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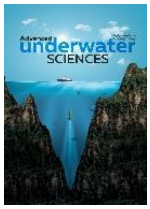

Toxic Effects of Copper Nanoparticles in Aquatic Organisms

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ABSTRACT

Although copper is an essential element for animal organisms, its potentially toxic effects have been known for centuries. In recent years, nanoparticles, which are widely used in many fields, have been added to the sources that allow copper to participate in aquatic ecosystems. The difference between nanoparticles containing Cu, Zn, Fe, Ag, Ti, and Al ions from known metal toxicity is not clear enough yet, and the toxic effects of metal oxide nanoparticles continue to be investigated. In this study, it was aimed to compile the toxic effects of copper nanoparticles in aquatic organisms living in different trophic zones of the food chain and with different organizational levels.

1. INTRODUCTION

1.1. Source of Environmental Pollution

Although environmental pollution caused by metals in the world is based on human history, technological development has also increased the sources of participation. Nanotechnology, which has an important place in human life and causes developments in health, engineering, and basic sciences, is among the new sources of pollutants. Metal oxide nanoparticles have entered human life in recent years with the increase in nanotechnology. These materials range in size from 1-100 nm and mostly contain metal ions such as Ag, Ti, Cu, Zn, Fe, and Al. Nanoparticles are used in many fields with their high surface-volume ratio, electronic properties, reactivity, surface structures, and crystal properties (Handy and Shaw, 2007). It took many years for the concept of nanotechnology, which was first used by Norio Taniguchi in 1974, to become widespread. The development of nanotechnological products has been divided into four generations. The second-generation products containing active nanostructures have started to take place in medical applications, and fourth-generation products containing molecular size tools and atomic design have been added to the applications in the last few years, and it is believed that it will make a greater contribution to the development in the health sector (Turgut et al., 2011).

The use of nanomaterials in many areas, from the use of contrast agents in medical imaging to gene transfer to specific cells, enables analyzes and treatments that cannot be carried out with a different application in medicine. On the other hand, developments in the energy, electronics, automotive, textile, food, and paint industries have also been applications that facilitate human life (Song et al., 2015). In addition to these positive effects, increased metal pollution with nanotechnology causes great concerns (Turgut et al., 2011).

Metals used as raw materials in the industry are discharged to aquatic ecosystems. In addition to agricultural practices, domestic wastes, increase in fossil fuel use are factors that increase metal incorporation into aquatic ecosystems. The long half-lives of these pollutants and the continuity of discharges pose a great threat to aquatic ecosystems.

1.2. Copper Toxicity in Animals

Although copper is an essential element for animal organisms, it is a potentially toxic substance. For this reason, a cellularly evolved homeostatic control mechanism has developed over time. Under the influence of a high concentration of copper through water and food, protein functions and homeostatic mechanisms are impaired due to cellular copper increase. Cellular copper increase leads to physiological disorders and toxicity. Increasing copper concentration in the environment is

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not a simple parameter that affects water quality but affects the bioavailability, physiology, and sensitivity of metal by organisms.

The redox potential of copper makes it essential for all aerobic organisms. Copper is involved in the structure of many enzymes such as cytochrome c oxidase. However, the redox potential causes the formation of reactive oxygen species (ROS) under the influence of high copper concentration.

1.3. Copper Nanoparticle Toxicity in Animals

Copper nanoparticles are a form of insoluble copper with a wide range of applications such as air and liquid filtration, wood protection, bioactive coatings, integrated circuits, coatings on batteries, thermal and electrical conductivity, textiles (Shaw and Handy, 2011; Gomes et al., 2011). Although insoluble in water, copper nanoparticles release copper ions into the surrounding water due to the small particle size. Nanoparticles and copper alone are toxic to aquatic organisms. Studies are trying to determine the difference between the toxic effects of CuO-NPs and the known metal toxicity.

In order to adequately understand the toxicity mechanism of copper nanoparticles, researches were carried out on aquatic organisms living in different trophic zones of the food chain and with different organizational levels, and cytotoxic effects were determined in bacteria (Vibrio fischeri, Escherichia coli, Staphylococcus aureus, Bacillus subtilis), protozoa (Tetrahymena thermophila), crustacean (Daphnia magna, Thamnocephalus platyurus, Daphnia pulex, Ceriodaphnia dubia), algae (Pseudokirchneriella subcapitata) and zebrafish (Danio rerio) (Gomes et al., 2011). It has been determined that copper nanoparticles cause the formation of reactive oxygen species and oxidative stress. It has been reported that lipid peroxidation, oxidative stress, antioxidant enzyme activity, and metallothionein synthesis increase in Mytilus galloprovincialis under the influence of CuO-NPs (Gomes et al., 2012).

1.4. Factors affecting tissue metal toxicity

Metabolically active tissues are responsible for the uptake, detoxification, and excretion of metals. Gills are target organs that function in ion regulation and interact directly with the environment (Evans et al., 2005). The uptake and accumulation of metals from the gills vary depending on the physical and chemical properties of the water, as well as the competition between metals for the attachment points in the gill epithelium. It is known that metals accumulate in the gills at high concentrations at the beginning of the effective period in aquatic organisms, causing hyperplasia, hypertrophy, and proliferation in gill epithelial cells, and inhibition of Na+/K+ ATPase activity (Evans, 1987; Xiong, et al., 2011; Zhao et al., 2011). Inhibition of enzyme activity, ammonia excretion from the gills, and a decrease in sodium uptake, resulting in impaired osmoregulation and mortality (Grosell et al., 2007).

In freshwater fish, the gills filter more water than in marine fish to provide osmoregulation. For this reason, they are more affected by the toxic substances in the environment. It has been reported that CuNP and copper nitrate cause deformations in the gill filaments and gill pavement cells in Oncorhynchus mykiss, Pimephales promelas, and Danio rerio, and the toxic effect varies depending on the sensitivity of the examined species (Song et al., 2015).

The initial effect of metals that accumulated in the gills is transmitted to the liver via the circulatory system by binding to carrier proteins when the carrying capacity of the gills is exceeded. Liver in vertebrates or hepatopancreas in invertebrates is a metabolically active tissue that functions in the conversion of nutrients absorbed from the intestine, the synthesis of bile salts that function in the digestion of fatty acids, and the storage of glycogen, which is the main source of blood glucose, and the regulation of heavy metals. In addition, It is one of the main synthesis sites of metallothionein and glutathione which functions in the elimination of toxic effects by binding heavy metals. Metallothionein is a cytoplasmic protein with low molecular weight, rich in cysteine, devoid of aromatic amino acids, soluble in acids, resistant to precipitation by alcohol, and trichloroacetic acid. Glutathione is a tripeptide containing large amounts of cysteine with a sulfhydryl group (Heath, 1995).

Increasing in exposure time, accumulation in liver tissue increases, and it is known that there is an increase in vacuolization, number of lysosomal vesicles, and gluconeogenic enzyme (Aspartate aminotransferase, Alanine aminotransferase) activity in liver cells in fish. It was determined that Cu+2 accumulated at higher concentrations in the hepatopancreatic tissue at the beginning of the exposure time in M. galloprovincialis compared to mussels under the influence of CuO-NP, but the concentration decreased due to the increase in the exposure time. In mussels exposed to CuO-NP, the accumulation showed a linear increase. The decrease in the concentration of Cu+2 in the hepatopancreas at the end of the experiment can be explained by detoxification (Gomes et al., 2012).

Cu-NPs tend to both release metal ions and form aggregates in water. Many studies have suggested that the toxicity of metallic NP suspensions may be due to the release of metal ions (Griffitt et al., 2009; Hoheisel et al., 2012). In M. galloprovincialis, CuO-NPs were found to accumulate twice as much in the hepatopancreas than in the muscle tissue. This was explained by the release of CuO from NPs and its transport to the hepatopancreas for detoxification (Gomes et al., 2012). It is also supported by previous research that copper ions can be released from the nanoparticle surface, thereby exposing fish to dissolved Cu as well as nano-form (Gomes et al., 2011; Shaw and Handy, 2011).

It was determined that CuO-NPs accumulated in tissues of Cyprinus carpio, respectively, in intestine>gill>muscle>skin & scale>liver>brain tissue. Intestines and gills may have accumulated in higher concentrations than other tissues since they constitute the main uptake route from food and the environment. Higher concentration in muscle tissue than liver may indicate a chronic effect of CuO-NP. Increased accumulation in the intestines due to an increase in exposure time may indicate that CuO-NPs are removed

from the body through feces. Meanwhile, in fish exposed to CuO-NP, it is thought that soluble Cu+2 ions accumulate in the brain and inhibit cholinesterase (ChE) activity and cause neurotoxicity (Zhao et al., 2011).

It has been reported that CuNPs have more negative effects on intestinal tissue and copper sulfate has more negative effects than gill and liver tissues of Epinephelus coioides (Hoseini et al., 2016). These findings show that the chemical form of the toxic substance, as well as the metabolic function of the tissues, is effective on the accumulation. In mammals, the liver, kidney, and spleen are target tissues for CuNPs and cause tissue damage, but micro Cu particles do not cause damage. This indicates that toxicity varies depending on particle size (Chen et al., 2006).

1.5. The toxicity effects of Cu-NPs and CuSO4 in animals

It was determined that the effect of Cu-NPs and CuSO4 in juvenile Oncorhynchus mykiss caused similar tissue damage in fish. Hyperplasia, aneurisms, and necrosis in the secondary lamellae, in gill; swelling of goblet cells, necrosis in the mucosa layer and vacuole formation, in the gut; hepatitis-like injury and cells with pyknotic nuclei, in the liver; damage to the epithelium of some renal tubules and increased Bowman's space, in the kidney; some mild changes were observed in the nerve cell bodies in the telencephalon, alteration in the thickness of the mesencephalon layers, and enlargement of a blood vessel on the ventral surface of the cerebellum, in the brain; changes in the proportional area of muscle fibers in skeletal muscle were observed (Al-Bairuty et al., 2013).

In the gill, CuSO4 and Cu-NPs were found to cause similar pathological changes in juvenile rainbow trout, but the damage was more due to the CuSO4 effect. Intestines are responsible for functional absorption, and it has been determined that damages are more severe under the influence of Cu-NPs. Effects in the brain are explained by indirect toxicity and/or hypoxia on neuroendocrine functions. It has been reported that Cu, Ag, and Al-NPs cause damage to the blood-brain barrier and edema formation in the brain in rodents. Enlarged blood vessels on the surface of the brain are consistent with an attempt to increase blood flow to offset the effects of hypoxia. It was emphasized that the thickening caused by Cu-NPs and CuSO4 in the midbrain layers could not be caused by different tissue growth, since the trial period (10 days) was not long, and the thickening was definitely associated with edema. Although more research is needed to adequately understand the etiology of Cu and Cu-NP pathology in the brain, the findings show that various physiological dysfunctions (osmotic, metal homeostasis, oxidative stress, vascular) may contribute to the pathology.

2. CONCLUSION

Consequently, copper toxicity in gills and viscera in water-borne exposures has a significant effect on cellular metabolism and respiration. Copper can promote mitochondrial dysfunction and exert detrimental effects on fish metabolic energy production (Linder and Hazegh-Azam, 1996; Braz-Mota et al., 2018). Identifying changes caused by copper toxicity in species living in different trophic zones of the water column and with different levels of the organization will be valuable in understanding the effects of copper nanoparticles and ions in more detail. The continuity of the discharge of metals and nanomaterials into the aquatic ecosystem causes an increase in the risk of toxicity in aquatic organisms. Investigating the toxic effect mechanisms of these substances is important for the environment and human health.

Author contributions

Merve Kolukısaoğlu: Investigation, Writing Reviewing and Editing.
Nuray Çiftçi: Conceptualization, Investigation, Writing-Original draft preparation, Writing Reviewing and Editing.
Deniz Ayas: Investigation, Writing- Original draft preparation, Writing-Reviewing and Editing.

Conflicts of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence this paper.

Statement of Research and Publication Ethics

The authors declare that this study complies with Research and Publication Ethics.

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