered alone might be associated with decreasing the bioavailability of selenium. This hypothesis was in line with results obtained by Wada et al. in 1976 who found that mercuric chloride inhibited GSH-Px in mice and that simultaneous administration of the same molar dose of sodium selenite eliminated any reduction in GSH-Px activity.

The exact cause of mercury toxicity is as complex and confusing as everything else associated with attempting to correlate selenium and mercury physiological effects. It had been assumed that cellular damage caused by mercury was directly related to the Hg generation of lipid peroxidation. However, Stacey and Kappus (1982) presented results which indicated that the Hg\textsuperscript{2+}-induced loss of cellular viability was not due to the associated lipid peroxidation. Their data (derived by dissociating lipid peroxidation and cellular toxicity) indicated that Hg\textsuperscript{2+} cytotoxicity was not mediated by the concurrently observed lipid peroxidation. These results were confirmed in experiments done by Heisinger and Scott 1985 who determined that Se GSH-Px activity, using H\textsubscript{2}O\textsubscript{2} as a substrate, was not inhibited in either the liver or kidney.

There are many studies showing that selenium has been able to reduce the toxicity of mercury. However the exact means by which this is accomplished remained unclear. Yamamoto in a 1985 study attempted to clarify this mechanism. Previous studies by Cherian and Goyer in 1978 had indicated the presence of metallothionein might be related to detoxification of heavy metals as well as their transport in the body. In his experiments with mice, Yamamoto thought that metallothionein could prevent toxic effects of mercury through synthesis of mercurithionein and went on to conclude: "The present study confirmed that the simultaneous administration of mercury and selenium reduced the percentage of mercury in metallothionein fraction by gel filtration of supernatant. Furthermore, increasing doses of selenium resulted in lower percentages of mercury. Therefore, it appears that synthesis of mercurithionein is suppressed by a decrease in the concentration of Hg\textsuperscript{2+} resulting from binding to selenium."

After inhalation mercury vapor (Hg\textsuperscript{0}) is distributed in the body and taken up by different organs. This initial distribution pattern is much different than that seen after injection of Hg\textsuperscript{2+}. Not only is there a high lung concentration observed, but there is a high uptake in the respiratory epithelium, kidney, myocardium, adrenal cortex and thyroid. The perivascular hepatocytes of the liver also show characteristic accumulations. Significantly, it appears that uptake in these organs takes place prior to oxidation to Hg\textsuperscript{2+}.

Khayat and Dencker (1983) in experiments with mice provided data on what effect pretreatment with selenium selenite had on the distribution and retention of inhaled mercury vapor. Some of their significant conclusions or findings: "selenium and mercury, after having entered cells independently (in particular in the lung after inhalation of Hg\textsuperscript{0}) can interact in such a way that they prolong each other's retention time in the cells."; "Hg\textsuperscript{0} inhalation results in an intracellular production of Hg\textsuperscript{2+} (by oxidation), where it may interact with the selenium available."; "This study shows that mercury is normally excreted more rapidly after injection of Hg\textsuperscript{2+} than after inhalation of Hg\textsuperscript{0}. Although we have no data on this, it may be speculated that a firmer intracellular binding of the mercury occurs.
after oxidation of Hg\(^0\) in the cells." Their data also indicated that Se/Hg\(^2+\) complexes formed in the serum had much greater retention than those presumed complexes formed intracellularly after oxidation of Hg\(^0\) to Hg\(^2+\).

In experiments with mice exposed to subtoxic doses of mercury vapor it was shown that excess selenium increased the mercury half-time and that selenium influenced the organ distribution of mercury without any noticeable effect on the elimination of mercury from the whole body. The researchers in these experiments postulated that in mercury vapor exposed mice there were at least two types of interactions between mercury and selenium. The first may influence the oxidation of mercury increasing only the lung and kidney accumulations without affecting whole body clearance. The second affects the clearance of mercury possibly through the formation of colloidal mercury selenide. (Hansen, Kristensen, Westergaard 1981).

Very few studies have been done on the correlation of Se/Hg in humans exposed to mercury vapor. In a 1983 paper Alexander, Thomassen and Aaseth describe the results of their study in which the content of selenium and mercury in the urine was measured in 28 mercury vapor exposed male workers from a chloralkali plant and compared to 21 unexposed male controls. The mean value of exposure for an 8 hour shift per year was 0.038 (range: 0.010-0.050) mg Hg m\(^{-3}\). The authors felt that the mean of 0.038 Hg mg m\(^{-3}\) was stoichiometrically comparable to the daily dietary intake of selenium in Norway which is approximately 0.4 umol per day. Although there was no significant correlation between mercury and selenium excretion, the mercury vapor exposed group excreted significantly more selenium in their urine than did the controls. If as reported, there is coaccumulation of selenium and mercury you would expect greater retention of both elements and less excretion of selenium. As their results differed from this rationale the authors hypothesized that: "Selenoprotein with complexed mercury in the kidney may be catabolized more rapidly than native proteins, leading to increased urinary excretion of selenium. Increased urinary selenium excretion might possibly reflect increased levels of selenium in the kidneys of mercury-exposed subjects, because the clearance of selenium from the kidney is not inhibited by mercury."

It is apparent that I could devote this whole issue to selenium and only scratch the surface regarding the complexities and interrelationships of this element. It is obvious from the data presented that selenium must be included in any detoxification protocol. A hidden or unplanned benefit would be selenium's ability to influence the absorption/excretion of other heavy metals other than mercury. One obvious benefit is the synergistic effect with other antioxidants such as vitamin E and C. There is also evidence that selenium is involved in the synthesis of Coenzyme Q which research is demonstrating to be significantly involved in periodontal disease, cardiovascular health, the immune system, etc etc.

There seem little doubt that selenium is an essential nutrient for man and that deficiencies or low dietary intakes have a bearing on mortality associated with several major disease states. Mercury's ability to complex with selenium, increase it's excretion and reduce its bioavailability for primary metabolic functions must have a deleterious impact on homeostasis. It remains to be seen what contribution the mercury from dental amalgams makes to selenium bioavailability.
It was also interesting to note that selenium is excreted in sweat and that excretion via this route may be high. This same route of excretion for mercury is also receiving more attention.


**ZINC**

It is apparent that our knowledge of minerals lags far behind that of vitamins. In the case of zinc this is especially true. It was in 1934 that Todd determined that zinc was essential in the diets of rats. However, although it was assumed to also be essential to humans this wasn't confirmed until 1963 when Prasad et al published the results of their 1961 work demonstrating a human deficiency of zinc. It took until 1974 before zinc was included in the RDA's. Since that time extensive research has associated zinc deficiencies in man with retarded growth, anorexia, hypogonadism, hypogeusia (diminished sense of taste), hyposomia (inadequate bodily development), dermatitis, koilonychia (dystrophy of the fingernails) and impaired wound healing. Zinc\(^{2+}\) is an essential component of approximately 100 different enzymes. These include alcohol dehydrogenase, carbonic anhydrase, DNA and RNA polymerases, and carboxypeptidase. The hormone insulin is stored as a zinc complex. It is also involved in metallothionein which is also involved in the storage or detoxification of Cd, Hg, and Cu. Moreover, Pearson 1968, and Williams 1976 have shown that zinc resembles cadmium and mercury in its ability to form complexes with thiols.

Zinc works synergistically with vitamin B6 and vitamin E. B6 increases zinc absorption significantly.

Experiments with rats by Day, Funk and Brady in 1984 provide data on the displacement of zinc from metallothionein by cadmium and mercury. They found that mercury could displace zinc in vivo and ex vivo consistent with the demonstrated in vivo affinities of metallothionein for divalent metals: Hg > Cu > Cd > Zn. Their study as well as many other studies consider zinc induced synthesis of metallothionein as perhaps the primary factor in reducing the toxicity of many heavy metals. During the stress of exposure to exogenous toxic metals, zinc is mobilized and its uptake increased in the liver which then results in the synthesis of metallothionein which continues until most of the exogenous metal has been bound. High hepatic zinc thionein levels did not alter tissue distribution of Hg(II) because exogenous mercury was distributed predominantly into metallothionein. Where hepatic zinc levels were low the exogenous mercury in the cytoplasm saturated the existing metallothionein while the remainder was bound to higher molecular weight proteins in the cytoplasm. Incorporation of mercury occurs by the displacement zinc.

In the rat, bile is considered to be an important excretory route for zinc. In a 1981 paper, Alexander, Aaseth and Refsvik, concluded that their results "strongly indicate that the biliary zinc excretion
is a glutathione-dependent process and probably makes use of GSH as a carrier molecule when excreted into bile. This might involve competition with the biliary excretion of other heavy metals (e.g. copper, cadmium, methyl mercury). Other substances using GSH in their metabolism might interfere with zinc homeostasis."

As you are well aware, the exact actions by which mercury exerts its toxic effect or causes pathological damage is not well defined. One action actively considered is the ability of mercury to stimulate lipid peroxidation. Yonaha et al. (1982) observed that lipid peroxidation occurred in the kidney of rats given mercuric chloride and that the urinary excretion of enzymes was increased. This was subsequently confirmed by Fukino et al. (1984). However, Fukino and his associates were able to reduce mercury induced lipid peroxidation and renal damage by zinc pretreatment. They offered two possible mechanisms by which zinc suppresses mercury toxicity. 1) zinc induces the synthesis of metallothionein which mercury then binds to and 2) that zinc pretreatment increases intracellular glutathione which in turn increases GSH-Px and G-6-PD activities in the rat kidney thus reducing mercury induced lipid peroxidation. They also hypothesized that because the zinc pretreatment caused a significant rise of GSH in the kidney and serum this might indicate that GSH synthesized in the liver could be mobilized to protect the kidney against the toxic effects of mercury.

In 1984, Gale conducted a study utilizing pregnant hamsters, designed to determine if zinc could modify the embryolethelal effects of inorganic mercury (mercuric acetate). The results of this experiment by Gale were consistent with other experiments that had demonstrated the ability of zinc to protect vertebrate embryo from the harmful effects produced by several different teratogenic agents. Unfortunately, exactly how mercury causes the great number of embryo abnormalities and how zinc affords protection against this damage remains speculative.

Bjorksten et al. (1980) found that the serum level of zinc in patients with Down's Syndrome was markedly reduced. Annneren et al. (1985) found the concentration of selenium in the erythrocytes of Down's Syndrome patients was higher than in controls. They also found a sex difference (higher values in females) both in GSH-Px activity and in plasma and erythrocyte selenium levels. Other research has shown sex differences for Hg status. Does mercury play some part in metabolic differences discerned in Down's Syndrome children?

There is evidence indicating that in some inflammatory diseases there are alterations in the concentration of essential and nonessential elements in various tissues and body fluids. For example, Aaseth et al. in 1981 observed increased concentrations of copper and decreased concentrations of zinc and selenium in the serum of patients with rheumatoid arthritis. A recent study by Alroth-Westerlund (1985) attempted to determine if there could be clinical significance in interpreting macro and trace element variations under various health conditions. Erythrocytes and granulocytes (venous blood from the patient group) had increased concentrations of mercury and strontium which was not present in the control group. The patient group also displayed a decreased zinc concentration in erythrocytes and neutrophil granulocytes. There seemed to be a general permeability disturbance in blood cells resulting in elevated calcium and strontium and lowered zinc concentrations.
Carmignani and Boscolo (1984) in a study designed to determine cardiovascular homeostasis in rats chronically exposed to mercuric chloride found that Hg exposure induced baroreflex hyposensitivity and produced a drastic alteration of the levels of copper and zinc in the brain and kidney. "The Hg-induced altered concentrations of copper and zinc may be related to the observed cardiovascular changes. This increase in essential metals may be explained by an augmented synthesis of metallothionein, a protein which may protect against the toxic effects of Hg (Cherian and Goyer 1978; Tandon et al. 1980). Increased brain copper following Hg exposure has not been previously reported. It remains to be established if the cardiovascular effects observed in the chronically exposed rats can be extrapolated to humans."

Perhaps the most important aspect of mercury's effect on zinc will be its inhibitory effect on zinc responsive enzymes and coenzymes. Hopefully future research will expand greatly on some of the preliminary work in this area. At present, we do know that mercury will inhibit the following zinc involved enzymes or coenzymes: alcohol dehydrogenase; delta-aminolevulinic acid dehydratase; carbonic anhydrase; alkaline phosphatase and aldolase.

One last thing before leaving zinc. The 1985 Nutrition Desk Reference has some interesting data on zinc: Serum zinc concentrations were found to have an inverse relationship to blood pressure, i.e. low serum zinc, high blood pressure; "58 patients with confirmed secondary immunodeficiency syndrome were tested for plasma copper and zinc levels. These patients had depressed cell-mediated immunity and were found to have a low serum zinc and elevated serum copper level.---Cellular immunity is known to be impaired by zinc deficiency."

All of this data presents a rather serious question, i.e. does the release of mercury vapor/ions from dental amalgams increase the overall body burden of mercury enough to represent a significant metabolic factor in the development of some of the imbalances outlined for selenium and zinc?

REFERENCES

SELENIUM

ZINC

ABSTRACT:
Experiments were performed to determine the effects of dietary selenium and/or vitamin E deficiency on cell-mediated cytotoxicity in the mouse. Natural killer cell-mediated cytotoxicity (NKCC) was depressed in 8 weeks on diets deficient in selenium and/or vitamin E. In contrast, antibody-dependent cell-mediated cytotoxicity (ADCC) was not affected by 8 week dietary deficiency of selenium and/or vitamin E. T-lymphocyte-mediated cytotoxicity (TCMC) was found to be depressed by combined selenium-vitamin E deficiency after 7 weeks on diets.


ABSTRACT:
This study examined the validity of evoked somatosensory potentials as a measure of subclinical neurological damage to workers chronically exposed to nonorganic mercury. In this study potentials were recorded along the somatosensory pathway from the periphery to the primary cortex in response to electrical and mechanical stimuli. The findings of this study indicate that such workers exhibit subclinical damage which manifested in a delay in nerve ending conduction times at the periphery, and an acceleration of the conduction from brain stem to cortex. These findings support the suggestion that evoked potentials may be a sensitive and reliable measure in the detection of subclinical neuropathic phenomena. They may consequently be utilized as an efficient early warning system in the prevention of clinical symptoms.

One other result, considered to be of great significance but not contained in the abstract is: "Clear, negative and significant correlation was found between time on the job and the level of mercury in the urine; R = -0.83, p < 0.005. In other words, the longer a worker is on the job, the less mercury is excreted into his urine, as measured during the year prior to the neurological examination in our laboratory."


ABSTRACT:
A consistent diurnal variation of urinary mercury concentration (expressed as nmol of mercury/mmol of creatinine) has been demonstrated in 36 occupationally exposed workers, the concentration being highest in the morning and lowest in the late evening. This variation is partly intrinsic and partly an artifact because creatinine excretion also varies diurnally, but in the opposite direction. The implications of these findings in relation to the biological monitoring of mercury workers is discussed.
EDITORIAL

ISN'T MERCURY A POISON?

The ADA developed and published in the January 2, 1984 issue of the ADA News, a fact sheet on the safety of dental amalgam for patients that was designed to quiet the growing public and media concern about the potential toxicity of mercury amalgam dental fillings. The fact sheet, in question and answer form, was designed to be cut out and duplicated and subsequently handed to any patient who voiced any fears about the safety of mercury amalgam fillings.

One of the major questions contained in the handout was: Isn't mercury a poison? The answer to that question contains perhaps the most irresponsible statement ever made concerning the amalgam controversy i.e. "When mercury is combined with the metals used in dental amalgam, its toxic properties are made harmless." Starting with Dr. Stock in 1926, the release of mercury vapor from mercury amalgam fillings has been measured and documented. Confirmation of this fact has recently been documented by researchers at the University of Iowa, University of Calgary, Oral Roberts University, as well as independent studies from Sweden and New Zealand. All of these scientists were measuring mercury vapor, not some non-toxic substance bearing the name of mercury but possessing none of it's toxic properties because it had previously been combined into amalgam. It is certainly true that once this highly poisonous mercury vapor enters the body it may complex with various substances that may alter its inherent toxicity. However, the mercury vapor being released from mercury amalgam dental fillings is the same poisonous mercury vapor that is measured during the invitro release from amalgam or in the atmosphere of the dental operatories of those dentists placing amalgam. A fact incidentally supported not only by the ADA itself but also by OSHA, and NIOSH. So it would seem logical and reasonable to assume that this particular type of mercury vapor emanating from set amalgam dental fillings has not defied all the laws of physics and chemistry and suddenly become something different.

In the face of overwhelming evidence to the contrary the ADA has modified its position. In their beautiful four color 1985 patient brochure on Dental Amalgam, the ADA put into final form the original patient fact sheet published in the January 1984 ADA News. However, they did modify the question and answer slightly i.e. "Is the mercury component of amalgam also safe?" "Yes. After more than a century of thorough testing, no scientifically reliable study has found the mercury component of amalgam to present a threat to the general health of dental patients. Indeed, for the great majority of patients, the benefits of using amalgam restorations far outweigh any risks. This is because mercury is made virtually harmless when it combines with the other metals used to produce amalgam." It would be extremely beneficial to the world scientific community if the ADA would inform them how they have made poisonous mercury vapor "virtually harmless".

At this particular point in time though I am more concerned with their statement that "for the great majority of patients, the benefits of using amalgam restorations far outweigh any risks". If you think that isn't new heights of "double speak" let me also quote from ADA pamphlet 3-0053 which is titled "You Owe It To Yourself!" and describes the ADA mercury testing service for dentists and their staffs. "The potential symptoms of mercury exposure are scarcely