Copper and its Role in Human Health: A Review

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Abstract: Copper is one of a tiny number of metallic elements that are required for human health. These minerals, as well as amino acids, fatty acids and vitamins, are necessary for proper metabolic activities. However, because the human body cannot produce copper, the human diet must provide consistent quantities for absorption. Copper has the following oxidation states: Copper(0) is a metallic copper with a temperature of Cu°. • Cu(I): cuprous ion; at neutral pH, Cu+ is unstable and is oxidized by air to Cu²⁺. • Copper(II): cupric ion; in alkaline pH water, stable Cu²⁺ generates Cu(OH)₂. Although, our bodies only require a little quantity of copper (the RDA for people in the United States is 0.9 mg), its importance to human health is obvious and it is just as important as calcium, iron and zinc. Copper which is present in our bodies from conception, aids in the development of an infant’s cardiac, skeletal and neurological systems as well as arteries and blood vessels. As we become older, copper continues to play an important part in keeping our hair and skin healthy as well as mending and maintaining connective tissue in our hearts and arteries. It also helps cells absorb and utilise iron as well as allowing them to use the energy found in carbs, proteins and lipids.

INTRODUCTION
Copper is a trace element (sometimes known as a micronutrient) that is essential for the health of plants, animals and humans[1]. It is also necessary for aerobic (oxygen-requiring) bacteria to operate normally. Copper is present in a range of proteins and metalloenzymes that conduct essential metabolic activities, Bone, cartilage and connective tissue are all examples of connective tissue and it is necessary for maintenance and development, as well as cholesterol and glucose metabolism[2]. Copper improves the ability of mechanism of defense to combat infections, repair damaged tissues and speed up recovery time. Copper can also aid in the neutralization of "Radical libertarians" are molecules that can harm cells. The importance of copper was first highlighted in 1928, when it was discovered that human fetuses, infants and children require copper in order to grow and develop correctly[3]. Copper levels in human milk are low, During the breast-feeding period, the neonates liver stores decrease soon after delivery, providing copper to the fast-growing body. These supplies are required for metabolic processes such as
melanin pigment, cellular respiration and connective tissue synthesis in newborns, as well as gene expression, free radical defense, iron metabolism and proper heart and immune system function. While permanent life long As processes mature, babies have unique biochemical systems for controlling copper levels in their body. If not enough copper is consumed in a short period of time, copper stores in the liver will be depleted. A copper health deficit might emerge if this depletion continues. An excess situation can occur if too much copper is consumed. Both shortage and excess of these nutrients can cause tissue damage and illness. Copper’s transport and metabolism in the act of living beings are now hot topics. The transfer of extracellular copper over the cell membrane and into the cell via. specialized transporters is referred to as copper transport at the cellular level. Other proteins in the circulatory system include albumin, ceruloplasmin and others. carry copper throughout the body. Ceruloplasmin binds the majority of copper in the blood (or serum copper their copper status, hormone cycle and season all have a role and copper status, in different people, the amount of ceruloplasmin-bound copper varies from 70-95%, among other factors. Metallochaperones are specialized proteins that transport intracellular copper to copper-requiring enzyme production sites and organelles. Copper is transported into sub-cellular compartments by another group of these transporters. Copper may be released from the cell through a variety of ways. Copper is not absorbed by the stomach or small intestine because it is insoluble in stomach acids. Many meals may also contain indigestible fiber that binds to coppe. Copper absorption can be severely hampered by a zinc deficit. Zinc deficiency can reduce copper absorption substantially. Copper absorption can be hampered by high doses of vitamin C or iron, reminding us that micronutrients must be eaten in a balanced ratio. This is one of the reasons why consuming large amounts of any one vitamin is not recommended. Even if they eat copper-rich meals, those with persistent digestive issues may be unable to absorb enough quantities of copper. Copper transporters that can transfer copper across cell membranes have been found. Other copper transporters in the intestine may exist. Ctr1 may accelerate copper absorption in the intestine. Ctr1 catalyzes the transfer of Cu+1 through the cell membrane and is found in all cell types studied thus far, including enterocytes. Copper generated by intestinal cells goes to the portal blood, where it binds to glutathione, albumin and amino acids. Evidence also exists for transcuprein, a tiny protein that has a transport role. Copper transport in the serum might be mediated by any or all of these copper-binding molecules. The liver absorbs the majority of copper from the portal circulation. Copper is either incorporated into copper-required proteins or removed from them or released into the bloodstream once it reaches the liver. The majority of copper discharged via means of the liver (70-95%) is absorbed into ceruloplasmin, the primary copper transporter in blood. Ceruloplasmin transports copper to extracellular tissues. The bile excretes albumin and amino acids, or albumin and amino acids are discharged in the urine. Extrahepatic copper is kept under homeostatic control by the liver by controlling copper release. Copper is excreted mostly through the bile which is necessary for maintaining liver copper levels. As new scientific evidence becomes available, these criteria are modified and updated on a regular basis. Standards range from country to country and organization to organization. The World Health Organization recommends a daily consumption of 1.3 mg as the minimum tolerable level. For the vast majority of the population, these values are deemed acceptable and safe. Copper is an important trace mineral that the human body cannot produce. It must be obtained through dietary means. Almost majority of the copper eaten by humans comes through food. Copper tubing used to transport drinking water can be a source of dietary copper in many parts of the world. Following that, on the interior of copper tubes, a protective layer develops which assists in the prevention of leaching. Copper intake that exceeds the World Health Organization’s recommendations, on the other hand, can be dangerous. The most prevalent cause of acute copper poisoning is accidental ingestion. The symptoms go away after the high copper dietary source is no longer eaten. According to the International Program on Chemical Safety, “a lack of copper consumption poses a larger risk of health consequences than an excess of copper intake” which is connected with the World Health Organization warned in 1996. Recent multi-route exposure surveys have backed up this finding. On the degree of insufficiency in the United States, there are contradictory accounts. According to one study, around 25% of adolescents, adults and persons over 65 do not consume enough copper to satisfy The RDA is a group of people that work together to (Recommended Daily Allowance) According to another source, a government assessment of food intake found that typical food and beverage Women and males over the age of 19 consumed 1.11 and 1.54 mg per day, respectively. Only 3% of males drank less than the estimated average requirement, whereas 10% of females
did copper deficiency is linked to osteoporosis, osteoarthritis, cardiovascular disease, rheumatoid arthritis chronic illnesses include cancers of the colon as well as bone, connective tissue, heart and blood vessel disorders. The immune system and the neurological system are closely connected[27, 28]. Copper deficiency interferes with the function of other antioxidant-active cellular components including iron, selenium and glutathione and so contributes to disease[29] defined by high levels of oxidative stress. A minor, or ‘mild’, Copper deficiency which is expected to be more prevalent than originally believed, can have a modest negative impact on human health[30, 31]. Vegetarians may have had lower copper intake, since, they ate plant diets with low copper bioavailability[32]. Low birth weights, muscular weakness and neurological abnormalities are more common in babies and fetuses born to severely copper deficient mothers. Anemia, bone disease abnormalities. Anemia manifests itself in these populations as delayed development, weight increase, frequent infections (colds, flu, pneumonia), impaired motor coordination and exhaustion[33], is the topic of a lot of current study. Copper excess In a typical situation, variables differ. people vs. those with greater vulnerability to harmful consequences and those with uncommon hereditary disorders, according to research[34]. As a result, health groups have made comments that may be misleading to the uninitiated. According to a report published by the United States Institute of Medicine. Copper consumption is significantly lower than recommended for a large majority of the population. In its study copper in drinking water, the National Research Council of the United States[35]. Copper toxicity is a problem among vulnerable groups, according to the report and further Copper-sensitive populations. Copper in excess must be identified and characterized via. research. consumption can result in stomach discomfort, nausea and diarrhea, as well as tissue harm and illness. Copper’s oxidation potential may be to blame for part of its toxicity in situations of excessive intake. Copper has been linked to oxidative stress in biological systems. at high doses, including lipid and other macromolecule peroxidation[36]. While the etiology and course of Alzheimer’s disease remain unknown research suggests that iron[37], aluminum and copper[38, 39] build up in Alzheimer’s patients’ brains, among other things. However, it is unclear if this build-up is there a reason or a symptom of the illness. Copper has already passed over the last two decades, researchers have looked at see if it is a causal or preventative factor in Alzheimer’s disease. Copper, for instance, possibly by disrupting a molecule that serves a purpose. in promoting protein clump development in Alzheimer’s disease brains, as a possible causal factor or a symptom of a metal homeostasis, removes the detrimental accumulation of amyloid beta (A) in the brain. disruption. potentially by causing damage to a molecule that eliminates harmful amyloid beta (A) accumulation in the brain[40]. There is a link between Alzheimer’s disease and a copper and iron-rich diet, as well as fat that has been saturated[41]. On the other side, research show that copper may play a helpful function in curing Alzheimer’s disease rather than causing it[42]. In humans, copper poisoning most commonly affects the liver. Other organs targeted include bone, the central nervous system and the immunological system[10]. When consumed in excess, copper can produce toxicity when it combines with additional nutrition[33]. Anemia is caused by copper toxicity which interferes with iron absorption, as well as metabolism and/or transport[4, 10]. nausea, vomiting and stomach discomfort are frequent symptoms of acute gastrointestinal discomfort after consuming drinking water with high copper concentrations (usually >3-6 mg L⁻¹). When the copper concentration in drinking water is lowered, the symptoms go away. There is no evidence that prolonged copper exposure in humans has systemic consequences other than liver damage. For three years, a young adult guy with no known family history of copper sensitivity took 30-60 mg of copper per day as a mineral supplement was diagnosed with chronic copper toxicity and liver failure. Individuals living in homes in the United States with there were no negative health consequences from drinking tap water with >3 mg L⁻¹ copper[44, 45]. Copper supplementation had no impact on serum liver enzymes, oxidative stress biomarkers, in healthy young human volunteers, following receiving daily copper dosages of 6-10 mg d⁻¹ for up to 12 weeks, or other biochemical endpoints[46-48]. The exposed infant group had somewhat greater serum ceruloplasmin. At 9 months, the homeostatic response was more mature than the controls, indicating homeostatic adaptation and/or maturation[49]. John Menkes initially reported Menkes disease, a hereditary disorder characterized by copper deficiency, in 1962. It’s an extremely rare X-linked disease that affects around 1/200,000 live births with men accounting for the vast majority of cases[46]. Menkes disease sufferer’s livers are unable to absorb copper which is required for survival. The majority of those who are affected die before they reach the age of ten, however, some people have lived into their teens and early twenties[50]. The Menkes gene produces a protein that transports copper across the mucosa of the Gastrointestinal Tract (GIT) and the blood-brain barrier[39, 51]. Copper becomes stuck in the lining of the
small intestine due to mutations in the copper ATPase gene. As a result, copper cannot be pumped into the circulation from intestinal cells where it might be transported to the liver and then to the rest of the body[52]. Despite adequate copper intake, the illness mimics a severe nutritional copper shortage. Early detection and treatment which includes daily visits, are recommended. Intraperitoneal and intrathecal injections of copper histidine into the central nervous system can avoid certain serious neurological diseases and extend life expectancy. Patients with Menkes disease, on the other hand, continue to have aberrant bone and connective-tissue abnormalities, as well as mild to severe mental impairment[53]. Menkes disease is typically deadly, even when diagnosed and treated early.

Wilson's disease is a rare autosomal recessive copper transport genetic syndrome that affects chromosome 13 that produces an accumulation of copper in the liver[54, 55]. This causes liver damage, as well as other signs and symptoms The illness is now curable. Mutations in a protein that transfers copper from the liver to the bile for elimination cause Wilson's disease[56]. The illness is caused by mutations that affect the activity of the Wilson copper ATPase and it causes inadequate copper incorporation reduced biliary copper excretion and conversion to ceruloplasmin. Copper toxicosis is caused by excess copper buildup which occurs mostly in the liver and brain but also in the kidneys, eyes and other organs to a lesser extent. Copper's function in angiogenesis has been studied in relation to many forms of malignancies[57]. Tetrathiomolybdate in pilot and clinical research. Copper, a trace metal has been discovered to promote tumor development[58, 59]. Tumors contain significant amounts of copper, according to data from animal models. In the meanwhile, excess copper has been discovered in several human malignancies[60, 61]. Therapeutic methods that target copper in the tumor have recently been proposed. Copper complexes would form in malignancies at a rather high level after treatment of a particular copper chelator. Tumor cells were destroyed by copper complexes which are frequently poisonous to cells but normal cells throughout the body survived at lower copper levels[62].

REFERENCES


