



# BIO-PROBE NEWSLETTER

Volume 3

April 1986

Issue 2

## **SPECIAL ARTICLE**

Detoxification. Sam Ziff

Part II. Selenium, Zinc.....2

## **REVIEWS/ABSTRACTS**

Antioxidant effects on cell-mediated immunity. Meeker et al.....11

Subclinical effects of exposure to inorganic mercury  
revealed by somatosensory-evoked potentials. Lamm O. and Pratt H....11

Diurnal variations in urinary mercury excretion. Calder et al.....11

## **EDITORIAL**

Isn't Mercury a Poison. Sam Ziff.....12

## **FORUM**

In memoriam to Dr. Carl W. Svare.....16

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The Bio-Probe Newsletter is published bi-monthly  
Editorial office located at 4401 Real Ct., Orlando, FL 32808  
Subscription price \$65.00 per year. Postage paid at Orlando

## 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 Foltz 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  $\text{Ag}^+$ ,  $\text{Hg}^{2+}$ ,  $\text{Cd}^{2+}$  and  $\text{Pb}^{2+}$  (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 (Ganther, 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