Copper

Sources and Physiological Functions

Copper is a chemical element that occurs naturally in rocks, soil, water, and at low levels in air. It is mined and recovered from scrap materials through smelting. Copper and its compounds are used in many products, including electrical wire, roofing, plumbing, alloys, jewelry, herbicides, pesticides, and antimicrobial products (U.S. Agency for Toxic Substances and Disease Registry 2024). Copper is the primary metal in brass and bronze. Copper sulfate pentahydrate is sometimes added to surface water for the control of algae (WHO 2022). Copper is an essential micronutrient for plants and animals, so is found in foods and supplements. People can be exposed to unusually high levels of copper by consuming acidic food or liquid that has been in prolonged contact with copper (e.g., from plumbing or malfunctioning soda fountains) (National Research Council 2000).

Human growth, lung elasticity, vascular function, neovascularization, neuroendocrine function, and iron metabolism depend on adequate copper intake. The body will adjust the absorption of dietary copper in the gastrointestinal tract to maintain copper homeostasis. ATP7A and ATP7B regulate copper metabolism, and almost all of the body's copper is bound to proteins (Johnson 2023). Copper is necessary for some enzymes involved in aerobic metabolism, including cytochrome c oxidase in the mitochondria, lysyl oxidase in connective tissue, dopamine monooxygenase in the brain, and ceruloplasmin (National Research Council 2000). It is a cofactor for apo-copper-zinc superoxide dismutase, protecting against free-radical damage. Copper is also involved in neurotransmitter biosynthesis (dopamine beta-hydroxylase), peptide hormone maturation (peptidyl-glycine alpha-amidating monooxygenase), pigmentation (tyrosinase), keratinization (sulfhydryl oxidase), and iron homeostasis (ceruloplasmin and hephaestin) (Møller 2022).

Health Effects

Dietary deficiency of copper is rare (Collins 2020). Abnormal genetic mechanisms, such as Menkes disease (mutant X-linked genes in male infants), can control copper metabolism (Johnson 2023; Prohaska 2012). In Menkes disease, intestinal copper absorption is blocked. Deficiency results in severe outcomes, including progressive nerve degeneration, because of reduced cuproenzyme activity in neurons (Collins 2020). Copper deficiency may increase the risk of infections and be associated with connective tissue disorders, osteoporosis, myelopathy, neuropathy (with

symptoms similar to vitamin B12 deficiency), hypochromic anemia not responsive to iron supplements, and increases in fat storage and alterations in lipid metabolism (Johnson 2023; Collins 2020). Some have theorized that sub-clinical deficiency of copper could be implicated in ischemic heart disease (Klevay 2016). Zinc intakes, well above the amount normally found in the diet, can decrease copper absorption in adults, and high iron intakes may interfere with copper absorption in infants (Institute of Medicine 2001).

Vomiting, nausea, diarrhea, and abdominal pain are the most reported effects after oral ingestion of high concentrations of copper. Excess copper exposure may adversely interact with certain heavy metals such as zinc. Individuals with Wilson disease inherit a rare genetic disorder of copper metabolism that results in accumulation of copper in the body and leads to damage in the liver, brain, and eyes (U.S. National Institutes of Health 2023). Jaundice, liver impairment, and renal failure are reported in cases where individuals ingested highly lethal doses of copper sulfate. Copper dust or spray irritates the respiratory system and eyes and causes skin discoloration. Copper has not been classified as a carcinogen by the U.S. Department of Health and Human Services (HHS), the International Agency for Research on Cancer (IARC), or the U.S. Environmental Protection Agency (EPA) (U.S. Agency for Toxic Substances and Disease Registry 2024).

Intake Recommendations

The Food and Nutrition Board (FNB) at the National Academy of Sciences, Engineering, and Medicine established Adequate Intakes (AIs) and Recommended Dietary Allowances (RDAs) for copper by life stage and sex group (Institute of Medicine 2001). RDAs are the average daily amount of copper needed to meet the nutrient requirement of nearly all (97–98%) healthy individuals. AIs are used when an RDA cannot be determined—they are based on observations and experiments that estimate nutrient intake.

For children from birth to age 12 months, the FNB established an AI for copper that is equivalent to the mean intake in healthy, breastfed infants (copper in cow milk is lower than that reported in human milk). From birth to age 6 months, the AI is 200 mcg (30 µg/kg/day). From ages 7–12 months, the AI is 220 mcg (24 µg/kg/day). The RDAs for copper for children and adults are as follows:

- 340 mcg (1–3 years)
- 440 mcg (4–8 years)
- 700 mcg (9–13 years)

- 890 mcg (14–18 years)
- 900 mcg (≥19 years)

The RDA during pregnancy for women ages ≥14 years is 1,000 mcg. During lactation, the RDA is 1,300 mcg.

The FNB established Tolerable Upper Intake Levels (ULs) by life stage and sex group. The UL is the highest average daily nutrient intake level that is likely to pose no risk of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the risk of adverse effects may increase. The FNB did not establish an UL for infants from birth to age 12 months, noting that intake should be from food and formula only. These are the ULs for copper for other age groups:

- 1 mg/day (1–3 years)
- 3 mg/day (4–8 years)
- 5 mg/day (9–13 years)
- 8 mg/day (14–18 years, including pregnancy and lactation)
- 10 mg/day (≥19 years, including pregnancy and lactation)

Individuals with Wilson's Disease, Indian childhood cirrhosis, and idiopathic copper toxicosis may be at increased risk of adverse effects from excess intake of copper (Institute of Medicine 2001).

The WHO International Programme on Chemical Safety (IPCS) has estimated an UL in the range of 2 or 3 mg/day for copper-contaminated drinking water among adults with normal copper homeostasis. IPCS was not certain about the proposed limit because concentration matters more than the total dose ingested in a day (IPCS 1998).

A nutritional cutoff point of 8 µmol/L (50.8 µg/dL) serum copper has been proposed in the literature as a clinical decision limit, i.e., the level below which mild symptoms may be expected. The proposed cutoff point was based on avoiding levels observed in patients with mild anemia resulting from high oral intake of over-the-counter zinc supplements (Andrew 2023).

Biochemical Indicators

Serum copper is typically used to assess copper status at the population level. Total ceruloplasmin protein may also be used to identify highly depleted individuals (Harvey 2009). Elevated serum copper may indicate excess copper exposure or disrupted copper homeostasis. Gastrointestinal symptoms and liver enzyme levels can be used to monitor and manage the toxic effects of copper on tissues (U.S. Agency for Toxic Substances and Disease Registry 2024).

Analytical Methods

Serum copper is often measured using highly specific inductively coupled plasma mass spectrometry (ICP-MS) or by atomic absorption spectrometry (AAS). A standard reference material (SRM 1598a Inorganic constituents in animal serum) is available from the National Institute of Standards and Technology (NIST) with certified values for copper. The New York State Department of Health's Wadsworth Center, College of American Pathologists, and Center for Toxicology in Quebec (CTQ) offer external quality assessment or proficiency testing programs for metals in biological matrices, which include copper in serum.

Findings from NHANES

The National Health and Nutrition Examination Survey (NHANES) is the only source for nationally representative data on serum copper for the U.S. population (Pfeiffer 2026). Serum copper data were collected in the U.S. population for participants ages 3 years and older from NHANES II (1976–1980) by an atomic absorption spectrometry (AAS) method. Serum copper was measured in the U.S. population for participants ages 6 years and older from NHANES 2011–2016 (from a 1/3 sample subset) and was generated by inductively coupled plasma mass spectrometry (ICP-MS). Using serum copper concentrations measured in NHANES 2011–2016, researchers determined recommended reference intervals that could be transferred for routine use in other clinical biochemistry laboratories (Andrew 2023). The mean and median serum copper concentrations for

supplement nonusers across these cycles were 113–114 µg/dL and 110–112 µg/dL, respectively. For supplement users, the mean and median concentrations were 115–116 µg/dL and 113–114 µg/dL, respectively. Serum copper concentrations were affected by age, gender, ethnicity, pregnancy, use of oral contraceptive pills, health status, and smoking (in males). The reference interval concentrations for all demographics were above the nutritional cutoff point of 8 µmol/L (50.8 µg/dL) proposed in the literature.

For more information about copper, see the Institute of Medicine's Dietary Reference Intake report (Institute of Medicine 2001) and fact sheets from the National Institutes of Health, Office of Dietary Supplements (https://ods.od.nih.gov/factsheets/list-VitaminsMinerals/).

Data in the 2026 tables

Data presented are from univariate analysis that was not adjusted for demographic variables (e.g., age, sex, race and Hispanic origin) or other blood concentration determinants (e.g., dietary intake, supplement use, smoking, BMI). Data for serum copper were available from three NHANES cycles between 2011 and 2016 for persons ages 6 years and older from a 1/3 subsample. The same inductively coupled plasma mass spectrometry (ICP-MS) method has been used throughout.

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