USANA Technical Bulletin

Copper

Technical Background

- Copper is essential for enzymes involved with metabolic reactions that consume oxygen or oxygen radicals.[1] Copper works as an antioxidant with the copper-requiring enzyme superoxide dismutase, which protects cell membranes from free radical damage. [2]
- Copper is involved in many essential processes in the body, including immune function, bone formation, red- and white-blood-cell maturation, lipid metabolism, iron transport, myocardial contraction, and neurological development.[3]
- Oxidation of ferrous iron to ferric iron (a reaction needed for hemoglobin synthesis) requires copper. Lysyl oxidase, another copper-requiring enzyme, helps to synthesize collagen and heal wounds.
- Copper is also needed in many reactions related to cellular respiration and release of energy. For example, cytochrome C oxidase is a copper-containing enzyme in the electron transport chain.
- Copper is also essential for brain function. It acts as a cofactor for dopamine-beta-hydroxylase and other proteins necessary for normal brain function, and is distributed throughout the entire central nervous sytem.[4]
- Because copper is involved in so many bodily functions, symptoms of copper deficiency are myriad, including anemia, neutropenia, and bone marrow abnormalities in children. All of these can be addressed with supplementation.[5] One study associated copper deficiency with congenital cataracts, severe muscular hypotonia, sensorineural hearing loss, and developmental delay. With supplementation, these symptoms disappeared.[6]

Sources and Recommended Intake

- The richest sources of copper are organ meats, shellfish, nuts, seeds, legumes, and water.
- The recommended dietary intake is 1.5 – 3.0 mg/day for an adult; children between the ages of 4 and 11 require 1.0 to 2.5 mg per day.[7]

Abstracts

Salviati L, Hernandez-Rosa E, Walker WF, Sacconi S, DiMauro S, Schon EA, Davidson MM. Copper supplementation restores cytochrome c oxidase activity in cultured cells from patients with SCO2 mutations. Biochem J. 2002 Apr 15;363(Pt 2):321-7. Human SCO2 is a nuclear-encoded Cu-binding protein, presumed to be responsible for the insertion of Cu into the mitochondrial cytochrome c oxidase (COX) holoenzyme. Mutations in SCO2 are associated with cardioencephalomyopathy and COX deficiency. Studies in yeast and bacteria have shown that Cu supplementation can restore COX activity in cells harbouring mutations in genes involving Cu transport. Therefore we investigated whether Cu supplementation could restore COX activity in cultured cells from patients with SCO2 mutations. Our data demonstrate that the COX deficiency observed in fibroblasts, myoblasts and

myotubes from patients with SCO2 mutations can be restored to almost normal levels by the addition of CuCl(2) to the growth medium.

References

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[4] Barnes N, Tsivkovskii R, Tsivkovskaia N, Lutsenko S. The copper-transporting ATPases, menkes and wilson disease proteins, have distinct roles in adult and developing cerebellum. J Biol Chem. 2005 Mar 11;280(10):9640-5. Epub 2005 Jan 5.

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