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Gill Damage Patterns in *Heteropneustes fossilis* Exposed to Cypermethrin: A Diagnostic Tool for Monitoring Freshwater Contamination

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Abstract
The widespread application of cypermethrin in agriculture leads to its persistent entry into natural waters, where it may compromise fish health. This work investigates the progression of structural disturbances in the gills of *Heteropneustes fossilis* exposed to sublethal cypermethrin concentrations for 24, 48, 72, and 96 hours. Gill samples were processed using standard histological techniques and examined to determine lesion patterns associated with exposure duration. Even the shortest exposure interval produced visible epithelial disruption, including swelling, local detachment, and initial fusion of secondary lamellae. As exposure time increased, lamellar deformation intensified, accompanied by hyperplasia of chloride cells, marked vacuolation, hemorrhagic regions, and distortion of pillar cell arrangement. After 96 hours of exposure, the gill framework showed severe necrosis and extensive fragmentation, suggesting functional collapse. The gradual escalation of tissue injury indicates that gill morphology can provide a sensitive measure of pesticide stress in aquatic organisms. These results confirm that cypermethrin impairs respiratory surfaces in *H. fossilis* and support the application of gill histopathology in monitoring chemical contamination in freshwater systems.

Keywords: pyrethroid toxicity; gill lesions; *Heteropneustes fossilis*; environmental contaminant monitoring; histopathology; respiratory tissue damage; biomarker

1. Introduction

Freshwater bodies frequently receive agricultural chemicals through runoff, accidental spills, and improper disposal practices. Among these contaminants, cypermethrin is routinely used as a crop protectant and is known to persist long enough to affect resident fish communities. Although the compound is regarded as relatively low-risk for mammals, numerous investigations have shown that it can severely impair aquatic organisms at concentrations far below those applied in the field (Brusle et al., 1996).
Fish are particularly suited to detecting detrimental changes in water chemistry because their physiological processes operate in constant interaction with the surrounding medium. The gills are both the first and one of the most heavily affected sites of toxicant interaction due to their role in gas exchange and ion balance and because of their delicate tissue architecture (Rankin & Bolis, 1982). Even minor structural deviations may interfere with respiratory efficiency and the maintenance of fluid and electrolyte balance.
Histological examination of gill tissues is widely recognized as an effective means of detecting early and intermediate responses to chemical exposure (Wester et al., 2002; Hinton & Lauren, 1990). Previous research on pyrethroids and related substances has highlighted common responses such as epithelial detachment, lamellar thickening, ruptured pillar cells, aneurysmal dilation, and breakdown of respiratory surfaces (Caliskan et al., 2003; Olufayo, 2012; Rakhi et al., 2013). *Heteropneustes fossilis* is a hardy freshwater catfish distributed across South Asia and routinely exposed to pesticide residues in agricultural zones, yet detailed records describing the temporal progression of cypermethrin-induced gill alterations remain limited.

The present study documents structural modifications in the gills of *H. fossilis* after controlled exposure to cypermethrin. By examining sequential exposure intervals, we provide a time-linked account of histopathological changes that may serve as indicators of pesticide stress in contaminated waters.

2. Materials and Methods

2.1 Fish Maintenance

Juvenile *H. fossilis* were sourced from a commercial hatchery and held for two weeks in aerated tanks to allow physiological adjustment. During this period, they were fed a routine pellet diet. Water conditions were maintained within narrow limits of temperature (approximately 26°C), pH (7.0–7.5), and oxygen concentration (>6 mg/L). Feeding was discontinued a day prior to exposure to reduce metabolic interference.

2.2 Exposure Protocol

Cypermethrin of analytical grade was dissolved to create a stock solution from which working concentrations were prepared. Levels selected corresponded to sublethal fractions of previously reported LC50 values for the species. Groups of fish were exposed for 24, 48, 72, and 96 hours under semi-static conditions. Test solutions were refreshed at 24-hour intervals to stabilize concentrations. Unexposed fish held under identical conditions served as controls.

2.3 Histological Processing

Following each exposure interval, specimens were euthanized humanely. Gill arches were removed carefully, fixed in buffered formalin, dehydrated using ethanol gradients, embedded in paraffin, and sectioned into thin slices (5–6 µm). Hematoxylin and eosin staining was applied prior to microscopic examination. Representative lesions were photographed for documentation.

2.4 Ethical Considerations

Procedures adhered to accepted standards for animal experimentation, minimizing distress throughout handling and sampling.

3. Results

3.1 Control Condition

Tissues from untreated individuals maintained a consistent organization: primary and secondary lamellae were evenly spaced, epithelial layers remained intact, pillar cells exhibited orderly alignment, and chloride and pavement cells were present in expected proportions. No degenerative alterations or hemorrhagic traces were detected.

3.2 Cypermethrin Exposure

24-hour exposure:

Initial morphological disruptions appeared as mild swelling of secondary lamellae, partial lifting of epithelial layers, and isolated fusion at adjacent lamellar edges. Hyperplasia of chloride cells began to emerge, while small cytoplasmic vacuoles were present within epithelial cells (Plate-1).

48-hour exposure:

Morphological changes became more pronounced. Epithelial lifting increased in extent, lamellar units fused more frequently, and pillar cell arrangement became irregular. Distinct vacuolation and localized hemorrhagic spots were observed, reflecting compromised structural integrity(Plate-1).

72-hour exposure:

Gill architecture showed marked deterioration: lamellae appeared compressed or collapsed, large vacuoles occupied epithelial spaces, and hemorrhage spread through broader regions of the filaments. Chloride cell hyperplasia intensified, indicating significant stress to ion-handling mechanisms(Plate-2).

96-hour exposure:

The cumulative effect of cypermethrin exposure resulted in extensive tissue breakdown. Primary and secondary lamellae were fragmented or detached, regions of necrotic tissue were common, epithelial surfaces were severely deteriorated, and widespread hemorrhage was evident. Overall organization of the gill was lost, implying respiratory impairment (Plate-2).

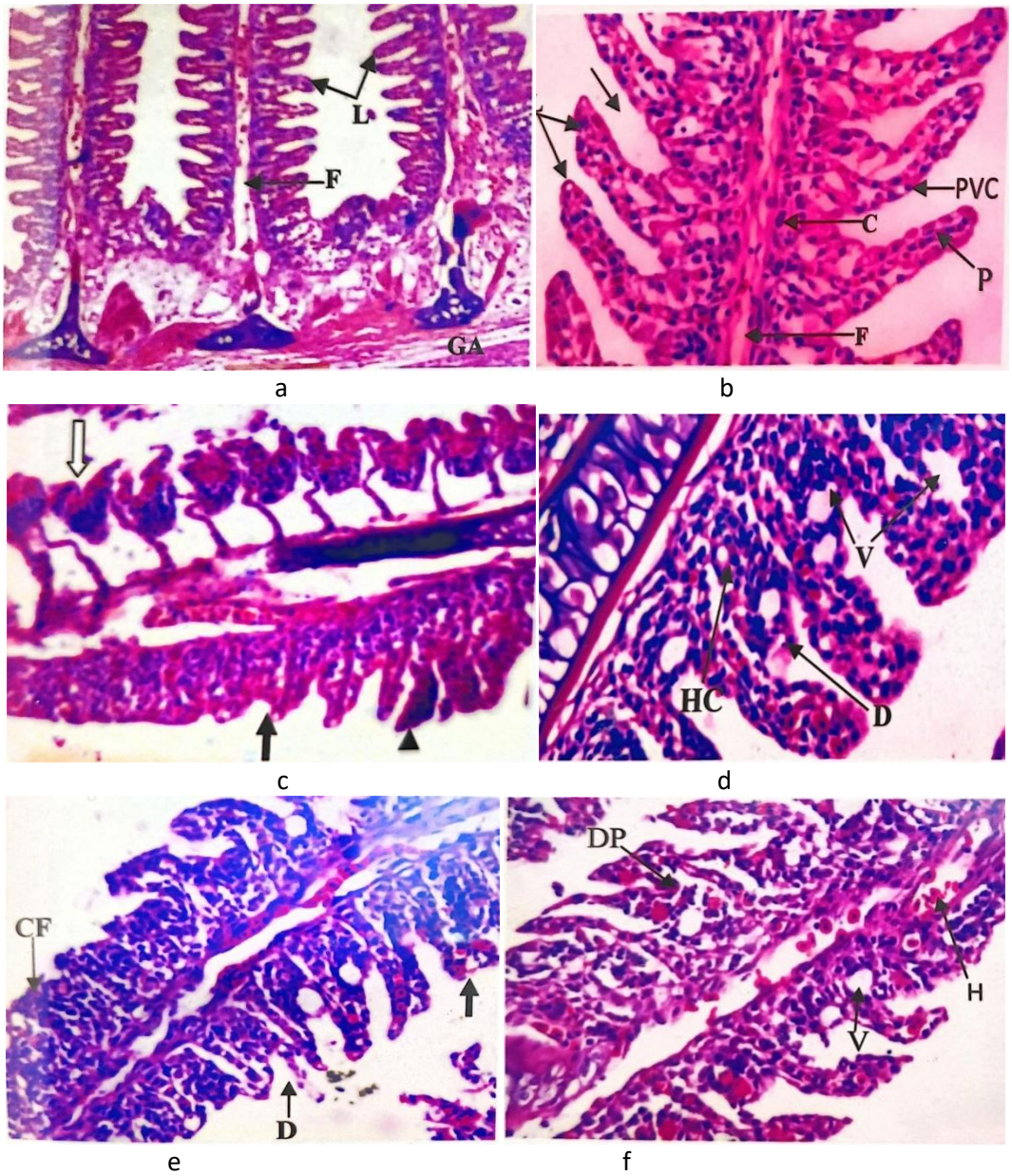
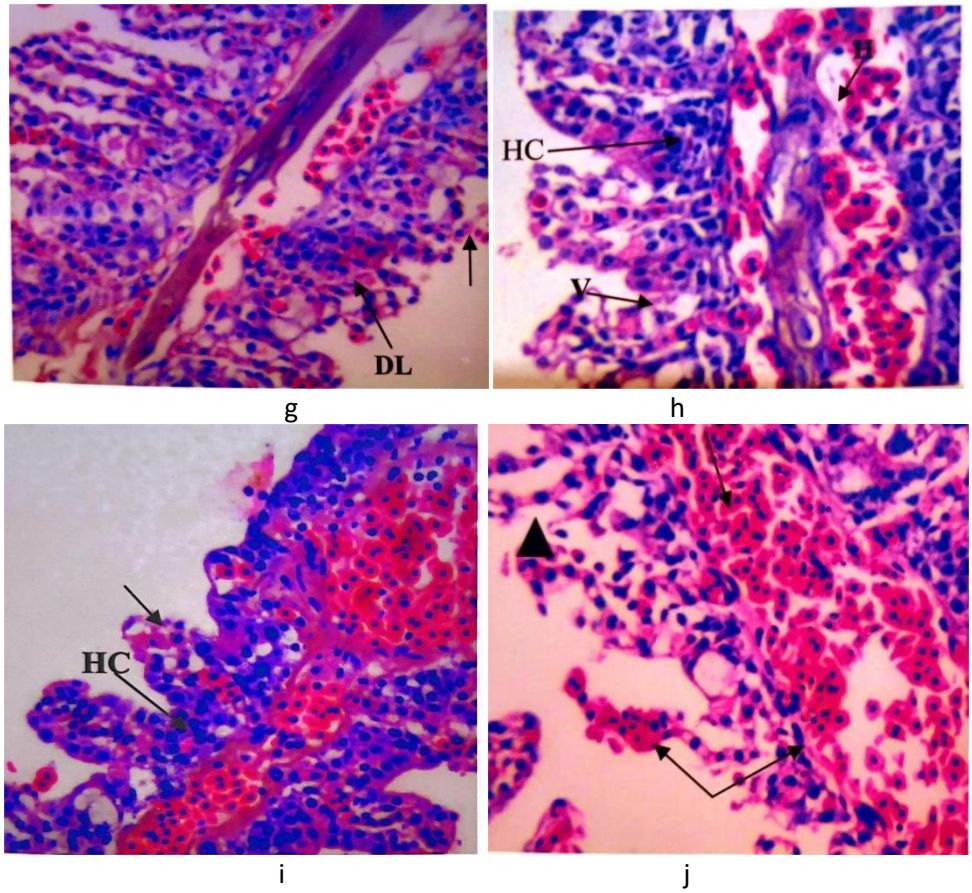


Plate:1. Photomicrograph of the gill of *H. fossilis*. (a) Control, showing primary lamellae or filament (F), secondary lammellae (L) and gill arch (GA), X100. (b) Control showing primary lamellae (F), secondary lammellae (L), pillar cell (P), Chloride cell (C), Pavement Cell (PVC) and water Channel (arrow), X400. (c) After 24 hours of cypermethrin exposure showing shortening and fusion of secondary lamellae (black arrow), swelling of secondary lamellae (white arrow) and lamellar aneurysm (arrow head), X100. (d) After 24 hours of cypermethrin exposure showing hyperplasia of chloride cells (HC), desquamation of epithelial lining (D) and Vacuole formation (V), X400. (e) After 48 hours of cypermethrin exposure showing epithelial lifting (arrow), desquamation (D) and complete fusion of secondary lamellae (CF), X400. (f) After 48 hours of cypermethrin exposure showing disorganized pillar cells (DP), vacuole formation (V) and haemorrhagic areas (H), X400.



g h

i j

Plate:2 Photomicrograph of the gill of *H. fossilis*. (g) After 72 hours of cypermethrin exposure showing lamellar disorganization (DL) and epithelial lifting (arrow), X400. (h) After 72 hours of cypermethrin exposure showing haemorrhagic areas (H), hyperplasia of chloride cells (HC) and vacuolation (V), X400. (i) After 96 hours of cypermethrin exposure showing hyperplasia of chloride cells (HC) and degeneration of epithelial cells (arrow), X400. (j) After 96 hours of cypermethrin exposure showing complete distortion of primary and secondary lamelae (arrow), severe haemorrhagic areas (H) and breakage of gill lamellae (arrow head), X400.

4. Discussion

The gradual escalation of gill damage observed in *H. fossilis* demonstrates the high sensitivity of respiratory tissues to cypermethrin. Early signs of epithelial lifting and swelling reflect attempts to reduce direct toxicant contact, a phenomenon also noted in other teleosts facing pesticide pressure (Caliskan et al., 2003). Disruption of pillar cells and lamellar aneurysm formation suggest harmful changes in blood flow regulation within the filaments, supporting earlier interpretations by Martinez and Sotelo-Mazon (2004).

Persistent chloride cell hyperplasia, which intensified over time, indicates efforts to counteract ion imbalance caused by toxicant interference (Fernandes & Mazon, 2003). As exposure continued, structural fusion and deformation of lamellae reduced the effective respiratory surface area, likely placing the fish under increasing metabolic strain. Comparable changes have been noted in cypermethrin-treated *Clarias gariepinus* and *Cyprinus carpio*, emphasizing that such lesions are consistent indicators of pyrethroid exposure (Velmurugan et al., 2009; Tilak et al., 2005).

In the final exposure interval, extensive necrosis and lamellar fragmentation signaled irreversible damage and correspond to end-stage alterations documented for chemically stressed fish gills (Wester et al., 2002). Taken together, the findings underscore the value of gill histopathology as an early-warning tool and further establish *H. fossilis* as a suitable model species for monitoring the ecological impact of agricultural runoff.

5. Conclusion

Cypermethrin exposure produced a clear sequence of structural lesions in the gills of Heteropneustes fossilis, beginning with epithelial lifting and swelling and advancing to widespread necrosis and lamellar fragmentation. The progressive nature of these changes highlights the organ's vulnerability to waterborne toxins and confirms the usefulness of gill morphology as a sensitive biomarker for assessing pesticide contamination in freshwater environments. Incorporating such histological endpoints into monitoring programs can improve detection of sublethal chemical stress before population-level impacts arise.

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