The role of copper in biological functions and its toxic effects on the body

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Cupric ion (Cu2+) is the predominant oxidation state of copper in biological systems. On consumption, the acidic conditions in the stomach release these ions from the food particles and are chelated with amino acids to increase solubility. Copper enters the bloodstream using the copper transport protein (ATP7B) that links it to the plasma protein ceruloplasmin. While ceruloplasmin transports copper to the tissues, ATP7B secretes copper into bile. Copper is a cofactor for several enzymes, mainly those performing oxidative or reductive activity, and is essential for enzymes responsible for aerobic metabolism. It is also an electron transport intermediate in redox reactions. Cytochrome c oxidase (Cox) enzyme which is involved in respiration processes, mainly contains three subunits and requires copper as a cofactor. Subunit I contains two copper atoms that transfer electrons from cytochrome c to the haem centre. Subunit II contains one copper atom and reduces oxygen. In addition, copper has a critical antioxidant function and provides defence against free radicals. Superoxide free radicals produced during metabolism damage the cell membrane, proteins and DNA, and there is a need to eliminate them. The Cu/Zn superoxide dismutase 1 (SOD1) contains copper in its binding site and gives the enzyme its functionality of binding to the substrate molecule. It acts as a catalyst in disproportionating superoxide anions (O2-). Copper itself solely demonstrates antioxidant activity.

Lysyl oxidase, another important copper enzyme, causes oxidative deamination of the lysine side chains, initiating a cascade of spontaneous reactions, eventually cross-linking newly formed collagen or elastin fibres. In addition, copper helps in producing myelin, haemoglobin and thyroxine. It is involved in angiogenesis and is a key component of tyrosinase (converts tyrosine to melanin) and dopamine ß-hydroxylase (converts dopamine to norepinephrine). Blood clotting factors (V and VIII) also contain copper atoms in each molecule. Further, copper performs several other functions. & nbsp: Copper ingested through the diet does not produce toxic effects. However, toxicity is induced by consuming unusually high amounts of copper salts. Acute toxicity effects include gastrointestinal mucosal ulcerations, bleeding, headache, jaundice, etc. Postpartum depression and anxiety are chronic effects of copper ingestion, which is caused mainly due to diminishing histamine levels through over-methylation. The mutations in ATP7B impairs copper transport and causes Wilson's disease, characterised by excess accumulation of copper in the liver and brain. A few other disorders related to copper metabolism include Indian childhood cirrhosis, caused by an extremely high concentration of copper within the liver, eventually causing death. The increased levels of copper also suggest the role it plays in cancer. The angiogenesis-inducing property of copper is exploited by cancer cells as angiogenesis is a hallmark of cancer. Copper generates oxygen radicals using a Fenton-type reaction and is also involved in arthritis and Menkes syndrome. Thus, consuming copper in the right amounts can be beneficial, while too much can be harmful to the body.

Keywords: Copper, Copper toxicity, Biological function, Copper deficiency, Antioxidant, Enzyme

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