



Cadmium Toxicity On Aquatic Ecosystem: A Review

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Abstract

Water is a crucial life-supporting element, vital for various purposes, but its pollution is a pressing concern due to rapid industrialization and population growth. This review article focuses on the toxicity of cadmium (Cd) in aquatic environments, impacting aquaculture and overall ecosystems. The article highlights Cd's sources, distribution, bioaccumulation, and its adverse effects on aquatic organisms, including fish and crustaceans. The aim is to raise awareness about the importance of effective management strategies to safeguard aquatic ecosystems.

Key words- Cadmium, Heavy metal, Fishes, Crustaceans, Aquatic ecosystem

1. Introduction

Water, a vital life-supporting element, plays a critical role in promoting overall well-being. Being a precious natural resource, it serves diverse purposes, including supporting aquaculture, industry, irrigation, and domestic needs. Our country is blessed with abundant water resources, but rapid industrialization, population growth, and irresponsible use of natural resources have resulted in a significant rise in water pollution. This problem has particularly affected India's major rivers, with the Ganga and Yamuna facing the most substantial impacts (Ajmal *et al.*, 1985; Jain and Sharma, 2001; Kaushik *et al.*, 2003; Kumar *et al.*, 2007).

Cadmium (Cd) is a widespread toxic contaminant in the environment, presenting a pressing and recurring global challenge (Gao *et al.*, 2018). Due to its persistence and high bioavailability, Cd poses a severe threat to ecosystems, especially aquatic environments (Chen *et al.*, 2019; Islam *et al.*, 2015). Anthropogenic activities are responsible for 90.4% of Cd released into water (Yuan *et al.*, 2019), making the aquatic environment the primary receptor for this toxic substance. Cd has been observed to cause various harmful effects on aquatic organisms, including growth inhibition and compromised pigment contents in microalgae (Zamani-Ahmadm Mahmoodi *et al.*, 2020), abnormal embryonic development in *Daphnia magna* (Perez and Hoang, 2018), retarded cell cycle and increased apoptosis in crab (Cheng *et al.*, 2021), disruption of energy metabolism in *Carassius auratus gibelio* (Cai *et al.*, 2020), and alterations in gut microbiota in Chinese toad larvae (Ya *et al.*, 2019).

Aquaculture, a vital component of the Indian economy and a livelihood for millions, heavily depends on the aquatic ecosystem. Significant advancements in fish culture techniques have led to substantial growth in fish production, contributing to a total fish production of 6.2 million metric tonnes in India in 2002-2003 (Dwivedi *et al.*, 2004). Fish not only provide essential nutrition but are also considered a delicacy across the country (Prasad and Kumar, 2007). However, the introduction of pollutants into the water has adverse effects on the aquatic ecosystem and ultimately impacts fish health, leading to economic losses for fish farmers. To

mitigate these losses and safeguard public health, it is crucial to monitor and comprehend the pathophysiology of various toxicants.

This review article delves into the toxicity of cadmium in aquatic environments, covering its sources, distribution, bioaccumulation, and potential health implications. The aim is to raise awareness about effective management and mitigation strategies to safeguard aquatic ecosystems.

2. What are heavy metal

The term “heavy metals” encompasses a group of metals and metalloids with an atomic density greater than 4000 kg/m³ (Edelstein & Ben-Hur 2018). Nearly all heavy metals are toxic to humans, even at low concentrations of metal ions (Saravanan *et al.* 2019;). Examples of heavy metals include copper, cadmium, zinc, chromium, arsenic, boron, cobalt, titanium, strontium, tin, vanadium, nickel, molybdenum, mercury, lead, and others.

Cadmium metal has specific properties that make it suitable for a wide variety of industrial applications. These include: excellent corrosion resistance, low melting temperature, high ductility, high thermal and electrical conductivity (u). It is used and traded globally as a metal and as a component in six classes of products, where it imparts distinct performance advantages. According to the US Geological Survey, the principal uses of cadmium in 2007 were: nickel–cadmium (Ni–Cd) batteries, 83%; pigments, 8%; coatings and plating, 7%; stabilizers for plastics, 1.2%; and other (includes non-ferrous alloys, semiconductors and photovoltaic devices), 0.8% (USGS, 2008, ATSDR, 2008).

While some heavy metals like copper, zinc, nickel, boron, iron, and molybdenum are essential for plant growth, excessive concentrations of these metals can harm both plants and animals. On the other hand, heavy metals such as lead, mercury, cadmium, and arsenic are not required for plant and animal growth. Soil pollution by heavy metals occurs due to the entry of industrial wastewater, sewage sludge, fertilizers, treated wastewater in land application, and soil mineral weathering (Edelstein & Ben-Hur 2018; Liu *et al.* 2018).

High exposure to heavy metals can lead to various diseases in humans, such as gastrointestinal and renal toxicity, cardiovascular problems, tumors, hematological disorders, depression, tubular and glomerular dysfunctions, and osteoporosis. Newborns, children, and adolescents are particularly vulnerable to heavy metal exposure, resulting in developmental difficulties and lower intelligence quotients. Many countries have set permissible limits for heavy metals in food to avoid their consumption. The major sources of heavy metal contamination are industrial wastewater from mining, pharmaceuticals, electroplating, rubber and plastics, metal finishing, tanneries, organic chemicals, pesticides, timber, and wood products (Manirethan *et al.* 2018). Runoff water carries heavy metals, polluting water sources due to industrial activities. Since all living organisms, including plants, animals, and microorganisms, rely on water, toxic metals can attach to microbial surfaces through bioaccumulation and even penetrate inside the cells, where they may undergo chemical changes during biochemical reactions involved in digestion.

3. Cadmium

Cadmium (Cd) has found extensive use in various industries such as batteries, ceramics, electronics, metal-finishing, pigments, insecticides, and more (Qiu *et al.* 2018b; Zheng *et al.* 2018). The elevated concentrations of cadmium in air, water, and soil are often observed near industrial sources, particularly nonferrous mining and metal handling activities. Cd is a highly toxic metal that can accumulate in the human body and cause irreversible harm to various biological systems, even at very low concentrations. It has been reported that Cd is more effectively retained in the respiratory system than in the gastrointestinal tract. Although the levels of cadmium in the air are generally below 10 ng/m³, it becomes a matter of significant concern in heavily industrialized areas (Schoeters *et al.* 2006).

Cadmium can enter the human body through diet and tobacco smoking. High concentrations of cadmium are found in mammal offal, such as kidneys and liver, as well as in certain types of fish, mussels, and clams from contaminated coastal areas. For example, edible crabs from polluted regions may contain Cd levels of around 30–50 mg/L (Overnell 1996). Non-smokers are highly affected by the consumption of cadmium-contaminated food materials, while tobacco smoking is a major source of exposure for smokers in non-

contaminated areas. A single cigarette contains approximately 1 to 2 µg Cd, with the actual intake dependent on the cigarette type, brand, and production location. Smokers consuming a pack per day may absorb around 1 µg of cadmium. Once inside the human body, Cd binds strongly to metallothioneins. Over 50% of Cd in the human body accumulates in the liver and kidneys due to their ability to coordinate metallothionein (Bernard 2008). Cd can eventually be eliminated through urine (Ikeda et al. 2018). However, the amount of Cd discharged daily in urine is very limited (0.005% to 0.01% of the total Cd content), leading to high retention in the body. Cd is known as a persistent toxin with a biological half-life of over 20 years. The kidney, as the primary storage organ for toxicants, is consistently the critical target organ that shows early signs of toxicity (Nordberg *et al.* 2007). Chronic exposure to elevated levels of cadmium can lead to liver damage, bone degeneration, blood disorders, and renal dysfunction. There is ample evidence in humans regarding the carcinogenic nature resulting from exposure to both Cd and Cd compounds (Chen 2012). The permissible limit of cadmium in drinking water, as recommended by the BIS, is 0.003 mg/L (BIS 1992).

4. Cadmium Global production

Cadmium, a widely but sparsely distributed element, can be found in the earth's crust at concentrations ranging from 0.1 to 5 ppm. It is primarily associated with zinc ores, zinc-bearing lead ores, and complex copper-lead-zinc ores. The annual cadmium refinery production in the United States declined between 2003 and 2006, and demand in the nickel-cadmium battery industry is increasing. However, primary production of cadmium may decrease as zinc prices rise. There were only a few companies producing primary cadmium in the United States in 2007, and the total cadmium recovery rate was relatively low, with a significant amount of cadmium being disposed of in waste or stockpiled. (UNEP 2008)

5. Effects of Cadmium in aquatic ecosystem

Cadmium enters the aquatic environment from numerous diffuse (eg. agricultural and urban run-off, atmospheric fall-out) and point sources, both natural and anthropogenic. Weathering and erosion of cadmium-containing rocks result in the release of cadmium not only to the atmosphere, but also to the soil and the aquatic system (directly and through the deposition of airborne particles) (UNEP 2008). Cadmium is released to the aquatic environment from a range of anthropogenic sources, including non-ferrous metal mining and smelting (from mine drainage water, waste water, tailing pond overflow, rainwater run-off from mine areas), plating operations, phosphate fertilizers, sewage-treatment plants, landfills, and hazardous waste sites (IARC, 1993).

Weathering and erosion are estimated to contribute 15000 tonnes of cadmium annually to the global aquatic environment, while atmospheric fall-out (of anthropogenic and natural emissions) is estimated to contribute between 900 and 3600 tonnes (UNEP, 2008).

6. Effect of Cadmium on fish

When fish are under stress due to the presence of Cadmium (Cd) in their aquatic environment, an abundance of reactive oxygen species (ROS) and reactive nitrogen species (RNS) radicals, surpassing their natural scavenging capabilities, accumulate in the fish's bodies. This accumulation leads to a cascade of oxidative damage reactions (Zheng *et al.*, 2017). The toxic effects of Cd on fish primarily manifest as oxidative stress caused by ROS, resulting in structural alterations in biological macromolecules, such as proteins and DNA. This leads to disruptions in DNA replication and repair processes, eventually culminating in pathological changes (Vaglio and Landriscina, 1999). In combination, these factors negatively impact enzymes responsible for regulating cell proliferation, differentiation, and apoptosis on the membrane, ultimately causing functional disturbances and bodily harm (Dai *et al.*, 2020).

The neurotoxicity induced by Cd stress poses a significant threat to the normal life activities of fish. Cd directly inhibits the biological activities of enzymes containing sulfhydryl groups when it binds to them. Additionally, Cd leads to a reduction in the content of various metabolic intermediates, resulting in severe damage to the fish's nervous system (Green and Planchart, 2018). Cd can enter the fish's brain tissue, affecting cerebellar functions, leading to balance issues during swimming. Moreover, Cd primarily causes

neuronal damage in the brain, inhibiting the formation of neural crest cells during early fish development, which leads to motor neuron damage and behavioral abnormalities (Monaco *et al.*, 2017).

A study on Nile tilapia demonstrated that increasing Cd levels in fish diets significantly decreased albumin concentrations and serum total protein ($p < 0.001$). Conversely, serum creatinine, glutamic-pyruvic transaminase (GPT), and aspartate aminotransferase (AST) levels increased significantly ($p < 0.001$). However, the concentration of urea-N was not significantly affected (Nogami *et al.*, 2000). Similarly, research on carp indicated that cadmium poisoning disrupts renal tubule reabsorption, leading to excessive calcium and phosphorus excretion in urine, resulting in decreased serum calcium and phosphorus levels. Furthermore, Cd can damage renal tubules, inhibit vitamin D activation, and negatively impact calcium and phosphorus metabolism (Ye, silbudak and Erdem, 2014). Another study on *Sparus aurata* found that Cd exposure altered the activity of GOT (glutamic-oxaloacetic transaminase) in the liver cytoplasm and serum (Vaglio and Landriscina, 1999).

In response to various stresses in the aquatic environment, fish exhibit significant inhibition or induction of stress-related genes in their bodies, allowing the toxic effects of stress to be alleviated through self-regulation (Petitjean *et al.*, 2019). Consequently, fish can modulate the generation of numerous antioxidant enzymes to reduce oxidative damage caused by ROS. Additionally, Cd induces abnormal expression of stress response protein genes, such as heat shock proteins and metallothioneins, as observed in studies on the liver of *Cyprinus carpio* and *Labeo rohita*. The expression levels of CYP1A, HSP47, HSP60, HSP70, HSP90, and MT-B genes significantly increased with prolonged Cd exposure time (Jiang *et al.*, 2016).

Cd can diminish the activities of various enzymes in fish, particularly antioxidant enzymes containing zinc and sulfhydryl groups. Moreover, Cd can bind to glutathione and metallothionein, essential thiol proteins responsible for antioxidant functions, thus reducing the body's ability to eliminate ROS and leading to oxidative damage (Cirillo *et al.*, 2012).

7. Effect of Cadmium on Crustaceans

Numerous studies have reported that Cd induces oxidative stress in most of the studied organisms, including crustaceans (Lin *et al.* 2017). When organisms encounter oxidative stress due to high concentrations of certain toxins like trace metals and xenobiotics, reactive oxygen species (ROS) are commonly produced (Ahmad *et al.* 2000). The production of ROS and free radicals increases with higher concentrations of metals or toxins, resulting in severe damages to lipids, DNA, and proteins (Wu *et al.* 2014). Structural responses in various invertebrate organs have proven to be valuable tools for characterizing the health status of organisms and assessing the impact of environmental contaminants in laboratory-exposed organisms (Sousa *et al.*, 2005; Sousa, 2003).

Das *et al.* (2018) observed decreased activities of antioxidant enzymes in the hepatopancreas, gills, and muscles of organisms exposed to higher concentrations of Cd. The hepatopancreas, an essential digestive organ, plays crucial roles in nutrient absorption, secretion, and the production of digestive enzymes in crustaceans (Wu *et al.* 2014). This organ is analogous to the liver in higher organisms (Chiodi Boudet *et al.* 2015), making it vulnerable to damage to its cellular structure or changes in the structure of epithelial cells, potentially affecting its function. Due to its role as the main depository organ for toxic metals, the hepatopancreas is extensively studied as a marker to diagnose harmful effects of pollutants or toxic chemicals (Zhou *et al.* 2017).

In their study, Das *et al.* (2018) compared the cellular structures of the hepatopancreas of healthy individuals (control group) with those treated with 5 mg/kg of cadmium. They found significant changes in the histological structure of the mud shrimp *A. Edulis*, including the disappearance of the boundary of epithelial cells and separation of cells from the basal lamina. Similar changes in epithelial cells have been observed in other studies examining the effects of exposure to noxious metals or toxic chemicals on the structure of the hepatopancreas in various crustaceans (Chiodi Boudet *et al.* 2015).

Revathi *et al.* (2011) investigated the effect of cadmium chloride (25 µg/l) on oogenesis in *Macrobrachium rosenbergii* prawns. Cadmium exposure resulted in decreased Gonado Somatic Index (GSI), disrupted

ovarian development, and abnormal cellular architecture in the gills, hepatopancreas, and ovaries. Intact prawns showed altered hormone secretion, affecting gonadal maturation, while eyestalk-ablated prawns exhibited changes in yolk material and oocyte membrane thickness after exposure. These findings emphasize the potential risks of cadmium in freshwater ecosystems and prawn populations.

Soegianto *et al.* (2013) discovered that cadmium exhibited acute toxicity to *Macrobrachium sintangese*, with a 96-hour LC50 value of 86 µg/L. The research also demonstrated the bioaccumulation of cadmium in different tissues, with the highest accumulation in the gills, followed by the hepatopancreas and abdominal muscle. Histopathological examination revealed severe damage to the gills and hepatopancreas due to cadmium exposure, with improvements noted after transferring the prawns to cadmium-free media. The study suggested that *Macrobrachium sintangese* could serve as a valuable test organism for toxicity assays due to its sensitivity to cadmium.

Wu *et al.* (2008) observed histopathological alterations in the hepatopancreas and induction of biochemical markers (hemolymphatic glutamate-oxalacetate transaminase and glutamate-pyruvate transaminase), indicating hepatopancreatic injury in *L. Vannamei*.

Boudet *et al.* (2014) assessed the effects of Cd on *P. Argentinus* shrimp from two lagoons with different environmental quality. Cd exposure led to histological changes and increased LPO levels in both populations, but shrimp showed recovery capacity after Cd removal...

Cadmium (Cd) is one of the most toxic heavy metals for humans; the main source of nonoccupational exposure to Cd includes smoking, air, and food and water contaminated by Cd (Nagata *et al.*, 2005). In addition, herbal medicine is another source of Cd. The World Health Organization (WHO) estimates that 4 billion people or 80 percent of the world population, presently use herbal medicine (Naithani *et al.*, 2010). Several articles have reported of adverse effects of these herbal preparations due to the presence of high level of heavy metals such as Cd, lead, chromium, nickel, etc. (Naithani *et al.*, 2010). Saeed *et al.* (2010) investigated twenty-five herbal products. The results revealed that the concentrations of some heavy metals, including Cd, were far greater than the permissible limits proposed by the International Regulatory Authorities for herbal drugs. Acute or chronic exposure of Cd causes respiratory distress, lung, breast and endometrial cancers, cardiovascular disorders and endocrine dysfunction (Åkesson *et al.*, 2008; Chang *et al.*, 2009; Messner *et al.*, 2009; Nagata *et al.*, 2005; Naithani *et al.*,).

In addition, Cd is a common inorganic contaminant of coastal sediments and waters due to anthropogenic pollution and natural sources (Ivanina *et al.*, 2008, 2010; Sokolova *et al.*, 2004). It can be accumulated in aquatic animals (eg. crabs, shrimps, oysters and mussels) after entering through different way such as respiratory tract, digestive tract, surface penetration etc. (Dailianis & Kaloyianni, 2004; Dailianis *et al.*, 2009; Ivanina *et al.*, 2008, 2010; Li *et al.*, 2008; Sokolova, 2004; Sokolova *et al.*, 2004; Wang L. *et al.*, 2001, 2008; Wang Q. *et al.*, 2003; Zhao *et al.*, 1995). It is seriously harmful to the growth of aquatic life and survival, resulting in decline of their populations. At the same time, as aquatic food products, these animals exposed to Cd might threaten human health.

8. Mechanism of Toxicity

Cadmium affects cell proliferation, differentiation, and apoptosis. These activities interact with DNA repair mechanism, the generation of reaction oxygen species (ROS) and the induction of apoptosis (Rani *et al.*, 2014.). Cadmium binds to the mitochondria and can inhibit both cellular respiration and oxidative phosphorylation at low concentration (Patrick, 2003).

It results in chromosomal aberrations, sister chromatid exchange, DNA strand breaks, and DNA- protein crosslinks in cell lines. Cadmium causes mutations and chromosomal deletions potentially (Joseph, 2009). Its toxicity involves depletion of reduced glutathione (GSH), binds sulfhydryl groups with protein, and causes to enhance production of reactive oxygen species (ROS) such as superoxide ion, hydrogen peroxide, and hydroxyl radicals. Cadmium also inhibits the activity of antioxidant enzymes, such as catalase, manganese-superoxide dismutase, and copper/zinc-dismutase (Filipic, 2012). Metallothionein is a zinc – concentrating protein that contains 33% cysteine. Metallothionein also can act as a free- radical scavenger. It scavenges

hydroxyl and superoxide radicals (Liu *et al*, 2009). Generally, the cells that contain metallothioneins are resistant to cadmium toxicity. On the other hand, the cells that cannot synthesize metallothioneins are sensitive to cadmium intoxication (Han *et al*, 2015). Cadmium can modulate the cellular level of Ca²⁺ and the activities of caspases and nitrogen-activated protein kinases (MRPKs) in the cells, in which these processes cause apoptosis indirectly (Brama *et al*, 2012).

9. Conclusion

Cadmium is a toxic heavy metal widely used in industries, leading to its release into the aquatic environment from various sources. The reviewed studies demonstrate that Cd poses significant threats to aquatic organisms, including fish and crustaceans, through oxidative stress and disruptions to biological processes. As a crucial component of the Indian economy, aquaculture faces economic losses due to water pollution caused by Cd and other contaminants. To safeguard public health and aquatic ecosystems, effective monitoring and mitigation strategies are imperative. This review article emphasizes the urgency to implement measures to reduce Cd contamination, preserve water quality, and ensure the sustainability of aquaculture and the environment.

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