Effect of Pyrethroid Insecticides on Freshwater Fish: A Mini-Review

Abstract

Synthetic pyrethroids are widely used because of their rapid biodegradability and non-persistent nature. However, these compounds frequently enter aquatic environments through agricultural run-off and spraying operations, where they adversely affect non-target organisms such as fish. This review summarizes findings on the toxicological effects of pyrethroids on freshwater fish, highlighting physiological, biochemical, histopathological, and endocrine impacts. Evidence shows that pyrethroids act as neurotoxins that induce hyperexcitation or direct cytotoxicity, with fish exhibiting high sensitivity due to slow metabolism and elimination. Sublethal exposures reduce growth, alter water quality, impair behavioural responses, decrease glycogen reserves, trigger oxidative stress, disrupt endocrine function, and damage vital organs. These findings emphasize the need for improved insecticide management to reduce risks to aquatic life, especially fish.

Introduction

Pyrethroid insecticides are synthetic derivatives of pyrethrins, the naturally occurring insecticidal compounds found in *Chrysanthemum cinerariaefolium*. They are widely used in agriculture owing to their biodegradability and relatively low environmental persistence. Despite this, they frequently enter freshwater ecosystems through run-off and aerial spraying, posing risks to non-target aquatic organisms (Morgan, 2012). Pyrethroids are now among the most extensively

used insecticide groups globally, and their presence in surface waters has been documented across multiple continents (Stehle & Schulz, 2015). Fish are particularly vulnerable, and understanding the full range of toxicological effects of pyrethroid exposure is important for both environmental protection and fisheries management.

Toxicity Mechanisms and Sensitivity of Fish

Pyrethroids primarily function as neurotoxins by interfering with voltage-gated sodium channels in the nervous system, resulting in prolonged sodium influx, hyperexcitation, and ultimately death (Farag et al., 2021). Type II pyrethroids such as deltamethrin, cypermethrin, and fenvalerate are particularly associated with severe neurological effects in fish, including tremors, loss of coordination, and convulsions (Kumar et al., 2015). Fish show extreme sensitivity to pyrethroids, with 96-hour LC_{50} values in laboratory tests generally falling below 10 μg/L (Kumar et al., 2015). This sensitivity is attributed to their relatively slow metabolism and inefficient elimination of pyrethroid compounds (Farag et al., 2021). Beyond the nervous system, the sodium channel is also distributed in cardiac and skeletal muscle tissues, which means pyrethroids can affect multiple organ systems simultaneously (Gao et al., 2020).

Sublethal and Physiological Effects

Sublethal exposure to pyrethroids has been associated with multiple physiological disruptions. In *Oreochromis niloticus*, exposure results in decreased growth, increased pH levels, and reduced dissolved oxygen concentrations (Baoteng et al., 2006; El-Sayed et al., 2007). Behavioural strength in *Daphnia magna* decreases progressively with increasing deltamethrin

concentrations (Ren et al., 2008). These behavioural changes likely reflect broader neurological disruption that impairs feeding, predator avoidance, and reproductive behaviour in exposed fish populations.

Cypermethrin exposure reduces glycogen content in the gills, muscle, brain, liver, and kidney of *Labeo rohita* (Naik et al., 2016). Long-term exposure of *Oreochromis niloticus* to 1.25 µg/L and 2.5 µg/L of cypermethrin over 90 days results in decreased growth and increased mortality compared to unexposed controls (Majumdar & Kaviraj, 2017). These findings are concerning because concentrations in this range have been detected in agricultural surface waters (Stehle & Schulz, 2015), suggesting that real-world exposures may already be sufficient to suppress growth in farmed and wild fish populations.

Oxidative Stress and Biochemical Effects

An important but often understated dimension of pyrethroid toxicity is the induction of oxidative stress. Deltamethrin, cypermethrin, and lambda-cyhalothrin all promote the production of reactive oxygen species (ROS) in the gills, liver, muscles, and kidneys of exposed fish, causing lipid peroxidation, DNA damage, and apoptosis (Gao et al., 2020). Antioxidant enzyme activity, including catalase and superoxide dismutase, is altered following exposure, reducing the fish's capacity to cope with additional environmental stressors (Gao et al., 2020). In *Clarias gariepinus*, deltamethrin exposure at concentrations as low as 0.75 µg/L significantly increases malondialdehyde levels in the liver, kidneys, and gills, while simultaneously reducing catalase activity (Amin & Hashem, 2012). These biochemical changes are important because they represent sublethal damage that may go undetected in standard lethality tests but can still compromise fish health and survival over time.

Histopathological Alterations

Pyrethroids induce marked histopathological damage in various freshwater fish. These alterations include hepatic cell atrophy in *Ctenopharyngodon idella*, degeneration of acidophilic cells, and gill deformation in *Clarias gariepinus* (Obiezue et al., 2015; Srinivasarao et al., 2018). In *Cyprinus carpio*, cypermethrin impairs ionic regulation and gill Na^+/K^+-ATPase activity, indicating compromised osmoregulation (Suvetha et al., 2010). Farag et al. (2021) further noted that gill damage is of particular concern because the gills are the primary point of pyrethroid entry into the fish body. Once absorbed, pyrethroids are distributed to the liver, kidney, and gonads, where they continue to exert toxic effects. These structural changes underscore the importance of assessing pyrethroid-induced tissue damage as part of ecological risk assessments for freshwater systems.

Endocrine Disruption and Reproductive Effects

An additional area of concern is the endocrine-disrupting potential of pyrethroids in fish. Pyrethroids are lipophilic compounds that accumulate in sediments and fish tissues, where they can interfere with hormonal signalling by mimicking, blocking, or synergising with endogenous hormones (Brander et al., 2016). This can lead to abnormal expression of reproductive proteins such as vitellogenin and choriogenin, altered sex ratios, feminisation of male fish, and impaired reproductive success (Brander et al., 2016; Kumar et al., 2015). Notably, pyrethroid metabolites have been shown to exhibit greater endocrine activity than their parent compounds, suggesting that the risk may not diminish as the insecticide degrades (Brander et al., 2016). These findings

highlight a gap in how pyrethroid risk is currently assessed, as standard acute toxicity tests do not capture these longer-term reproductive and developmental effects.

Conclusion

Across multiple studies, pyrethroid insecticides have been shown to inhibit growth, disrupt metabolic function, alter behaviour, induce oxidative stress, impair endocrine signalling, and cause significant organ damage in freshwater fish. Run-off from agricultural fields and effluents from manufacturing industries contribute to elevated concentrations of pyrethroids in aquatic environments, leading to high mortality in severe cases. What is particularly troubling is that even at sublethal concentrations commonly detected in surface waters, pyrethroids are capable of causing cumulative, long-term harm that standard toxicity evaluations may not fully capture.

To minimise these risks, careful application of pyrethroids and responsible management of industrial waste are essential. Regulatory frameworks should also be updated to account for sublethal, oxidative, and endocrine effects, not just acute lethality. Reducing contamination of inland waters is critical for preserving aquatic life, particularly fish.

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