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Toxicological Impact of Lambda-Cyhalothrin on Hepatic and Intestinal Tissues of *Anabas Testudineus:* An Ultrastructural (TEM) Assessment

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Abstract: Lambda-Cyhalothrin (LCT), a widely used pyrethroid insecticide, frequently accumulates in aquatic environments and poses substantial risks to non-target organisms, including freshwater fish. The present work examines the toxic effects of LCT on the liver and intestine of Anabas testudineus using Transmission Electron Microscopy (TEM). Ultra structural evaluation revealed pronounced cellular alterations in exposed fish, indicating impaired metabolic, detoxification, and absorptive capacities. These observations highlight the vulnerability of A. testudineus to pesticide contamination and emphasize the necessity of regulating agrochemical inputs into aquatic systems.

Keywords: Anabas testudineus, Lambda-cyhalothrin, Liver, Intestine.

Introduction

The extensive use of pesticides in agricultural fields and forested areas often results in the release of toxic compounds into the environment. These substances can easily enter water bodies such as reservoirs, streams, and rivers and negatively affect aquatic life, including fish (John and Prakash, 2003).

Lambda-cyhalothrin (LCT) is a broad-spectrum pyrethroid insecticide widely employed to manage diverse insect pests across multiple crop types.Insecticides formulated with pyrethroids are commonly employed across agriculture, public health programmes, and household settings to manage a wide range of insect pests (Amweg and Weston 2005).Lambda-cyhalothrin is frequently sprayed on rice fields to manage insect pests, residues can easily enter surrounding water bodies and sediments. This runoff poses a toxic threat to various aquatic organisms, including mosquitofish, shrimps, crabs, and clams(Lawler et al., 2003).Pyrethroids act as axonic neurotoxins, disrupting normal nerve function by attaching to the proteins that control voltage-gated sodium channels. Under normal conditions, these channels open briefly to trigger a nerve impulse and then close to stop the signal. They serve as routes for ion movement into the axon, allowing excitation to occur. When pyrethroids keep these channels open for too long, the nerve fires repeatedly, ultimately leading to loss of coordination and paralysis (Bradbury and Coats 1989; Shafer and Meyer 2004).Fish, being highly sensitive to waterborne pollutants, serve as useful bio indicators for ecotoxicological studies (Singh and Srivastava, 1999).

There is limited experimental information regarding the histopathological effects of LCT on fish tissues. The liver and intestine are two major organs involved in detoxification, nutrient assimilation, and metabolic regulation. The liver serves as a central site for storage, biotransformation, and excretion of pesticides. The intestine is the first organ to interact with pesticide-contaminated food particles. These organs are well-recognized indicators of environmental pollution (Hinton and Lauren, 1990). Transmission Electron Microscopy provides a powerful tool to identify sub-cellular lesions that may not be visible under light microscopy. The present study evaluates the ultrastructural effects of LCT on hepatic and intestinal tissues of *A. testudineus*, providing insights into the toxicological mechanisms triggered by this insecticide.

Materials and Methods

For this study, *A. testudineus* were procured from Local area Pound of Bhagalpur District Bihar. This source was specifically chosen because it is free from nearby agricultural or industrial activities, minimizing prior chemical exposure. The fish had an average weight of 20-25 ± 0.66 g and measured 8-10 cm in length.

The experiment was conducted in University Department of Zoology, Tilka Manjhi Bhagalpur University, Bhagalpur, Bihar, India. Prior to the study, the fish were acclimated for at least 15 days under laboratory conditions in a 50 L glass aquarium containing dechlorinated water. Water quality parameters were assessed following APHA (1995) methods. The mean values for the test water were: temperature 26.4 ± 1.2°C, pH 7.1 ± 0.23, dissolved oxygen 6.1 ± 0.45 mg/L, alkalinity 285 ± 1.8 mg/L, and total hardness 396 ± 4.5 mg/L. The fish were fed daily to satiation with commercial fish feed (Hashimai, China) and maintained under a 12:12 h light/dark photoperiod.

Commercial-grade LCT (Mustang by Sulpher Mills Limited, Mumbai, India) was used for the exposures. Toxicity test, following APHA (1995) guidelines, was conducted to determine the 96-hour LC_{50} for *A. testudineus*, which was found to be 0.020 mg/20L. Based on this, two sub-lethal concentrations were selected for the study: 0.001mg/20L (Dose-1) and0.003mg/20L (Dose-2), ensuring the doses remained below lethal levels.

The fish were divided into three groups of 10 individuals each, housed in separate aquaria. Group I was maintained in pesticide-free water to serve as the control, while groups II and III were exposed to the sub-lethal concentrations of lambda-cyhalothrin.

Liver and intestinal tissues were fixed in glutaraldehyde for ultrastructural studies, tissue samples were fixed and processed by standard TEM procedures. Ultrathin sections were obtained and examined under a TEM. Ultrastructural changes were compared between control and exposed fish.

Results

Hepatic Ultrastructural Alterations

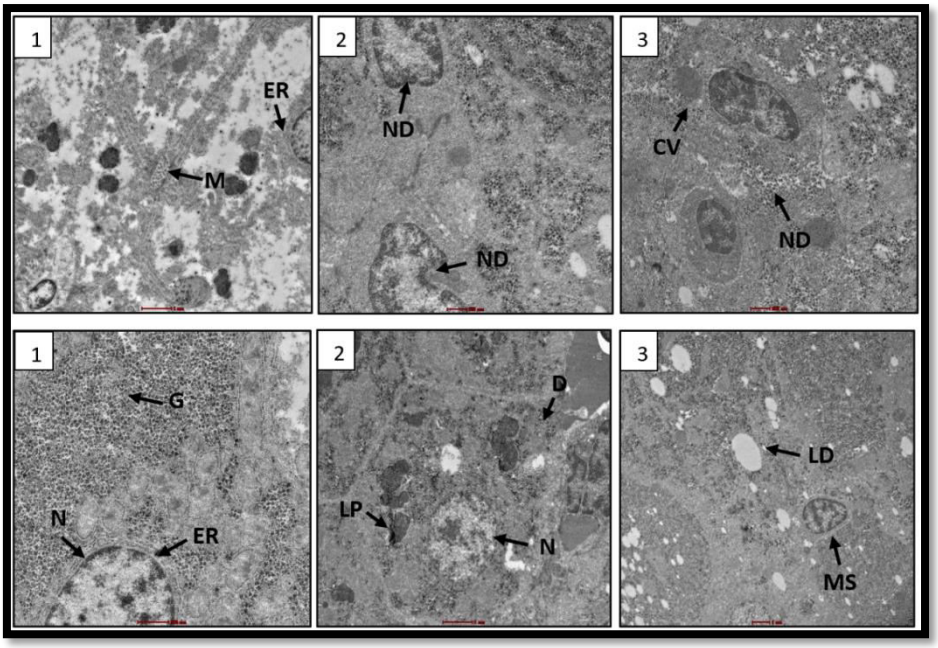
TEM analysis of control liver sections showed normal hepatocytes with distinct nuclei, well-organized endoplasmic reticulum (ER), glycogen, and intact cell membranes (Fig.1).

In the LCT-exposed fish, the liver tissues showed several marked abnormalities,fig.2 (Dose-1)& 3 (Dose-2). The mitochondria in many hepatocytes were swollen, and their cristae appeared ruptured or disorganized, indicating that normal oxidative metabolism had been severely disrupted. The endoplasmic reticulum was noticeably dilated and fragmented, suggesting that both protein synthesis and detoxification processes had been impaired. The nuclei also showed clear signs of damage, taking on irregular shapes and, in some cases, discontinuities in the nuclear membrane. The cytoplasm contained numerous vacuoles, reflecting heightened cellular stress and the early stages of degeneration. Together, these alterations demonstrated the intense hepatocellular stress brought about by lambda-cyhalothrin exposure.

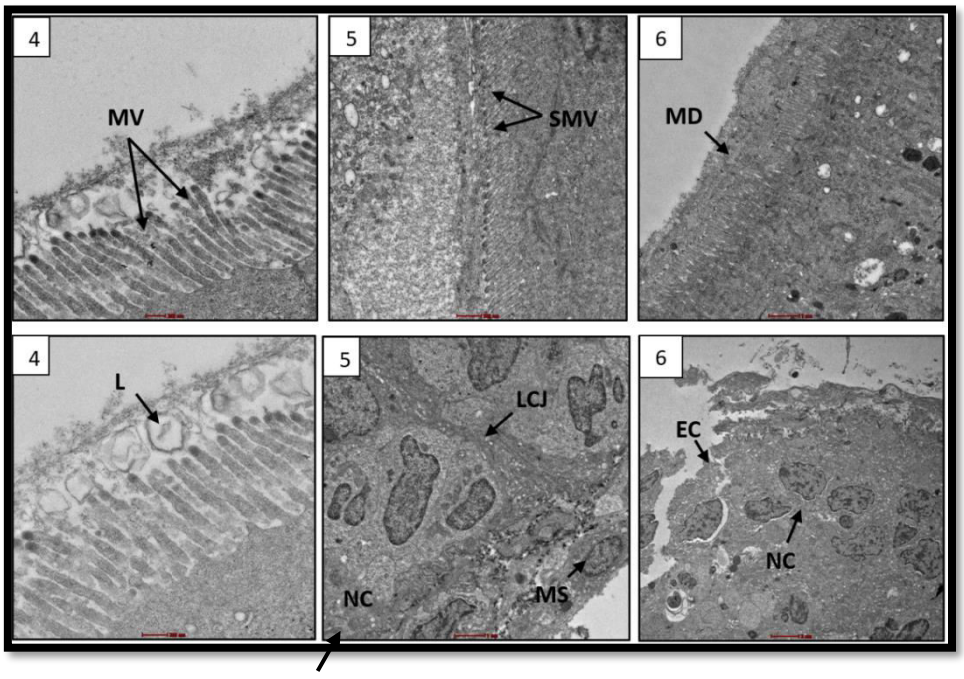
Intestinal Ultrastructural Alterations

TEM sections of the control intestine showed closely arranged microvilli, intact epithelial cells, well-structured mitochondria, and continuous tight junctions connecting adjacent cells (Fig.4).

Exposure to LCT resulted in clear structural damage to the intestinal tissue,fig.5 (Dose-1)& 6 (Dose-2). The microvilli along the apical surface were often shortened, fused, or completely worn away, suggesting that nutrient absorption had been significantly compromised. The epithelial cells themselves showed marked injury, with vacuolated cytoplasm, distorted mitochondria, and disrupted endoplasmic reticulum commonly observed in the enterocytes. The junctions between neighbouring epithelial cells also appeared loosened or missing, which could have increased the permeability of the intestinal lining. In more severe cases, the mucosal surface showed extensive disruption, including sloughing of the epithelial layer and distorted villus architecture. Altogether, these findings indicate that LCT severely damaged both the structure and function of the intestinal barrier.



Figs.1-3. Fig. 1 Liver Tissue of control fish, Fig. 2exposed to 0.001mg/20L, and Fig. 3exposed to 0.003mg/20L for 28 days. G (Glycogen), N (Nucleus),ER (Endoplasmic Reticulum), D (Degenerative Plasma membrane), ND (Nuclear deformation), LP(Lysosomal proliferation), CV (Cytoplasmic Vacuolation), MS (Mitochondrial swelling), LD (Lipid droplets), M (Mitochondria).



Figs.4-6.Fig. 4 Intestine Tissue of control fish, Fig.5 exposed to 0.001mg/20L, and Fig. 6 exposed to 0.003mg/20L for 28 days. EC (Damage Epithelial Cells), L (Lipid droplets), LCJ (Loosening of Cell Junctions), MD (Mucosal Disruption), MS (Mitochondria Swelling), MV (Microvilli), NC (Necrosis), SMV (Shortened Microvilli).

Discussion

The ultrastructural damage observed in *A. testudineus* demonstrates the toxic impact of LCT on organs crucial for metabolism and immunity. Mitochondrial degeneration in the liver suggests impaired ATP production, while alterations in ER point to cellular stress and disrupted detoxification. The liver is one of the most severely affected organs in fish exposed to pesticides, as it serves as the primary site for detoxification (Dutta et al., 1993). Muthukumaravel et al., 2013 observed that in *Oreochromis mossambicus* exposed to 0.0025 ppm of cyhalothrin for 10 and 20 days, the liver displayed marked pathological changes, including dilated blood sinusoids, vacuolization, and disrupted cell boundaries. These findings further highlight the toxic nature of cyhalothrin to aquatic organisms.Structural alterations in the liver are often reliable indicators of prior exposure to environmental pollutants. Gill et al. (1990) reported various hepatic lesionssuch as hypertrophy, vacuolization, nuclear pyknosis, karyolysis, and fatty degeneration in *Pontius conchoids* following chronic exposure to sub-lethal doses of three pesticides. Similarly, Cengiz et al. (2001) observed degeneration, hypertrophy, sinusoidal enlargement, hemorrhage, nuclear pyknosis, cytoplasmic vacuolization, and mononuclear lymphocyte infiltration in the liver of pesticide-exposed fish. In *Corridors palates* treated with methyl parathion, cloudy swelling, bile stagnation, focal necrosis, atrophy, and vacuolization have also been documented (Fanta et al., 2003). Cengiz and Unlu (2006) further reported hepatocyte hypertrophy, increased Kupffer cell numbers, circulatory disturbances, sinusoidal narrowing, nuclear pyknosis, fatty degeneration, and focal necrosis in *Gambusia affine* exposed to deltamethrin.

Similarly, intestinal erosion and microvilli loss indicate reduced digestive and absorptive efficiency.The intestine, being the first organ to encounter food-borne contaminants (Braunbeck and Appelbaum, 1999), also exhibits notable damage under pesticide stress. Mandal and Kulshrestha (1980) described villus lesions in *Claries batrachus* exposed to summation. Fish such as *Channa striatus* and *Heteropneustes fossilis* living in polluted waters showed degenerative changes in the serosa, mucosa, and submucosa, along with focal necrosis, epithelial proliferation, and desquamation of villi (Kumari and Kumar, 1997). Braunbeck and Appelbaum (1999) also noted that exposure to endosulfan disrupts the intestinal epithelial lining, indicating impaired nutrient absorption. Additional intestinal abnormalitiesincluding edema, degeneration, lymphocyte accumulation in the lamina propria, nuclear pyknosis, and necrosiswere documented in *G. affinis* exposed to endosulfan (Cengiz et al., 2001). Necrosis and infiltration of lymphocytes and eosinophils were further observed in the intestine of *G. affine* treated with delta methrin (Cengiz and Unlu, 2006).Such lesions may lead to reduced growth, weakened immunity, and increased susceptibility to environmental stressors. The findings agree with earlier studies indicating that pyrethroids interfere with membrane integrity, generate oxidative

stress, and disturb enzymatic functions in fish. TEM data thus provide direct visual evidence of sub-cellular impairment caused by LCT exposure.

Overall, the histopathological findings from this study show that sub-lethal exposure to LCT leads to extensive damage in the liver and intestinetissues of *A. testudinous*. Such alterations, consistent with previous research, can result in significant physiological disruptions that may ultimately prove fatal to the fish.

Conclusion

LCT induces profound ultrastructural alterations in the liver and intestine of *Anabas testudineus*. The observed lesions,ranging from mitochondrial swelling to epithelial erosion,highlight the insecticide's capacity to disrupt normal physiological processes. These findings underscore the ecological risks associated with pyrethroid contamination and reinforce the need for strict monitoring of pesticide use in aquatic environments.

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Conflict of Interest

I confirm that there are no conflicts of interest.

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