ALBION®
HUMAN NUTRITION

RESEARCH NOTES

A COMPILATION OF VITAL RESEARCH UPDATES ON HUMAN NUTRITION

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A Few Words about Copper

Copper has been established as an essential nutrient. Research has revealed an extensive list of vital functions for which the body requires copper as a key component. It is essential to the formation of copper-zinc superoxide dismutase, which will be discussed in greater detail below, and as such, is involved in fighting the negative effects of free radicals, infections, and inflammatory problems.

Copper also plays a key role in the utilization of iron for the formation of hemoglobin, and thus has been increasingly employed as a component of many hematinic formulations. It aids in the utilization of tyrosine for formation of the pigments found in hair and skin. As part of several enzyme systems, copper is involved in protein metabolism, wound healing, and phospholipid synthesis. Vitamin C and Copper work together in the formation of muscle elastin. However, high Vitamin C intake will inhibit inorganic copper absorption. Research over the last 10 years has shown that not only is copper involved in bone formation, but it also helps inhibit bone resorption. Lack of copper can lead to elevated cholesterol levels; however, several studies have shown that too much free copper in the serum can cause of exacerbate atherosclerotic changes.

A recent government survey (CSFII), found that the average intake of copper in the United States was below the Estimated Safe and Adequate Daily Dietary Intake recommended for men, women, and children. Because of the important role that copper plays, and in light of the survey findings, it is safe to assume that the majority of the U.S. populace should probably supplement their diets with copper. No recommended dietary allowance (RDA) for copper has been yet established. Nevertheless, the chart below shows the Estimated Safe and Adequate Daily Dietary Intake for copper.

Estimated Safe and Adequate Daily Dietary Intake for Copper

Infants	0-6 mos.	0.4-0.6 mg
	6 mos.-1 yr	0.6-0.7 mg
Children	1-3 yrs	0.7-1.0 mg
	4-6 yrs	1.0-1.5 mg
	7-10 yrs	1.0-2.0 mg
	11+ yrs	1.5-2.5 mg
Adults		1.5-3.0 mg

Superoxide Dismutase

Superoxide dismutases are metalloenzymes which are capable of catalytically scavenging superoxide radicals throughout the body. As such, they are essential for the aerobic survival of all forms of life. There are three types of superoxide dismutase. One contains manganese, another iron, and a third copper and zinc. The copper-zinc form is composed of two identical subunits, each containing one atom of copper and one atom of zinc.

The copper participates in the catalytic activity of superoxide dismutase, whereas the zinc only plays a structural role.

Superoxide dismutase provides protection from oxygen toxicity as well as against compounds that cause exacerbation of oxygen toxicity. It also protects against ionizing radiation and the damage to tissue resulting from prolonged inflammation, such as in the case of arthritis.[1]

1. Hassan, H.M., "Superoxide dismutases," in Evered, D. and Lawrensan, G., eds., Biological Roles of Copper (Amsterdam; Excerpta Medica) 125, 1980.

Increasing SOD Activity with Copper Amino Acid Chelates

In a study conducted at Oklahoma State University, Professor Robert Kropp equally divided a group of forty heifers. Half received a daily supplement containing 226 mg of copper as copper sulfate. The other half received 199 mg of copper as an amino acid chelate from Albion Laboratories. The chelate group received 12% less copper each day. At the beginning of the study blood samples were obtained from each animal in each group, and the red blood cells assayed for SOD activity.

The animals receiving the copper amino acid chelate had a 12.1% increase in superoxide dismutase activity (even though they received 12% less copper than the sulfate group) compared to a 3.7% drop in SOD activity in the copper sulfate group (which actually received more copper in their diets.)[1]

1. Kropp, J.R., "The Role of Copper in Beef Cattle Fertility," in Ashmead, H.D., The Roles of Amino Acid Chelates in Animal Nutrition, (Park Ridge, NJ: Noyes) 154, 1993.

Intestinal Uptake of Copper

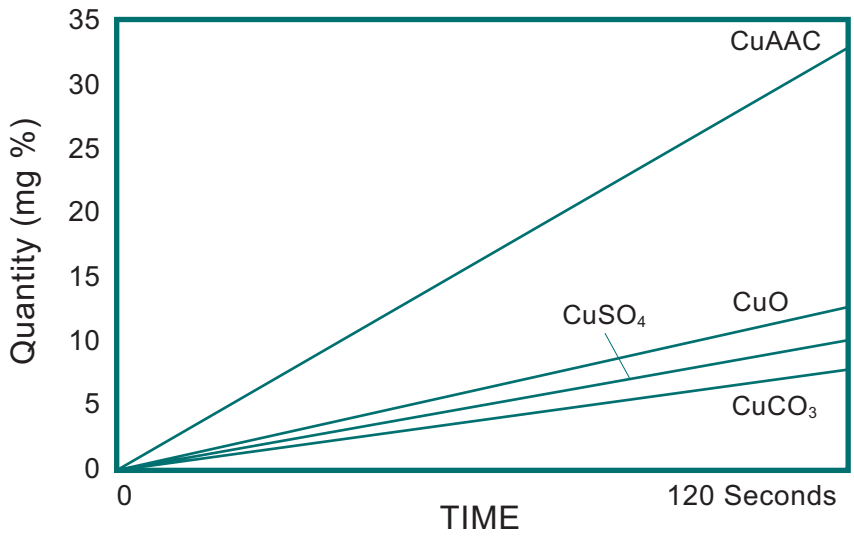
Why does Albion's copper amino acid chelate have the effectiveness it does in elevation SOD? One of the chief reasons may be its high bioavailability. In work done at Weber State University by Professor Darrell Graff, the copper amino acid chelate was absorbed more rapidly and in greater quantities into intestinal tissue (see graph below).

In vitro studies[1] have shown Albion's copper amino acid chelate was absorbed at rates that were:

- 218% higher than copper oxide
- 337% higher than copper sulfate
- 483% higher than copper carbonate

From these numbers, it can be seen why Albion's copper chelates offer significant advantages over other copper forms.

1. Graff, D.J., et al., "Absorption of Minerals Compared with Chelates from Various Protein Sources into Rat Jejunal Slices in Vitro." Paper presented at Utah Academy of Arts, Letters, and Sciences, 1970.



Cardiomyopathy of Copper Deficiency: Effect of Vitamin E Supplementation

A copper deficient diet results in reduces activity of the copper containing enzyme, superoxide dismutase. Superoxide dismutase plays an important role in the protection of tissues against lipid per oxidation. In copper deficiency, its reduced activity has been associated with the enhancement of cell damage. Vitamin E has been seen to ameliorate the cytotoxic effects of free radicals. In one study, copper deficient and copper adequate rats were fed a diet containing 62% carbohydrate as fructose. Half of the copper deficient rats were injected daily with Vitamin E. The Vitamin E treated group was found not to be protected from the lethal consequences of copper deficiency. The cardiomyopathy of copper deficiency cannot be ameliorated by Vitamin E supplementation.[1]

1. Fields, et al., J. Amer Col Nutr II, (3) 330-333, 1992.

Copper Amino Acid Chelates and SOD Activity

In the 1970's, some classic in vitro studies in Germany[1,2] showed that the administration of low molecular weight copper amino acid chelates gave rise to significant increases in superoxide dismutase activity. A chelate of copper and lysine yielded the highest increases in SOD activity. A human study conducted by Dr. DiSilvestro[3] reported in 1992, found that Albion's Copper Chelazome® induced significant elevations in erythrocyte SOD levels.

1. Joester, K., Jung, G., Weber, U., and Wester, U.: "Superoxide Dismutase Activity of Cu-Amino Acid Chelates," FEBS Letters 25(1); 25-27; September 1972.

2. Brigelius, R., Spotti, R., Bors, W., Lengfelder, E., Saran, M., and Wester, U: "Superoxide Dismutase Activity of Low Molecular Weight Cu-Chelates Studied by Pulse Radiolysis," FEBS Letters 47(1); 72-75; October 1974.

3. DiSilvestro, R., et al., "Effects of Copper Supplementation on Cerulplasmin and Copper-Zinc Superoxide Dismutase in Free Living Rheumatoid Arthritis Patients," J Amer Col Nutr II (2); 177-180, 1992.

Effects of Copper Supplementation on Ceruloplasmin and Copper-Zinc Superoxide Dismutase in Free Living Rheumatoid Arthritis Patients.

Animal studies have indicated that stress, as seen in inflammatory diseases, induces acute phase elevations in serum concentrations of the copper containing protein ceruloplasmin. Experimental inflammation in animals results in decreased copper-zinc superoxide dismutase, even when fed copper at concentrations suggested by the American Institute for Nutrition. The depression in copper-zinc SOD was absent when the animals were fed copper at levels 2.5 times that suggested by the Institute, implying that acute stress increased the amount of copper needed to maintain normal copper enzyme activities.

In a human study, the effects of copper supplementation – using Albion's patented Copper Chelazome® - were studied in a group of normal and rheumatoid arthritic patients. Blood samples were taken before and after the copper supplementation period. Copper (as Albion's Copper Chelazome®) was administered orally at a dosage of 2 mg per day for four weeks. The individuals suffering from rheumatoid arthritis showed a 21 percent increase in erythrocyte SOD levels after receiving the Copper Chelazome®. They were the individuals with the lowest pre-supplement SOD levels, and exhibited the greatest increases! The researchers felt that individuals suffering from inflammatory diseases may have an increased metabolic need for copper[1].

1. DiSilvestro, R., et al., J Amer Col Nutr II (2); 177-180, 1992.

Inhibition of Copper Absorption by Zinc

Effect of Histidine

Copper and zinc are known to interact at the intestinal mucosal level, affecting copper absorption. Researchers investigated these concepts using a duodenal-jejunal single-pass perfusion process in rats. Copper absorption was decreased with increasing zinc presence[1].

Due to the unique chemical composition of Albion's copper amino acid chelate, it has been observed over many years that the co-administration of Albion's copper amino acid chelate with other minerals that tend to interfere with the absorption of copper when both are in the inorganic salt form, or in an improperly chelated form, does not occur. Unlike other forms of copper, copper amino acid chelate from Albion is absorbed at dipeptide absorption sites, and in fact is handled in the same way as dipeptide absorption. Due to this, it does not suffer from the typical dietary interferences seen with other copper forms[2].

1. Wapnir, R.A., and Balkman, C.: "Biological Trace Element Research," 29 (3); 193-202, June 1991.

2. Coffey, R.: "The Use of Amino Chelates to Enhance the Immune System," in Ashmead, H.D., ed., The Roles of Amino Acid Chelates in Animal Nutrition (Park Ridge, NJ: Noyes) 117, 1993.

Copper Toxicity

Copper is one of the trace minerals that are considered to have a narrow margin of safety. Although it is extremely important for a number of biological functions, its excess has been implicated in a wide variety of mental and physical problems. Research has shown Albion's copper amino acid chelate to be less toxic than typical copper salts or complexes. Professor Austen Larson, at the University of Utah, conducted LD50* studies on Albion's copper amino acid chelate and copper sulfate. The results of the testing showed that copper sulfate had an LD50 of 300 mg/kg (117 mg copper/kg), while Albion's copper amino acid chelate had an LD50 of 1300 mg/kg (335 mg copper/kg). It took over three times more copper from the amino acid chelate to cause toxicity than it did from the inorganic metal salt. That is an extremely large difference in acute toxicity results for a substance with a supposedly narrow safety margin[1].

**LD50 is the dose of a substance needed to kill 50% of the animals in the study and is normally expressed in mg/kg animal body weight.*

1. Larson, A.E.: "L.D. 50 Studies with Chelated Minerals," in Ashmead, H.D., ed., Chelated Mineral Nutrition in Plants, Animal and Man (Springfield: Charles, C. Thomas) 163, 1982.

Albion Human Nutrition

100 Maple Park Blvd., Suite 110
St. Clair Shores, Michigan 48081 USA
[P] 586•774•9055 | [TF] 800•222•0733
[F] 586•774•8838
[e] info@AlbionMinerals.com