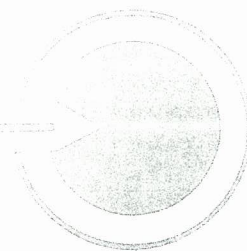


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# NEWSLETTER



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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 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

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

(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 assement 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)