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Pesticide Pollution and its Histopathological Effects on Carps: An Overview

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ABSTRACT: The pervasive use of pesticides in agriculture has led to the contamination of aquatic ecosystems, posing significant threats to non-target organisms such as carps. This review synthesizes current research on histopathological alterations observed in various carp species—including *Labeo rohita, Catla catla, Cirrhinus mrigala,* and *Cyprinus carpio*—exposed to sub-lethal concentrations of pesticides. Notable tissue-level changes include epithelial hyperplasia, lamellar fusion, and necrosis in gills; vacuolar degeneration and necrosis in liver hepatocytes; nuclear damage and tubular degeneration in kidneys; mucosal desquamation and lymphocytic infiltration in intestines; myocardial anomalies in heart tissues; and neuronal necrosis with vascular congestion in the brain. These histopathological biomarkers are critical for assessing the sub-lethal impacts of pesticide exposure on fish health and can inform environmental monitoring and management strategies. The findings underscore the urgent need for implementing eco-friendly pest control measures to safeguard aquatic biodiversity and ensure the sustainability of aquaculture practices.

Keywords: Pesticide**,** *Labeo rohita, Catla catla, Cirrhinus mrigala,* bioindicators, Ecotoxicology.

INTRODUCTION

The extensive application of pesticides in modern agriculture has inadvertently led to the contamination of aquatic ecosystems, primarily through surface runoff and leaching. This environmental issue poses significant risks to non-target aquatic organisms, notably freshwater fish species such as carps (*Cyprinus carpio, Labeo rohita, Catla catla, and Cirrhinus mrigala*), which hold substantial ecological and economic importance.

Carps, being integral to aquaculture and serving as bioindicators of aquatic health, are particularly susceptible to the toxic effects of pesticide exposure. Research has documented various histopathological alterations in these species upon exposure to sub-lethal concentrations of pesticides. For instance, studies have reported epithelial hyperplasia, lamellar fusion, and necrosis in gill tissues; vacuolar degeneration and necrosis in liver hepatocytes; nuclear damage and tubular degeneration in kidneys; mucosal desquamation and lymphocytic infiltration in intestines; myocardial anomalies in heart tissues; and neuronal necrosis with vascular congestion in the brain (Özdemir *et al.*, 2017).

These tissue-level changes can impair vital physiological functions, leading to reduced growth, compromised immunity, and increased mortality rates. Understanding these histopathological impacts is crucial for assessing the sub-lethal effects of pesticide exposure on fish health. This review aims to consolidate current research findings on the subject, highlighting the need for stringent environmental monitoring and the adoption of sustainable pest management practices to safeguard aquatic biodiversity and ensure the sustainability of aquaculture operations.

Pesticide Use and Environmental Concerns: The persistent and chemically stable nature of pesticides, coupled with their widespread and often injudicious use in agriculture, has gradually intensified the degradation of aquatic ecosystems. As terrestrial pollutants ultimately converge in water bodies, they compromise water quality and pose a serious threat to aquatic life—especially to fish species, which are highly sensitive bioindicators of ecosystem health.

Efficiency of Pesticide Application and Ecotoxicological Impact: According to Rand and Petrocelli (1984), only a negligible fraction (approximately 0.1%) of pesticides reach their intended targets, while the remainder disperses into surrounding environments, exacerbating ecological harm. Acute toxicity assessments by Johnson & Finely (1980); Mehrle & Mayer (1985) underscore the vulnerability of aquatic organisms, particularly fish, to these contaminants.

Modes of Action and Accumulated Biological Effects: Pesticides exhibit diverse mechanisms of toxicity, disrupting physiological and cellular processes in non-target species. The accumulation of non-biodegradable pesticides such as insecticides, herbicides, and

fungicides in aquatic food chains leads to a cascade of harmful outcomes—including oxidative stress, histopathological and biochemical changes, neurobehavioral impairments, and developmental anomalies.

Ecological Disruption and Habitat Alteration: Anthropogenic influences—such as runoff, drainage, chemical leaching, habitat fragmentation, and invasive species—have placed freshwater organisms at increasing risk of extinction (Cerejeira *et al.,* 2003). Pesticides, designed to lethally target biological life, emerge as particularly insidious contaminants with profound ecological consequences. Freshwater comprises a mere 0.01% of the planet's total water and spans only 0.8% of Earth's surface (Gleick, 1996). Alarmingly, these ecosystems are among the most threatened globally. Sala *et al*. (2000) highlighted that biodiversity losses in freshwater habitats surpass those in the most degraded terrestrial biomes.The entry of pesticides into aquatic habitats alters ecosystem dynamics. These pollutants interrupt fish food webs and habitat conditions, particularly during rainfall or irrigation events that facilitate their widespread dissemination (Maskaoui *et al*., 2005). The consequences include altered reproductive cycles, stunted growth, and even skeletal deformities in aquatic species.

Pesticide Usage Trends in India: India contributes roughly 3% to global pesticide consumption, with usage rising by 2–5% annually (Bhadbhade *et al.,* 2002). In the Indian context, pesticide usage is dominated by insecticides (70%), followed by fungicides (22%), with herbicides and other formulations comprising the remainder. These rising figures mirror growing ecological risks to aquatic ecosystems and their biodiversity.

Classification of Pesticides: Pesticides are broadly classified into inorganic (e.g., borates, fluorides), natural organic (e.g., nicotine, rotenone), and synthetic organic types (e.g., organophosphates, carbamates). Functionally, they are categorized based on target organisms—as insecticides, herbicides, fungicides, algaecides, and rodenticides. Though agriculturally valuable, these substances pose grave ecological and toxicological concerns due to their persistence and toxicity.

Broader Implications of Insecticide and Fungicide Use: Insecticides, while controlling crop pests, negatively impact aquatic life by triggering neurological disorders, behavioral anomalies, and even genetic damage in fish populations. Meanwhile, herbicides—comprising the largest share of pesticide use—and fungicides, essential for managing fungal outbreaks in crops, significantly contribute to freshwater contamination (Fisher *et al*., 2012), further endangering aquatic biodiversity and ecosystem stability. Surface water contamination by pesticides is a global concern. Once introduced, pesticides almost inevitably infiltrate aquatic systems—complex environments where tracing their pathways and fate is challenging. Insecticides such as organochlorines and organophosphates primarily disrupt nervous system function and tissue integrity (Delorenzo *et al*., 2001).

Chronic Pesticide Exposure and Its Significance in Fish Health Monitoring: In their natural aquatic habitats, fish are continuously subjected to chronic exposure from a myriad of pesticide residues. Many of these chemical agents possess lipophilic properties, enabling their bioaccumulation in various tissues over time. The structural alterations occurring in these tissues serve as pivotal indicators in toxicological assessments and are increasingly employed as sensitive biomarkers for environmental monitoring. Histopathological evaluations not only reveal the extent of cellular and organ damage but also provide essential insights into the physiological integrity and overall health status of aquatic organisms. The resultant impairments in organ systems often translate into diminished growth rates, reproductive inefficiencies, and reduced survival potential, thereby impacting population dynamics and ecosystem health.

ORGAN-SPECIFIC HISTOPATHOLOGICAL EFFECTS OF PESTICIDES

Histological Disruptions in Key Organs: This paper presents a comprehensive review of the histological perturbations observed in major organs—namely, the gills, liver, kidneys, intestine, heart, and brain—of fish exposed to diverse classes of pesticides. Numerous studies have consistently reported the presence of pesticide residues in fish inhabiting agricultural runoff zones. The pathological manifestations induced by these contaminants have been meticulously documented by researchers over several decades (King, 1962; Cope, 1966; Eller, 1971; Razani *et al*., 1986; Mukhopadhyay *et al.*, 1987; Bruno & Ellis 1988; Narain & Singh 1991; Mercy *et al.*, 1996).

Liver Alterations: The liver, a central organ in xenobiotic metabolism and detoxification, frequently exhibits a range of structural anomalies upon pesticide exposure. Documented hepatic alterations include cytoplasmic granularity, disorganization of hepatic cords (loss of radial arrangement), and focal atrophy or shrinkage of hepatocyte clusters—signs indicative of cellular degeneration and impaired metabolic function. Sarkar *et al*. (2005) assessed the histological impacts of carbamate pesticide carbofuran and pyrethroid pesticide cypermethrin on *Labeo rohita* over a 28-day exposure period. At low carbofuran concentrations (0.06 mg/l), the liver exhibited diffuse necrosis and vacuolation, with a complete loss of nuclei in affected hepatocytes. A higher dose (0.15 mg/l) disrupted the normal arrangement of hepatocytes, severely damaging the central veins and inducing hyperplasia. Cypermethrin exposure also led to significant liver damage. At 0.16 µl/l, the structure of the portal triad and central veins

became disordered, with small furrows and lesions appearing in affected regions. At the highest concentration (0.40 µl/l), the liver exhibited focal coagulative necrosis, with mild aggregation of mononuclear cells around necrotic zones and pronounced hyperplasia, indicating a more severe response to this pesticide. Liver alterations were only apparent at the higher concentration (10.0 mg/l) of Glyphosate, where congestion of a few sinusoids was noted, and early signs of fibrosis were seen in some areas. Despite notable alterations in the gills and liver, no histopathological changes were detected in the kidney, even with extended exposure to sub-lethal glyphosate concentrations. Hepatic alterations included vacuolation, nuclear enlargement, sinusoidal dilation, and atrophic regions, dur to phenol exposure (Tilak *et al*., 2006). Exposure to dichlorvos (0.91–1.82 ppm) in *Cirrhinusmrigala* resulted in severe aneurysms and liver histology revealed cloudy swelling, karyolysis, karyorrhexis, vacuolar degeneration, sinusoidal dilation, and nuclear hypertrophy, suggesting hepatocellular breakdown (Velmurugan *et al*., 2009). Pal *et al*. (2012) documented liver lesions in *Cyprinus carpio* post-chlorpyrifos exposure, including nuclear hypertrophy, cytoplasmic vacuolation, cellular rupture, nuclear degeneration, and pyknosis. Singh (2013) observed in C. carpio exposed to dimethoate (0.40 mg/L) a disruption in hepatic architecture, vascular rupture, hemorrhage, indistinct cellular boundaries, chromatin condensation, and progressive necrosis over time. Butchiram *et al*. (2013) further noted phenol-induced vacuolization, nuclear enlargement, sinusoidal dilation, and the presence of melanomacrophages at lethal doses, severely impairing liver architecture.

Renal Disruptions: The posterior kidney, integral to osmoregulation and excretion, reveals conspicuous pathological changes in its glomeruli. These include pyknotic nuclei, cytoplasmic vacuolization, and cellular atrophy—hallmarks of nephrotoxicity and compromised renal clearance mechanisms. In the kidney, mild degenerative changes were observed at the lower concentration of HCH, with necrotic alterations becoming more pronounced at the higher concentration, characterized by karyorrhexis and karyolysis of the tubular epithelial cells. The kidney tubules were dilated, and the interstitial space showed significant infiltration by mononuclear cells (Das and Mukherjee 2000). Phenol exposure is seen to cause renal damage ranged that ranged from tubular degeneration and nuclear membrane deformation at sub-lethal levels to cloudy swelling, hyaline droplet degeneration, and tubular lumen occlusion under lethal exposure. These impairments ultimately jeopardize vital organ functionality and survival. Pal *et al*. (2012) reported kidney damage in *C. carpio* from chlorpyrifos, including cellular and nuclear hypertrophy, tubular degeneration, glomerular disruption, and hemorrhage in Bowman's capsule. Velisek *et al*. (2012), examining simazine exposure, found hyaline degeneration and atrophy in caudal renal tubules, though liver and cranial kidneys remained unaffected. Butchiram *et al*. (2013) recorded progressive nephrotoxicity in *L. rohita* exposed to phenol, where sub-lethal concentrations induced tubular degeneration and vacuole formation, while lethal levels caused occluded lumens, hyaline droplet degeneration, and cloudy swelling.

Gill Pathologies: As multifunctional respiratory and excretory structures, gills represent primary interfaces with the aquatic milieu. Studies, including those by Mallatt (1985), have established gills as the initial and often most severely affected tissues following exposure to waterborne toxins. Common lesions include lamellar necrosis, epithelial hyperplasia, hypertrophy, fusion of lamellae, rupture of delicate tissues, increased mucous cell proliferation, and alterations in chloride and vascular cells. These modifications severely disrupt respiratory efficiency and ionic balance. Das and Mukherjee (2000) explored the effects of hexachlorocyclohexane (HCH) on Labeo rohita, a species of freshwater fish, at varying concentrations. At sub-lethal levels of 0.35 ppm, mild congestion of blood vessels was noted in the primary lamellae, but at the higher concentration of 1.73 ppm, more severe histopathological changes emerged, including fusion of primary lamellae, hyperplasia of branchial arches, and swelling of hepatocytes. Areas of diffuse necrosis were also evident in the liver, with marked damage to the sinusoids and central veins due to endothelial degeneration. The architecture of the liver was severely disrupted, reflecting systemic toxicity. Neskovic *et al*. (1996) investigated the histopathological consequences of organophosphate pesticide glyphosate on Cyprinuscarpio, a species commonly known as the common carp. At a concentration of 5.0 mg/l, significant epithelial hyperplasia and sub-epithelial edema were observed in the gills, signaling early stress responses. When the exposure level was raised to 10.0 mg/l, these changes became more pronounced, with additional signs of leukocyte infiltration, hypertrophy in chloride cells, and partial rupturing and detachment of the respiratory epithelium along certain secondary lamellae. Tilak *et al*. (2006) investigated tissue anomalies in *Labeo rohita, Cirrhinusmrigala, and Catla catla* under sub-lethal and lethal phenol exposure. Gills exhibited lamellar fusion, epithelial hyperplasia, hypertrophy, telangiectasia, edema, necrosis, and mucous hypersecretion. At lethal doses, degeneration and disorganization of primary and secondary lamellae were profound. In *Cirrhinus mrigala* exposed to fenvalerate (1.5–3.0 ppb), Velmurugan *et al*. (2007) noted marked gill pathologies: epithelial necrosis, desquamation, lamellar fusion, edema, and lamellar curling. Renal histology revealed pyknotic nuclei, epithelial hypertrophy, glomerular contraction, and Bowman's space expansion. Sub-lethal exposure (0.3–0.6 ppb) of *Cirrhinus mrigala* to lambda-cyhalothrin

caused severe gill anomalies including aneurysms, epithelial lifting, necrosis, desquamation, and lamellar shortening. Saravanan *et al.* (2010) highlighted endosulfan-induced damage in *Labeo rohita*: deformed gill lamellae, interlamellar epithelial debris, red blood cell congestion, hematomas, and aneurysms. After 50 days, liver tissues showed vacuolation, cellular enlargement, histolysis, and necrosis (Saravanan *et al*., 2010). Pal *et al*. (2012) observed profound histopathological alterations in the gills of *Cyprinus carpio* exposed to sub-lethal chlorpyrifos (100 µg/L, 14 days), including epithelial hyperplasia, hypertrophy, vascular congestion, marginal channel dilation, epithelial lifting, lamellar fusion and disorganization, aneurysm, rupture of pillar cells, and necrosis. Maharajan *et al.* (2013) reported similar effects in *Catla catla* under sub-lethal profenofos exposure (0.0008–0.0016 ppm), marked by lamellar degeneration, fusion, epithelial necrosis, haemorrhage, and irregular lamellar morphology. Butchiram *et al.* (2013) noted in *Labeo rohita* that phenol exposure (5.2 mg/L sub-lethal; 25.9 mg/L lethal) caused escalating gill damage—from epithelial hyperplasia and lamellar necrosis to complete disorganization at lethal doses.

Vascular and Cardiac Abnormalities: Large blood vessels and cardiac tissues are not immune to damage. Methoxychlor-induced vascular lesions have been particularly well-characterized in bluegill (Kennedy *et al*., 1970), illustrating the systemic impact of pesticide exposure across circulatory structures. Histopathological changes were also observed in the heart, where myocardial muscle fiber fragmentation, polymorph infiltration, and fluid accumulation were prominent on exposure to HCH in *L. rohita.*

Neurological alterations: Furthermore, HCH exposure caused neurological damage in the brain, with mild vacuolar lesions in the cerebrum at lower doses, progressing to severe neuronal necrosis at the higher concentration.

Histopathological Biomarkers: Indicators of Sublethal Stress: Histopathological changes in fish serve as valuable biomarkers that mirror cumulative sublethal stress imposed by environmental toxicants. These structural aberrations are integrally linked to a spectrum of cellular and biochemical stress responses. According to Hinton (1992), such biomarkers reveal pollutant-driven metabolic disturbances that ultimately precipitate cellular dysfunctions. Their relevance extends beyond organ-specific pathology, acting as integrative endpoints in ecotoxicological risk assessment.

CONCLUSIONS

The reviewed studies collectively illustrate the profound histopathological consequences of pesticide exposure on freshwater teleosts, particularly Indian major carps such as Cyprinuscarpio, *Labeo rohita, Catla catla,* and *Cirrhinus mrigala*. Exposure to organophosphates, phenols, pyrethroids, and other agrochemicals, even at sub-lethal concentrations, results in severe tissue-level anomalies that compromise physiological functioning and survival.

The gills—being the primary site for respiration and osmoregulation—exhibit consistent pathological features such as epithelial hyperplasia, lamellar fusion, aneurysms, desquamation, and necrosis. These changes impair gaseous exchange and disrupt ion regulation, rendering the fish vulnerable to hypoxic stress and secondary infections. Hepatic tissues, critical for detoxification and metabolism, show marked vacuolation, sinusoidal dilation, cellular degeneration, and necrosis—reflecting systemic toxicity and compromised metabolic function. The kidney, an essential organ for excretion and osmoregulation, also shows degenerative changes such as tubular atrophy, glomerular damage, and interstitial hemorrhage, which can ultimately impair renal function and fluid balance.

These findings underscore the bio-indicator potential of histopathological endpoints in aquatic toxicology. They also highlight the urgency of re-evaluating pesticide use near aquatic ecosystems, advocating for eco-friendly pest control alternatives and stricter regulations on agrochemical discharge. Persistent sub-lethal exposure not only threatens fish health and biodiversity but also risks collapsing aquaculture productivity and aquatic food chains, thereby impacting socio-economic structures dependent on freshwater resources.

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