BHARATI INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY RESEARCH & DEVELOPMENT (BIJMRD)

(Open Access Peer-Reviewed International Journal)

DOI Link : https://doi.org/10.70798/Bijmrd/03120003

Available Online: www.bijmrd.com|BIJMRD Volume: 3| Issue: 12| December 2025| e-ISSN: 2584-1890

Nickel Toxicity in Freshwater Fish: Biochemical and Histopathological Insights with Special Reference to *Channa punctatus*

Pralay Mahapatra

Research Scholar, DR. C. V. Raman University, Vaishali, Bihar.

Abstract:

Heavy metal contamination of freshwater ecosystems has emerged as a major environmental concern due to its persistence, bioaccumulative nature, and adverse biological effects. Among these metals, nickel (Ni) occupies a unique position as an essential trace element at low concentrations but becomes highly toxic when present beyond permissible limits. Industrial effluents, mining activities, agricultural runoff, and fossil fuel combustion are primary contributors to nickel enrichment in aquatic environments. Fish, owing to their ecological relevance and sensitivity to pollutants, serve as reliable bioindicators of metal-induced toxicity. Channa punctatus, a widely distributed freshwater teleost in the Indian subcontinent, has been extensively employed in ecotoxicological investigations due to its adaptability and well-characterized physiology.

This mini review synthesizes current knowledge on the toxic effects of nickel in freshwater fish, with particular emphasis on biochemical alterations and histopathological damage reported in Channa punctatus. The review highlights the influence of physicochemical water parameters on nickel bioavailability, summarizes reported LC_{50} values, and discusses oxidative stress–mediated biochemical disruptions in liver tissues alongside structural damage in gills. By integrating biochemical and histopathological biomarkers, this review underscores their utility in early detection of sub-lethal nickel toxicity and environmental monitoring. The paper also identifies key research gaps and future directions for developing biomarker-based frameworks for freshwater ecosystem risk assessment.

Keywords: *Nickel Toxicity, Freshwater Fish, Channa punctatus, Oxidative Stress, Histopathology, Aquatic Ecotoxicology.*

1. Introduction

Aquatic ecosystems are among the most vulnerable components of the biosphere, continuously exposed to a wide array of anthropogenic contaminants arising from rapid industrialization, urban expansion, and intensified agricultural practices. Among these pollutants, heavy metals pose a particular threat due to their non-biodegradable nature, long environmental persistence, and tendency to bioaccumulate and biomagnify within aquatic food webs (Burger &Gochfeld, 2000; Tchounwou et al., 2012). Chronic exposure to heavy metals can disrupt physiological homeostasis in aquatic organisms, leading to impaired growth, reproduction, and survival, ultimately destabilizing freshwater ecosystems.

Nickel (Ni) is widely used in electroplating, alloy manufacturing, battery production, pigment industries, and fossil fuel combustion, making it a common contaminant of freshwater environments (Cempel& Nikel, 2006; Gupta & Rajamani, 2020). Although nickel is considered an essential trace element involved in enzymatic regulation and metabolic processes, its beneficial role is strictly concentration-dependent. At elevated levels, nickel becomes highly toxic, inducing oxidative stress, enzyme inhibition, and cellular damage in aquatic organisms (Pandey et al., 2020). Once released into water bodies, nickel exists in dissolved and particulate forms, and its bioavailability is strongly influenced by physicochemical parameters such as pH, temperature, dissolved oxygen, hardness, and organic matter content (Campbell, 1995; Adhikari & Ayyappan, 2020).

Fish are extensively employed as sentinel organisms in aquatic toxicology because of their ecological relevance, economic value, and high sensitivity to waterborne pollutants. They readily accumulate metals through gills, skin, and diet, making them reliable indicators of environmental contamination (Farag & Oshima, 2020). Among freshwater teleosts, *Channa punctatus* (spotted snakehead) has emerged as a preferred experimental model in ecotoxicological studies, particularly in the Indian subcontinent. Its wide geographical distribution, hardiness, and well-characterized physiology allow detailed investigation of sub-lethal toxic effects under controlled laboratory conditions (Sharma et al., 2017; Sinha et al., 2021).

Nickel toxicity in fish has been shown to elicit a broad spectrum of biochemical disturbances, particularly in metabolically active organs such as the liver. The liver serves as the principal site of detoxification and biotransformation, and is therefore one of the earliest organs to exhibit biochemical alterations following metal exposure. Studies have consistently reported nickel-induced oxidative stress characterized by elevated lipid peroxidation and disrupted antioxidant defense systems, including altered activities of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) (Kumar et al., 2019; Gupta & Rajamani, 2020). These biochemical perturbations act as sensitive early-warning biomarkers of sub-lethal metal stress.

In addition to biochemical changes, histopathological alterations provide direct morphological evidence of metal-induced toxicity. The gills, being the primary interface between fish and the aquatic environment, are particularly susceptible to waterborne nickel exposure. Structural changes such as epithelial lifting, lamellar fusion, hyperplasia, and necrosis have been widely documented in nickel-exposed fish, reflecting impaired respiratory and osmoregulatory functions (Mishra et al., 2018; Pathak & Gupta, 2019). When integrated with biochemical biomarkers, histopathological assessments offer a comprehensive understanding of the mechanistic pathways underlying nickel toxicity.

Despite substantial progress in heavy metal ecotoxicology, studies on nickel have received comparatively less attention than those on cadmium, lead, or mercury, especially with respect to chronic and sub-lethal exposure scenarios. Moreover, many existing studies assess biochemical or histopathological endpoints in isolation, limiting their ecological interpretability. An integrated synthesis of available literature focusing on *Channa punctatus* is therefore essential to consolidate existing knowledge, identify research gaps, and support the development of biomarker-based frameworks for freshwater environmental monitoring.

Table 1. Summary of Representative Studies on Nickel Toxicity in Freshwater Fish

Author(s) & Year	Fish Species	Nickel Form / Concentration	Exposure Duration	Major Findings	Organs Studied
Sharma et al., 2017	*Channa punctatus*	$Ni(NO_3)_2$ (0.5–2.5 mg/L)	21 days	Elevated SGPT and ALP; hepatocellular damage	Liver
Javed & Usmani,	*Catlacatla*	$NiCl_2$ (1.0–4.0	30 days	Reduced protein levels; increased lipid	Liver,

2019		mg/L)		peroxidation	gills
Mishra et al., 2018	*Heteropneustes fossilis*	$NiSO_4$ (1.5 mg/L)	28 days	Lamellar fusion and epithelial lifting	Gills
Gupta & Rajamani, 2020	*Oreochromis mossambicus*	$NiCl_2$ (2.0 mg/L)	14 days	Suppressed antioxidant enzymes; oxidative stress	Liver
Rajesh et al., 2021	*Labeorohita*	$NiCl_2$ (0.75–3.0 mg/L)	28 days	Vacuolization and necrosis of hepatocytes	Liver
Sinha et al., 2021	*Channa punctatus*	Ni-contaminated effluent	20 days	Oxidative stress and tissue damage	Liver, gills

2. Sources and Environmental Fate of Nickel in Aquatic Systems

Nickel (Ni) enters aquatic environments through a combination of natural processes and anthropogenic activities, with the latter contributing most significantly to elevated concentrations in freshwater systems. Natural inputs include weathering of nickel-bearing rocks, volcanic emissions, and soil erosion, which typically release nickel at low background levels that rarely pose ecological risks (Alloway, 2013; Cempel& Nikel, 2006). In contrast, anthropogenic sources often result in localized but substantially higher nickel loads, leading to chronic contamination of rivers, lakes, and reservoirs.

Industrial activities represent the dominant source of nickel pollution in freshwater ecosystems. Effluents from electroplating, alloy and stainless-steel manufacturing, mining and metallurgical operations, battery production, and pigment industries frequently contain soluble nickel salts that are discharged into surface waters with inadequate treatment (Gupta & Rajamani, 2020; Tchounwou et al., 2012). In developing countries, rapid industrial expansion coupled with weak enforcement of environmental regulations has intensified nickel inputs into aquatic systems. Agricultural runoff and municipal sewage further contribute to nickel loading through the application of phosphate fertilizers, sludge disposal, and urban waste discharge (Singh et al., 2022). Atmospheric deposition from fossil fuel combustion and vehicular emissions also acts as a diffuse but persistent source of nickel, particularly in urban and peri-urban water bodies (Cempel& Nikel, 2006).

Once introduced into aquatic environments, the environmental fate of nickel is governed by complex physicochemical interactions that determine its mobility, speciation, and bioavailability. Nickel occurs in water in both dissolved and particulate forms, with the dissolved fraction generally considered more bioavailable to aquatic organisms (Campbell, 1995). The speciation of nickel is strongly influenced by pH, redox potential, water hardness, and the presence of organic ligands. Under acidic conditions, nickel remains largely in its free ionic form (Ni^{2+}), which is readily taken up by aquatic biota and exhibits higher toxicity. In contrast, increased water hardness and alkalinity promote the formation of nickel–carbonate or nickel–hydroxide complexes, reducing its bioavailability and acute toxicity (Adhikari & Ayyappan, 2020).

Sediments play a critical role in regulating the long-term fate of nickel in aquatic ecosystems. Nickel readily adsorbs onto suspended particulate matter and settles into bottom sediments, where it may remain sequestered for extended periods (Rainbow, 2002). However, changes in environmental conditions, such as shifts in pH, oxygen depletion, or resuspension during flooding, can remobilize sediment-bound nickel back into the water column, reintroducing it into the biologically active phase (Froehlich & Riget, 2014). This dynamic exchange between water and sediment compartments complicates risk assessment, as short-term measurements of dissolved nickel may underestimate long-term ecological exposure.

Biologically, nickel present in the water column can be taken up directly through the gills or indirectly through dietary intake. The gills, owing to their large surface area and direct contact with water, serve as a primary route for nickel absorption, particularly in its ionic form. Once absorbed, nickel is transported via the bloodstream and preferentially accumulates in metabolically active organs such as the liver, kidney, and gills (Farkas et al., 2018). The liver functions as a major site for metal sequestration and detoxification, while the gills reflect early exposure due to their continuous interaction with the surrounding medium.

The persistence of nickel in aquatic systems and its potential for remobilization underscore the importance of understanding its environmental fate rather than relying solely on concentration-based assessments. Long-term exposure to even low, sub-lethal concentrations of nickel can exert cumulative toxic effects on aquatic organisms, particularly fish, through oxidative stress, metabolic disruption, and tissue damage (Pandey et al., 2020). Therefore, a comprehensive evaluation of nickel pollution must integrate information on sources, chemical speciation, sediment interactions, and biological uptake to accurately assess ecological risk and inform effective management strategies.

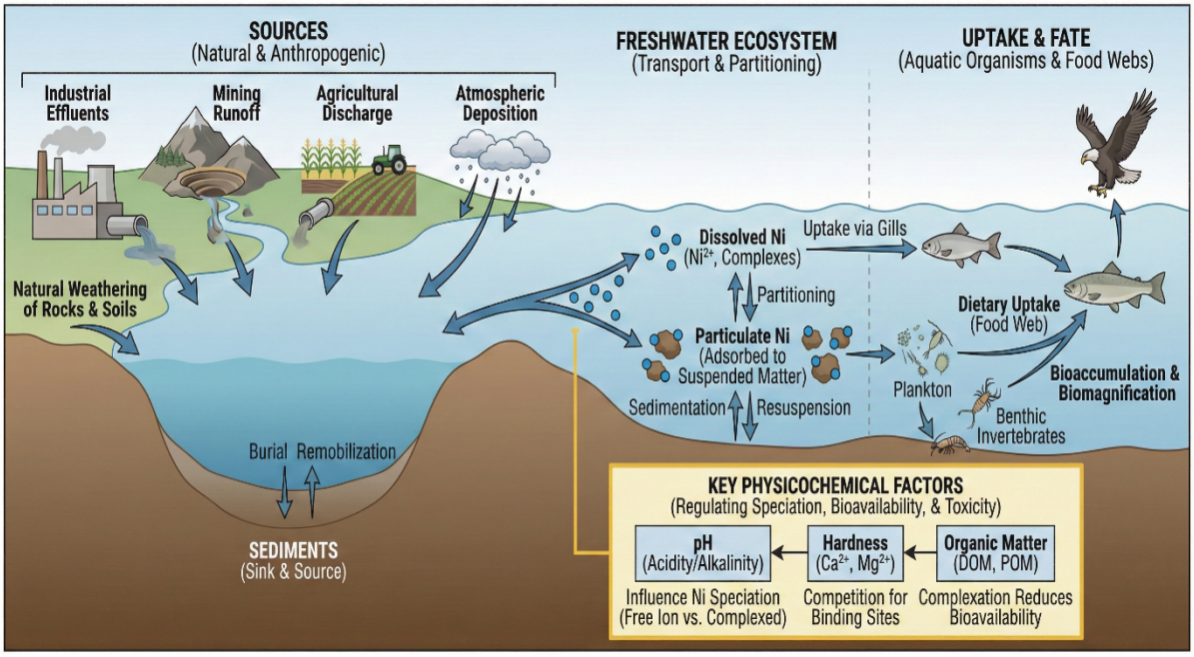

Figure 1. Sources, transport, and environmental fate of nickel in freshwater ecosystems

3. Nickel Uptake and Bioaccumulation in Fish

Nickel uptake in fish occurs primarily through direct contact with contaminated water and indirectly through the food chain. Owing to their continuous interaction with the surrounding aquatic environment, fish readily absorb dissolved nickel through the gills, integument, and gastrointestinal tract, making them particularly susceptible to metal exposure even at sub-lethal concentrations (Burger &Gochfeld, 2000; Rainbow, 2002). The extent of nickel uptake is strongly influenced by its chemical speciation, exposure duration, and environmental conditions, as well as species-specific physiological traits.

The gills represent the principal route of nickel entry due to their large surface area, thin epithelial barrier, and high perfusion rate. Dissolved nickel ions (Ni^{2+}) can cross gill epithelia via passive diffusion or through ion transport pathways that normally regulate essential metals such as calcium and magnesium (Campbell, 1995). Competitive interactions between nickel and calcium at gill binding sites have been shown to modulate metal uptake, with increased water hardness reducing nickel absorption and toxicity (Adhikari & Ayyappan, 2020). Consequently, gill tissues often exhibit early accumulation of nickel and serve as sensitive indicators of recent exposure.

Dietary uptake constitutes another important pathway for nickel bioaccumulation, particularly in benthic and omnivorous fish species. Nickel bound to suspended particles or sediments can enter the food web through algae, plankton, and benthic invertebrates, which are subsequently consumed by fish (Froehlich & Riget, 2014). Compared to waterborne exposure, dietary uptake generally results in slower but more sustained accumulation, contributing significantly to chronic toxicity. The relative importance of dietary versus branchial uptake depends on feeding habits, trophic position, and habitat characteristics.

Once absorbed, nickel is transported via the bloodstream and preferentially accumulates in metabolically active organs such as the liver, kidney, and gills. The liver plays a central role in metal sequestration and detoxification through binding with metallothioneins and other metal-binding proteins (Rainbow, 2002). Several studies have reported higher nickel concentrations in liver tissues compared to muscle, reflecting the organ's role in metabolic regulation and xenobiotic processing (Farkas et al., 2018; Gupta & Rajamani, 2020). Muscle tissue, although less metabolically active, is of particular concern from a human health perspective due to its direct consumption.

Bioaccumulation of nickel in fish is often tissue-specific and concentration-dependent. Experimental studies have demonstrated that prolonged exposure to low nickel concentrations can result in significant accumulation without causing immediate mortality, thereby masking ecological risk (Javed & Usmani, 2019). In *Channa punctatus*, nickel accumulation has been consistently reported in the order liver > gills > kidney > muscle, highlighting the liver and gills as primary target organs for toxicological assessment (Sharma et al., 2017; Sinha et al., 2021). This tissue-specific accumulation pattern is closely associated with subsequent biochemical and histopathological alterations.

Importantly, nickel does not biomagnify to the same extent as certain other heavy metals such as mercury or cadmium. However, its ability to bioaccumulate and persist within tissues can still lead to chronic physiological stress, oxidative damage, and impaired organ function (Pandey et al., 2020). Therefore, bioaccumulation data, when combined with biochemical and histopathological endpoints, provide critical insights into the long-term ecological consequences of nickel pollution in freshwater ecosystems.

Table 2. Nickel Uptake and Bioaccumulation Patterns in Freshwater Fish

Author(s) & Year	Fish Species	Exposure Route	Nickel Concentration / Duration	Primary Accumulation Sites	Key Observations
Farkas et al., 2018	*Abramis brama*	Waterborne	1.0 mg/L, 28 days	Liver > gills > muscle	Tissue-specific accumulation linked to metabolic activity
Sharma et al., 2017	*Channa punctatus*	Waterborne	0.5–2.5 mg/L, 21 days	Liver > gills > kidney	Elevated hepatic nickel associated with enzyme disruption
Javed & Usmani, 2019	*Catlacatla*	Water + diet	1.0–4.0 mg/L, 30 days	Liver, gills	Chronic exposure led to significant bioaccumulation without acute mortality
Gupta & Rajamani, 2020	*Oreochromis mossambicus*	Waterborne	2.0 mg/L, 14 days	Liver > kidney > gills	Metallothionein-mediated sequestration in liver

Sinha et al., 2021	*Channa punctatus*	Effluent-based	Ni-contaminated effluent, 20 days	Liver, gills	Bioaccumulation correlated with oxidative stress biomarkers
Froehlich & Riget, 2014	Multiple species	Dietary	Field-based	Liver, muscle	Dietary uptake contributed to long-term tissue retention

4. Acute and Sub-Lethal Toxicity of Nickel (LC_{50} and Threshold Effects)

Acute toxicity assessments provide a foundational understanding of the lethal effects of contaminants and are widely used to establish benchmark concentrations for environmental risk evaluation. In aquatic toxicology, the median lethal concentration (LC_{50}), defined as the concentration causing 50% mortality of test organisms within a specified exposure period, is a standardized metric for comparing the relative toxicity of pollutants across species and environmental conditions (OECD, 2019). For nickel (Ni), LC_{50} values in freshwater fish vary considerably depending on exposure duration, chemical form of nickel, water chemistry, and species-specific physiological sensitivity.

Several laboratory studies have demonstrated that nickel exhibits moderate acute toxicity to freshwater fish, with reported 96-hour LC_{50} values typically ranging from <1 mg/L to >5 mg/L. This variability reflects differences in experimental design, water hardness, pH, and acclimatization conditions (Vutukuru, 2005; Kumar et al., 2019). In general, fish exposed to acutely toxic concentrations of nickel display pronounced behavioral alterations, including erratic swimming, loss of equilibrium, increased opercular movement, and reduced feeding activity, preceding mortality. These behavioral responses are considered important early indicators of acute stress and neurophysiological disruption.

In *Channa punctatus*, acute exposure studies have reported 96-hour LC_{50} values within the lower mg/L range, suggesting moderate sensitivity to nickel compared to other freshwater teleosts (Sharma et al., 2017; Sinha et al., 2021). Acute toxicity is primarily associated with respiratory distress resulting from gill damage, impaired ion regulation, and disruption of oxygen uptake. Nickel ions can interfere with branchial ion transport mechanisms, leading to osmotic imbalance and metabolic exhaustion under high exposure conditions (Campbell, 1995).

While LC_{50} values provide useful benchmarks, they represent extreme exposure scenarios and do not adequately capture the ecological consequences of chronic, low-level nickel contamination. Sub-lethal toxicity, often occurring at concentrations well below LC_{50}, is increasingly recognized as environmentally more relevant. Sub-lethal exposures may not cause immediate mortality but can induce significant physiological, biochemical, and histopathological alterations that compromise organismal fitness and population sustainability over time (Pandey et al., 2020). Consequently, many studies adopt fractions of the 96-hour LC_{50} (typically 10–30%) to evaluate chronic toxicity under controlled conditions.

Sub-lethal nickel exposure has been consistently linked to oxidative stress, metabolic disruption, and tissue damage in fish. Biochemical endpoints such as increased lipid peroxidation, altered antioxidant enzyme activities, and elevated serum transaminases have been reported even at low nickel concentrations, indicating early cellular stress (Javed & Usmani, 2019; Gupta & Rajamani, 2020). These biochemical responses often precede visible histopathological changes and thus serve as sensitive biomarkers of threshold effects.

Histologically, sub-lethal nickel concentrations can induce progressive structural damage, particularly in gills and liver. Gill tissues may exhibit epithelial lifting, lamellar fusion, and hyperplasia, which reduce

respiratory efficiency and ion exchange capacity. In the liver, vacuolization, hepatocyte degeneration, and focal necrosis have been documented, reflecting impaired detoxification and metabolic functions (Mishra et al., 2018; Pathak & Gupta, 2019). Importantly, the severity of these alterations generally increases with both concentration and duration of exposure, underscoring the cumulative nature of nickel toxicity.

The concept of toxicity thresholds is central to understanding nickel's ecological impact. Threshold concentrations represent levels below which adverse effects are minimal or reversible and above which toxic responses become pronounced. However, defining universal threshold values for nickel is challenging due to interspecific variability and the strong influence of environmental parameters such as water hardness and organic matter content (Adhikari & Ayyappan, 2020). Therefore, LC_{50}-derived sub-lethal thresholds must be interpreted cautiously and integrated with site-specific environmental data.

Overall, acute toxicity studies establish essential reference points for nickel hazard characterization, while sub-lethal assessments provide deeper insights into chronic stress mechanisms and long-term ecological risk. Integrating LC_{50} data with biochemical and histopathological endpoints enables a more comprehensive evaluation of nickel toxicity and supports the development of environmentally relevant water quality criteria for freshwater ecosystems.

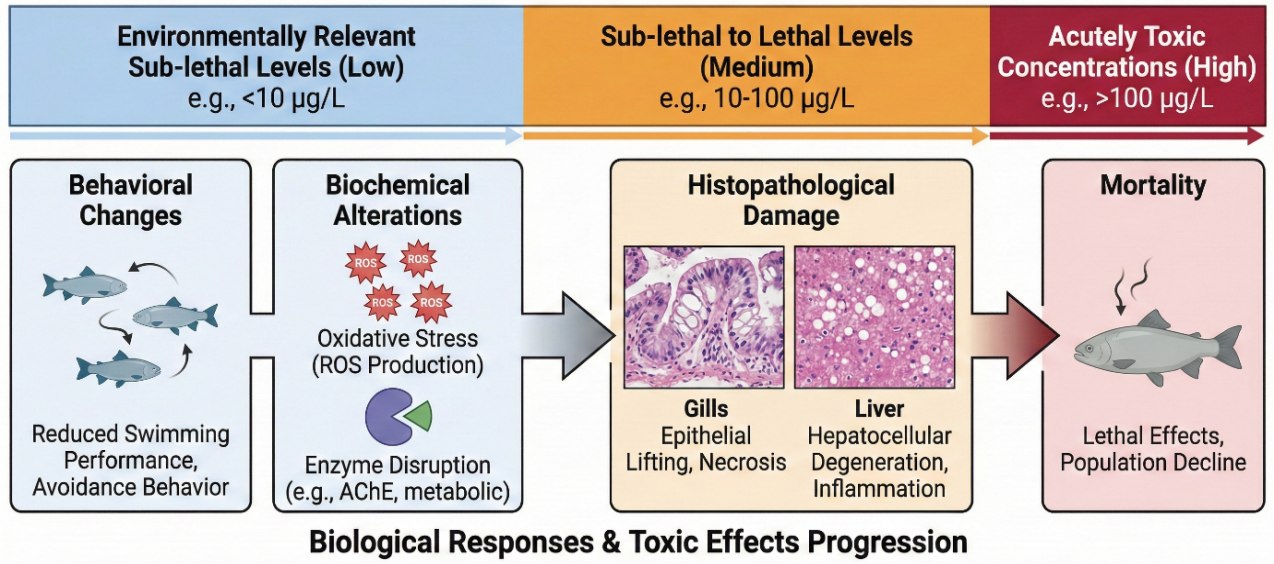

Figure 2. Conceptual relationship between acute (LC_{50}) and sub-lethal toxicity of nickel in freshwater fish.

5. Biochemical and Oxidative Stress Responses to Nickel Exposure

Biochemical responses constitute some of the earliest and most sensitive indicators of metal-induced stress in fish, often preceding overt histopathological damage or mortality. Nickel (Ni), though essential in trace amounts, disrupts cellular redox homeostasis when present at elevated concentrations, leading to oxidative stress–mediated toxicity. Oxidative stress arises from an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defense systems to neutralize them, resulting in damage to lipids, proteins, and nucleic acids (Pandey et al., 2020; Zheng & Zhang, 2017).

Nickel exposure has been shown to enhance ROS production either directly, through redox cycling and interference with mitochondrial electron transport, or indirectly, by inhibiting antioxidant enzymes and depleting cellular reducing equivalents. In fish, the liver is particularly vulnerable to oxidative damage due to its central role in metabolism and detoxification. Numerous studies have reported significant increases in lipid peroxidation, commonly measured as malondialdehyde (MDA) levels, in the liver tissues of nickel-exposed fish, indicating membrane destabilization and cellular injury (Kumar et al., 2019; Gupta & Rajamani, 2020).

Antioxidant enzymes form the primary defense against ROS-induced damage, and their modulation is a hallmark of nickel toxicity. Superoxide dismutase (SOD) catalyzes the conversion of superoxide radicals into hydrogen peroxide, which is subsequently detoxified by catalase (CAT) and glutathione peroxidase (GPx). Alterations in the activities of these enzymes have been consistently reported following nickel exposure, although the direction and magnitude of change may depend on exposure concentration and duration. Short-term or low-level exposure often induces an initial upregulation of antioxidant enzymes as an adaptive response, whereas prolonged or higher exposure leads to enzyme inhibition due to oxidative damage and protein denaturation (Javed & Usmani, 2019; Pandey et al., 2020).

In *Channa punctatus*, nickel-induced oxidative stress has been associated with a decline in antioxidant enzyme activities and a concomitant rise in lipid peroxidation, reflecting a breakdown of cellular defense mechanisms under sustained exposure (Sharma et al., 2017; Sinha et al., 2021). These biochemical perturbations are frequently accompanied by alterations in protein metabolism, including reduced total protein content in liver tissues. Such reductions may result from impaired protein synthesis, enhanced proteolysis, or oxidative modification of structural and enzymatic proteins (Rajesh et al., 2021).

In addition to oxidative stress markers, serum biochemical parameters provide valuable insights into systemic toxicity and organ dysfunction. Elevated activities of serum enzymes such as serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase (SGOT), and alkaline phosphatase (ALP) have been widely reported in nickel-exposed fish. These enzymes leak into the bloodstream following membrane damage and hepatocellular injury, serving as reliable indicators of liver dysfunction (Sharma et al., 2017; Pathak & Gupta, 2019).

Importantly, biochemical responses to nickel exposure are often concentration- and time-dependent, reinforcing their value in defining sub-lethal toxicity thresholds. Unlike mortality-based endpoints, biochemical biomarkers can detect early stress at environmentally relevant nickel concentrations, making them particularly useful for ecological risk assessment and biomonitoring programs. When integrated with bioaccumulation data and histopathological observations, oxidative stress biomarkers provide a mechanistic framework for understanding nickel toxicity in freshwater fish and predicting long-term ecological consequences.

Table 3. Biochemical and Oxidative Stress Responses to Nickel Exposure in Freshwater Fish.

Author(s) & Year	Fish Species	Nickel Concentration / Duration	Biochemical Parameters Assessed	Major Findings
Kumar et al., 2019	*Channa punctatus*	0.5–2.0 mg/L, 21 days	MDA, SOD, CAT, GPx	Increased lipid peroxidation; reduced antioxidant enzyme activities
Javed & Usmani, 2019	*Catlacatla*	1.0–4.0 mg/L, 30 days	Protein, MDA, SOD	Decline in protein content; elevated oxidative stress
Sharma et al., 2017	*Channa punctatus*	0.5–2.5 mg/L, 21 days	SGPT, SGOT, ALP	Elevated serum enzymes indicating hepatocellular damage
Gupta & Rajamani, 2020	*Oreochromis mossambicus*	2.0 mg/L, 14 days	CAT, SOD, lipid peroxidation	Suppressed antioxidant defense; oxidative injury
Rajesh et al., 2021	*Labeorohita*	0.75–3.0 mg/L, 28 days	Protein, MDA, GPx	Protein depletion and oxidative stress in liver

Sinha et al., 2021	*Channa punctatus*	Ni-contaminated effluent, 20 days	MDA, SOD, CAT	Oxidative stress correlated with tissue damage

6. Histopathological Alterations in Target Organs

Histopathological evaluation provides direct morphological evidence of metal-induced toxicity and serves as a crucial link between biochemical disturbances and functional impairment in fish. Unlike biochemical biomarkers, which reflect early cellular stress, histopathological changes represent cumulative and often irreversible damage resulting from prolonged or repeated exposure to contaminants. In the context of nickel (Ni) toxicity, organs that are either directly exposed to the aquatic environment or actively involved in detoxification and metabolism, particularly the gills and liver, are considered primary targets for pathological assessment (Mishra et al., 2018; Pathak & Gupta, 2019).

6.1 Gill Pathology

The gills constitute the first line of interaction between fish and waterborne nickel and are therefore highly sensitive to metal exposure. Histological alterations in gill tissues are commonly reported even at sub-lethal nickel concentrations and are indicative of impaired respiratory and osmoregulatory functions. Typical nickel-induced gill lesions include epithelial lifting, lamellar fusion, hyperplasia of epithelial cells, aneurysm, and focal necrosis (Mishra et al., 2018; Rajesh et al., 2021). These structural changes reduce the effective surface area available for gas exchange and disrupt ion transport processes, leading to hypoxia and ionic imbalance.

In *Channa punctatus*, several studies have documented progressive gill damage with increasing nickel concentration and exposure duration. Epithelial lifting and lamellar fusion are often interpreted as defensive responses aimed at reducing metal uptake; however, prolonged exposure compromises this adaptive mechanism and results in severe tissue degeneration (Sharma et al., 2017; Sinha et al., 2021). The severity of gill pathology is frequently correlated with nickel accumulation levels and oxidative stress biomarkers, highlighting the interdependence of biochemical and structural endpoints.

6.2 Liver Pathology

The liver is a central organ for metabolism, detoxification, and storage of xenobiotics and is therefore particularly vulnerable to nickel-induced injury. Histopathological examination of liver tissues in nickel-exposed fish has revealed a range of degenerative changes, including hepatocellular vacuolization, nuclear pyknosis, cytoplasmic degeneration, sinusoidal dilation, and focal necrosis (Pathak & Gupta, 2019; Gupta & Rajamani, 2020). These alterations reflect impaired metabolic activity, disrupted cellular architecture, and compromised detoxification capacity.

In *Channa punctatus*, nickel exposure has been associated with disorganization of hepatic cords, swelling of hepatocytes, and loss of normal lobular architecture (Sharma et al., 2017). Such changes are often accompanied by elevated serum transaminase activities and oxidative stress, reinforcing the link between histopathological damage and functional liver impairment. The extent of hepatic injury generally increases with both concentration and duration of exposure, indicating a dose-dependent pathological response.

6.3 Integrative Significance of Histopathological Biomarkers

Histopathological endpoints provide valuable insights into the severity and progression of nickel toxicity that cannot be inferred from chemical measurements alone. While gill lesions primarily reflect direct waterborne

exposure and acute stress, liver pathology is more closely associated with chronic accumulation and metabolic burden. The combined assessment of these organs enables a comprehensive evaluation of both immediate and long-term toxic effects.

Importantly, histopathological alterations often persist even after external nickel concentrations decline, underscoring the potential for delayed or residual toxicity. When integrated with bioaccumulation and biochemical data, histopathological findings strengthen the ecological relevance of laboratory studies and support the use of fish as sentinel organisms in environmental monitoring programs (Farag & Oshima, 2020). Thus, tissue-level biomarkers are indispensable for understanding the mechanistic pathways of nickel toxicity and for assessing the health status of freshwater ecosystems.

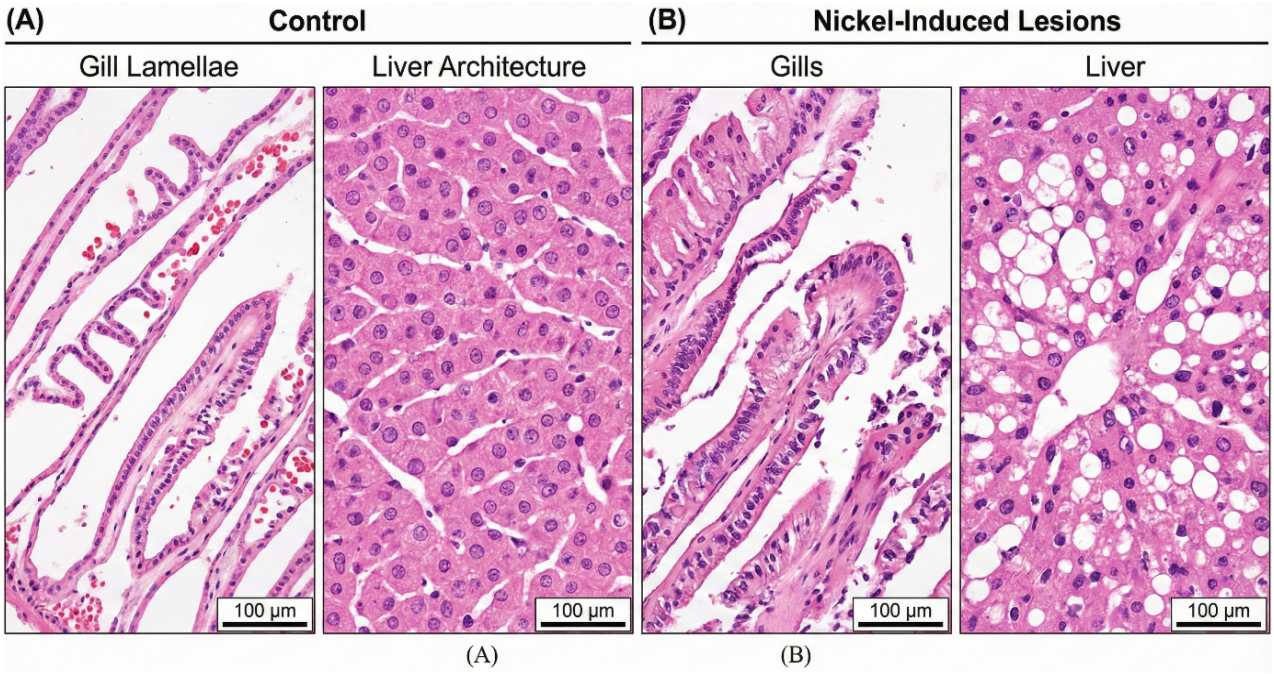

Figure 3. Representative histopathological alterations in gills and liver of freshwater fish exposed to nickel.

7. Integrative Biomarker Approach and Environmental Relevance

Traditional assessments of metal pollution in aquatic ecosystems have relied heavily on chemical measurements of contaminant concentrations in water and sediments. While such measurements provide valuable information on the presence and magnitude of contamination, they do not adequately reflect biological impact or ecological risk. In recent years, integrative biomarker approaches, combining bioaccumulation data with biochemical and histopathological endpoints, have emerged as powerful tools for evaluating the sub-lethal and chronic effects of heavy metals such as nickel (Ni) on aquatic organisms (Farag & Oshima, 2020; Zheng & Zhang, 2017).

Biochemical biomarkers represent early warning signals of nickel toxicity, reflecting cellular and molecular disturbances that precede visible tissue damage. Parameters such as lipid peroxidation, antioxidant enzyme modulation, and alterations in serum transaminases provide sensitive indicators of oxidative stress and metabolic disruption at environmentally relevant nickel concentrations (Pandey et al., 2020; Kumar et al., 2019). However, biochemical responses alone may be transient or reversible, particularly under fluctuating exposure conditions. Therefore, their interpretation is strengthened when coupled with histopathological evidence of structural damage.

Histopathological biomarkers offer tissue-level confirmation of toxic effects and integrate exposure over time. Gill lesions reflect direct waterborne exposure and acute stress, whereas liver pathology indicates

chronic accumulation and detoxification burden (Mishra et al., 2018; Pathak & Gupta, 2019). When evaluated together, biochemical and histopathological endpoints provide a mechanistic continuum of nickel toxicity—from oxidative imbalance and enzyme disruption to cellular degeneration and organ dysfunction. This integrative framework enhances the ecological relevance of laboratory toxicity studies by linking molecular responses to organismal health.

Bioaccumulation data further strengthen biomarker-based assessments by establishing internal exposure levels and tissue-specific metal burdens. In freshwater fish, including *Channa punctatus*, preferential accumulation of nickel in liver and gills has been consistently correlated with oxidative stress intensity and histopathological severity (Sharma et al., 2017; Sinha et al., 2021). Such correlations support the use of these organs as target tissues in routine biomonitoring programs and facilitate the interpretation of biomarker responses in relation to environmental contamination.

From an environmental management perspective, integrative biomarker approaches are particularly valuable for detecting sub-lethal effects that may not be apparent through mortality-based endpoints or short-term chemical analyses. Chronic exposure to low nickel concentrations can impair growth, reproduction, and disease resistance in fish populations, ultimately affecting trophic dynamics and ecosystem stability (Burger &Gochfeld, 2000; Tchounwou et al., 2012). Biomarkers therefore provide an essential link between contaminant exposure and long-term ecological consequences.

Moreover, biomarker-based frameworks can support regulatory decision-making by complementing water quality guidelines and toxicity thresholds derived from LC_{50} values. By incorporating biological responses, environmental assessments become more site-specific and ecologically meaningful, accounting for local physicochemical conditions that influence nickel bioavailability and toxicity (Adhikari & Ayyappan, 2020). As such, integrative biomonitoring using fish biomarkers represents a pragmatic and scientifically robust approach for freshwater ecosystem health assessment and sustainable aquatic resource management.

Table 4. Integrative Biomarkers Used for Assessing Nickel Toxicity and Environmental Relevance

Biomarker Category	Representative Parameters	Target Organs	Ecological Significance	Key References
Bioaccumulation	Tissue Ni concentration	Liver, gills, muscle	Indicates internal exposure and chronic metal burden	Sharma et al., 2017; Sinha et al., 2021
Oxidative Stress	Lipid peroxidation (MDA)	Liver, gills	Early indicator of cellular damage	Kumar et al., 2019; Pandey et al., 2020
Antioxidant Defense	SOD, CAT, GPx	Liver	Reflects adaptive or impaired stress response	Gupta & Rajamani, 2020
Serum Biochemistry	SGPT, SGOT, ALP	Blood/liver	Marker of hepatocellular injury	Pathak & Gupta, 2019
Histopathology	Lamellar fusion, necrosis, vacuolization	Gills, liver	Confirms cumulative and structural damage	Mishra et al., 2018
Integrated Assessment	Correlation of all endpoints	Whole organism	Improves ecological risk assessment	Farag & Oshima, 2020

8. Conclusions and Future Perspectives

Nickel contamination in freshwater ecosystems poses a sustained ecological risk due to its persistence, bioaccumulativebehavior, and capacity to induce toxicity at relatively low concentrations. The evidence synthesized in this mini review demonstrates that nickel exposure in fish triggers a progressive cascade of adverse effects, beginning with uptake and tissue accumulation, followed by oxidative stress–mediated biochemical disruption, and culminating in structural damage to critical organs such as the gills and liver. While acute toxicity indices such as LC_{50} values remain useful for hazard benchmarking, they inadequately represent the ecological significance of chronic, sub-lethal nickel exposure that predominates under natural environmental conditions.

Biochemical responses, particularly alterations in antioxidant defense systems, lipid peroxidation, and serum enzyme activities, emerge as sensitive early indicators of nickel-induced stress. These responses are closely aligned with histopathological lesions, including epithelial damage in gills and degenerative changes in hepatic tissue, which collectively impair respiratory, osmoregulatory, and metabolic functions. The convergence of these endpoints highlights the cumulative nature of nickel toxicity and underscores the importance of integrative biomarker approaches over mortality-based assessments alone. In this context, freshwater fish, especially *Channa punctatus*, serve as effective sentinel organisms for evaluating nickel pollution and ecosystem health.

Future research should prioritize long-term and multigenerational exposure studies to better understand the chronic and population-level consequences of nickel toxicity. Greater attention is also required to assess the combined effects of nickel with other environmental stressors, such as co-occurring metals, organic pollutants, and climate-related variables, which may amplify toxic outcomes. Advancements in biomarker integration, including the linkage of biochemical, histopathological, and emerging molecular indicators, offer promising avenues for improving sensitivity and ecological relevance in environmental monitoring. Incorporating such biologically informed approaches into routine assessment frameworks will be essential for developing protective water quality criteria and ensuring the sustainable management of freshwater ecosystems.

References :

- Adhikari S, Ayyappan S (2020) Heavy metal contamination in aquatic ecosystems and their impact on fish physiology. *Environmental Monitoring and Assessment*192(3):152. https://doi.org/10.1007/s10661-020-8154-7
- Alloway BJ (2013) *Heavy metals in soils: trace metals and metalloids in soils and their bioavailability*, 3rd edn. Springer, Dordrecht. https://doi.org/10.1007/978-94-007-4470-7
- Burger J, Gochfeld M (2000) Effects of heavy metals on fish: a review. *Environmental Research*83(2):110–122. https://doi.org/10.1006/enrs.2000.4071
- Campbell PGC (1995) Interactions between trace metals and aquatic organisms: a critique of the free-ion activity model. *Marine Environmental Research*40(1):81–108. https://doi.org/10.1016/0141-1136(94)00032-L
- Cempel M, Nikel G (2006) Nickel: a review of its sources and environmental toxicology. *Polish Journal of Environmental Studies*15(3):375–382

- Farag AM, Oshima Y (2020) Biomarkers in fish for assessing heavy metal pollution. *Ecotoxicology*29:1–17. https://doi.org/10.1007/s10646-020-02256-3
- Farkas A, Salánki J, Specziár A (2018) Relationship between growth and heavy metal concentration in fish organs. *Hydrobiologia*506:249–254. https://doi.org/10.1023/A:1026444913456
- Froehlich H, Riget F (2014) Bioaccumulation and biomagnification of metals in freshwater fish. *Environmental Science & Technology*48(11):6143–6150. https://doi.org/10.1021/es500232c
- Gupta N, Rajamani S (2020) Heavy metal pollution in freshwater ecosystems: sources, impacts, and remediation. *Environmental Toxicology and Pharmacology*78:103370. https://doi.org/10.1016/j.etap.2020.103370
- Javed M, Usmani N (2019) Nickel toxicity induces biochemical alterations in *Catlacatla*. *Toxicology Reports*6:64–72. https://doi.org/10.1016/j.toxrep.2019.01.007
- Kumar P, Sharma R, Singh R (2019) Nickel-induced oxidative stress and biochemical alterations in freshwater fish *Channa punctatus*. *Ecotoxicology and Environmental Safety*169:8–15. https://doi.org/10.1016/j.ecoenv.2018.11.045
- Mishra R, Singh S, Tiwari A (2018) Histopathological effects of nickel exposure on fish gills. *International Journal of Fisheries and Aquatic Studies*6(4):230–236
- OECD (2019) *OECD guidelines for the testing of chemicals: Fish acute toxicity test (TG 203)*. Organisation for Economic Co-operation and Development, Paris
- Pandey R, Singh V, Kumar S (2020) Mechanisms of heavy metal-induced oxidative stress in fish. *Environmental Science and Pollution Research*27:11234–11252. https://doi.org/10.1007/s11356-020-08123-8
- Pathak R, Gupta D (2019) Liver histopathology and oxidative stress biomarkers in *Labeorohita* exposed to nickel. *Journal of Environmental Biology*40(2):227–233. https://doi.org/10.22438/jeb/40/2/MRN-123
- Rainbow PS (2002) Trace metal bioaccumulation: models, metabolic availability and toxicity. *Environmental International*28(6):545–554. https://doi.org/10.1016/S0160-4120(02)00014-9
- Rajesh G, Pradhan S, Reddy M (2021) Biochemical and histopathological responses of *Labeorohita* to nickel chloride toxicity. *Aquatic Toxicology Reports*3(1):12–20. https://doi.org/10.1016/j.atoxrep.2021.03.001
- Sharma A, Singh N, Verma R (2017) Hepatic alterations and enzyme activity in *Channa punctatus* exposed to nickel nitrate. *Proceedings of the Zoological Society*70(1):53–61. https://doi.org/10.1007/s12595-017-0187-2
- Singh R, Verma P, Sharma S (2022) Agricultural runoff as a source of heavy metals in freshwater ecosystems. *Journal of Environmental Management*302:113997. https://doi.org/10.1016/j.jenvman.2021.113997
- Sinha A, Roy R, Chakraborty P (2021) Toxicological responses of *Channa punctatus* to sub-lethal exposure of industrial effluents. *Journal of Environmental Biology*42(3):534–542. https://doi.org/10.22438/jeb/42/3/MRN-987

- Tchounwou PB, Yedjou CG, Patlolla AK, Sutton DJ (2012) Heavy metal toxicity and the environment. *EXS*101:133–164. https://doi.org/10.1007/978-3-7643-8340-4_6
- Vutukuru SS (2005) Acute toxicity of heavy metals to fish. *Research Journal of Environmental Toxicology*1(2):82–87. https://doi.org/10.3923/rjet.2005.82.87
- Zheng W, Zhang X (2017) Heavy metal-induced oxidative stress in freshwater fish: mechanisms and biomarkers. *Aquatic Toxicology*193:128–136. https://doi.org/10.1016/j.aquatox.2017.07.009

Citation: **Mahapatra. P.,** (2025) "Nickel Toxicity in Freshwater Fish: Biochemical and Histopathological Insights with Special Reference to *Channa punctatus*", *Bharati International Journal of Multidisciplinary Research & Development (BIJMRD)*, Vol-3, Issue-12, December-2025.