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Cadmium and Nickel Contamination in the Kali River and Its Ecotoxicological Impact on Hepatic and Hematological Health of *Channa punctatus* (Bloch.)

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ABSTRACT

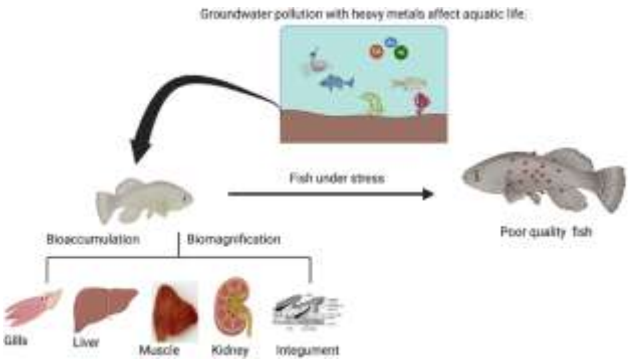
Cadmium and nickel contamination in freshwater ecosystems has become a significant environmental issue, mainly because these metals stick around for a long time, accumulate in living organisms, and can be toxic to aquatic life. The Kali River in western Uttar Pradesh, India, is particularly affected, receiving a mix of industrial, agricultural, and municipal waste that raises the levels of these metals. This review pulls together existing research on where these metals come from, how they spread, and their harmful effects on *Channa punctatus*, a fish often used as a bioindicator. It highlights how these metals accumulate in the fish, the oxidative stress they cause in the liver, and the changes in blood health due to exposure. Cadmium and nickel can mess with the fish's antioxidant defenses, increase harmful reactive oxygen species, and lead to liver damage, while also causing anemia and weakening the immune system. These health issues pose risks not just to the fish but also to the stability of their populations and the overall health of the ecosystem. Plus, the accumulation of these metals in edible fish tissues raises concerns for human consumers. The review emphasizes the need to include biochemical and hematological markers in regular monitoring to better understand heavy metal pollution and improve river management strategies.

Introduction

Freshwater ecosystems are facing increasing threats from contamination by heavy metals, a problem that's largely driven by rapid industrialization, urban growth, and agricultural practices. Cadmium (Cd) and nickel (Ni) stand out as particularly concerning pollutants due to their toxicity, persistence in the environment, inability to break down, and their tendency to accumulate in aquatic life (Jaishankar *et al*., 2014; Tchounwou *et al*., 2012). Rivers that receive untreated industrial waste and municipal runoff often become dumping grounds for these metals, leading to long-lasting damage to the ecosystem. The Kali River, located in western Uttar Pradesh, India, is unfortunately recognized as one of the most polluted river systems in the area. Discharges from electroplating industries, metal processing units, textile factories, battery manufacturing, and domestic sewage have been shown to raise cadmium and nickel concentrations in the river water and sediments beyond acceptable levels (Singh *et al*., 2018; CPCB, 2020). When environmental conditions change, the metals associated with sediments can be released back into the water, making them more accessible to aquatic life. Fish are commonly used as bioindicators for heavy metal pollution because they are directly exposed to these harmful substances, are ecologically significant, and can accumulate metals in their essential organs. *Channa punctatus* (Bloch), a prevalent freshwater fish, is particularly well-suited for ecotoxicological studies thanks to its bottom-feeding behavior, high tolerance for polluted environments, and its value in commercial fisheries (Authman *et al*., 2015). The liver is vital for detoxifying and storing metals, making it especially prone to oxidative damage from metal exposure. Cadmium and nickel can lead to the production of reactive oxygen species (ROS), which in turn causes oxidative stress, lipid peroxidation, and disrupts the body's antioxidant defenses (Flora *et al*., 2008). Hematological parameters serve as sensitive indicators of physiological stress. Changes in red and white blood cell counts can signal disruptions in oxygen transport, immune function, and the overall metabolic health of fish that have been exposed to heavy metals (Fazio, 2019). Thus, evaluating hepatic oxidative stress along with hematological changes gives us a thorough understanding of how cadmium and nickel contamination affects the Kali River ecosystem.

Sources and Distribution of Cadmium and Nickel in the Kali River

In western Uttar Pradesh, the Kali River is under heavy strain from human activities, making it a hotspot for cadmium (Cd) and nickel (Ni) pollution. The primary sources of these metals are a mix of industries, such as electroplating, metal finishing, alloy manufacturing, battery production, textile dyeing, sugar mills, distilleries, and municipal sewage that flows into the river (Singh *et al*., 2018; CPCB, 2020). On top of that, agricultural runoff filled with phosphate fertilizers and pesticides further increases the cadmium levels in the river (Jaishankar *et al*., 2014). Once they're released, cadmium and nickel can be found in both dissolved and particulate forms. Because they have a strong attraction to organic matter and fine sediments, these metals tend to build up mainly in river sediments, which serve as long-term storage sites (Varol & Şen, 2012). Factors like seasonal changes in flow, pH, redox potential, and organic content play a big role in how these metals move around and how available they are for living organisms, especially during the monsoon and post-monsoon seasons. When contaminated sediments get stirred up, it increases the uptake of these metals by bottom-dwelling creatures and fish like *Channa punctatus*. Studies on the spatial distribution of the Kali River have consistently shown that there are higher levels of cadmium (Cd) and nickel (Ni) in the downstream areas and those affected by industrial activities. These levels often surpass the safe limits recommended for freshwater ecosystems (Singh *et al*., 2018). This ongoing contamination brings about serious ecological concerns and promotes the transfer of these metals through the aquatic food web.



Bioaccumulation of Cadmium and Nickel in *Channa punctatus* (bloch.)

The bioaccumulation of cadmium (Cd) and nickel (Ni) in freshwater fish is a well-known result of long-term exposure to polluted aquatic environments. In river systems like the Kali River, these metals make their way into the bodies of *Channa punctatus* mainly through the gills, gastrointestinal tract, and skin. This process depends on factors like the type of metal, its concentration, and how long the fish are exposed (Authman *et al*., 2015). Additionally, bottom-feeding species tend to accumulate more metals through their diet by ingesting contaminated sediments and benthic organisms. When it comes to different tissues, the liver stands out as the one that accumulates the most cadmium and nickel, with the kidney, gills, and muscle following behind (Jezierska & Witeska, 2006). This tendency is mainly due to the liver's crucial functions in detoxifying substances, processing nutrients, and storing metals. It also produces metallothioneins, which are small, cysteine-rich proteins that help bind heavy metals and lessen their toxicity (Roesijadi, 1992). However, if exposure to these metals continues for a long time, it can overwhelm the liver's ability to detoxify, resulting in cellular damage caused by the metals. Cadmium has a notable tendency to bind with sulfhydryl groups, leading to disruptions in calcium homeostasis. In contrast, nickel can hinder enzymatic functions and compromise membrane integrity (Tchounwou *et al*., 2012). Environmental elements like water pH, hardness, dissolved organic matter, and sediment traits significantly impact the bioavailability and accumulation of these metals. Additionally, seasonal fluctuations in the Kali River, especially during low-flow conditions, may intensify the metal burdens found in tissues. When cadmium and nickel accumulate in *Channa punctatus*, it not only affects the fish's bodily functions but also enables these toxins to transfer through the food web, leading to ecological dangers and possible health risks for humans consuming contaminated fish.

Cadmium and Nickel–Induced Hepatic Oxidative Stress

Mechanisms of Oxidative Stress

Cadmium (Cd) and nickel (Ni) may not be redox-active metals, but they still manage to cause some serious trouble in the liver by indirectly triggering oxidative stress. These metals mess with the mitochondrial electron transport chain, which leads to electron leakage and an overproduction of reactive oxygen species (ROS) (Flora *et al*., 2008; Jaishankar *et al*., 2014). On top of that, cadmium and nickel can deplete the body's internal antioxidants by binding to sulfhydryl groups, which means there's less of those protective molecules like glutathione available. When antioxidant enzymes are inhibited, it only makes the ROS problem worse, resulting in lipid peroxidation, protein oxidation, DNA damage, and destabilization of membranes. All of this ultimately disrupts normal liver function in fish that are exposed to these metals.

Alteration of Antioxidant Defense Systems

In *Channa punctatus*, exposure to cadmium and nickel has been shown to significantly disrupt the liver's antioxidant defense system. Both experimental and field studies have reported a decrease in the activity of superoxide dismutase (SOD), along with a reduction in catalase (CAT) and glutathione peroxidase (GPx)—all crucial enzymes for detoxifying reactive oxygen species (ROS) (Livingstone, 2001). Additionally, there have been notable drops in reduced glutathione (GSH) levels, which suggests a disturbance in redox balance. At the same time, increased levels of malondialdehyde (MDA), a by-product of lipid peroxidation, act as a reliable marker for oxidative damage to liver membranes (Lushchak, 2011).

Histopathological Correlates

Oxidative stress-related liver damage in *Channa punctatus* is often linked to noticeable changes in tissue structure. These include the degeneration of liver cells, the formation of vacuoles in the cytoplasm, dilation of blood vessels, cell death, and the shrinking of cell nuclei. Such alterations indicate significant metabolic and functional issues within the liver, which hinder its ability to detoxify and perform its essential functions, especially when exposed to chronic levels of cadmium and nickel.Hematotoxic Effects of Cadmium and Nickel

Cadmium (Cd) and nickel (Ni) exposure can have a significant impact on the blood health of freshwater fish, indicating the stress they face from metal contamination. In *Channa punctatus*, chronic exposure to these metals often leads to a substantial decrease in red blood cell (RBC) count, hemoglobin (Hb) concentration, and hematocrit (PCV) values, which suggests the onset of anemia (Fazio, 2019). These changes could be a result of inhibited red blood cell production, increased destruction of these cells, or disrupted iron metabolism due to the metals interfering with the tissues that produce blood. Cadmium disrupts the synthesis of heme and increases the fragility of red blood cell membranes due to oxidative damage, while nickel affects the enzymes that are vital for normal blood cell production (Tchounwou *et al*., 2012). Additionally, changes in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) have been noted, indicating shifts in red cell shape and their ability to carry oxygen effectively. Leukocyte responses are key indicators of stress caused by metals. When white blood cell (WBC) counts rise, it often signals an acute stress reaction. However, if exposure continues over time, it can lead to leukopenia and changes in the types of leukocytes present, such as lymphopenia and neutrophilia, which indicate a weakened immune system (Authman *et al*., 2015). These changes in blood health can hinder oxygen transport, reduce immune function, and ultimately affect the overall health of fish populations, making them less likely to survive and adapt in polluted environments. Therefore, monitoring these blood parameters is crucial for evaluating the toxicity of cadmium and nickel in freshwater ecosystems.

Ecotoxicological and Environmental Implications

Chronic pollution of freshwater ecosystems with cadmium (Cd) and nickel (Ni) brings about serious ecotoxicological challenges for aquatic organisms and the environment as a whole. In rivers such as the Kali River, the steady flow of these metals leads to bioaccumulation and biomagnification at different trophic levels. This can result in negative effects on the survival, growth, and reproductive capabilities of fish populations (Jaishankar *et al*., 2014). For example, the oxidative stress in the liver and blood disorders seen in *Channa punctatus* highlight systemic toxicity that can lower their fitness, change their behavior, and increase their risk of disease, ultimately threatening the stability of their populations. At the community level, heavy metal pollution can really disrupt the balance of species composition and biodiversity. It tends to favor those species that can withstand the pollution while driving out the more sensitive ones, which in turn destabilizes aquatic food webs (Authman *et al*., 2015). Cadmium and nickel that settle in sediments act as lasting sources of contamination, extending ecological stress even after efforts to cut down on effluent discharge. This ongoing exposure can compromise vital ecosystem services, including the productivity of fisheries and the quality of our water. From a health standpoint, *Channa punctatus* is a popular fish that many people eat, but there's a concerning issue with cadmium and nickel building up in its tissues. Eating fish that are contaminated over time can lead to serious health problems, including kidney and liver damage, as well as an increased risk of cancer (Tchounwou *et al*., 2012). This makes it crucial to monitor metal levels, biological responses, and assess risks effectively. We need to implement strong wastewater treatment solutions, enforce pollution control policies, and carry out regular biomonitoring to help prevent environmental harm and protect public health.

Conclusion

This review focuses on the serious ecological threat that cadmium and nickel contamination poses to the Kali River and its impact on the health of *Channa punctatus*. The continuous release of these heavy metals from industrial, agricultural, and domestic sources has caused them to linger in the water and sediments, allowing them to accumulate in fish tissues, particularly in the liver. The way cadmium and nickel preferentially build up in liver tissues disrupts the balance of cellular redox, leading to an overproduction of reactive oxygen species and a breakdown of antioxidant defenses. These processes ultimately result in oxidative stress and damage to liver cells, as indicated by various biochemical and histopathological changes. In addition to harming the liver, cadmium and nickel significantly affect blood health, causing anemia, changes in red blood cell parameters, and immune system issues. These blood-related disturbances can compromise oxygen delivery, reduce metabolic efficiency, and weaken disease resistance, ultimately lowering the overall fitness and survival rates of fish in polluted environments. On an ecosystem scale, these physiological challenges can lead to population declines, shifts in species diversity, and disruptions in aquatic food webs. The presence of cadmium and nickel in *Channa punctatus*, a fish that many people enjoy eating, raises alarming concerns about health risks for humans through their diet. In this regard, markers of oxidative stress in the liver and various blood parameters are emerging as reliable and sensitive tools for tracking heavy metal pollution. The results of this study stress the urgent need for strict pollution control measures, regular monitoring, and effective management of the Kali River to restore its ecological health and ensure the safety of both aquatic life and public health.

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