Copper

Copper is necessary for the proper functioning of every cell in the body, the main function is in the area of hematopoiesis (ceruloplasmin), ceruloplasmin oxidase activity in the plasma is essential for the oxidation of Fe^{2+} to Fe^{3+} → Fe mobilization and incorporation into heme.

- It is part of respiratory and antioxidant enzymes;
- is important in the formation of hair and pigments;
- it is important for the correct course of immune reactions;
- lysyl oxidase is required for collagen and elastin cross-linking.

Copper is resorbed, bound to albumin and incorporated into **ceruloplasmin** in the liver. Main excretion – bile. Nutritional deficit is rare, rather as part of malnutrition. *The function of copper is closely related to the function of zinc (optimal ratio Zn:Cu = 7:1, when both trace elements act synergistically)*. Sources of copper are eggs, meat, legumes. The recommended daily dose of copper is **2-2.5 mg**.

Deficiency symptoms

Acquired deficit

- Microcytic, hypochromic anemia, leukopenia, osteoporosis;
- anemia does not respond to administration of Fe, decreased ceruloplasmin;
- more sensitive indicator – decrease in Zn, Cu-SOD activity in erythrocytes;
- immune disorders;
- hair and nail growth disorders.

Congenital deficiency

Menkes disease (trichopoliodystrophia, "kinky-hair" syndrome) is an X-linked hereditary disease caused by a mutation of the gene encoding **Cu^{2+}-transporting ATPase**. This leads to the inability of intestinal mucosal cells to transport Cu across the serous membrane into the blood circulation. It manifests itself in male infants in the first few weeks; those with disabilities usually die within three years of birth. The disease is characterized by severely delayed mental development and growth, a peculiar appearance of hair (tiny curls on short fine gray hair - "kinky" or "steely" hair), scorbutic bone changes, cerebral gliosis with cystic degeneration, temperature instability and arterial tortuosity (twisting). Clinical symptoms are the result of reduced activity of Cu-containing enzymes such as ceruloplasmin, cytochrome c oxidase, superoxide dismutase, lysyl oxidase, dopamine-β-hydroxylase (DBH). In the biochemical findings, there is a significant decrease in the level of copper in the plasma, a decrease in S-ceruloplasmin, a decrease in the content of Cu in the tissues and in the hair, with the exception of the duodenal mucosa, which contains an abnormally increased amount of Cu. Anemia occurs, usually hypochromic and normocytic, is neutropenia; also osteoporosis and bone fractures, irregularities in the formation of the metaphysis. In the pathogenetic mechanism of the disease, the main role is probably played by the reduced activity of Cu-metalloenzymes: lysyl oxidase (impaired biosynthesis of collagen and elastin leads to changes in the bones and vascular wall), and also cytochrome c-oxidase, dopamine-β-hydroxylase and superoxide dismutase, which leads to degeneration neurons and demyelination of brain tissue. Parenteral administration of Cu (immediately after birth) can prevent severe changes. A decrease in DBH activity also causes a different concentration of catecholamines in plasma and cerebrospinal fluid: a high level of DOPA, DOPAC and dopamine, a low value of dihydroxyphenylglycol (DHPG). An increased index of DOPA/DHPG and DOPAC/DHPG is a suitable diagnostic marker of Menkes disease.

Toxicity and disease

- Copper is relatively toxic, inhaled - "metal fever" - like Zn;
- in the serum – icterus, damage to the liver, kidneys, often fatal;
- **Wilson's disease**.

  Limitation of Cu incorporation into ceruloplasmin and limited hepatic excretion.
  Accumulation of copper in the liver, unbound copper rises, more goes out through the kidneys, is deposited - in the cornea (Kayser-Fleischer ring), in the brain (mainly the basal ganglia).
  Symptoms resemble cirrhosis, there is also rigidity, tremor.

🔎 *For more information see Wilson's disease.*

Links

Related articles

- Trace elements
- Wilson's disease

Source

- BENEŠ, Jiří. *Studijní materiály* [online]. ©2007. [cit. 2009]. <http://www.jirben.wz.cz/>.

Bibliography

- SCHNEIDERKA, Petr, et al. *Kapitoly z klinické biochemie.* 2. edition. Karolinum, 2004. ISBN 80-246-0678-X.
- MASOPUST, Jaroslav – PRŮŠA, Richard. *Patobiochemie metabolických drah.* 2. edition. Univerzita Karlova, 2004. 208 pp. pp. 190−192.