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Assessment of the ecotoxicity of heavy metal contaminants on aquatic organisms: A review on the impact of Lead, Chromium, Cadmium, Zinc and Mercury

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Abstract:

Heavy metal ecotoxicity has been recognized to be a major threat to aquatic organisms and there are several health risks associated with it. This in-depth review investigation explores the critical issue of heavy metals pollution, concentrating on the hazardous effects of chromium, cadmium, lead, mercury, and zinc oxide nano particles on aquatic species. The balance of nature suffers as industrialization, urbanization and unsustainable land use changes spreads throughout the earth, with heavy metals released by industries causing destruction on the environment. These contaminants build up in living things, damaging their metabolism, immunological system as well enzymatic functions. The significance of the global ecological problem is clear, particularly in developed nations with a significant concentration of businesses. The present review also provides an analysis of each heavy metal's toxicity patterns, considering things like age, species, and abiotic circumstances. Moreover, research is also done on how heavy metals affect different organ systems, such as the brain, gills, liver, kidneys, and reproductive system of aquatic species. Histopathological abnormalities, oxidative stress in cells altered metabolism, and even endocrine disturbance are just a few of the reported alterations. The toxicological consequences of zinc oxide nano particles are also examined in the present review, emphasizing the possible harm they may do to many aquatic species. The present review also emphasizes the necessity of tackling this significant issue and advances our understanding of the damaging effects of toxic metals on aquatic environments.

Keywords: Aquatic organism; ecotoxicity; lead; chromium; cadmium; mercury; zinc oxide

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1. Introduction

Environmental contamination is one of the biggest problems that modern society is dealing with (Yarsan&Yipel, 2013). The natural ecology gets destroyed as a result of industrialization as the world develops day by day (Mahboob et al., 2014). The ecology is destroyed by heavy metals emitted by factories (Saleh & Marie, 2015). The majority of heavy metals are derived from residential, commercial, and agricultural waste, as well as from mining. Once organisms absorb these metals, they accumulate in their bodies and harm them. Heavy metals in the soil also reduce soil fertility and have an adverse effect on plant growth (Jeziarska&Witeska, 2006). The heavy metals are easily transmitted to the bodies of live species when they are dissolved in an aquatic environment. From there, they move up the food chain and damage all the higher living things that consume them (Varanka et al., 2001). According to Mahboob et al. (2014), heavy metals have a significant impact on all cell organelles, metabolism, immunological function, and enzymatic functions. Due to the abundance of enterprises, environmental pollution problems are more prevalent in developed nations (Arellano et al., 1999). Heavy metals impair important biological processes and cause broad ecological imbalances, making the ecological cost of human progress more and more obvious. This review paper offers a thorough analysis of the toxicity of particular heavy metals, illuminating the complex interactions between age, species, abiotic variables, and their effects. This analysis highlights the urgent need for sustainable practises to limit the negative effects of environmental pollution by exploring the complicated relationships among these toxic metals and marine life (Eyckmans et al., 2001, Varanka et al., 2001). In the present review has been discussed about the toxicity of chromium, cadmium, lead, mercury, zinc and their probable remediation process.

2. Toxicity of metals

2.1 Chromium

Chromium is a crystalline metal with a steel-gray hue and a proton number (Z) of 24. It is mostly found in the forms of Cr III and Cr VI (Sridharet al., 2000). Although chromium III plays a role in the metabolism of lipids and carbohydrates, it also has hazardous effects (Ghosh 2002). Chromium VI is more hazardous and can enter many cells as well as induce stomach cancer (Kaviraj 1983). In addition to age and species of aquatic ecosystem, chromium toxicity is also influenced by abiotic factors including pH, temperature, and oxidative forms. Any change in these parameters can make chromium more harmful

(Lushchak et al., 2009). Chromium VI is distributed differently in different species, however the overall pattern that has been seen is: Gills>Liver>Skin>Muscles (Ahmed et al., 2013).

According to research (Van der Putte et al., 1981), a decrease in pH causes more chromium to accumulate, and the amount of chromium in gills is higher than that in other organs without a change in pH.

Chromium exposure also alters fish behaviour, and the effect was studied on *Labeorohita*, whose body balance was lost after exposure. In order to predict any toxic impact of a xenobiotic agent on aquatic organisms, it is necessary to expose organisms to that agent for a brief period of time before measuring the effect. Another incidence involved a 24-hour exposure to a concentration of 28.99 mg/litre fingerlings of *H.fossilis* after 24 hours, they noticed that the activeness had somewhat risen and that the chromium concentration was 56.59 mg/litre (Vera-Candioti et al., 2011).

Acute effect of chromium concentration on different fish species

Different fish species were subjected to chromium concentrations, which had a variety of impacts. For instance, *Salmo gairdneri*'s ability to reproduce was affected by exposure to 0.005 mg/L of chromium. When subjected to 0.098 mg/L of chromium, *Tilapia sparrmanii* showed a shorter blood clotting time. Additionally, *Tilapia sparrmanii* showed decreases in white blood cell count, the number of red blood cells count, haemoglobin concentration, and ALA-D activity at a somewhat lower quantity of 0.0098 mg/L (pH 7.4–9.0). As a result, one becomes more vulnerable to infections caused by bacteria (Vera-Candioti et al., 2011).

Different physiological alterations were seen in *Sccobranchus fossilis* after exposure to chromium concentrations between 0.1 and 3.2 mg/L. Spleen-to-body ratio, haemoglobin levels, white blood cell number packed volume of cells, the number of red blood cells, average corpuscular volume, and splenocyte count were among the modifications that occurred. However, the fish's ability to produce antibodies dropped, and they were more susceptible to bacterial infections (Ackermann, 2010).

When subjected to chromium concentrations between 5 and 15 mg/L, *periophthalmus dipes* showed a decline in ion-dependent ATPase activity. The glycogen, total lipid, and total protein content of the muscular tissue, gill, and liver tissues of *Labeorohita*, on the other hand, decreased after exposure to a concentration of 39.40 mg/L (96-hour LC50) (Lushchak et al., 2009).

When exposed to 250 M of chromium, *Carassius auratus* produced more reactive oxygen species (ROS) and had lower cell viability. Similar to how *Colisafasciatus* behaved, it produced more glucose after being exposed to 60 mg/L of chromium (Lushchak et al., 2009).

Chronic effect of exposure of chromium in different species

In live testing with *Oreochromis mossambicus*, exposure to 7.5 g/fish Cr(VI) and 100 g/fish Cr(III) resulted in lower splenic weight, lower lymphocyte count, and impaired antibody production (Ackermann, 2010). *Oncorhynchus tshawytscha* was used in laboratory experiments where exposure to chromium concentrations of 24-120 g L⁻¹ and 54-266 g L⁻¹ caused DNA damage and decreased growth and survival rates (Abbasi et al., 1995). Research using *Cyprinus carpio* at 1010 g L⁻¹ for 38 weeks revealed reduced humoral responses and a 25% drop in blood protein levels (Abbasi et al., 1995). According to Luschak et al. (2009), *Salmo gairdneri* was exposed to 0.2 mg L⁻¹ at pH 6.5 and 2 mg L⁻¹ at pH 7.2, which increased mortality and had an impact on embryo hatching. According to Ackermann (2010), *Clarias gariepinus* treated to 36 mg L⁻¹ and 11 mg L⁻¹ showed lower embryo survival and larval growth. Fin and fin ray degradation was shown in in vitro studies with *Nuria denricus* in the concentration range of 0-100 mg L⁻¹ (Lushchak et al., 2009). According to Vera-Candiotti et al. (2011), *Channa punctatus* treated to 2.6 mg L⁻¹ for 60 and 120 days had raised blood and muscle lactic acid, decreased liver lactic acid and glycogen, inhibited LDH activity in the liver and kidney, and changed PDH and SDH activities in a variety of tissues.

2.2 Mercury (Hg)

Mercury exposure is more harmful and causes immediate harm to living organisms (Malleth et al., 2015). Due to its bioaccumulation and neurotoxic effects, mercury contained in organic farms is highly harmful (Dave & Xiu, 1991).

Gills are the primary organ involved in gaseous exchange and also have a little regulatory purpose (Zulkipli et al., 2021), but they also serve as a channel for the entry of some hazardous heavy metals because of mercury pollution that impairs the gills' capacity to breathe. According to the study, the yellow Fin sea bream lost its capacity for gaseous exchange at mercury concentrations of 0.01 and 0.02 mg/L. Lesions caused by mercury poisoning also appeared on the fish's gills. According to Ung et al. (2010), the liver is the primary organ engaged in detoxification, metabolic regulation, antioxidant generation, and hormone regulation. Therefore, the liver is where mercury accumulates the most because of its role in detoxifying. Mercury accumulation also differs by gender in zebrafish; male zebrafish accumulate mercury more than female zebrafish when they are exposed to HgCl₂.

Mercury in the liver can result in lesions, necrosis, liver cell enlargement, lipidosis, and bile doldrums (Zhang & Wong, 2007).

The mercury poisoning of the water has an equal impact on the kidneys as it does on the other organs (Zulkipli et al., 2021). Because the kidney is involved in the osmoregulatory process and regulates body fluid, heavy metals also accumulate in the kidney and can lead to lesions such as necrosis and inflammation of the kidney cells. Complete kidney function can also be lost as a result of mercury contamination.

Both organic and inorganic forms of mercury can cause the central nervous system to deform, which can also have an effect on the brain. Granular cells responded noticeably to exposure to MeHg at levels between 0.8 and 13.5 g/g in a study on the brains of *Danio rerio* (Zebrafish). Cell density was decreased, nuclear area was reduced, and chromatin was condensed (Ung et al., 2010). Similar significant observations were made in the case of *Salmo salar* (Atlantic salmon) after exposure to CH₃-HgCl and HgCl₂ at dosages of 1, 10, and 100 mg/kg. According to Mallesh et al. (2015), these included the existence of vacuolation, necrotic cells, and tissue edoema, which clearly damaged the tissue's gross architectural structure and were associated with oxidative injury.

Table 1. The previous studies on the different organs of the fish.

Organ	Impact on Fish spp.	References
Gill	Changes in the structure of the respiratory lamellae that are large enough to diminish the capacity for gas exchange cause gill discoloration and respiratory problems that impair the ability to detect water quality due to tissue and cellular damage.	(Eisler .,1986)
Liver	Stressful situations include starving durations, cold-related deaths, and impacts of direct hypothermia. increased cellular metabolism as the first and most important reaction	(Bakshi., 2016)
Kidney	Severe stress on kidney during deaths from the cold, hunger crises, and the effects of direct hypothermia Increased renal filtration rate acting as a protective strategy against toxic stress, circulating Hg accumulation, and ensuing cellular damage	(Langard&Norseth ., 1979)
Brain	Pyramidal and Purkinje cells are lost as a result of damage to the posterior part of the brain, and astrocyte proliferation is increased.	(Doudoroff, &Katz ., 1953)

2.3 Zinc Oxide- Nanoparticles

However, zinc oxide nanoparticles are more significant due to their wide range of uses in a variety of industries, including rubber, textile, pharmaceutical, and cosmetics (Chaari&Matoussi, 2012). Numerous investigations have demonstrated that the degenerative alterations caused by zinc oxide nanoparticles in fish also impact the central nervous system and induce lipid peroxidation(Dhara .,2014). The effects of zinc oxide nanoparticles on fish embryos and young fish are similar to those on adult fish, but they vary depending on the species (Grobler et al., 1989).

Table 2 The previous studies on the different fishes.

Species	Zebrafish (<i>Danio rerio</i>)	Common Carp (<i>Cyprinus carpio</i>)	Nile Tilapia (<i>Oreochromis niloticus</i>)	
Effects	- Larval body length and hatching rate were restricted	- Acute toxicity was evident at 4.897 mg/L.	- caused the gills to severely vacuolate and swell.	(Vinodhini& Narayanan ., 2009)
	- Smaller NPs displayed more toxicity.	- Acute toxicity comparable to zebrafish at 4.92 mg/L	- Disrupted gill cells, decreased contact surface	(Chaari&Matoussi ., 2012).
	- reduced enzyme activity and glutathione content	Affecting physical and chemical characteristics	- Mucus with black blocks, NPs penetrating through membrane	(Dineley et al ., 2003)
	- adverse effect on reproduction	It also show the decrease in reproduction	Types of Gill injuries: Necrosis and Hyperplasia	(Obasohan .,2007)
Findings	- ZnO-NPs inhibited growth and hatching.	- The study of gills histology showed structural changes	- Necrosis and severe gill vacuolation	(Grobler et al ., 1989)
	- Catalase and other enzyme activity had an impact.	- Gill show alterations due to ZnO-NPs toxicities	- Ion exchange problems, gill cell disruption, and edoema	(Fernández et al ., 2013)

2.4 Cadmium (Cd)

The typical concentration of cadmium (Cd), a trace metal, in the crust of the Earth is between 0.1 and 0.5 ppm. It frequently goes hand in hand with ores of zinc, copper, and lead. According to Barjhoux et al. (2012), the average concentration in ocean water ranges from 5 to 110 mg/L, whereas it is typically less than 1 g/L in surface water and groundwater. Cadmium is frequently found in compound forms, such as cadmium chloride, cadmium oxide, and others, rather than in its elemental form. Natural causes like volcanic activity and rock weathering as well as human activities like burning fossil fuels and industrial processes all contribute to the release of this element into aquatic environments (Liu et al., 2022).

The effects of cadmium on aquatic life are alarming. It enters the food chain after being ingested by plants and animals in different ways. Some fish directly absorb the free ionic form of cadmium through their gills, digestive tracts, and skin, whereas others can assimilate water-soluble or sediment-based cadmium compounds (Ebrahimi et al., 2020).

Fish are hazardous to cadmium because it is unnecessary. It messes with their cells' internal workings, causing DNA damage and the formation of reactive oxygen species. According to Tamele and Vázquez (2020), exposure to cadmium causes histopathological abnormalities in organs including the liver and kidneys as well as other effects on fish health. Anaemia and abnormal haematological responses result from this alteration. Cadmium also interferes with metabolic processes, which alter glucose levels and glycogen stores.

Due to its endocrine-disrupting properties, cadmium interferes with fish reproductive processes like vitellogenesis. Even fish larvae are affected, resulting in deformities and lower survival rates (Tamele & Vázquez, 2020).

Due to the fact that cadmium accumulates largely in organs including the liver, kidney, and gills, its slow excretion rate creates environmental issues. Gills among them are crucial in the detoxification of cadmium. Cadmium is one of the most hazardous heavy metals for aquatic creatures due to its high rate of bioaccumulation (Atchison et al., 1987).

Lead (Pb)

Hazardous heavy metal lead occurs naturally in the environment and is frequently coupled with other substances including PbS, PbSO₄, and PbCO₃. Lead concentrations have dramatically increased as a result of human activities including metal mining, burning of coal, oil and petrol, battery production and use of lead-based products like paint and

insecticides (Lindqvist et al., 1991). Lead is released into aquatic habitats through industries, agriculture, runoff, dust, and wastewater, damaging aquatic life. The solubility of lead in water is influenced by variables like pH, salinity, and hardness. In soft, acidic water, it dissolves the most (Ebrahimi et al., 2020). Lethal lead concentrations for fish occur between 10 and 100 mg/L, while sublethal exposure results in impotence, behavioural abnormalities, and growth problems. According to Katti, prolonged low-level exposure to lead nitrate changed the lipid and cholesterol levels of *Clariasbatrachus* organs. When exposed to lead, African catfish (*Clariasgariepinus*) developed deformed gill and liver tissues. *Mastacembeluspancalus* with lead exposure demonstrated ovarian tissue alterations. According to Atchison et al. (1987), fish exposed to lead exhibited parenchyma cell necrosis, hepatic cord and connective tissue fibrosis, diminished growth, collapsed blood vessels, and lower body weight.

When exposed to lead, Nile tilapia (*Oreochromis niloticus*) showed decreased haemoglobin levels, red blood cell counts, and hematocrit. In fish, lead-induced oxidative stress led to synapse loss and problems with neurotransmitters. Due to fatal and sublethal lead exposure, tench (*Tincatinca*) showed altered immunological parameters (Tamele & Vázquez, 2020).

Fish bioaccumulate lead, which primarily damages the gills, liver, spleen, and kidney. *Acipensersinensis*, a Chinese sturgeon, experienced mobility issues and malformations as a result of lead buildup (Mondal et al., 2018).

2.3 Bioaccumulation of metals in different organs of fishes

Significant scientific interest has been generated by the occurrence of heavy metal bioaccumulation in the tissues and organs of several fish species. Intriguing patterns in the accumulation of certain heavy metals, including chromium, cadmium, lead, mercury, and zinc, have been discovered by research on these substances (Figueiredo-Fernandes et al., 2007).

Chromic accumulation showed a notable hierarchy, with the kidney showing the highest concentration and the heart, muscle, and gills following. This was seen in species including *Clariasgariepinus*, *Hydrocynusforskahlia*, and *Hydrocynusbebeooccidentalis*. The liver and kidney, followed by the gills and muscle, were the predominant locations for chromium buildup in *Coregonuslavaretus*. Contrarily, in diverse species including *Pleuronectesplatessa*, *Raja clavata*, and several *Cyprinus carpio* populations, cadmium accumulation followed varied patterns (Lushchak et al., 2009).

Another heavy metal of concern, lead, showed a variety of bioaccumulation patterns. The muscle and gills of animals like *Hydrocynusforskahlia* and *Hydrocynusbebeooccidentalis* showed greater accumulation than the heart and kidney. *Coregonuslavaretus*, on the other

hand, showed more lead buildup in the muscle, liver, kidney, and gills. The accumulation pattern of heavy metals like cadmium, lead, and zinc differed according to the element in question, as was interestingly shown by the study on *Pelteobagrus fulvidraco* (Ebrahimi et al., 2020).

Mercury, known for its harmful effects, showed its own distinct patterns of buildup. Mercury buildup in *Coregonus lavaretus* was arranged in a hierarchy, with the kidney, liver, muscle, and gills being the most noticeable locations. A different pattern was shown in *Cyprinus carpio*, where the gills, kidney, muscle, liver, and intestine all contributed significantly to mercury buildup. On the other hand, *Oreochromis niloticus* demonstrated how mercury tended to build up higher in the kidney, liver, muscle, and skull (Zulkipli et al., 2021).

Last but not least, research into zinc buildup uncovered some fascinating patterns. The sequence of the gills, kidney, liver, and intestine in zinc accumulation in *Pleuronectes platessa* was evident. According to Chaari and Matoussi (2012), *Channa punctatus* showed a variety of trends, with zinc accumulation concentrated mostly in the muscle, liver, kidney, intestine, and gills.

3. Conclusion

The information discussed in this review emphasizes how dangerous heavy metal contamination in aquatic ecosystems is and how seriously it affects aquatic life. Chromium, cadmium, lead, mercury, and zinc have all been shown to have toxic effects that change organ systems, metabolic processes, and even reproductive functions. Due to the cumulative effects of these heavy metals and their propensity for bioaccumulation, a serious situation is present that needs to be addressed. Prioritizing sustainable practices that reduce the release of heavy metals into ecosystems is crucial as industrialization develops. The complex web of ecological relationships necessitates a comprehensive strategy to handle the numerous problems caused by heavy metal poisoning. We can create the conditions for a healthier and more balanced aquatic ecosystem by implementing continuous monitoring system, upholding strict rules, and implementation of cutting-edge technologies. Finally, the present analysis serves as a wake-up call to acknowledge the gravity of the problem and ignite group efforts to protect our priceless aquatic ecosystems for coming generations.

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