OVERVIEW OF COPPER TOXICITY TO AQUATIC LIFE

Okocha RO and Adedeji OB

Veterinary Public Health and Preventive Medicine University of Ibadan, Nigeria
roobee27_okocha@yahoo.com +23428183240

ABSTRACT: Copper is an essential element required by all living organisms, but it can be toxic to aquatic species when present at elevated concentrations. This document is an overview of copper toxicity and its effects on aquatic organisms. The subtopics include history: the copper age; general sources and uses; basic copper chemistry; environmental factors; effects on aquatic organisms; public health implication; proposed criteria for the protection of aquatic life and human health and recommendations.
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1. INTRODUCTION

A wide range of contaminants are continuously introduced into the aquatic environment mostly associated with industrial, agricultural and domestic wastes run-off (Lima et al., 2008). Among these contaminants, heavy metals constitute one of the main dangerous groups, because they are toxic, persistent and not easily biodegradable. The species and concentrations of metals in water are determined by geochemical processes and large scale releases into the aquatic environment by human activities (anthropogenic activities) (Wittmann, 1979). Rapid industrial developments as well as the use of metals in production processes have led to the increased discharges of heavy metals into the environment (Koli et al., 1977). According to Förstner and Prosi (1979) the harmful effects of heavy metals as pollutants result from incomplete biological degradation. Therefore, these metals tend to accumulate in the aquatic environment. Since heavy metals are non-biodegradable, they can be bio-accumulated by fish, either directly from the surrounding water or by ingestion of food (Patrick and Loutit, 1978; Kumar and Mathur, 1991). In addition, Heath (1987) indicates that when metals reach sufficiently high concentrations in body cells they can alter the physiological functioning of the fish.

Copper is an essential trace nutrient that is required in small amounts (5-20 micrograms per gram (µg/g)) by humans, other mammals, fish and shellfish for carbohydrate metabolism and the functioning of more than 30 enzymes. It is also needed for the formation of haemoglobin and haemocyanin, the oxygen-transporting pigments in the blood of vertebrates and shellfish respectively. However, copper concentrations that exceed 20 micrograms per gram (µg/g) can be toxic, as explained by Bradl and Heike (2005) and Wright and Welbourn (2002). Copper has been known to humans for at least 6000 years. Its uses in alloys, tools, coins, jewelry, food and beverage containers, automobile brake pads, electrical wiring and electroplating reflect its malleability, ductility and electrical conductivity. The use of copper to kill algae, fungi and molluscs demonstrates that it is highly toxic to aquatic organisms. In fact, copper is one of the most toxic metals to aquatic organisms and ecosystems. This is just one of the reasons that environmentally sensitive mining practices are so important. Fish and crustaceans are 10 to 100 times more sensitive to the toxic effects of copper than are mammals. Algae, especially blue-green algae species, are 1,000 times more sensitive to the toxic effects of copper than are mammals, as several authors, including Forstner and Wittman (1979), Hodson (1979) and Wright and Welbourn (2002), have demonstrated. This is an exception to the general principle that aquatic animals are more sensitive than aquatic plants to the toxic effects of metals.

2. History: The Copper Age

Copper occurs naturally as native copper and was known to some of the oldest civilizations on record. It has a history of use that is at least 10,000 years old, and estimates of its discovery place it at 9000 BC in the Middle East; (CSA – Discovery Guides, 2008) a copper pendant was found in northern Iraq that dates to 8700 BC (Rayner, 2007). There is evidence that gold and meteoric iron (but not iron smelting) were the only metals used by humans before copper. (Copper, 2008) The history of copper metallurgy is thought to have followed the following sequence: 1) cold working of native copper, 2) annealing, 3) smelting, and 4) the lost wax method. In southeastern Anatolia, all four of these metallurgical techniques appear more or less simultaneously at the beginning of the Neolothic 7500 BC (Renfrew and Colin1990). However, just as agriculture was independently invented in several parts of the world (including Pakistan,

China, and the Americas) copper smelting was invented locally in several different places. It was probably discovered independently in China before 2800 BC, in Central America perhaps around 600 AD, and in West Africa about the 9th or 10th century AD (Cowen, 2009). Investment casting was invented in 4500–4000 BC in Southeast Asia (CSA – Discovery Guides, 2008) and carbon dating has established mining at Alderley Edge in Cheshire, UK at 2280 to 1890 BC (Timberlake and Prag 2005). Ötzi the Iceman, a male dated from 3300–3200 BC, was found with an axe with a copper head 99.7% pure; high levels of arsenic in his hair suggest his involvement in copper smelting (CSA – Discovery Guides, 2011)

Experience with copper has assisted the development of other metals; in particular, copper smelting led to the discovery of iron smelting. Production in the Old Copper Complex in Michigan and Wisconsin is dated between 6000 and 3000 BC (Pleger and Thomas 2002, Emerson et al., 2009). Natural bronze, a type of copper made from ores rich in silicon, arsenic, and (rarely) tin, came into general use in the Balkans around 5500 BC. Previously the only tool made of copper had been the awl, used for punching holes in leather and gouging out peg-holes for wood joining. However, the introduction of a more robust form of copper led to the widespread use, and large-scale production of heavy metal tools, including axes, adzes, and axe-adzes.

2.1. General Sources and Uses

The United States is the major world producer and consumer of copper and its compounds. Most of the copper produced is used to manufacture electrical equipment, pipe, and machinery. Copper releases to the global biosphere—which may approach 1.8 million metric tons per year—come mostly from anthropogenic activities such as mining and smelting, industrial emissions and effluents, and municipal wastes and sewage sludge. Copper compounds are widely used as biocides to control nuisance algae and macrophytes, freshwater snails that may harbor schistosomiasis and other diseases, ectoparasites of fish and mammals, marine fouling organisms, and mildew and other diseases of terrestrial crop plants. Copper compounds are also used in agricultural fertilizers, in veterinary and medical products, in the food industry, and as a preservative of wood and other materials (Eisler, 1998).

2.2. Basic Copper Chemistry

Copper is a heavy metal that can be found naturally in a number of different forms. The form of copper that is most effective for algae and parasite control is the positively charged copper with a 2+ charge, also known as "Cu^{2+}." This is the form that is found in "bluestone" copper sulfate (more properly known as "copper sulfate pentahydrate" because it is attached to 5 water molecules).

When copper sulfate is dissolved into water, copper sulfate splits into separate copper (Cu^{2+}) and sulfate (SO_4^{2-}) ions (and water). Because this "Cu^{2+}" is the "active ingredient" of "bluestone" copper sulfate, this is the ion that must remain in solution and which must be measured. For susceptible marine parasites, including *Amyloodinium* (Reed and Floyd 1994) and *Cryptocaryon* (Yanong 2009), the target concentration is 0.15–0.20 mg/L Cu^{2+}.

Maintaining target concentration levels of copper can be challenging. Keeping copper concentrations high enough is difficult for many reasons. Water has numerous dissolved compounds (for example, bicarbonate ion (HCO_3), which can readily "combine" with copper and remove copper from solution. Carbonates—which are part of dolomite, crushed coral, oyster shell, and other common marine substrates—dissolve in the water and complex (or bind) with copper, affecting the level of copper in solution. Copper can also be taken up by living organisms, including bacteria, algae, and brine shrimp, and it can bind to substrates in the system (including activated carbon) (Cardeilhac and Whitaker 1988).

Still other factors can cause the copper concentration to rise too high. Increases in salinity will decrease the binding (adsorption) of copper to surfaces. In salt water at more neutral pH (e.g., pH of around 7), copper is surrounded by chloride molecules. Decreases in pH will release previously bound copper, and increase levels in solution, thereby increasing the risk of toxicity. Also, if some live foods, such as brine shrimp, are present during copper treatments, they may bioaccumulate enough copper to be toxic to fish that eat them (Cardeilhac and Whitaker 1988).

3. Environmental Factors

A number of factors will determine the toxicity of copper in water: a) the amount of free copper (Cu2+) in the water; b) the sensitivity of the fish or invertebrate species exposed; c) the age of the fish; d) the acclimation time to target concentration; e) the presence of substrates, especially those made of calcium or magnesium carbonate (including dolomite, oysters shell, and coral), that may remove copper from the water by adsorption; f) the presence of dissolved substances that may bind with copper and reduce its activity, including carbonates; g) the presence of "live foods" that may absorb and bioaccumulate (biologically concentrate) copper in their bodies;

and h) the tank water pH (Cardeilhac and Whitaker 1988). Because copper levels can vary over time--for instance, they may suddenly increase with a drop in pH--copper concentration should be measured at least twice a day and adjusted accordingly.

3.1. Effects on Aquatic Organisms

Copper is an essential compound for aquatic organisms in small quantities. However, copper becomes toxic when biological requirements are exceeded. The effects of copper on aquatic organisms can be directly or indirectly lethal. Different species, and even organisms within the same species, can exhibit different sensitivities to elevated copper levels in the water column. Organisms have different mechanisms by which they cope with and process copper. Some organism bioaccumulate and store copper, whereas others actively regulate its levels. In general, copper is actively regulated in fish, decapod crustaceans, and algae and stored in bivalves, barnacles, and aquatic insects (Brix and Deforest 2000). Therefore, to properly evaluate the copper-related effects on aquatic life, one must understand the factors that affect the biological fate of copper and the mechanisms by which it acts to produce its toxicity.

Copper undergoes complex speciation in natural waters; some species are bioavailable (free Cu2+ and Cu+ ions), while others are not. Only bioavailable forms of copper are considered to be toxic to exposed organisms. The reference to "copper" and "free copper" in the following discussion refers to its bioavailable form. The bioavailability, biodistribution to various parts of the organism, and bioaccumulation of copper are dramatically influenced by water chemistry. Therefore, water pH, hardness, organic content, and salinity play important roles in copper-induced toxicity.

The majority of studies in which the toxicity of copper has been addressed were performed on freshwater species. Copper is generally more toxic to organisms in freshwater than in saltwater. One of the reasons for this difference is that freshwater lacks cations, which compete with Cu2+ at the biological action sites, thus reducing copper toxicity (Brooks et al. 2007). Consequently, pH and water hardness play more important roles in freshwater than in saltwater environments. Increased pH accentuates copper toxicity because of the reduced competition between copper and hydrogen ions at the cell surface (Wilde et al. 2006). Cations that are involved in water hardness (i.e., Ca2+ and Mg2+) also compete with Cu2+ for biological binding sites (Boulanger and Nikolaidis 2003). Therefore, Cu2+ is less bioavailable in hard water than in soft water.

Although water pH and hardness protect organisms against Cu toxicity to some degree, the DOC content is among the most important factors in reducing copper toxicity to both fresh- and salt-water species. DOC forms organic complexes with copper and thereby reduces copper's bioavailability. According to McIntyre et al. (2008), water hardness and pH are unlikely to protect fish from copper-induced sensory neurotoxicity. However, water that contains high DOC concentrations does diminish the toxic effects of copper on the peripheral olfactory nervous system in Coho salmon (Oncorhynchus kisutch) (McIntyre et al. 2008). High DOC levels also significantly decrease acute copper toxicity to the freshwater flea, Daphnia magna, and the estuarine copepod, Eurytemora affinis (Hall et al. 2008; Kramer et al. 2004). Study results show that the water salinity gradient can also significantly affect the biological fate of copper.Water salinity influences the biodistribution and bioaccumulation of copper and can affect its toxicity as well (Amiard-Triquet et al. 1991; Blanchard and Grosell 2005; Grosell et al. 2007; Hall et al. 2008). The biodistribution of copper throughout the gill, intestine, and liver of the common killifish, Fundulus heteroclitus, is salinity dependent (Blanchard and Grosell 2005).

According to these authors, the gill and the liver are important target organs for copper accumulation at low salinities, whereas the intestine is a target organ at high salinities. In addition, the liver is a major organ involved in copper homeostasis and accumulates the highest amounts of copper. For this reason, the liver may be a potential target organ during chronic copper exposure. Water salinity influences the biodistribution and the toxicity of copper. Grosell et al. (2007) found killifishes to be most tolerant to copper exposure at intermediate salinities, and the acute toxicity was significantly higher in the lowest and highest salinity water. Increased fish sensitivity at both salinity extremes can be attributed to two factors: changes in copper speciation and changes in fish physiology in changing aquatic environments.

In general, water salinity may be more important to species that actively regulate internal osmotic pressure. The majority of invertebrates, however, are osmoconformers. Hence, to them the salinity gradient may be less important. Although in bivalves, the biological fate of copper was salinity dependent, in copepods (Eurytemora affinis) the toxicity of copper correlated better to DOC content than water salinity (Hall et al. 2008). In oysters, copper accumulation was inversely related to salinity (Amiard-Triquet et al. 1991). Some species can adapt to tolerate higher pollutant levels. Damiens et al. (2006) described adult oysters that lived in polluted water, wherein their larvae become less sensitive to pollution over time. Phytoplankton species have different sensitivities to copper toxicity: cyanobacteria

appear to be most sensitive, coccolithophores and dinoflagellates show intermediate sensitivity, and diatoms are resistant to copper (Brand et al. 1986; Beck et al. 2002).

In many aquatic animals, copper causes toxicity by impairing osmoregulation and ion regulation in the gill (Blanchard and Grosell 2005; McIntyre et al. 2008). When bioavailable Cu2+ enters the cell, it is reduced to Cu+. This copper oxidation state has a high affinity to sulfhydryl groups that are abundant within ATPase enzymes (Katranitsas et al. 2003; Viarengo et al. 1996). The best studied copper toxicity pathways involve the inhibition of ATP-driven pumps and ion channels. Katranitsas et al. (2003) discovered that, in brine shrimp, copper inhibited Na/K ATPase and Mg2+ ATPase enzyme activity. Similarly, in the mussel, Mytilus galloprovincialis, copper interfered with Ca2+ homeostasis in the gill, causing disruptions in Na/K ATPase and Ca2+ ATPase (Viarengo et al. 1996). In an in vitro study, Corami et al. (2007) investigated lysosomal activity and found that copper acted at two different sites: the proton pump and Cl– selective channels. Therefore, copper acts by inhibiting enzymes, ATP-driven pumps, and ion channels, resulting in cell toxicity from disruption of cell homeostasis and leading to changes in internal pH balance, membrane potential, and osmosis.

In addition to inhibiting ATPase enzymes and disrupting ion flow, copper toxicity can be induced by generating reactive oxygen species (ROS) (Bopp et al. 2008; Viarengo et al. 1996). ROS can lead to different outcomes: genotoxicity via DNA strand break and impaired cell membrane permeability via lipid peroxidation. Both pathways compromise normal cell functions.

A less understood effect of copper is neurotoxicity to fish olfaction. There is evidence that exposure to sublethal copper levels results in the loss of chemosensory function, which affects predator-avoidance behavior (McIntyre et al. 2008). The exact mechanisms are not yet completely understood and are still under investigation. Tilton et al. (2008) revealed that copper depresses the transcription of key genes within the olfactory signal transduction pathway.

The environmentally relevant copper levels that interfere with fish chemosensory mechanisms are very low. TDCu concentrations in the range of 0–20 ppb affected sensory capacity and behavior in salmon (Sandahl et al. 2007). At higher levels, copper caused a degeneration of the sensory epithelium (Bettini et al. 2006; Hansen et al. 1999). These effects were observed within hours of exposure. Hence, fish olfaction is a vulnerable endpoint that should be considered in environmental risk assessment.

The developmental stage of fish during their exposure to elevated copper levels may be a critical factor in their sensitivity. Carreau and Pyle (2005) showed that exposure to copper during embryonic development can lead to permanently impaired chemosensory functions. In contrast, fish that are exposed to elevated copper later in life can recover their chemosensory ability after the toxicant is removed.

Copper is stored and transported inside an organism as inorganic and organic complexes. In killifishes, copper bioaccumulates in target organs primarily as copper carbonate (CuCO3) and, to a lower extent, as copper hydroxide (CuOH+ and Cu(OH)2) (Blanchard and Grosell 2005).

Bivalves accumulate considerable amounts of copper that is associated with a cytosolic protein called metallothionein (Claisse and Alzieu 1993; Damiens et al. 2006). Although copper bioaccumulates and biodistributes to different organs, it is an internally regulated essential micronutrient. Therefore, according to Brix and Deforest (2000), there is an inverse relationship between metal concentrations in the water and in the organism. Hence, the bioconcentration factor (BCF) is not a suitable concept to describe the bioconcentration of copper.

Toxicity data for aquatic species for copper oxide, selected from the U.S. EPA ECOTOX database, are summarized in Table 1 (U.S. EPA 2009a). The table is divided into sections for freshwater and saltwater organisms. Data are presented for fish, invertebrates, and plants. The toxicity endpoints are also presented in the table, as is the chemical concentration that was lethal (LC50) or produced an effect (EC50). There is a large range in copper toxicity values for different freshwater algae.

4. Public Health Implication

Copper is an essential element in human nutrition as a component of metalloenzymes in which it acts as an electron donor or acceptor. Conversely, exposure to high levels of copper can result in a number of adverse health effects (Bremner, 1998). Acute copper toxicity is generally associated with accidental ingestion; however, some members of the population may be more susceptible to the adverse effects of high copper intake due to genetic predisposition or disease (International Programme on Chemical Safety, 1998). Copper status has also been associated indirectly with a number of neurological disorders, including Alzheimer's disease and prion diseases, including bovine spongiform encephalopathy (BSE) (Llanos & Mercer, 2002).

Table 1 Copper (I) oxide toxicity to aquatic organisms

Toxicity to freshwater aquatic organisms Species name: scientific/common	Endpoint	Duration/effects	Concentration μg/L (ppb)	Purity (%)
Fish				
Danio rerio/Zebra danio	LC50	96h/mortality	75	100
Invertebrates:				
Daphnia similis/Water flea	EC50	48 h/mortality	42	100
Biomphalaria glabrata/Snail	LC50	48 h/mortality	179	100
Plants:				
Pseudokirchneriella subcapitata/Green	EC50	30 min, 35min	90, 1900, >4500	Not reported
algae physiology/photosynthesis	EC50	96 h/physiology/	1300, 1600	Not reported
Photosynthesis	EC50	96 h/population/	30, 60, 230	Not reported
Abundance				

Toxicity to saltwater aquatic organisms Species name: scientific/common	Endpoint	Duration/effects	Concentration μg/L (ppb)	Purity (%)
Fish:				
Cyprinodon variegates/Sheepshead minnow	LC_{50}	96 h/mortality	>173	93
Melanogrammus aeglefinus/Haddock	LT_{50}	4.5 h, 5.7h Mortality	1800	100
Invertebrates:				
Americamysis bahia/Opossum shrimp	LC_{50}	96h/mortality	69.7	97
Balanus improvisus/Barnacle	LC_{50}	12 h/mortality	700	Not reported
	LC_{50}	24 h/mortality	500	Not reported
	LC_{50}	48h/mortality	350	Not reported
	LC_{50}	72h/mortality	140	Not reported
	LC_{50}	96h/mortality	20	Not reported

Source: U.S. EPA (2009a) ECOTOX database (accessed: 11/05/09)

Exposure of humans to copper occurs primarily from the consumption of food and drinking water. The relative copper intake from food versus water depends on geographical location; generally, about 20–25% of copper intake comes from drinking water. Georgopoulos et al. (2001) conducted a review of issues that affect the ability to assess and quantify human exposures to copper from various environmental media with a primary consideration being exposure to copper from potable water supplies. Populations living near sources of copper emissions, such as copper smelters and refineries and workers in these and other industries may also be exposed to high levels of copper in dust by inhalation. Copper concentrations in soils near copper emission sources could be sufficiently high to result in significantly high intakes of copper in young children who ingest soil.

Copper is readily absorbed from the stomach and small intestine. After nutritional requirements are met, there are several mechanisms that prevent copper overload. Excess copper absorbed into gastrointestinal mucosal cells induces the synthesis of and binds to the metal binding protein metallothionein. This bound copper is excreted when the cell is sloughed off. Copper that eludes binding to intestinal metallothionen is transported to the liver. It is stored in the liver bound to liver metallothionen, from which it is ultimately released into bile and excreted in the feces. Although copper homeostasis plays an important role in the prevention of copper toxicity, exposure to excessive levels of copper can result in a number of adverse health effects including liver and kidney damage, anemia, immunotoxicity, and developmental toxicity. Many of these effects are consistent with oxidative damage to membranes or macromolecules. Copper can bind to the sulfhydryl groups of several enzymes, such as glucose-6-phosphatase and glutathione reductase, thus interfering with their protection of cells from free radical damage.

In case reports of humans intentionally or accidentally ingesting high concentrations of

copper salts (doses usually not known but reported to be 20–70 g copper), a progression of symptoms was observed including abdominal pain, headache, nausea, dizziness, vomiting and diarrhea, tachycardia, respiratory difficulty, hemolytic anemia, hematuria, massive gastrointestinal bleeding, liver and kidney failure, and death.

Episodes of acute gastrointestinal upset following single or repeated ingestion of drinking water containing elevated levels of copper (generally above 3–6 mg/L) are characterized by nausea, vomiting, and stomach irritation; symptoms resolve when the drinking water source is changed. Most reported cases have not provided good estimates of the copper levels that induce these effects. Three experimental studies of high quality were conducted (Araya et al., 2001, 2003a; Pizarro et al., 1999a, 1999b) that demonstrate a threshold for acute gastrointestinal upset of approximately 4–5 mg/L in healthy adults, although it is not clear from these findings whether symptoms are due to acutely irritant effects of copper and/or to metallic, bitter, salty taste. In another experimental study with healthy adults, the average taste threshold for copper sulfate and chloride in tap water, deionized water, or mineral water was 2.5–3.5 mg/L (Zacarias et al., 2001), which is just below the experimental threshold for acute gastrointestinal upset.

The long-term toxicity of copper has not been well studied in humans, but it is infrequent in normal populations not having some hereditary defect in copper homeostasis (Olivares & Uauy, 1996). Chronic copper poisoning leading to liver failure was reported in a young adult male with no known genetic susceptibility who consumed 30–60 mg/d of copper as a mineral supplement for 3 yr (O'Donohue et al., 1999). Pratt et al. (1985) reported no evidence of liver damage or gastrointestinal effects in a double-blind study of 7 healthy subjects given 10 mg/d of copper gluconate in capsules for a period of 12 wk. Further, there is some evidence that individuals are able to adapt to elevated copper concentrations in drinking water. Exposures of children under the age of 6 yr, totaling 64,124 child-years, to drinking water containing 8 mg/L of copper produced no deaths associated with any form of liver disease (Scheinberg & Sternlieb, 1996). Similarly, individuals residing in U.S. households supplied with tap water containing >3 mg/L of copper exhibited no adverse health effects (Buchanan et al., 1999).

Dermal exposure has not been associated with systemic toxicity, but copper was reported to occasionally induce allergic responses in sensitive individuals (International Programme on Chemical Safety, 1998). Workers exposed to high air levels of copper (resulting in an estimated intake of 200 mg Cu/d) developed signs suggesting copper toxicity (e.g., elevated serum copper levels, hepatomegaly); however, other co-occurring exposures to pesticidal agents or in mining and smelting may contribute to these effects (International Programme on Chemical Safety, 1998).

4.1. Proposed Criteria and Recommendations

Proposed copper criteria for the protection of aquatic life and human health are summarized in Table 2.

Table 2: Proposed copper criteria for the protection of aquatic life and human health

Resources, criteria, and other variables	Effective copper concentration	Reference[a]
Aquatic life, fresh water		
Sediments		
Great Lakes		
Non polluted	<25 mg/kg DW	1
Moderately polluted	25 to 50 mg/kg DW	1
Heavily polluted	>50 mg/kg DW	1
Reduced abundance of benthos	480 to 1,093 mg/kg DW	3
Toxic to benthos	>9,000 mg/kg DW	3
Tissue concentrations; rainbow trout, Oncorhynchus mykiss: ratio of zinc to copper in gill or operclula		
Normal	Ratio> 1.5	4
Probably copper-poisoned	Ratio 0.5 to 1.5	4
Acute copper poisoning	Ratio<0.5	4
Water		
Safe. No adverse effects on rainbow trout exposed from fertilization through 4 days after hatching		

In soft or medium water	2 to 5 μg/L	5
In hard water	5 to 8 μg/L	5
Death or teratogenicity in eggs of sensitive species of fishes and amphibians	5 to 10 μg/L	5
United States		
Safe; total recoverable copper; 24h average	<5.6 μg/L	6
Maximum allowable concentration at 50mg $CaCO_3$/L	12 μg/L	6
Maximum allowable concentration at 100mg $CaCO_3$/L	22μg/L	6
Maximum allowable concentration at 200mg $CaCO_3$/L	43μg/L	6
Inhibits fish growth and ability of fish to discriminate prey	18 to 28μg/L	7
The Netherlands; total recoverable copper; maximum allowable concentration	<50μg/L	8
Aquatic life, marine		
Seawater		
Safe. Total recoverable Copper, 24h average	<4.0 μg/L; not to exceed 23 μg/L at anytime	6
Safe. Maximum concentration	<5.0 μg/L	9
Avoidance by clams	>5 mg/kg DW	10
Clam burrowing ability inhibited (water concentrations of 113 to120 μg Cu/L)	>15 mg/kg DW	10
Not polluted	<40 mg/kg DW	9
Moderately polluted	40 to 60 mg/kg DW	9
Very polluted	>60 mg/kg DW	9
Reduced species diversity; sensitive species absent	>200 mg/kg DW	11
Human health		
Air		
Montana	<0.26 μg/m^3 for 8h; <1.57 μg/m^3 for 24h	2
Massachusetts	<0.54 μg/m^3 for 24h	2
Connecticut, North Dakota	<2μg/m^3 for 8h	2
Florida	<4 μg/m^3 for 8h	2
Nevada	<5μ g/m^3 for 8h	2
Virginia	<16 μg/m^3 for 24h	2
New York	<20 μg/m3 for 1 year	2
United States; workplace; 8 h daily Fumes	<10.1 <0.2 mg/cm^3	2

A,1,NAS 1977; 1, Beyer 1990; 2, ATSDR 1990; 3, Mackenthun and Cooley 1952; 4, Carbonell and Tarazona 1993; 5, Birge and Black 1979; 6, USEPA 1980; 7, Sandheinrich and Atchison 1989; 8, Enserink et al. 1991; 9, Fagioli et al. 1994; 10, Roper and Hickey 1994; 11, Bryan and Langston 1992.

Proposed criteria to protect most species of freshwater aquatic life from copper toxicity or deficiency include maximum water concentrations over a 24-h period of 12 µg Cu/L in soft water and 43 µg/L in hard water, sediment concentrations less than 480 mg Cu/kg DW, and, in rainbow trout, a zinc/copper ratio in gill or opercle greater than 1.5 (Table 2).

However, the proposed maximum water concentration range of 12-43 µg Cu/L exceeds the 5-10 µg/L range that is lethal or teratogenic to sensitive species of fishes and amphibians (Birge and Black 1979) and overlaps the 18-28 µg/L range that inhibits growth and ability to discriminate prey for other species (Sandheinrich and Atchison 1989). Some scientists state that laboratory studies tend to overestimate the adverse effects of copper on freshwater abundance and diversity and suggest more research on field mesocosms receiving water directly from the system under investigation (Clements et al. 1990). In marine ecosystems, copper concentrations should not exceed 23 µg Cu/L at any time, and sediments should contain less than 200 mg Cu/kg DW (Table 2). But adverse sublethal effects of copper to representative species of estuarine algae, mollusks, and arthropods frequently occur at less than 10 µg Cu/L (Bryan and Langston 1992). Also, extrapolation of laboratory data on copper and marine benthos to actual field conditions is difficult because of changing environmental conditions such as thermosaline regimes and the nature of the sediment substrate (Ozoh 1992c).

The proposed domestic drinking water criterion of less than 1.0 mg Cu/L for the protection of human health is not based on copper toxicosis but on the unpleasant taste which develops with higher levels of copper in drinking water (USEPA 1980). Increased copper levels (>1.3 mg Cu/L) in household water supplies caused by corrosion of copper plumbing materials may adversely affect infants and young children among residents of newly constructed or renovated homes (Knobeloch et al. 1994). Human groups at greatest risk to copper toxicosis now include young children subjected to unusually high concentrations of copper in soft or treated water held in copper pipes or vessels, medical patients with Wilson's disease, medical patients treated with copper-contaminated fluids in dialysis or parenteral administration, people with a glucose-6-phosphate dehydrogenase (G-6-PD) deficiency (about 13% of the Afro-American male population has a G-6-PD deficiency) who drink water containing greater than 1.0 mg Cu/L, and occupationally exposed workers (USEPA 1980).

5. Conclusion

Copper is plentiful in the environment and essential for the normal growth and metabolism of all living organisms. Its discharges to the global biosphere are due primarily to human activities, especially from the mining, smelting, and refining of copper and from the treatment and recycling of municipal and industrial wastes. It is important to note that copper deficiency, though not within the scope of this subject, can produce adverse health effects. Its toxicity as a result of its excess also does, as outlined above. Abiotic factors such as temperature, pH and hardness and others have shown to influence the bioavailability of metals to the aquatic organisms. Through routine monitoring of the aquatic environment, these abiotic factors should be maintained at constant and, if possible, acceptable levels. However, if this is not achieved, it is highly probable that an increase in copper accumulation and toxicity would result which, ultimately, could be detrimental to the health of the aquatic organisms and humans via consumption of such organisms.

Correspondence to:
Adedeji Olufemi B.
Department of Veterinary Public Health and Preventive Medicine
University of Ibadan.
Oluadedeji2001@yahoo.com

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