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Structural and Histopathological Responses of Gill Tissue in *Channa punctatus* under Chlorpyrifos Stress

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Abstract
Freshwater fishes are continuously exposed to pesticide residues released into aquatic ecosystems through agricultural runoff, posing serious risks to non-target organisms. Chlorpyrifos, a commonly used organophosphate insecticide, is known to induce toxic effects in fish, particularly in organs directly interacting with the aquatic environment. The present study investigates the structural and histopathological responses of gill tissue in *Channa punctatus* following exposure to chlorpyrifos, with emphasis on tissue-level alterations as indicators of toxic stress. Gill tissues from control and chlorpyrifos-exposed fish were examined using standard histological techniques. Control specimens exhibited well-organized primary and secondary lamellae with intact epithelial lining and distinct interlamellar spaces, reflecting normal gill architecture. In contrast, fish exposed to chlorpyrifos displayed pronounced histopathological alterations. Sub-lethal exposure resulted in shortening and partial fusion of secondary lamellae, epithelial degeneration, bulging of filament tips, and cytoplasmic vacuolization, indicating early cellular stress. Lethal exposure caused severe disruption of gill structure, characterized by extensive lamellar fusion, curling and clubbing of lamellae, epithelial lifting, hyperplasia, hypertrophy, necrosis, vacuolization, and nuclear abnormalities such as pyknosis. Many of the observed changes appear to represent adaptive defense mechanisms aimed at reducing toxicant entry, while prolonged exposure led to irreversible pathological damage. These structural alterations significantly compromise respiratory efficiency and ionic regulation, ultimately affecting fish health and survival. The study highlights the sensitivity of gill histopathology as a reliable biomarker for assessing chlorpyrifos toxicity and underscores the need for controlled pesticide usage to safeguard freshwater ecosystems.

Keywords: Channa punctatus; Chlorpyrifos; Gill histopathology; Organophosphate toxicity; Aquatic pollution; Biomarkers

Introduction

Aquatic organisms, particularly fish, are continuously exposed to environmental contaminants due to their close association with the surrounding water. Increasing agricultural and industrial activities have led to the extensive use of pesticides, which frequently enter freshwater ecosystems through runoff and leaching. These contaminants exert chronic chemical stress on aquatic organisms, triggering a range of physiological, biochemical, and structural disturbances. Among the various biomarkers employed to assess toxic stress, histopathological alterations are considered highly sensitive and reliable, as they often appear earlier than behavioral or biochemical changes. Histological examination allows direct visualization of tissue-level responses to toxicants and provides valuable insight into the overall health status of fish. Structural alterations in tissues reflect both adaptive and pathological responses and can be correlated with functional impairments induced by environmental stressors (Marchand *et al*., 2009).

Chlorpyrifos as an Environmental Toxicant

Chlorpyrifos is a widely used organophosphate insecticide applied in agriculture to control a broad spectrum of insect pests. Despite its effectiveness, chlorpyrifos frequently contaminates aquatic ecosystems and poses serious risks to non-target organisms. Its primary mode of action involves inhibition of acetyl cholinesterase, resulting in neurotoxicity and physiological dysfunction in fish. In addition to neurotoxic effects, chlorpyrifos induces oxidative stress, disrupts cellular membranes, and alters metabolic processes. These effects collectively compromise vital organs, particularly those directly exposed to the aquatic environment, such as the gills.

Significance of Gills as Target Organs

Gills are among the most sensitive organs in fish due to their constant exposure to waterborne pollutants. They perform essential functions including respiration, osmoregulation, acid–base balance, and excretion of nitrogenous wastes. The large surface area, thin epithelial lining, and rich vascularization make gills especially vulnerable to toxicants dissolved in water.

Even minor structural damage to gill tissues can impair respiratory efficiency and ionic regulation, leading to physiological stress and reduced survival. Consequently, histopathological analysis of gills is widely used as a reliable indicator of aquatic pollution.

Normal Histology of Fish Gill

In teleost fishes, the gill apparatus consists of five pairs of gill arches, of which four pairs bear functional gill filaments. Each gill arch supports two rows of filaments that extend posteriorly and are joined basally by a gill septum. Each filament is composed of a primary gill lamella that supports numerous secondary gill lamellae arranged bilaterally.

The secondary lamellae are thin, flattened respiratory structures separated by interlamellar regions that facilitate efficient water flow and gas exchange. The gill epithelium is composed primarily of pavement cells that provide protection, along with mitochondria-rich chloride cells involved in ion regulation. Pillar cells form the structural framework of the secondary lamellae and regulate blood flow through the lamellar channels.

In the present study, control specimens of *Channa punctatus* showed well-organized primary and secondary lamellae, intact epithelial lining, and clearly defined interlamellar spaces, indicating normal gill architecture after 24 h exposure (Plate 1.I, Fig. A).

Histopathological Alterations in Gills Exposed to Chlorpyrifos

Due to their delicate structure and direct contact with the aquatic environment, gills are among the first organs to exhibit pathological changes following exposure to toxicants. In the present investigation, exposure of *Channa punctatus* to chlorpyrifos resulted in marked structural alterations in both primary and secondary gill lamellae.

At sub-lethal concentrations, common histopathological lesions included shortening and partial fusion of secondary lamellae, degeneration of the lamellar epithelium, bulging of filament tips, and cytoplasmic vacuolization. These changes indicate early cellular stress and disruption of normal gill function (Plate 1.I, Fig. B).

Fish exposed to lethal concentrations of chlorpyrifos exhibited severe and extensive damage to gill architecture. Prominent pathological features included clubbing and curling of secondary lamellae, complete fusion of adjacent lamellae, necrosis of primary gill lamellae, epithelial lifting, and marked hyperplasia and hypertrophy of epithelial cells. Nuclear abnormalities such as pyknosis were frequently observed, along with extensive vacuolization and degeneration of epithelial and pillar cells (Plate 1.I, Fig. C).

Defensive and Adaptive Responses of Gill Tissue

Several histopathological changes observed in chlorpyrifos-exposed fish appear to represent adaptive defense mechanisms. Epithelial lifting, hyperplasia, hypertrophy, and fusion of secondary lamellae increase the diffusion distance between the external environment and the bloodstream, thereby reducing the entry of toxicants. Similar protective responses have been reported in fish exposed to pesticides and other environmental pollutants (Mallatt, 1985).

Increased mucus secretion on the gill surface is another common response to toxic stress. While mucus provides a protective barrier, excessive accumulation can interfere with gas exchange by thickening the respiratory surface and reducing oxygen diffusion.

Vascular and Cellular Damage

Severe toxic stress disrupts the integrity of pillar cells, leading to blood congestion, dilation of marginal channels, and aneurysm formation within secondary lamellae. Such vascular damage impairs blood flow and reduces oxygen transport efficiency. Cytoplasmic vacuolization and degeneration of epithelial cells observed in the present study may result from metabolic disturbances and oxidative stress induced by chlorpyrifos exposure. These cellular alterations further compromise the functional capacity of gill tissues.

Functional Implications of Gill Damage

Structural damage to gill tissues has direct consequences on respiratory and metabolic functions. Fusion and shortening of secondary lamellae reduce the surface area available for gas exchange, while epithelial thickening increases diffusion distance. Curling and distortion of lamellae disrupt normal water flow patterns, leading to reduced oxygen uptake and impaired metabolic performance.

Such functional impairments can result in hypoxia, reduced growth, altered behavior, and increased susceptibility to disease. In natural ecosystems, these effects may negatively impact fish survival and population stability.

Comparative Studies and Indian Context

Several studies on Indian freshwater fishes have reported similar histopathological alterations following pesticide exposure. Veeraiah and Tilak documented epithelial lifting, lamellar fusion, and necrosis in the gills of fishes exposed to organophosphate pesticides. Tilak and co-workers also reported severe gill damage in fishes collected from pesticide-contaminated water bodies in Andhra Pradesh and Telangana.

These findings support the present observations and emphasize the usefulness of histopathological biomarkers in monitoring pesticide pollution in Indian freshwater ecosystems.

Mechanism of Chlorpyrifos-Induced Gill Toxicity

Organophosphate pesticides such as chlorpyrifos exert their toxic effects primarily through inhibition of acetylcholinesterase; however, gill damage is not solely mediated by neurotoxicity. Direct contact of chlorpyrifos with the gill epithelium disrupts membrane permeability and alters ion transport mechanisms. Mitochondria-rich chloride cells are particularly vulnerable, leading to impaired regulation of sodium, chloride, and calcium ions.

Chlorpyrifos exposure has also been shown to induce oxidative stress in gill tissues by generating reactive oxygen species. These free radicals cause lipid peroxidation of cellular membranes, protein denaturation, and DNA damage, ultimately resulting in cellular degeneration and necrosis. Degeneration of pillar cells disrupts the integrity of blood channels, leading to vascular abnormalities and impaired gas exchange. Similar mechanisms have been reported in freshwater fishes exposed to organophosphate insecticides by Tilak and Veeraiah.

Gill Histopathology as a Biomarker of Aquatic Pollution

Histopathological changes in fish gills are increasingly recognized as reliable biomarkers of aquatic pollution due to their sensitivity and integrative nature. Unlike biochemical markers that may fluctuate rapidly, tissue-level alterations reflect cumulative exposure to toxicants over time. Gill histopathology therefore provides a clear link between environmental contamination and biological effects.

Several investigators have emphasized the usefulness of gill lesions such as epithelial lifting, lamellar fusion, hyperplasia, and aneurysm formation as diagnostic indicators of pesticide contamination in freshwater ecosystems. In Indian rivers and reservoirs receiving agricultural runoff, histopathological monitoring of fish gills has been suggested as a cost-effective and ecologically relevant tool for environmental assessment.

Ecological and Environmental Significance

Gill damage in fish has implications beyond individual health, affecting population dynamics and ecosystem stability. Impaired respiration reduces swimming performance, feeding efficiency, and predator avoidance, making affected fish more vulnerable in natural habitats. Chronic exposure to pesticides may therefore lead to reduced population density and altered species composition in freshwater ecosystems.

In regions with intensive agriculture, such as many parts of India, contamination of ponds, tanks, and rivers with chlorpyrifos poses a significant threat to native fish species. *Channa punctatus*, being a hardy and widely distributed freshwater fish, serves as an excellent bioindicator species for monitoring pesticide pollution in such environments.

Relevance to Human Health and Fisheries

Fish constitute an important source of protein for human populations, particularly in rural and semi-urban areas. Histopathological damage in fish tissues may not only affect fish health but also raise concerns regarding food quality and safety. Although gill lesions themselves are not consumed, chronic pesticide exposure may reflect systemic contamination of edible tissues.

From a fisheries perspective, pesticide-induced stress can lead to reduced growth rates, increased disease susceptibility, and higher mortality, ultimately affecting fish yield and economic returns. Understanding the histopathological effects of chlorpyrifos is therefore essential for sustainable fisheries management and environmental conservation.

Management Implications and Future Perspectives

The findings of the present study highlight the need for stricter regulation and judicious use of pesticides such as chlorpyrifos. Adoption of integrated pest management strategies and promotion of environmentally friendly alternatives can significantly reduce pesticide runoff into aquatic ecosystems.

Future studies should focus on long-term exposure scenarios, recovery potential of gill tissues, and combined effects of multiple pollutants. Integration of histopathological data with biochemical and molecular biomarkers would provide a more comprehensive understanding of pesticide toxicity in fish.

Conclusion

The present study demonstrates that chlorpyrifos induces significant histopathological alterations in the gills of *Channa punctatus*. The observed changes reflect a combination of adaptive defense mechanisms and pathological damage. While initial responses may serve to limit toxicant entry, prolonged exposure leads to severe structural disruption, ultimately impairing respiratory function and overall physiological fitness. Histopathological examination of gill tissues thus serves as a sensitive and reliable indicator of chlorpyrifos toxicity and highlights the need for controlled pesticide usage to protect aquatic life.

Plate 1.I 24 h Gill

Fig. A. Control: Normal gill lamellae of *Channa punctatus* after 24 h.

PGL: Primary gill lamellae
ILR: Inter lamellar region
SGL: Secondary gill lamellae

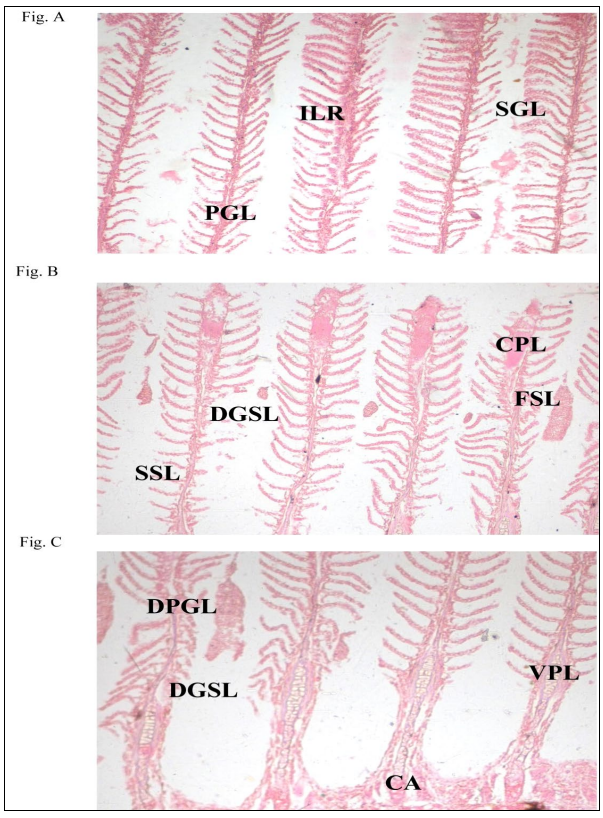
Fig. B. Sub-lethal: Gill lamellae of *Channa punctatus* exposed to sub-lethal concentration of chlorpyrifos for 24 h.

SSL: Shortened secondary lamellae
DGSL: Degenerated secondary lamellae
FSL: Fusion of secondary lamellae
CPL: Clubbing of primary lamellae

Fig. C. Lethal: Gill lamellae of *Channa punctatus* exposed to lethal concentration of chlorpyrifos for 24 h.

DPGL: Degenerated primary gill lamellae
DGSL: Degenerated secondary gill lamellae
CA: Central Axis
VPL: Vacuolization of primary lamellae

Plate 1.I 24 h Gill



Fish exposed to lethal concentrations of chlorpyrifos exhibited severe and widespread damage to gill architecture. Prominent pathological features included clubbing of secondary lamellae, extensive fusion of adjacent lamellae, necrosis of primary gill lamellae, curling and distortion of

secondary filaments, and marked hyperplasia and hypertrophy of epithelial cells. Nuclear abnormalities such as pyknosis, along with extensive vacuolization and degeneration of epithelial and pillar cells, were consistently observed across all exposure periods. Additionally, lifting of the epithelial layer from the underlying lamellae was evident, suggesting compromised structural integrity and impaired respiratory function (Plate I.I-I Fig. A; II Fig. B; III Fig. C).

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