ALBION® MINERALS

Human Nutrition

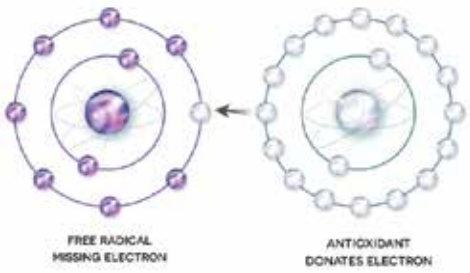
RESEARCH NOTES

A COMPILATION OF VITAL RESEARCH UPDATES ON HUMAN NUTRITION

September, 2017 Volume 26, No 3

Trace Minerals' Fight Against Free Radicals

Unchecked, free radicals are known to give rise to a number of physical disorders that have been referred to as Free Radical Pathologies. What is a free radical? Free radicals are unstable toxic byproducts of oxygen metabolism. The main targets of free radicals are the polyunsaturated fatty acids (PUFAs), which are the main components of the cell membranes. Free radicals can also cause extensive damage to the body's cells and tissues through the process of oxidative stress. While they cause damage to PUFAs through a process called lipid peroxidation. Now, we have substantial evidence implicating oxygen free radicals as agents of inflammation and tissue destruction in many painful disorders. Free radicals have been shown to be causative factors behind a variety of diseases, as well as aging itself. The following diseases have resulted from free radical damage: ischemic heart disease, atherosclerosis, cataracts, arthritis, retinopathy, emphysema, and damaging DNA leading to cancer. Free radicals can make it more difficult to fight off infection. Genes in which DNA is damaged by free radicals make ill formed proteins, which cause a decline in cell function, a decline in the ability to produce energy, increased incidence of disease, and finally aging and death. There are certain vitamins and minerals that can help fight against free radicals. These substances are known as antioxidants.



ANTIOXIDANT TRACE MINERAL FINDINGS

Copper Finding – acts as an antioxidant

AA-DiSilvestro RA, et al. J Trace Elements Med Biol .02.03

Marginal copper deficiency has been proposed to occur frequently, but the benefits of correction remain largely uncharacterized. Two benefits could be reduced oxidant stress and better crosslinking of collagen in bone. Copper intake was increased in 8 female university students by supplementation with copper glycinate (2 mg of copper/day) for 8 weeks. Supplementation improved copper status based on serum activity of 2 copper enzymes, ceruloplasmin and diamine oxidase (9% and 75% mean increase, respectively). No effect was seen for erythrocyte copper-zinc superoxide dismutase. Supplementation produced a 39% mean decrease in plasma F2α-isoprostanes (a marker of oxidative stress), and gave a 62% increase in the urine ratio of collagen crosslinks to a measure of total collagen. None of the supplementation effects were duplicated for 8 women given placebo. In conclusion, this pilot study found that in young adult women, increased copper intake can alter biochemical parameters relevant to copper function.

Zinc Finding – acts as an antioxidant

AA-Z. Suntrees and Ed M.K. Lui: Chemico-Biological Interactions, Vol. 162 Iss.1:2006

This study was concerned with the role of zinc (Zn) and zinc Metallothionein (Zn-MT) in oxidative stress. Hydrogen peroxide-induced oxidative injury and examined in Ehrlich ascites tumour cells isolated from control host mice, mice pretreated with 10mg/kg $ZnSO_4$ to increase cellular Zn/Zn-MT levels, and mice exposed to Zn deficient diet

to reduce the cellular Zn/Zn-MT levels. The results of the present study showed that Ehrlich cells with seven-fold differences in Zn-MT concentrations could be obtained by manipulating the Zn status of host mice and that high Zn and Zn-MT levels can make Ehrlich cells more resistant to H2O2-induced oxidative injury (cell viability, lipid peroxidation, [Ca2+]i) while cells with reduced Zn/Zn-MT levels were more susceptible to this treatment. H2O2 treatment resulted in oxidation of MT thiolate groups and loss of its metal binding capacity with translocation of Zn released from oxidized MT to other cellular sites. Preincubation of Ehrlich cells with $ZnSO_4$ in vitro also conferred some degree of resistance of H2O2 toxicity, suggesting the inherent antioxidative property of Zn ions. These data suggest that Zn-MT can be considered as an antioxidant by virtue of its thiolate groups and its Zn ions that are released in the presence of oxidative stress.

Manganese finding – acts as antioxidant

According to Oregon State's Linus Pauling Institute's Micronutrient Information Center (on-line publication), Manganese superoxide dismutase (MnSOD) is the principal antioxidant enzyme in the mitochondria. Because mitochondria consume over 90% of the oxygen used by cells, they are especially vulnerable to oxidative stress. The superoxide radical is one of the reactive oxygen species produced in mitochondria during ATP synthesis. MnSOD catalyzes the conversion of superoxide radicals to hydrogen peroxide, which are then reduced to water by other antioxidant enzymes.

Manganese Finding – antioxidant (pancreas protector)

AA-Keen,C., et al. Present Knowledge in Nutrition Ed.7, 1996 pp334-341

Although manganese has received a paucity of attention in diabetes research, defects in carbohydrate metabolism have been reported in manganese deficiency. Animal studies have shown that manganese deficiency results in severe pancreatic abnormalities, leading to aplasia and hypoplasia of all pancreatic cell components. Glucose challenge to manganese deficient animals has been followed by a diabetic-type glucose tolerance curve. Manganese supplementation completely reverses the abnormalities in pancreas and glucose tolerance seen in these animals. Additional animal research has shown that manganese deficiency results in depressed pancreatic insulin synthesis, enhanced intracellular insulin degradation, as well as depression in the insulin secretory process. Manganese production of MnSOD may protect pancreatic Beta-cells from destruction by high concentration of superoxide radicals. Manganese deficiency has been linked to a reduction in the number of glucose transporters in adipose tissue.

Selenium Finding – Antioxidant

AA- Baraboi, VA Shestakova, EN. Ukr Biokhim Zn [1999] 2004 Jan-Feb;76[1]:23-32

Selenium is essential trace element, Sulphur analogue with high chemical activity, component of some selenoproteins and enzymes: glutathione peroxidase and other peroxidases, blood and tissue proteins. As to their biological reaction mechanism, selenium and its compounds are antioxidants. Selenium is an active immunomodulatory, a much more potent antioxidant than vitamins E, C, and A, beta carotene, but much more toxic. It takes part in thyroxine conversion to triiodothyronine in thyroid hormone biosynthesis. As sperm antioxidant, selenium protected its motility and fertility. Selenium is a serious factor of biological antioxidant protection of vascular endothelium, of low density lipoproteins, protector of DNA, and chromosomes. As a food component, selenium is an exceptional agent of protection from atherosclerosis, coronary ischemic disease and cancer. Some hydrobionts: liver, kidney, meal, corn and garlic, onion, cabbage, broccoli are dietary products of high selenium content.

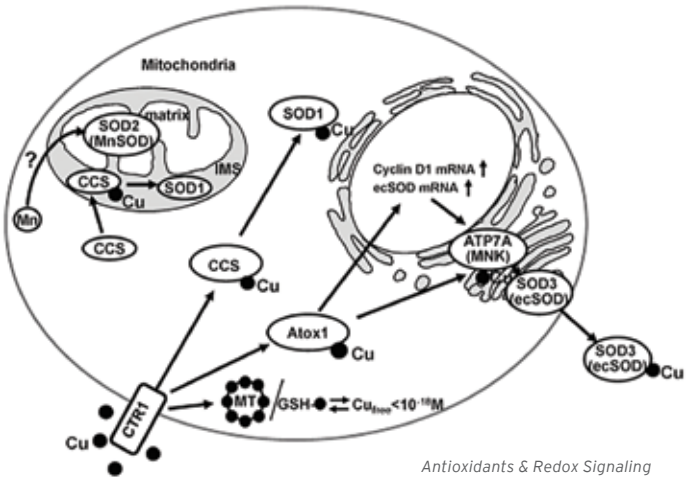
TRACE ELEMENTS - KEY FACTORS AGAINST FREE RADICALS

As one can see from the findings or conclusions of these abstracts on copper, zinc, manganese and selenium – these trace elements play large roles in protecting the body from the potential health hazards caused by free radicals. Each of these can act as antioxidants on their own, however, copper, zinc, and manganese can be even more effective when they are incorporated into their associated Superoxide Dismutase compounds. As reviewed (*Fukai, T. [Antioxidants & Redox Signaling 2011, 15, 6]*), it has been seen that excessive reactive oxygen species, especially the superoxide anion (O_2^-), play major roles in

the pathogenesis of many cardiovascular diseases, including hypertension and atherosclerosis. Superoxide dismutases (SODs) are the main antioxidant defense systems against the superoxide anions. The SODs consist of three isoforms of SOD: the cytoplasmic CU/Zn SOD (SOD1), the mitochondrial MnSOD (SOD2), and the extracellular Cu/Zn SOD (SOD3). All of these require a catalytic metal for their activation, either copper or manganese. Evidence has indicated that in each subcellular location, SODs stimulate the conversion of O_2^- into hydrogen peroxide and oxygen, which are then converted to water and oxygen via the action of catalase or glutathione peroxidase (see figure 2). The SODs also play a critical role in inhibiting oxidative deactivation of nitric oxide, thus preventing peroxynitrite formation as well as endothelial and mitochondrial dysfunction. There are other enzymatic sources of reactive oxygen species (ROS), all of which can cause damage, but the inadequate removal of ROS, especially superoxide anion, leads to oxidative stress, which are implicated in the pathogenesis of many cardiovascular diseases, including hypercholesterolemia, atherosclerosis, hypertension, diabetes, and heart failure.

Selenium supplementation has been shown to give rise to an increase in glutathione peroxidase (GSH-Px), which plays a major role in the body's antioxidant defense, and also helps maintain proper glucose homeostasis (*Biol Trace Elem Res. [2007] 118:217-226*). GSH-Px functions to scavenge and inactivate hydrogen and lipid peroxides, and thereby protect against oxidative stress. It is one of the most important antioxidant enzymes in the human body.

Figure 2.



Mn: Manganese
MT: Metallothionein
IMS: Intermembrane space

Antioxidants & Redox Signaling
Fukai, T. 2011, Sep 15 15(6): 1583-11606

ADDITIONAL FINDING

It has been clearly demonstrated that homocysteine has negative impacts on health. It is known to promote the production of free radicals, resulting in oxidative stress. As seen in this newsletter, oxidative stress from free radicals can give rise to a number of health problems or disease states. Choline, a vitamin-like essential nutrient and a methyl donor, is involved in a wide variety of physiological processes, including the normal metabolism and transport of lipids, methylation reactions, and neurotransmitter synthesis. Its deficiency can cause muscle damage and abnormal fat accumulation in the liver – even non-alcoholic fatty liver disease. In addition, choline has been seen to directly reduce the homocysteine in the human body. In doing so, choline has an antioxidant-like impact on the body, by decreasing the oxidative stress from homocysteine.

CONCLUDING REMARKS

As stated in this newsletter, the trace minerals Copper, Zinc, Manganese, and Selenium all have roles as antioxidants. As such, they help the body fight against the health dangers that can come from free radical damage. In addition, Choline helps fight against the oxidative stress imposed by homocysteine, and therefore plays a related antioxidant role.

Balchem® offers excellent forms of all of these trace minerals, as well as an excellent form of choline in the Vita-Choline®. The trace minerals offered by Balchem are:

- Copper Bisglycinate Chelate
- Zinc Bisglycinate Chelate (also in a Taste Free form)
- Manganese Bisglycinate Chelate
- Selenium Glycinate Complex

If you are looking for the best in trace minerals and choline forms, look no further than Balchem!

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67 South Main Street, Suite 100
Layton, UT 84041

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