SPECIAL ARTICLE
Detoxification
Part III. Vitamin C. Sam Ziff

REVIEW/ABSTRACTS
Differences in the effects of Hg(II) on DNA repair induced in Chinese hamster ovary cells by ultraviolet or X-rays. Christie

Retardation of experimental oral cancer by topical vitamin E. Odukoya

Beta-carotene levels in exfoliated mucosa cells of population groups at low and elevated risk of oral cancer. Stich

A study on the placental transfer of mercury in pregnant women. Nakano

The hepatotoxicity of mercury vapours in the light of biochemical, scintigraphic and morphological data. Cholewa

Mercury vapour released during the removal of old amalgam restorations. Richards

Effect of vitamins A, E, K on the glutathione antiperoxidase system in the gingival tissue in parodontosis. Khmelevsky

EDITORIAL
Debating the Amalgam Controversy

FORUM
Annual Meeting of the International Academy of Oral Medicine and Toxicology. Scientific Symposium on Mercury Toxicity

Debate between Dr. Victor Penzer and Dr. Robert Baratz

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DETOXIFICATION - PART III
VITAMIN C
Sam Ziff

One word, "controversial", sums up the status of vitamin C in the minds of scientists, professional health care providers and the lay public. The controversy covers everything from the amount of vitamin C recommended as the daily dose to its biochemical and pharmacological role in disease and in health. This same plight exists with regard to the role of vitamin C in the detoxification of heavy metals.

I certainly didn’t visualize any problems in finding documentation to support the inclusion of vitamin C in any detoxification protocol related to reducing mercury body burdens. I say that because from the outset of my research into the mercury/amalgam phenomenon the one thing that there didn’t seem to be any question about was the fact that vitamin C was specified in every detoxification protocol. Although there are thousands of research papers on vitamin C, much to my surprise, there is very little published on any direct detoxification relationship to mercury.

One usually identifies vitamin C as the antiscorbutic vitamin because its discovery was related to prevention and treatment of the disease scurvy. The antiscorbutic factor of the fruits used to treat and prevent scurvy was isolated from lemon juice by Szent-Gyorgi in 1928 and in 1933 the name of this factor (hexuronic acid) was changed to ascorbic acid. The symptoms of clinical scurvy include swollen joints, muscular aches and bone pain, edema, weakness, fatigue, anemia and hyperkeratosis (especially around hair follicles), and impaired wound healing and possibly a breakdown of scar tissue. Behavioural changes may include apathy, depression and emotional disturbances. There are also a number of characteristics probably related to a weakening of the walls of blood vessels such as swollen and bleeding gums, ocular hemorrhages, bruising, and varicosities of small blood vessels which are seen under the tongue. (1)

Although frank clinical scurvy is rarely seen today, there is evidence that chronic subclinical vitamin C deficiency may exist in a large segment of the population. This subclinical deficiency has metabolic and clinical aspects and symptoms different from clinical scurvy but can lead to impaired health and increased susceptibility to other disease. (2,3) It is this aspect that provides the biochemical interrelationships with mercury and detoxification protocols.

The known physiological functions of vitamin C are: Synthesis of polysaccharides and collagen; Formation of cartilage, dentine, bone, and teeth; antioxidant; absorption of iron; cold tolerance, maintenance of the adrenal cortex; metabolism of tryptophan, phenylalanine and tyrosine; growth; wound healing; and maintenance of capillaries. (4) There is also considerable evidence that vitamin C is directly involved in: proline and lysine hydroxylation; carnitine synthesis; and Dopamine hydroxylation and that vitamin C affects drug and cholesterol breakdown; sulphation; lymphocyte and neutrophil function; and folate reduction. (1)
In 1977 Kallner et al. demonstrated that there is an 80-90% absorption of dietary ascorbic acid from the intestine. (5) However, the absorption of vitamin C also occurs in the stomach and buccal mucosa with the uptake into the buccal mucosa being pH-dependent. Uptake increases the longer a solution containing vitamin C is held in the mouth. It is thought that the buccal absorption of vitamin C occurs by passive diffusion through the membrane of the buccal mucosal cells. The rate and extent of diffusion being determined by the initial concentration of vitamin C in the buccal cells and by its rate of passage from the cells into the blood in mucosal capillaries. (6)

Ascorbic acid is present in the plasma and is ubiquitously distributed in the cells of the body:

**Vitamin C Content of Adult Human Tissues**

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Vitamin C (mg/100 g wet tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pituitary gland</td>
<td>40-50</td>
</tr>
<tr>
<td>Adrenal glands</td>
<td>30-40</td>
</tr>
<tr>
<td>Eye lens</td>
<td>25-31</td>
</tr>
<tr>
<td>Brain</td>
<td>13-15</td>
</tr>
<tr>
<td>Liver</td>
<td>10-16</td>
</tr>
<tr>
<td>Spleen</td>
<td>10-15</td>
</tr>
<tr>
<td>Kidneys</td>
<td>5-15</td>
</tr>
<tr>
<td>Heart muscle</td>
<td>5-15</td>
</tr>
<tr>
<td>Lungs</td>
<td>7</td>
</tr>
<tr>
<td>Skeletal muscle</td>
<td>3</td>
</tr>
<tr>
<td>Testes</td>
<td>3</td>
</tr>
<tr>
<td>Thyroid</td>
<td>2</td>
</tr>
<tr>
<td>Leucocytes</td>
<td>35</td>
</tr>
<tr>
<td>Plasma</td>
<td>0.4-1.0</td>
</tr>
</tbody>
</table>

(Adapted from *Vitamin C in Health and Disease* by T.K. Basu and C.J. Schorah, 1982)

Vitamin C concentrations in leukocytes are sometimes taken to represent those in tissue and appear to be less susceptible to depletion than is the plasma. Sauberlich (1975) demonstrated that plasma vitamin C reflected recent dietary intake rather than necessarily indicating tissue reserves. Plasma vitamin C is rapidly affected by acute illness. This can also be true for leukocytes. Hume and Weyers (1973) found a significant fall in leukocyte vitamin C status during the first few days of respiratory infections and oral intakes of 2 grams a day (3 days) were required to restore the leukocyte vitamin C status to pre-cold values.

The vitamin C Saturation Test can provide an assessment of tissue status. If vitamin C reserves in the body are low, than an oral dose will not increase plasma vitamin C sufficiently to allow loss of ascorbic acid in the urine. This is predicated on the fact that a number of studies have indicated that the ability of the renal tubule to reabsorb vitamin C filtered through the glomerulus is exceeded when plasma vitamin C rises to 0.75-1.0 mg/100ml, and this leads to rapid loss of the vitamin in the urine. The lower the body saturation or the greater the rate of vitamin C metabolism, the larger will be the oral intake required to bring about a loss of a significant quantity of vitamin C in the urine. (1)
As stated at the outset, the rationale for use of vitamin C in detoxification protocols is not very clear, based on the literature that has been published. This fact notwithstanding, the problem can be approached from the aspect of physiologic functions of vitamin C in the body.

One of the major functions of vitamin C relates to assisting the body to handle chemical and physical stress. Chemical stresses would include hazards we are exposed to through contact, breathing, eating and smoking or toxins from infection and bacteria.

Vitamin C apparently has a protective effect against mercury poisoning. In experiments with guinea pigs done in 1951, Vauthey demonstrated that a specific dose of mercury cyanide injected into the guinea pigs killed all of the animals within one hour. However, if the guinea pigs were given high levels of ascorbic acid (equivalent to 35 grams a day for a human weighing 154 pounds) prior to the injection of the same dose of mercury cyanide, 40% survived the mercury poisoning. This same protective effects were demonstrated against other forms of mercury by Mavin in 1941 and by Mokranjac and Petrovic in 1964.(8,9,10)

For many years, mercury diuretics were used extensively by the medical profession. The toxicity of these mercurial diuretics could be reduced if the patient was given ascorbic acid prior to or simultaneously with the mercurial diuretic.(11)

How the ascorbic acid reduced the toxicity of mercury was not determined. However, there is a possible biochemical pathway that would in theory be plausible. When vitamin C is metabolized part of it is metabolized to vitamin C-sulphate with the sulphate being derived from sulphur-containing amino acids, such as cysteine. Vitamin C competes with certain drugs for sulphate conjugation which could affect the pharmacological activity and toxicity of drugs.(12)

Basu (1977) demonstrated that a dose of 3 grams of vitamin C per day would reduce the excretion of cysteine in the urine to 50% of the pre-vitamin C values. Basu postulated that cysteine is used to metabolize vitamin C.(13) Experimental and clinical evidence (Wokes 1958 and Smith 1961, suggest that the detoxification of cyanide takes place by its conversion to a sulphur-containing metabolite thiocyanate, and that the reaction may require cysteine.(14)

Furthermore, Basu (1977) demonstrated that the urine levels of thiocyanate were markedly decreased by administration of high doses of vitamin C and that concomitant administration of 10 mg/day of cysteine restored the urinary thiocyanate to normal levels.(13) Basu also states: "When excess vitamin C is ingested and the protein intake is limited, it is possible that cysteine would be monopolized for sulphate conjugation by the vitamin, and as a consequence render one of the body's detoxification mechanisms less effective.

As outlined in Bio-Probe Newsletter vol 3, issue 1, 1986, mercury competes very actively for the sulphur-containing amino acids cysteine and methionine as well as the cysteine molecule of the tripeptide
glutathione. Unfortunately, the only consideration regarding vitamin C and cysteine that we were previously aware of, related to the need to insure a three to one ratio of vitamin C to cysteine to prevent its conversion to cystine. Consequently it would seem, at least from the data presented, that cysteine should be a primary nutrient in any detoxification protocol to offset the depletion caused by the mercury being released and inhaled from amalgam fillings and the environment as well as that being ingested from dietary sources. When megadose vitamin C therapy is included in the protocol, without supplemental cysteine, the problem of cysteine depletion is additionally aggravated. This particular problem could be further compounded by the presence of lead, arsenic and cadmium which are all thiol sensitive.

Vitamin C also serves as an antioxidant and free radical scavenger. It is in this capacity that another biochemical pathway is evident. There is scientific evidence that mercury causes lipid peroxidation/free radicals. In a recent paper Dr. Robert F. Cathcart, III one of the world's leading authorities on the clinical use of vitamin C, discussed the role of ascorbate in the free radical scavenging pathway: "In general free radical scavenging occurs through complex metabolic pathways involving many steps which are rate-limited. Deficiencies of nutrients, vitamins and minerals, which make up the enzymes and coenzymes of these systems can slow down or halt certain pathways." (14)

Dr. Cathcart than goes on to give an example of the complexity and why it is rate-limited utilizing the glutathione pathway as an example: "When for example, a superoxide radical must be destroyed, superoxide dismutase can catalyze its conversion to $\ce{O2}$ and $\ce{H2O2}$. Ascorbate, nonenzymatically, also converts superoxide to $\ce{Eo 2\ce{H2O2}}$, but is oxidized in the process to the ascorbate free radical and dehydroascorbate. The ascorbate free radical and the dehydroascorbate are reduced back to ascorbate either by NADH (catalyzed by semidehydroascorbate reductase and forming NAD) or reduced glutathione (GSH) (catalyzed by dehydroascorbate reductase and forming oxidized glutathione (GSSG). Some of the peroxide can be converted to oxygen and water by catalase but most will be destroyed by a glutathione-requiring enzyme system. GSH (catalyzed by glutathione peroxidase) reduces the peroxide to water but in the process is oxidized to GSSG. The resulting GSSG is reduced by NAD(P)H (catalyzed by glutathione reductase). The resulting NAD is reduced back to NADH by way of the Krebs cycle or resulting NADP is reduced back to NADPH by the hexose monophosphate (HMP) pathway. It is thought that commonly the rate-limiting step in the last series of reactions is that catalyzed by glutathione peroxidase and its cofactor selenium, but other substances which could limit all this are the vitamin E, vitamin C, vitamin B2, vitamin B3, Cysteine, etc. Note: the ascorbate used in this example is as in the vitamin C sense; the small amount available is oxidized to dehydroascorbate and then must be reduced back to ascorbate by the pathway described, to be reused as ascorbate. One can easily see how this mechanism and similar mechanisms could be overwhelmed by a toxic pathogen liberating free radicals or by an inflammatory cascade regardless of its cause." (14)
It is evident from the example given by Dr. Cathcart that mercury in addition to generating free radicals, also adversely affects the availability of many of the key nutrients, vitamins and minerals involved in the complete scavenging cycle, i.e., selenium, glutathione, cysteine, ascorbate, and vitamin E.

Mercury is also known to inhibit collagen synthesis (15). The synthesis of collagen is impaired in vitamin C deficiency. This appears to be due to lowered ability to hydroxylate lysine and proline. There is considerable evidence that the reducing agent in both hydroxylation of lysine and proline is the reduced form of vitamin C. Consequently, in vitamin C deficiency, it is believed that the amount of effective collagen fibre present in connective tissue is reduced (1). "Swelling, hemorrhages, and secondary bacterial infections of the gingival margins are common in severely scurvy patients. The deficiency of vitamin C does not of itself cause the inflammation but rather impairs the normal defensive responses of the mucous membranes. Thus, the massive gingival enlargement so characteristic of scurvy results from the combined effects of lack of vitamin C and nonspecific inflammation." (19)

In periodontal disease there is documented scientific evidence showing mercury as one of the etiological factors. Autoradiographic studies with tritiated proline show a constant renewal of collagen in the periodontium, which relies on vitamin C. (16) Glickman in 1948 demonstrated that in vitamin C deficient guinea pigs the periodontal membrane experienced structural disruption with the severest changes in the area adjacent to the alveolar bone. (17). In another paper in 1948 Glickman applied a 10% solution of silver nitrate to the labile gingiva (for 30 seconds) to both vitamin C depleted guinea pigs and control animals. In the vitamin C deficient animals, the non-irritated tissue showed the same breakdown of periodontal membrane and indentation seen in the scurvy state, but the irritated tissue showed even more severe collagen breakdown in the membrane. In the control animals the collagen degeneration was only slight in the irritated area. These findings support the argument that while vitamin C deficiency does not cause gingival inflammation, the local irritating factor which does produce it will cause increased destruction when there is vitamin C deficiency.

Most textbooks and toxicology books outline gingivitis or stomatitis as definitive symptoms of mercury toxicity related to chronic exposure to mercury vapor. The patient with amalgam fillings and poor nutriture, especially low ascorbate values, is then in double jeopardy of developing periodontal problems. Use of vitamin C as a primary nutrient in any detoxification protocol takes on added significance when considered in light of the biochemical pathways related to chemical stress, free radical generation and collagen synthesis outlined above.

In addition to chemical stress there is physical and mental stress. The usual response to stress is increased secretion of the hormones of the adrenal glands. I find it fascinating that the adrenals are one of the target glands of mercury deposition and also contain the body's 2nd highest tissue levels of vitamin C.
Physical and mental stress increases adrenal activity which in turn depletes ascorbic acid from the gland. In mammals which produce their own ascorbic acid, this depletion is rapidly replenished. Humans who don’t produce ascorbic acid, attempt to replenish the adrenal stores of ascorbic acid by taking it from other stores in the body. If tissue values of ascorbic acid are low, there may be insufficient available to replenish or satisfy the requirements of the adrenals. Under these conditions normal adrenal hormone response may become inadequate.(20)

Physical stress also includes exposure to heat or cold. High environmental temperatures accelerate the destruction of ascorbic acid and increase the physiological need for it. The rate of depletion of ascorbic acid in blood serum was significantly higher in summer than in winter.(21,22,23). In animal studies investigating response to cold temperatures similar conclusions were reached.

Here again, the person with mercury/amalgam fillings who is being exposed to chronic intakes of mercury vapor would also be subjecting the adrenals to depletion by chemical stress. Unfortunately this relationship of mercury and vitamin C is not a new discovery. I would like to quote from a paper by Blackstone et al.: "Shun mercury as poison" was Kramer’s advice to scorbatic patients according to George Budd a London physician in 1840. Budd himself claimed that in cases of scurvy "...mercury in every form should be religiously avoided [as] we have met with instances in which the scorbatic symptoms seemed to have been much aggravated by mercury taken before the scurvy made its appearance."..."The observations of Budd indicated that low tissue levels of ascorbic acid increased a person’s susceptibility to mercury poisoning (or, conceivably, that an increased intake of mercury exacerbated the scorbatic condition).”(24)

In the paper cited above, Blackstone and his associates determined that mercury did induce adrenal hypertrophy and that this could be prevented by large doses of ascorbic acid. Biochemically they felt that thiol groups have a direct role in the preservation of tissue ascorbic acid and its reduced form and that any diminution in the biological reducing capacity of tissue thiols would presumably result in lower levels of tissue ascorbic acid. One surprising finding of this study was that large doses of ascorbic acid resulted in an increased deposition of mercury in the liver and kidney. The authors concluded: "Subjects exposed to higher-than-average concentrations of environmental mercury should perhaps avoid "megavitamin therapy".

With regard to the above caution, the mercury chloride was given in water and the authors felt that the lowered tissue levels of ascorbic acid could reflect mercury interference with absorption of ascorbic acid from the gastro-intestinal tract. However, other studies with mercury have shown a marked difference in the way the body responds to ingested versus parenterally administered mercury. In the case of mercury amalgam fillings, mercury vapor is being inhaled and entering the blood, which would more closely parallel the results shown for parenterally administered mercury, which does not show increased deposition. This fact was demonstrated in experiments.
conducted by Murray and Hughes in 1976. "The results of Experiment 1 indicated that a high ascorbic acid intake increased the tissue levels of orally-administered Hg but was without effect on injected Hg." (25)

REFERENCES


Although the primary emphasis of this article was to define the relationship between vitamin C and mercury/amalgam there are many current research papers on vitamin C containing significant information that should be brought to your attention:


Several epidemiological studies have demonstrated that high ascorbic acid intake is associated with low mortality rates of cerebrovascular disease (CVD). Hypertension is known to be the most important risk factor of the disease and ascorbic acid has been reported to be the most deficient nutrient in hypertensives compared with normotensives in the analysis of standardized mean differences in nutrient intake between the two groups. ....

It has been suggested that oral ascorbic acid administration enhances calcium absorption in the intestinal tract by its chelating effect on calcium independent of its biological function as a vitamin. In fact, the most pronounced effect of orally administered ascorbic acid in rats is an increased calcium excretion in the urine, showing that calcium absorption is enhanced.....ascorbic acid must affect blood pressure through its effect on calcium metabolism and hormones related to calcium metabolism, such as parathyroid and 1,25-dihydroxycholecalciferol. Citric acid has also been said to prevent blood pressure elevation in SH rats (unpublished). Therefore, ascorbic acid may not be a specific organic acid affecting blood pressure.


Summary: Ascorbic acid (246 mg/kg body weight/day) was administered orally to 9-week old female guinea pigs of the Hartley strain over a period of 20 months. The controls received 40 mg/kg body weight per day of ascorbic acid in the diet. Observations were made on body weight, food and water consumption, plasma ascorbic acid, and the total calcium and ionic calcium levels at various times during the growth of these animals. A second experiment was carried out when the guinea pigs were 18 months old. In addition to the oral intake, they received intraperitoneally 632 mg/kg body weight/day of sodium ascorbate for 6 weeks. With this treatment, the ascorbic acid intake for the test animals was 20 times that for the controls. The plasma ascorbic acid and calcium levels of these animals were measured during the treatment.

In the ascorbic acid-treated animals, there was a significant elevation in plasma ascorbic acid level in comparison with the controls, but no substantial differences were observed in the body
weight, total calcium or ionic calcium levels in the plasma. The results suggest that the administration of large quantities of ascorbic acid does not affect total calcium or ionic calcium levels in the plasma of these animals.


Summary: The effects of a high dose of ascorbic acid superimposed on a low magnesium diet were studied for the first time. Young male guinea pigs were fed for six weeks two diets containing 3000 or 600 ppm magnesium; half in each group was supplemented with a daily oral dose of either 3 mg or 100 mg ascorbic acid per 100 g body weight. No treatment effects were found in serum copper and ceruloplasmin, spleen copper, bone calcium, kidney magnesium, and brain calcium and magnesium contents. Both bone copper and brain ascorbic acid contents of the group fed the normal ascorbic acid/low magnesium diet were lower (p < 0.01) than the combined means of the other three groups; the high ascorbic acid/low magnesium treatment resulted in normalization of bone copper and brain ascorbic acid levels. Irrespective of ascorbic acid level, the low magnesium diet decreased the bone magnesium and increased the kidney calcium contents (p < 0.01); this effect on kidney was nearly doubled by the high ascorbic acid intake (p < 0.01). The results indicated that the main effects were due to magnesium deficit. (My comment: Mercury is known to inhibit certain magnesium sensitive enzymes. This paper places added emphasis on the need to insure inclusion of magnesium in any detox protocol.)


Abstract: A prolonged daily dose (100 mg/mouse for 20 days) of vitamin C completely prevented the growth of transplanted Sarcoma 180 in mice and these mice survived up to 72 days without any toxicity. However a single dose of 400 and 500 mg/mouse proved toxic and all the treated animals died with 1 and 1/2 hour respectively. The authors conclude their study with the following: "The results of the present study are encouraging enough to explore further the efficacy of vitamin C in treatment of malignant growth in experimental animals as well as in humans. As the growth of ascites tumour in mice was completely prevented by 2 g vitamin C further higher dose was not thought necessary." (100 mg/day for 20 days)


Summary: Studies were conducted to evaluate the blood levels of ascorbic acid, dehydroascorbic acid, glutathione, and histamine in patients with gastric carcinoma, tuberculous enteritis and nonspecific ulcerative colitis. Leucocyte ascorbic acid, urinary excretion of
total ascorbic acid and ascorbic acid saturation test were also carried out in order to assess the ascorbic acid status of these patients. It was observed that the plasma and leucocyte content of ascorbic acid was significantly lower with markedly decreased urinary excretion in these patients. Further urinary excretion of ascorbic acid after a test dose was also found to be subnormal. Decreased levels of glutathione and significantly higher levels of histamine reflect an overall reducing status of the body that is markedly deranged. (My comment: I find it intriguing how similar biochemical pathways and mercury toxicity symptomatology can be visualized or overlayed on the data presented)

RECOMMENDATIONS


SUMMARY: "The effect of relatively nontoxic levels of HgCl₂ on semi-conservative DNA synthesis and on DNA repair induced following treatment of intact cells with X-ray or ultraviolet (UV) light has been studied in cultured Chinese hamster ovary cells. In the presence of 1 um HgCl₂, the repair of DNA strand breaks induced by 450 rads of X-rays was reduced by 37%. If a treatment of 2.5 um HgCl₂ was given to cells for only 15 min prior to a 450 rad irradiation, the rate of repair was reduced even further with only 25% of the breaks being repaired in the first hour following irradiation. When comparable treatments of HgCl₂ were given to Chinese hamster ovary cells in conjunction with UV irradiation there was no significant effect on either the number of initial strand scission events or the return to high molecular weight DNA following completion of repair. Only after exposure of cells to toxic levels of Hg(II) (higher concentrations or longer treatments) was there measurable inhibition of UV-induced repair as evidenced by a reduced rate of ligation of DNA to a high molecular weight form. Inhibition of the endonuclease step of UV repair was not observed since Hg(II)-treated cells exhibited the same level of strand scission immediately following UV as cells not treated with Hg(II). The observed differences in the effects of Hg(II) on two pathways for DNA repair indicate that the potential for synergistic action between Hg(II) and other DNA damaging agents will be determined in part by the repair pathways induced by each agent. Additionally it was found that inhibition of semi-conservative synthesis also occurs at low concentrations of HgCl₂ similar to those affecting X-ray induced repair. The presence of Hg-DNA adducts in the DNA at these concentrations may cause a reduction in normal replication to facilitate DNA repair."

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ABSTRACT: "Forty-eight young male and female golden hamsters (Mesocricetus auratus) were divided into four groups of 12 animals each. The left buccal pouches of Group 1 and 2 animals were painted 3 times weekly with a 0.5% solution of 7,12-dimethylbenz(a)anthracene (DMBA) in heavy mineral oil for 7 weeks. At the end of this period, the left buccal pouches of Group 2 animals were painted 3 times weekly with vitamin E (DL-a-tocopherol, in pure form) for an additional 4 weeks. Group 3 animals were painted with vitamin E only, for 4 weeks. Group 4 animals were untreated controls. Group 2 animals demonstrated a significant delay in tumor formation in comparison with Group 1 animals. Gross observation revealed fewer and smaller tumors in the Group 2 animals; microscopic examination revealed smaller tumors with better cellular differentiation and less invasion. No tumors were observed in Group 3 and Group 4 animals. These observations were similar to those made in previous studies of oral carcinogenesis using systemic vitamin E to delay tumor formation."


ABSTRACT: "Beta-carotene was estimated in exfoliated oral mucosa cells in groups of individuals at various risks for oral cancer. Approximately \(4 \times 10^8\) exfoliated cells were collected from each subject by brushing the oral mucosa. Cell pellets were hydrolyzed with pronase and then with KOH/methanol. Beta-carotene was extracted with hexane, separated by reverse-phase HPLC, and detected at 450 nm. Mean beta-carotene levels in exfoliated cells were 0.08 ng/10^6 cells for 56 heavy consumers of alcoholic beverages (150 g or more per week), 1.36 ng/10^6 cells for 55 lacto-vegetarians of the International Society for Krishna Consciousness (ISKK) (abstainers from alcohol and tobacco), and 1.08 ng/10^6 cells for 61 representatives of a "Western" lifestyle pattern (64% consumed the equivalent of at least one bottle of wine or 7 bottles of beer per week, and all were non-smokers). If the heavy alcohol consumers (males) are matched to non-drinking males of comparable age, the mean beta-carotene values are 0.08 ng versus 1.24 ng/10^6 cells. The possible involvement of the low levels of beta-carotene in the mucosa of heavy alcohol drinkers in increased sensitivity towards the carcinogenic and genotoxic activity of cigarette smoking plus alcohol ingestion is discussed."


"To clarify the maternal-fetal transfer of mercury across the placenta, inorganic and organic mercury was determined in 41 paired samples of maternal blood, placenta, umbilical cord blood and umbilical cord obtained from pregnant women who had no particular exposure to mercury compounds in their history. Both inorganic and organic mercury were detected in all the
samples but the ratio of organic mercury to total mercury concentrations was much higher than that of inorganic mercury to total mercury. The concentrations of inorganic and organic mercury in the umbilical cord blood were significantly higher than those in the maternal blood, strongly indicating the maternal-fetal transfer of mercury via placenta. The ratio between placental concentration and maternal blood concentration of the two mercury forms was significantly higher in the inorganic than in the organic form, indicating that inorganic mercury does preferentially accumulate in the placental tissues. On the other hand, the ratio between umbilical cord blood concentration and placental concentration of the two mercury forms was significantly higher in the organic than in the inorganic forms, indicating that there was a preferential transfer of the organic mercury across the placenta. In addition, no significant difference was observed between the forms of mercury regarding the ratio of umbilical cord blood concentration to maternal blood concentration, suggesting a possibility that both forms of mercury may be transferred to the fetus with similar efficiency. However, the correlations between maternal blood and umbilical cord blood between maternal blood and placenta and between placenta and umbilical cord blood in terms of the concentration of the two forms of mercury suggest that the placenta is less permeable to inorganic mercury and that the organic mercury reaching the fetus through the transplacental route may be metabolized into an inorganic form."


ABSTRACT: "This report compares the mercury-induced embryotoxicity among one noninbred (LVG) and five inbred (CB, LHC, LSH, MHA, PD4) strains of hamsters. A single dose of mercuric acetate (15 mg/kg, sc) was injected into pregnant hamsters on the morning of the 8th gestation day. Treated and control animals were killed on either the 12th or 15th gestation day and studied for the types and frequency of external and internal abnormalities as well as the incidence of resorption sites. The hamster strains exhibited significant resorption rates as well as a variety of abnormalities including edema, retardation, ventral wall defects, pericardial cavity distention, cleft palate, hydrocephalus, and heart defects. Significant but varied interstrain differences were observed for most of these indicators of mercury-induced embryotoxicity. The results of this study were compared with prior work in which the same hamster strains were exposed to cadmium or lead."


SUMMARY: In a group of 83 workers with occupational exposure to mercury vapour (mean Hg in air 0.064 mg/m³) the following biochemical tests were performed: aspartate aminotransferase (AspAt), Alanine
aminotransferase (AlAt), sorbitol dehydrogenase, alkaline phosphatase (AP), cholinesterase (ChE), prothrombin index and bilirubin level determination.

Abnormal SDH, AlAt, AspAt, AP and prothrombin values were significantly more frequent in the exposed group than in the control group. Scintigraphy of the liver gave pathological results in 57 cases. In a group of 17 subjects, exhibiting abnormalities in biochemistry or scintigraphy, the liver biopsy was performed.

In 15 cases fatty degeneration and inflammatory reaction were found in liver tissue. No history of alcoholism or hepatitis was elicited in both groups."


"Glutathione reductase is activated and the content of glutathione sulfhydryl groups is increased in the gingival tissue of patients afflicted with parodontosis. The degree of alterations depends on the degree of the development and character of the disease. Application of antioxidant vitamin therapy (vitamin A, E, and K) locally and per os normalizes the parameters under study and improves the status of the parodontium."


ABSTRACT: "The hazards associated with the use of mercury metal in the preparation of amalgam fillings in dental surgeries have been well documented.

It has always been assumed that the loss of mercury from the surface of old amalgam fillings in the mouth was negligible, or at least unmeasurable, because the vapour pressure of mercury from a solid surface of formed amalgam was insignificant. The removal of old amalgam during cavity preparation was thought to present no risk from mercury exposure as it did not produce a significantly large amount of mercury containing vapour which could be inhaled by the operator. Nixon et al. measured this in a large number of surgeries and found mercury concentration in the breathing zone of their dental operators at or near the threshold limit value (TLV) for mercury; TLV = 0.05 mg/m³ air. These values surprised us, as we had assisted in some observations, made by a Specialist Inspector of the Health and Safety Executive, of mercury levels in the breathing zone of dentists following a number of procedures, including the removal of amalgam with high speed drills. These unpublished observation indicate that high levels of mercury vapour can be released during the cutting of cavities in teeth previously filled with amalgam, when minimal aspiration and cooling water are used. This paper describes some experiments performed to clarify the position and if possible measure and quantify any exposure to mercury vapour during the preparation and restoration of cavities."
Selected quotations from results of the tests: "Removal of amalgam by a high speed bur not water cooled and with no aspiration produces a large amount of mercury containing vapour... The mercury concentration in the air was greater than the maximum reading of the instrument (i.e. 1 mg/m³ air).... The use of drills with adequate water cooling and aspiration does not produce any significant mercury vapour." (My comment: It is interesting to note that the article made no mention of the exposure to the patient, only to the dentist.)

EDITORIAL
DEBATING THE AMALGAM CONTROVERSY

On October 6 and 7, 1986, the Calgary and Edmonton District Dental Societies sponsored debates on the use of dental amalgam. Dr. Murray J. Vimy and Dr. Michael P. Ziff, President and Secretary of the International Academy of Oral Medicine and Toxicology, opposed Dr. Sheldon Newman, head of the Restorative Dentistry Department at the University of Colorado Dental School.

Dr's. Vimy and Ziff delivered dynamic scientific presentations challenging all of the widely accepted arguments exposed in the defense of continued use of dental amalgam. Dr's. Vimy and Ziff had also debated Dr. Robert Baratz, Dr. Nelson W. Rupp, and Dr. Mark Wolff at the Greater Long Island Dental Society meeting in April, 1986.

The dramatic success of the anti-amalgam presentations in these debates was based upon the presentation of valid scientific data firmly addressing the specific issues of controversy. Those of our readers who are confronted with similar debate challenges or public presentations may well profit from the following guidelines:

1. Present scientific data demonstrating the release of mercury vapor from dental amalgams as a result of function, tooth brushing and hot liquids.
2. Present information regarding the unique toxicity of mercury in the vapor form.
3. Provide documentation negating the validity of urine or blood mercury measurements as a means of determining the safety of dental amalgam.
4. Provide information on Threshold Limit Values (TLV) clearly demonstrating their lack of validity in relation to dental amalgam mercury.
5. Present documentation on the effects of mercury and dental amalgam on periodontal structures.
6. Present data demonstrating the contribution to body burden of mercury from dental amalgams. (i.e. Vimy & Lorschieder 1985)
7. Cite documentation from the ADA and FDA that places ultimate responsibility upon the practitioner for the effects of the dental materials used.
8. Above all, do your homework, stick to scientific data and have in your possession the scientific articles that form the basis
of your presentation. Do not make claims that cannot be substantiated or rely on anecdotal evidence.

Experience has shown that the pro-amalgam presentors are forced to rely on attacks on you or subjective evaluations of the information you are presenting, in lieu of a counter-scientific defense of their position. Prior experience has also shown a distortion of the facts, out-of-context quotations, unreferenced opinions, and reference to issues totally unrelated to dental amalgam.

Be prepared, know the literature, stick to the scientific facts. Remember the cardinal rule: A good offense is your best defense.

FORUM

ANNUAL I.A.O.M.T. MEETING
SCIENTIFIC SYMPOSIUM ON MERCURY TOXICITY

Date: Friday-Sunday, November 28-30, 1986.

Location: Sheraton Centre Hotel
811 7th Ave. at 52nd St.
New York, NY 10019
Phone #: 800-223-6550.

Room Rates:
Single = $130.00 per night
Double = $155.00 per night
(Specify IAOIMT Meeting.
Ask for a special Thanksgiving weekend rate if available.)

Registration: No registration fee for Friday session.
Saturday and Sunday sessions: Doctors = $250.00 (Staff = $100.00)
Advance Registrations received prior to Nov. 18, 1986 include Saturday and Sunday buffet luncheons.

Speakers: David Eggleston, D.D.S.
Mats Hanson, Ph.D. (Sweden)
Isadore Kleinberg, D.D.S., Ph.D.
Ben C. Lane, O.D.
Aaron J. Rynd, Ph.D.
Robert E. Reeves, J.D.
David C. Kennedy, D.D.S.
Murray J. Vimy, D.M.D.
Michael F. Ziff, D.D.S.

Dr. Victor Penzer will debate Dr. Robert Baratz on the subject of the safety of mercury/amalgam dental fillings on Monday, November 17, 1986. The debate will take place at the Sturdy Memorial Hospital in Attleboro, Massachusetts. Anyone desiring more information, please contact Dr. Penzer at 617-332-1234.

DR. LARS FRIBERG (WORLD MERCURY AUTHORITY) HAS RECENTLY TOLD THE PRESS IN SWEDEN THAT AMALGAM FILLINGS SHOULD NOT BE PLACED IN PREGNANT WOMEN