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PATIENT EXPOSURE TO MERCURY FROM DENTAL AMALGAM FILLINGS
by
Michael F. Ziff & Sam Ziff

INTRODUCTION

For some of your patients, the chronic unstimulated release of mercury vapor from amalgam dental fillings may constitute an unrecognized risk to their health not previously considered.

Existing published scientific studies have concentrated on determining and quantifying the amount of mercury vapor released from amalgam fillings during function or other conditions stimulating the release of mercury vapor. These studies have evaluated the amount of mercury vapor contained in exhaled air under both unstimulated and stimulated conditions, or measured the amount of mercury vapor present intra-cranially under both conditions. Only the "stimulated" values derived from these studies has been extrapolated and compared to an exposure standard. No one has previously quantified the total daily intake and possible contribution to total body burden resulting from the "unstimulated" release of mercury vapor from amalgam fillings 24 hours per day, 365 days per year, during the entire lifetime of such fillings. These unstimulated intake values represent the barest minimum daily intake before any consideration of function is added.

Defenders of dental amalgam fillings have admitted that patients are exposed to mercury from amalgam fillings, but claim that the mercury exposure is too small to cause any harm. It is apparent that this arbitrary and capricious position is based solely on a flawed assessment of existing stimulated release data without any consideration being given to other factors or the daily contribution of chronic unstimulated release of mercury vapor from amalgam fillings. Moreover, these defenders of amalgam fillings are unable to provide scientific documentation to support their position of harmlessness.

Consequently, responsibility to the interest of public health dictates that a risk assessment evaluation be made of the documented minimum mercury exposure from dental amalgam fillings in comparison to established mercury exposure standards. This investigation clearly determines that patient exposure to mercury from dental amalgam fillings constitutes a significant portion of the maximum intake and exposure standards established by the United States Environmental Protection Agency (E.P.A.) for protection of the general public from the effects of inhaled mercury vapor and total daily intake of all forms of mercury.

A number of research investigations published since 1979 have clearly established that mercury vapor is released from
Intact dental amalgam fillings throughout the lifetime of these fillings. (1-6) These studies have confirmed the early results found by Stock in 1926. (7) It has also been confirmed that the release of mercury from amalgam fillings will be greatly increased by various intra-oral stimulation factors, such as chewing (1,2,4,5), toothbrushing (6), and hot fluids (8). Moreover, other investigations have revealed that mercury released from dental amalgam fillings will dissolve in saliva (9-13) and is also found in soft (14) and hard (15-17) tissues surrounding the fillings. Autopsy studies have confirmed that the mercury released from dental amalgam fillings does in fact contribute to the body burden of mercury in patients. (18-19)

No large scale, definitive studies investigating the effects of this mercury released from dental amalgam fillings have ever been published. The significance of the mercury exposure to patients bearing these fillings is, therefore, wholly unknown, purely speculative, and subject to considerable and distinctive personal bias. In view of the thoroughly acknowledged severe toxic nature of certain forms of mercury, callous neglect of the potential effects of this source of mercury on patients could forebode ominous consequences to the dental profession and its practitioners, as well as the recipients of these fillings.

Because of the lack of pathological and epidemiological investigations and the scientific documentation establishing that mercury is released from dental amalgam fillings and does enter the patient's body, the question of effects on the patient revolves around two questions; 1) how much mercury intake is harmless to patients, and 2) how much mercury from dental amalgam fillings is contributed to the body burden of patients?

**HOW MUCH MERCURY INTAKE IS HARMLESS TO PATIENTS?**

This question is thoroughly discussed in a previous issue of the Bio-Probe Newsletter. (20) Reference to that issue, which thoroughly reviewed the scientific literature and documents of organizations establishing exposure and intake standards for mercury in humans, clearly established that mercury (particularly in the vapor form) is so toxic that no toxic threshold can be determined. Even the United States Environmental Protection Agency so states in its document. (21) However, in view of the adamant refusal of amalgam advocates to acknowledge– or apparently even read– the scientific documentation, it would be helpful and revealing to select a reference point for investigating the harmful effects of patient exposure to dental amalgam mercury.

The previously mentioned issue of the Bio-Probe Newsletter set forth clearly and unarguably that scientifically and medico-legally there is only one established mercury intake and/or exposure standard that is applicable to this controversy in the U.S.A., that of the United States Environmental Protection Agency. (21) The E.P.A. has established a MERCURY INTAKE
STANDARD of 30 micrograms per day of mercury for the 70 kilogram adult for all mercury from all sources, and 20 micrograms per day of mercury for the 70 kilogram adult for all mercury from all sources other than food. The E.P.A. has also established a NESHAP (National Emission Standard as a Hazardous Air Pollutant) for mercury of 1.0 microgram of mercury per cubic meter as a MERCURY VAPOR EXPOSURE STANDARD. The Agency stated that this standard "is intended to protect the public health from the effects of inhaled mercury vapor". (21)

Any attempt to dispute the legality or scientific validity of this reference point, rather than the admittedly arbitrary occupational exposure standards, in relation to intra-oral mercury exposures would certainly require valid scientific studies rather than biased personal opinion. Indeed, even the E.P.A. standards clearly must be viewed with a caveat as they were based on studies investigating only the clinically observable signs and symptoms of mercury poisoning; they, therefore, do not guarantee prevention of sub-clinical pathology. The E.P.A. document itself repeatedly states that the amount of mercury vapor that is harmless to humans is unknown.

Nonetheless, a reasonably defensible reference point is necessary for the discussion of potential risk from mercury released from dental amalgam fillings. For this discussion, the following E.P.A. Standards are referenced: (21)

MERCURY VAPOR EXPOSURE = No more than 1.0 microgram of mercury per cubic meter as an average concentration.

MERCURY INTAKE = Not to exceed 20 micrograms of all forms of mercury per day per 70 kilograms (154 pounds) of body weight from all sources other than food.

HOW MUCH MERCURY FROM DENTAL AMALGAM FILLINGS IS CONTRIBUTED TO THE BODY BURDEN OF PATIENTS?

Any discussion attempting to quantitate the amount of mercury contributed by dental amalgam fillings to the body burden of patients must include mention of the various pathways by which this mercury can exit the fillings and enter the subject’s body. There are four potential pathways by which patients may be exposed to mercury from dental amalgam fillings: 1) Ingestion of abraded particles; 2) passage of ionic mercury and/or mercury vapor into oral hard and soft tissues and possibly through nasal mucosa; 3) ingestion of mercury dissolved in saliva or other fluids or mixed with food; and 4) inhalation of released mercury vapor. Quantitative assessment of each of these pathways shall be individually addressed.

1. INGESTION OF ABRADED PARTICLES:
This phenomenon is frequently mentioned in the scientific literature. However, no attempt has been made to determine the amounts of mercury involved, so no data are available. In view of the low gastrointestinal absorption rate of metallic mercury and the relatively low absorption rates of the various inorganic mercury compounds this pathway is not likely to be as significant as the others, although this is purely conjectural. Moreover, the effect of exposure to gastric hydrochloric acid and likelihood of bioconversion to organic mercury cannot be ignored.

II. PASSAGE OF IONIC MERCURY AND/OR MERCURY VAPOR INTO ORAL HARD AND SOFT TISSUES AND POSSIBLY THROUGH NASAL MUCOSA:

A number of studies have demonstrated the presence of mercury from dental amalgam in tooth dentin (16), dental pulp (17), and periodontal tissues (14-15). Quantification of the amount of mercury found in the dentin and pulp tissue has not been accomplished as yet.

Freden and associates, in 1974, biopsied gingival tissues in contact with silver amalgam fillings and control tissues and analysed the tissues with flameless atomic absorption spectrophotometry for mercury content. (14) The tissues contacting amalgams contained 19-380 micrograms/gram of mercury, with a mean of 147 mcg/gm. The control tissues contained 0-10 mcg/gm of mercury, with a mean of 3.0 mcg/gm.

In 1978 Till and Maly analysed the mercury content of tooth roots and the surrounding alveolar bone. (15) They recorded mercury contents up to several hundred mcg/gm for teeth containing amalgam fillings, and over 1200 mcg/gm for teeth having gold crowns covering amalgam cores.

Stortebecker (22) has thoroughly investigated and discussed the passage of mercury from dental amalgam directly to the brain via the axon transport system of nerves and the valve-less cranial venous system. Stortebecker and Stock (7) have both pointed out the potential for mercury passage from the nasal mucosa directly to the brain, particularly the pituitary gland. Nylander found high contents of mercury in the pituitary glands of deceased dentists. (23)

Other than the periodontal tissue measurement studies, there are no data on the quantities of mercury involved in these pathways and what relationship they might have to contribution to total body burden or existent threshold standards. However, the information that is available, particularly Nylander’s autopsy results, is sound enough to present grave cause for concern.

III. INGESTION OF MERCURY DISSOLVED IN SALIVA OR OTHER FLUIDS OR MIXED WITH FOOD:

A number of studies have investigated the amount of mercury dissolved from set dental amalgam in vitro. Unfortunately, only one of these studies attempted to duplicate conditions experienced by the fillings in practical use, that of Brune and
Evje (13) in 1985.

In 1976 Mayer and Diehl suspended amalgam from a string into synthetic saliva at 34 degrees C. They found that mercury dissolved from the amalgam at rates of 1.0-1.2 micrograms/day at one day and 0-0.02 micrograms/day at ten days. (9)

Further studies, released in 1985, totally refuted the results of Mayer and Diehl by comparing in vitro methodology to conditions naturally experienced in the oral cavity.

Nemali and associates (10) measured the mercury dissolved from seven amalgam products in human and synthetic saliva for 24 hours at 37 degrees C. The samples were suspended from string in the solutions and not subjected to functional stimulation. With all of the amalgam alloys tested, the mercury dissolution in human saliva was higher than that in the artificial saliva under identical experimental conditions. The values obtained were 2.5-5.0 micrograms of mercury/ml in human saliva and 1.6-2.2 mcg of mercury/ml in artificial saliva.

In an apparent companion investigation Roa and associates (11) found no significant difference in the dissolution of mercury from high-copper and conventional amalgams. Their results were also reported as 2.5-5.0 micrograms of mercury/ml at 24 hours.

Takaku (12) also compared the mercury dissolution rates from dental amalgam in various solutions. He also suspended the samples from string into the solutions and did not subject them to functional stimulation. He found that the mercury release from amalgam was 30 times higher in human saliva than in distilled water and 10 times higher in saliva than in synthetic saliva. He calculated the amount of daily mercury ingestion via swallowed saliva by subjects with amalgams to be 2.1 micrograms/day.

Brune and Evje (13) were the first investigators to measure mercury dissolved from dental amalgam under static conditions and under cyclic loading to simulate the influence of chewing, as well as rest conditions. They calculated that 20 amalgam fillings, each with a mean geometrical surface area of 0.3 square centimeters and subjected to three daily chewing periods, released 180 micrograms of mercury per day. Considering the gastrointestinal absorption rate of inorganic mercury, they calculated the daily INTAKE of ionic mercury from 20 amalgam fillings to be 18 micrograms.

It is patently obvious that suspending samples of amalgam from string into synthetic test solutions does not accurately portray the conditions to which dental amalgam fillings are subjected in the oral cavity. Indeed, one is compelled to suspiciously wonder why modern investigators fail to consider the in vivo conditions. In his recent dissertation (24) Okabe voluminously discusses the release of mercury from the various phases of amalgam, points out that the research did not consider intra-oral stimulation factors, laments the lack of data for in vivo dissolution of mercury, then blithely concludes that the release of mercury from amalgam fillings is not significant or a
matter for concern.

On the other hand Brune and Evje, the only investigation that attempted to simulate in vivo conditions, calculated the daily intake of mercury in subjects with 20 amalgam fillings to be 18 micrograms/day, which is 90% of the E.P.A. maximum intake standard. Although 20 amalgam fillings is a high number, it is not a rare occurrence. Moreover, the Brune and Evje data does not include consideration of other intra-oral stimulation factors, such as toothbrushing, snacks, hot fluids, or galvanic corrosion; nor does it include intake of inhaled mercury vapor. One must obviously conclude that ingestion of saliva-dissolved mercury from dental amalgam fillings alone represents a major, if not critical, contribution to the body burden of patients as determined by comparison to the E.P.A. standard for maximum daily intake of mercury from all sources other than food!

IV. INHALATION OF RELEASED MERCURY VAPOR:

The greatest cause for concern over the mercury released from dental amalgam fillings is the chronic inhalation of the vapors of mercury over extended periods of time. In 1967 Magos (25) pointed out that mercury vapor is fat soluble and has no electrical charge and, therefore, readily passes cell membranes while in this highly diffusible form. He also pointed out that the oxidation of mercury vapor to ionic mercury is a slow process compared to the circulation time of one passage of blood through the body. Magos stated that the stay of mercury vapor in the blood is transient, due to its rapid passage into the body tissues. It has also been well established that mercury vapor readily penetrates the blood-brain barrier and the placental membrane.

It is also well established that the urinary elimination of mercury from the body is a very slow process; numerous investigators have so determined and stated. Clarkson (26), perhaps, best stated this when he described three separate half-times for the urinary elimination of a single dose of mercury. These half-time phases extended over 133 days. An obvious conclusion would therefore be that multiple repeated daily doses of even small amounts of mercury vapor would result in nothing other than a slow accumulation of mercury within the body over time. This conclusion is easily defensible with the existing autopsy studies. (18-19)

An additional factor regarding exposure to mercury vapor must be considered; that of the minimal body detoxification encountered by inhaled mercury vapor as compared to ingested mercury in any form, including the highly toxic methylmercury. Ingested mercury enters the body through the intestinal mucosa and passes first to the liver via the portal circulation before entering the general circulation to the body. Detoxification and elimination procedures are conducted in the liver, thereby
negating a portion of the mercury intake. Inhaled mercury vapor, on the other hand, enters the body directly into the general circulation and passes to body cells before being subjected to liver detoxification processes.

How, then, does this daily exposure of mercury vapor from dental amalgam fillings relate to the standards for maximum exposure and intake established by the E.P.A.

There have been published studies documenting release of mercury vapor from dental amalgam fillings in vivo. Some of these studies have considered factors that stimulated release of increased levels of mercury vapor from the fillings. However, in this discussion we will not include consideration of these factors, or the numerous other stimulating factors that have not been investigated. We will consider only the unstimulated baseline levels reported in these studies and compare them to the established E.P.A. standards. To do so, we must define and reference various conversion factors that are utilized.

A. CONVERSION OF E.P.A. INTAKE STANDARD TO VARIOUS BODY WEIGHTS:

E.P.A. Mercury Intake Standard = 20 micrograms of mercury per day per 70 kilograms of body weight of all forms of mercury from all sources other than food. (21)

One kilogram = 2.2 pounds.

50 lbs = 6.49 mcg/day
75 lbs = 9.74 mcg/day
100 lbs = 12.99 mcg/day
125 lbs = 16.23 mcg/day
150 lbs = 19.48 mcg/day
154 lbs = 20.0 mcg/day
175 lbs = 22.73 mcg/day
200 lbs = 25.97 mcg/day


C. RELATIONSHIP OF E.P.A. STANDARD TO MERCURY AVAILABLE IN ONE AVERAGE AMALGAM FILLING:

20 micrograms/day x 365 days = 7.3 milligrams/year.
7.3 milligrams = less than 1% of 780 milligrams (the amount of mercury available in one average size amalgam filling).

D. RESPIRATORY FACTORS: (28)

Respiratory rate = 12 breaths per minute. (Average resting rate)
Tidal volume = 500 ml (0.5 liter).
Respiratory volume = 6 liters per minute. (Average at rest)

Oral/nasal breathing ratio = 35% oral (while not chewing). (29–31)
E. ORAL CAVITY VOLUME: An extensive search failed to uncover references for the volume of the oral cavity. In lieu of reference, twenty individuals filled their mouths naturally with water and delivered these volumes into a calibrated beaker. One individual exhibited a volume of 45 ml; the remainder exhibited volumes from 50 to 85 ml. Accordingly, a conservative estimate of 50 ml has been selected for the volume of the oral cavity.

F. VOLUMETRIC CONVERSION:

1 milligram (mg) = 1000 micrograms (mcg).
1 microgram (mcg) = 1000 nanograms (ng).
1 liter (l) = 1000 milliliters (ml).
1 cubic meter = 1000 liters.

Micrograms/cubic meter = nanograms/liter.
(1 microgram = 1000 nanograms. 1 cubic meter = 1000 liters.)

G. ORAL INHALATIONS PER DAY:

1. Respiratory rate at rest = 12 per minute.
2. Minutes per day = 1440 (60 x 24).
3. Inhalations per day = 17,280 (12 x 1440).
4. Oral breathing ratio = 35%.
5. Oral inhalations per day = 6048 (35% of 17,280).

H. CALCULATION OF MERCURY INTAKE FROM Ng/BREATH READINGS:

1. Tidal volume = 500 ml.
   Oral volume = 50 ml.
   Oral volume = 10% of tidal volume. (10% of the air in one
   inhalation is intra-oral air).
2. Oral inhalations per day = 6048.
3. Daily mercury intake (HgIn) = Mercury vapor reading x 0.1
   (10%) x 6048. [FORMULA H]

I. CALCULATION OF MERCURY INTAKE FROM Ng/LITER READINGS:

A. Readings from expired air:
   1. Nanograms per liter is one half of tidal volume = 0.5.
   2. Oral volume is 10% of tidal volume = 0.1.
   3. Oral inhalations per day = 6048.
   4. Daily mercury intake (HgIn) = Mercury vapor reading x 0.5 x
      0.1 x 6048. [FORMULA I-1]

B. Readings from intra-oral air:
   1. Amount of mercury contained in intra-oral air:
      1) Micrograms/cubic meter = nanograms/liter.
      2) One liter = 1000 ml.
      3) Oral volume = 50 ml = 5% of one liter.
      4) Amount of mercury in intra-oral air (inhaled with each
         breath) = mercury vapor reading x 0.05.
2. Oral inhalations per day = 6048.
3. Daily mercury intake ($\text{HgIn}$) = Mercury vapor reading $\times 0.05 \times 6048$. [FORMULA 1-2]

MERCURY VAPOR RELEASE STUDIES
(Reporting unstimulated baseline values)

I. Gay DD, Cox RD, Reinhardt JW. (1)
"Chewing releases mercury from fillings."

Study measured mercury in exhaled air.
Baseline values = 14.22 nanograms Hg/10 breaths.
Stimulated values = 64-244 nanograms Hg/10 breaths.

Conversion to Intake of mercury [FORMULA H]:

$$\text{HgIn} = 1.422 \text{ ng/breath} \times 0.1 \times 6048 = 860 \text{ ng} = 0.86 \text{ mcg/day}.$$  

Percentage of E.P.A. Intake Standard (154 lbs) = 4.3%.
Percentage of E.P.A. Intake Standard (50 lbs) = 13.25%.

II. Svare CW, et al. (2)
"The effect of dental amalgams on mercury levels in expired air."

Study measured mercury in exhaled air.
Baseline values = 0.88 mcg Hg/cubic meter. (MEAN).
Stimulated values = 13.74 mcg Hg/cubic meter. (MEAN).

Conversion to Intake of mercury [FORMULA I-1]:

$$\text{HgIn} = 0.88 \text{ ng/l.} \times 0.5 \times 0.1 \times 6048 = 266 \text{ ng} = 0.27 \text{ mcg/day}.$$  

Percentage of E.P.A. Intake Standard (154 lbs) = 1.35%.
Percentage of E.P.A. Intake Standard (50 lbs) = 4.16%
Percentage of E.P.A. Exposure Standard = 88%.

III. Reinhardt JW, et al. (3)
"Exhaled mercury following removal and insertion of amalgam restorations."

Study measured mercury in exhaled air.
Baseline values = 1.24 mcg Hg/cubic meter. (AVERAGE).

Conversion to intake of mercury [FORMULA I-1]:

$$\text{HgIn} = 1.24 \text{ ng/l.} \times 0.5 \times 0.1 \times 6048 = 375 \text{ ng} = 0.375 \text{ mcg/day}.$$  

Percentage of E.P.A. Intake Standard (154 lbs) = 1.88%.
Percentage of E.P.A. Intake Standard (50 lbs) = 5.78%.
Percentage of E.P.A. Exposure Standard = 124%.

IV. Abraham JE, et al. (4)
"The effect of dental amalgam restorations on blood mercury levels."

Study measured mercury in intra-oral air.
Baseline values = 2.24 ng Hg/15 seconds. (MEAN).
Stimulated values = 18.97 ng Hg/15 seconds. (MEAN).

Conversion to intake of mercury [FORMULA H]:

2.24 ng/15 sec. divided by 15 = 0.15 ng/sec.
\( \times 5 \) seconds/breathe = 0.75 ng/breathe.
HgIn = 0.75 ng/breathe \( \times 0.1 \times 6048 = 454 \) ng = 0.454 mcg/day.

Percentage of E.P.A. Intake Standard (154 lbs) = 2.27%.
Percentage of E.P.A. Intake Standard (50 lbs) = 7%.

V. Vimy MJ and Lorscheider FL. (5)
"Intra-oral air mercury released from dental amalgam."

Study measured mercury in intra-oral air.
Baseline values = 4.91 mcg Hg/cubic meter. (AVERAGE).
Stimulated values = 29.10 mcg Hg/cubic meter. (AVERAGE).

Conversion to intake of mercury [FORMULA I-2]:

HgIn = 4.91 ng/l. \( \times 0.05 \times 6048 = 1485 \) ng = 1.48 mcg/day.

Percentage of E.P.A. Intake Standard (154 lbs) = 7.4%.
Percentage of E.P.A. Intake Standard (50 lbs) = 22.8%.
Percentage of E.P.A. Exposure Standard = 491%.

VI. Patterson JE, et al. (6)
"Mercury in human breath from dental amalgams."
Bull Envir Contam Toxicol. 1985: 34: 459-68.

Study measured mercury in exhaled air.
Baseline values = 3.1 ng Hg/liter. (MEAN).
Stimulated values = 8.2 ng Hg/liter. (MEAN).

Conversion to intake of mercury [FORMULA I-1]:

HgIn = 3.1 ng/l \( \times 0.5 \times 0.1 \times 6048 = 937.4 \) ng = 0.94 mcg/day.

Percentage of E.P.A. Intake Standard (154 lbs) = 4.7%.
Percentage of E.P.A. Intake Standard (50 lbs) = 14.5%.
Percentage of E.P.A. Exposure Standard = 310%.

By consideration of only the unstimulated baseline values reported in these six studies, a minimum average exposure to mercury vapor from dental amalgam fillings may be compared to the E.P.A. Standard for maximum mercury vapor exposure for the general population. Moreover, an estimation of the minimum daily intake of mercury resulting from these unstimulated baseline values may be calculated and compared to the E.P.A. Standard for maximum daily intake of all forms of mercury per 70 kilograms of body weight.

It is found that the values expressed in four of these studies could be compared to the E.P.A. mercury vapor exposure standard. These were found to be 88%, 124%, 310%, and 491% of the exposure standard.

An estimation of minimum daily mercury intake could be made from all six studies and compared to the E.P.A. mercury intake standard. These were found to be 4.3%, 1.35%, 1.88%, 2.27%, 7.4%, and 4.7% of the maximum daily intake for the 154 pound individual and 13.25%, 4.16%, 5.78%, 7%, 22.8%, and 14.5% of the maximum daily intake for the 50 pound individual.

Since the values utilized were only the unstimulated baseline values, this investigation does not include consideration of the increased mercury vapor exposure and intake stimulated by chewing, toothbrushing, hot or acidic fluids and foods, oral and personal habits, or other potential stimulating factors. These findings, therefore, represent the minimum exposure and daily intake that can be expected from mercury vapor released from dental amalgam fillings.

CONCLUSIONS

It is obvious that reference to scientific documentation permits deduction of several conclusions:

1. By virtue of the documents of the agencies themselves, the OSHA and NIOSH occupational threshold limit values for mercury vapor exposure are not applicable for evaluation of potential risks to patients from mercury released from dental amalgam fillings.

2. The E.P.A. standards for mercury vapor exposure and mercury intake for the general population are the only standards in the U.S.A. that are scientifically or medico-legal valid for evaluation of the potential risk to patients from the release of mercury from dental amalgam fillings. Even these standards are qualified by the E.P.A. statements that no amount of exposure to mercury vapor can be considered harmless.

3. There are no scientific studies that provide data allowing
inclusion of consideration of mercury intake from amalgam fillings by the routes of ingestion of abraded particles or passage of ionic mercury into oral and nasal soft and hard tissues.

4. There is only one research study that simulated in vivo conditions and estimated the intake of ionic mercury from dental amalgam fillings via dissolution of mercury in saliva and subsequent ingestion. That study concluded the ionic intake of mercury to be 18 micrograms per day, which is 90% of the E.P.A. standard for maximum daily intake.

5. There are four studies which provide data on unstimulated baseline values for exposure to mercury vapor from dental amalgam fillings that are expressed in terms which can be compared to the E.P.A. standard for maximum mercury vapor exposure for the general population. The average baseline values in three of these studies exceeded the E.P.A. standard (124%, 310%, and 491%). The average baseline value in the fourth study was 88% of the E.P.A. standard.

6. There are six studies which provide data on unstimulated baseline values for exposure to mercury vapor from dental amalgam fillings which can be utilized to estimate minimum daily intake from that source and compared to the E.P.A. standard for maximum daily intake per 70 kilograms of body weight of all forms of mercury from all sources other than food. The estimated daily intake of mercury vapor from these six studies were 4.3%, 1.35%, 1.88%, 2.27%, 7.4%, and 4.7% of the maximum daily intake for the 154 pound individual and 13.25%, 4.16%, 5.78%, 7%, 22.8%, and 14.5% of the maximum daily intake for the 50 pound individual.

7. This discussion does not include consideration of increased exposures and intakes of mercury resulting from meals, snacks, toothbrushing, or the numerous other intra-oral stimulation factors. The findings represent the minimum exposure and daily intake that can be expected from mercury vapor released from dental amalgam fillings. The unstimulated values must also be considered as additive to the stimulated values and therefore, would over time, greatly exacerbate the potential health risks related to mercury intake.

8. The final inescapable conclusion is that patients, especially children, bearing amalgam fillings are being continuously exposed to the dissolution of mercury and the release of mercury vapor from such fillings. Moreover, the resulting chronic intake of this mercury from amalgam fillings constitutes a potential health risk of somber, and as yet, undefined proportions requiring the most intelligent, serious and immediate consideration by the entire dental, medical and scientific communities. Valid scientific data to contradict this conclusion simply does not exist at this time.
REFERENCES


ABSTRACTS/REVIEWS


ENGLISH SUMMARY:

Blood serum levels of the selected glycoproteins (IgG, IgM, IgA, α1AT, α2M, transferrin, haptoglobin, curiolplasmin, C3 and C4 complement compounds) were determined in 15 women occupationally exposed to metallic mercury at the dentists' clinics and in 11 non-exposed control subjects. At the same time, total mercury in the samples of both scalp and pubic hair of the same women was assayed. Statistically significantly lower levels of α2M were found in blood serum of the exposed women (p < 0.005). Total mercury was significantly higher in both the scalp and pubic hair of the exposed women in comparison to non-exposed control subjects. Reversely negative correlation of hair mercury and blood serum IgG in the exposed women was highly significant (p < 0.001).

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SUMMARY:

Selenium deficiency in rats impairs the ability of neutrophils and peritoneal macrophages to kill Candida albicans organisms in vitro. In contrast, killing of Salmonella typhimurium and Staphylococcus aureus organisms is unaffected by the deficiency. Survival of rats after intraperitoneal injection
of $8 \times 10^7$ S. aureus organisms was not affected by Se deficiency, but a 5-fold increase in the dose ($4 \times 10^8$ S. aureus organisms) led to a significantly greater mortality in the Se deficient rats.

Bio-Probe comments: This article demonstrates a viable biochemical pathway that might partially explain the proliferation of candida albicans in some individuals with amalgams. Bio-Probe 3(2) April 1986 details some of the literature demonstrating the ability of mercury to deplete available selenium. It would also provide scientific support for the efficacy of eliminating dental amalgams as a chronic source of mercury in the Candida albicans treatment protocol being employed by Dr. A.V. Zamm in obstinate cases. (see J. Orthomol Med. 1(4):261-266, 1986).


ABSTRACT:

Severe protein-calorie malnutrition is common in patients with AIDS (acquired immunodeficiency syndrome). These nutritional deficits are likely to further impair immune responses and other organ functions vital for recovery from serious infectious diseases. Since selenium deficiency is known to be associated with oral candidiasis and abnormal phagocytic function in animals and depressed helper T-cell numbers in man, we evaluated both selenium status and other nutritional parameters in 12 patients with AIDS compared to 27 healthy controls. Selenium was measured by a spectrofluorometric method. The mean (±SD) plasma selenium level in AIDS was $0.043 \pm 0.01 \text{ ug/ml}$ vs $0.095 \pm 0.016 \text{ ug/ml}$ in controls ($p < 0.001$). Whole blood selenium and red blood cell selenium levels were also significantly reduced in AIDS ($p < 0.005$). The mean weight loss in AIDS patients was $35.5 \pm 21.2$ pounds. Serum albumin was significantly ($p < 0.01$) lower in AIDS patients compared to controls. A good correlation between serum albumin and plasma selenium was noted ($r = 0.77; p < 0.001$). We conclude that selenium deficiency is a common component of the malnutrition seen in AIDS patients. Therefore, aggressive nutritional support, including attention to selenium status should be considered an integral part of the therapy of AIDS patients.

Bio-Probe comment: See Bio-Probe comments on the previous article above. Although not published as yet, we are aware of ongoing research demonstrating lower than normal levels of selenium and glutathione peroxidase in persons with amalgam fillings. Moreover, 5 months of therapeutic supplementation with selenium and glutathione were required to restore selenium and glutathione peroxidase values to their norms. After obtaining
normal levels, the placement of one amalgam filling had an immediate effect of reducing selenium and glutathione peroxidase levels to their pre-supplementation values.

There presently exists a vast body of scientific data demonstrating the ability of mercury to affect the biochemical availability of both selenium and glutathione and their derivative enzyme systems. There is also scientific literature demonstrating selenium plays an important role in the humoral and cellular immune system of animals. It is interesting to note that mercury also has the ability to suppress the immune system including reducing available helper T cells and changing the helper to suppressor T cell ratios. (See Bio-Probe Vol 1, Issue 1 & 3, 1984 and Eggleston, J Prosthet Dent. 51(5):617-623, 1984). Is the reduction of available selenium the mechanism by which mercury suppresses the immune system and changes critical T cell ratios????

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SUMMARY:

Diets specifically deficient in selenium and/or vitamin E or adequate in both nutrients were given to chickens from the time of hatching. Lymphoid organs (bursa, thymus and, in some instances, spleen) were collected from chickens 7-35 days old. Growth and lymphoid organ growth were measured. The development of the primary lymphoid organs was further assessed by histological techniques. Specific deficiencies of Se or of vitamin E significantly impaired bursal growth as did a combined deficiency. Thymic growth was impaired only by the combined deficiency diet. Severe histopathological changes in the bursa resulted from the combined deficiency and these were detectable by 10-14 days after hatching. These changes were characterized by a gradual degeneration of the epithelium and an accompanying depletion of lymphocytes. Similar changes, although slower to develop and less severe, were observed in the thymus as a result of the combined deficiency. Vitamin E and Se in serum and tissue were rapidly and independently depleted by the specific deficiency diets. The data suggest that the primary lymphoid organs are major targets of Se and vitamin E dietary deficiencies and provide a possible mechanism by which immune function may be impaired.

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SUMMARY:
Of 12 patients with asthma and food and aspirin intolerance 10 had low or very low glutathione peroxidase (GSH-Px) activity and the frequency of low GSH-Px activity in that group was significant compared with values in age- and sex-matched healthy controls.


SUMMARY:
Mean serum selenium concentration in 20 adults, 19 to 34 years old with cystic fibrosis (CF), including 2 with cancer, was 0.81 ± 0.16 umol/litre; the value in controls was 1.41 ± 0.20 umol/litre. Mean serum vitamin E concentration in patients was 4.4 umol/litre. It is suggested that low concentrations of circulating Se are associated with increased risk of carcinoma especially where serum vitamin E concentration is low. Older patients with CF may be at increased risk of developing carcinoma.

BIO-PROBE COMMENT:
SELENIUM HOMEOSTASIS = ETIOLOGICAL FACTOR TO GOOD HEALTH
MERCURY = DISRUPTION/DEGRADATION OF SELENIUM HOMEOSTASIS
AMALGAM FILLINGS = CONTRAINDICATION TO GOOD HEALTH


ABSTRACT:
Mercuric chloride induces in Brown-Norway rats an autoimmune disease due to a T dependent polyclonal activation of B cells. Various autoantibodies and a striking increase in total serum IgE level are observed as consequences of this polyclonal activation. The aim of this study was to investigate the in vitro response of autologous syngeneic normal lymphocytes to lymphocytes exposed in vivo or in vitro to HgCl₂. These experiments demonstrate that HgCl₂ induces autoreactive T cells and suggest that these cells may be responsible for the autoimmune disease.


SUMMARY:
The treatment of male rats with Hg²⁺ resulted in significant alterations in heme and hemoprotein metabolism in the adrenal gland which, in turn were reflected in abnormal steriodogenic
activities and steroid output. It is suggested that Hg2+ directly caused a defect in adrenal steroid biosynthesis by inhibiting the activity of 21 α-hydroxylase. The apparent physiological consequences of this effect included lowered plasma levels of corticosterone and elevated concentrations of progesterone and dehydroepiandrosterone. This abnormal plasma steroid profile is indicative of 21 α-hydroxylase impairment.


ABSTRACT:

Adult Wistar rats of both sexes were exposed to mercuric chloride (HgCl₂) through drinking water (20 mg HgCl₂ liter⁻¹ distilled water) ad libitum during an 8-month period. Animals were subsequently sacrificed and coronal sections of the brain and cervical spinal cord were examined according to a histochemical technique based on a physical development process which renders mercury deposits visible. Mercury was found unevenly distributed in the brain and spinal cord with the heaviest deposits found within the motor nuclei of the rhombencephalon. In cerebral cortex, the highest concentration of mercury was found in the striate area. Mercury was also localized within the deep nuclei of cerebellum; none was found within Purkinje cells. A proportionately high amount of mercury was additionally found in the anterior horn motorneurons of the spinal cord. In general, mercury was found primarily within neurons but it was also observed in the cytoplasm of glia and ependymal cells.


SUMMARY:

Metals were estimated by atomic absorption spectrophotometry in 27 double samples of canned or bottled beer of different types, nearly all German. Mean values per litre were for copper 0.19 mg and for chromium, mercury, cadmium and lead 39.0, less than 1, 1.1 and 13.7 ug based on, respectively, 52, 47, 10, 54 and 54 samples. Beer consumption, representing 17.3% by weight of food and beverage intake of the average German citizen contributed only 5 to 9% of dietary intake of harmful metals and was not considered a health hazard. Heavy metal content was not related to type of beer, brewing method, packaging material or use of copper or chrome steel equipment. Existing heavy metal limits for drinking water could be applied to beer.

SUMMARY:

Chromium and nickel were estimated in 95 European wines by flameless atomic absorption spectrometry. Values were 13-186, mean 35.4 and 14-300, mean 51.7 ng/g, respectively. There was a significant correlation between Cr and Ni values. The Cr:Ni ratio was 0.68 and agreed well with the ratio of released Cr:Ni by stainless steel in wine but the amount of released Cr and Ni was too small to explain the high values of these in wine. The calculated daily Cr intake from wine was 1.7 ug in Belgium, 1.6 in the Netherlands, 8.0 in Spain, 8.7 in Italy and 11.2 ug in France.


ABSTRACT:

Lead induces peripheral nerve segmental demyelination in rats. Arsenic and thallium produce a peripheral neuropathy characterized by axonal degeneration in humans. Mercury and thallium appear to damage both the peripheral and the central nervous system. It is not known whether this difference in effect is due to different molecular forms of the elements, to differential access to various compartments of the nervous system, or to intrinsically different properties of the elements. Using an in vitro model system of dorsal root ganglion neurons and morphometry of neurite outgrowth and myelination, we demonstrated that mercury and arsenic produce 50% inhibition of neurite outgrowth at 3.9 and 9.6 X 10^-6 M, respectively, whereas the same degree of inhibition is produced by 1.3 X 10^-4 M thallium and 3.3 X 10^-4 M lead. Lead also produces complete inhibition of myelination at 1 X 10^-6 M, suggesting that a primary effect on myelination is present in this model system as well as in the intact rodent.


SUMMARY:

Changes in the morphology of the tongue, olfactory epithelia, trachea and inner ear were studied by electron microscopy in guinea pigs given a diet free from vitamin A for 90 days. Papillae of the tongue were shorter than in controls and had many squamous cells covering the whole epithelium. Taste buds were not detectable beneath the dense layer of squamous cells which may explain the loss in taste sensitivity reported in vitamin A deficiency. In the olfactory and respiratory epithelia there was atrophy and focal degeneration of ciliae. In the ear there was vacuolization of the outer hair cells indicating that the tight junction of outer hair cells may be affected allowing intermixing of inner ear fluids.