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Acute and Chronic Toxicity of Deltamethrin on the Hepatopancreas of Freshwater Crab *Barytelphusa cunicularis*: A Histopathological Study

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Abstract

Deltamethrin, a commonly used synthetic pyrethroid insecticide, often contaminates freshwater ecosystems through agricultural runoff, which may pose risks to non-target aquatic organisms. However, information on its chronic toxicity in freshwater crabs remains limited. The present study examined acute and chronic deltamethrin-induced histopathological changes in the hepatopancreas of *Barytelphusa cunicularis*, an ecologically important freshwater crustacean. Acute toxicity assessment using probit analysis revealed 24 h, 48 h, 72 h, and 96 hour LC50 values of 0.0125 ppm (95% CI: 0.008–0.014), 0.0075 ppm (95% CI: 0.005–0.010), 0.0060 ppm (95% CI: 0.002–0.009), and 0.0049 ppm (95% CI: 0.001–0.007), respectively. Crabs were exposed to deltamethrin at 0.00075 ppm (1/10 of 48-hour LC50) for 10, 20, and 30 days in chronic exposure. The hepatopancreatic tissues were stained with hematoxylin and eosin for histological analysis. The changes in the tissues were evaluated by a semi-quantitative lesion scoring system. Histopathological analysis revealed progressive exposure-dependent tissue damage characterised by vacuolation, tubular disorganisation, lumen alteration, nuclear pyknosis and necrosis. Acute exposure resulted in significant degeneration of hepatopancreatic tubules, while chronic exposure led to severe disruption of tissue architecture and extensive cellular damage. Semi-quantitative scoring showed a significant increase in severity of lesions with increasing duration of exposure and concentration (Kruskal–Wallis test, $p < 0.0001$), indicating a clear time- and dose-dependent toxic response.

Major Findings: The results show that deltamethrin causes significant hepatopancreatic damage in *B. cunicularis* and describe histopathological alterations as sensitive biomarkers of pyrethroid exposure in freshwater crustaceans under experimental conditions.

Keywords: *Barytelphusa cunicularis*, Deltamethrin, Ecotoxicology, Freshwater Crustaceans, Hepatopancreas, Histopathology, Pesticide Toxicity

1. Introduction

Barytelphusa cunicularis (Decapoda: *Gecarcinucidae*) is a freshwater crab species, widely distributed in the Indian subcontinent and occurs commonly in rivers, ponds, streams, reservoirs and associated wetland ecosystems[1,2]. The species exhibits characteristic burrowing behaviour in the Godavari River basin of Maharashtra and occupies an important ecological niche as a benthic omnivore in nutrient recycling and organic matter turnover in freshwater habitats[2].

The use of synthetic pesticides in modern agricultural practices has resulted in increased potential for contamination of freshwater resources through surface run-off, leaching and spray drift[3]. Such contamination may have adverse effects on non-target aquatic organisms such as crustaceans, fish, amphibians and benthic invertebrates, thus disturbing the ecosystem structure and ecological balance[3,4]. Among these contaminants, synthetic pyrethroid insecticides are of particular interest due to their extensive usage in agriculture and high toxicity to aquatic organisms, although they are relatively less toxic to mammals[4,5].

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Deltamethrin[(S)-α-cyano-3-phenoxybenzyl(1R,3R)-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropane carboxylate] is one of the most widely used type-II synthetic pyrethroids for the control of agricultural and veterinary pests[6]. Despite the high effectiveness against target insects, deltamethrin is known as one of the most toxic pyrethroids to aquatic organisms, especially crustaceans and fish, even at very low environmental concentrations[4,6]. Recent data indicate that long-term exposure to deltamethrin causes oxidative stress, mitochondrial dysfunction, immunotoxicity, cellular degeneration and tissue damage in aquatic organisms, via overproduction of Reactive Oxygen Species (ROS) and interference with antioxidant defence systems[7,8].

In aquatic invertebrates, the hepatopancreas is a multifunctional organ that is involved in processes of digestion, absorption of nutrients, storage of reserve materials, detoxification, and regulation of metabolism[9,10]. The hepatopancreas is considered to be one of the most sensitive target organs for ecotoxicological evaluation because of its direct involvement in xenobiotic metabolism and detoxification and its susceptibility to pesticide-induced injury[9,11]. Histopathological changes in the hepatopancreas, such as vacuolation, tubular degeneration, epithelial necrosis, lumen distortion and nuclear pyknosis, are widely accepted as sensitive markers of environmental contamination and toxic stress in crustaceans[11,16].

Several studies have reported damage to the hepatopancreas after pesticide exposure in freshwater crabs, prawns and shrimp species[12,16]. Studies on *Macrobrachium nipponense* and *Macrobrachium rosenbergii* have demonstrated that deltamethrin exposure leads to severe histopathological lesions, oxidative stress, immunosuppression, and impairment of hepatopancreatic function[7,17]. Similarly, pyrethroids are reported to cause tissue degeneration, inflammation, and cell disruption in several species of aquatic vertebrates and invertebrates, indicating the ecological significance of pesticide contamination in freshwater ecosystems[4,5,18].

Despite increasing knowledge regarding pyrethroid ecotoxicity, information concerning chronic deltamethrin-induced hepatopancreatic toxicity in freshwater crabs remains limited. In particular, there is a scarcity of data regarding both acute and chronic histopathological responses of *B. cunicularis* to deltamethrin exposure. Furthermore, few studies have attempted to quantify lesion severity using semi-quantitative histopathological scoring systems in this species.

Therefore, the present study was undertaken to evaluate acute and chronic deltamethrin toxicity in *B. cunicularis* through detailed histopathological examination of the hepatopancreas. The study hypothesises that deltamethrin exposure would induce concentration- and time-dependent tissue alterations characterised by vacuolation, tubular disorganisation, nuclear pyknosis, lumen alteration, and necrosis. Histopathological lesions were assessed microscopically and quantified using a semi-quantitative scoring approach. This is among the first studies to investigate both acute and chronic deltamethrin-induced hepatopancreatic toxicity in *B. cunicularis* using a semi-quantitative lesion scoring system. The findings provide baseline ecotoxicological data for an ecologically important Indian freshwater crustacean and contribute to environmental risk assessment of pyrethroid contamination in freshwater ecosystems.

2. Materials and Methods

2.1 Collection and Acclimatisation of Experimental Animals

Adult intermolt *B. cunicularis* of both sexes were collected from the Godavari River basin near Kopargaon, Ahmednagar District, Maharashtra, India (19.8820° N, 74.4760° E). Species identification was confirmed using standard taxonomic keys[1,2]. Following collection, crabs were transported to the laboratory in aerated containers and acclimatised for 7 days in plastic troughs containing dechlorinated tap water. During acclimatisation, animals were fed freshly cut earthworms (*Pheretima posthuma*) *ad libitum*. Water was renewed every alternate day to maintain optimal environmental conditions and reduce accumulation of metabolic wastes. Feeding was discontinued 24 h before the initiation of toxicity experiments. Water quality parameters were monitored during the acclimatisation and exposure period and maintained at pH 6.8 ± 0.2, temperature 24 ± 1°C, hardness 210 ± 10 mg/L as Calcium Carbonate ($CaCO_3$), dissolved oxygen 6.5–7.2 mg/L and photoperiod 12 h light: 12 h dark.

All experimental procedures were conducted with attempts to minimise animal stress and suffering during handling, exposure and tissue collection.

2.2 Preparation of Deltamethrin Solutions

The study was conducted using a commercial emulsifiable concentrate formulation of deltamethrin, Decis® 100 EC

(100 g/L deltamethrin; Bayer CropScience Ltd., India). The formulation was used as the source of the active ingredient. A stock solution was prepared by diluting the commercial formulation with dechlorinated tap water, followed by serial dilution to obtain the required experimental concentrations.

For the acute toxicity experiments, the final nominal concentrations of deltamethrin were 0.0125, 0.0075, 0.0060 and 0.0049 ppm, corresponding to the 24-, 48-, 72- and 96-h LC_{50} values, respectively. For the chronic toxicity experiment, a sublethal concentration of 0.00075 ppm, equivalent to one-tenth of the 48-h LC_{50} (0.0075 ppm), was used for exposure periods of 10, 20 and 30 days.

2.3 Acute Toxicity Assessment and LC_{50} Determination

Acute toxicity experiments were carried out to establish the median lethal concentration (LC_{50}) of deltamethrin in *B. cunicularis*. Healthy crabs of approximately uniform size (carapace width 3.5-4.5 cm; body weight: 25-35g) were randomly allocated to experimental groups (n=10 per group).

Mortality was recorded after 24, 48, 72, and 96 h of exposure. LC_{50} values and their corresponding 95% confidence intervals were estimated using Finney's Probit Analysis.

For histopathological assessment, separate groups of crabs (n = 10 per group) were exposed to the respective LC_{50} concentrations corresponding to each exposure duration (Table 1).

- Control group (pesticide-free water)
- 24 h exposure group (0.0125 ppm)
- 48 h exposure group (0.0075 ppm)
- 72 h exposure group (0.0060 ppm)
- 96 h exposure group (0.0049 ppm)

Mortality observations and behavioural responses were recorded throughout the exposure period. Exposure solutions were renewed every alternate day to maintain concentration stability.

2.4 Chronic Exposure Experiment

For chronic toxicity assessment, crabs were randomly divided into four groups (n = 10 per group):

- Control group
- 10-day exposure group

Table 1. Relative toxicity of Deltamethrin pesticide when freshwater crab *Barytelphusa cunicularis* is exposed to acute concentrations (24 to 96 h)

Sr. No.	Exposure (h)	LC_{50} Value (ppm)	Regression Equation	Lower Confidence Limit	Upper Confidence Limit
1	24	0.0125	y = 119.07x + 3.5117	0.008	0.014
2	48	0.0075	y = 111.76x + 3.9989	0.005	0.010
3	72	0.0060	y = 143.31x + 4.0993	0.002	0.009
4	96	0.0049	y = 177.6x + 4.092	0.001	0.007

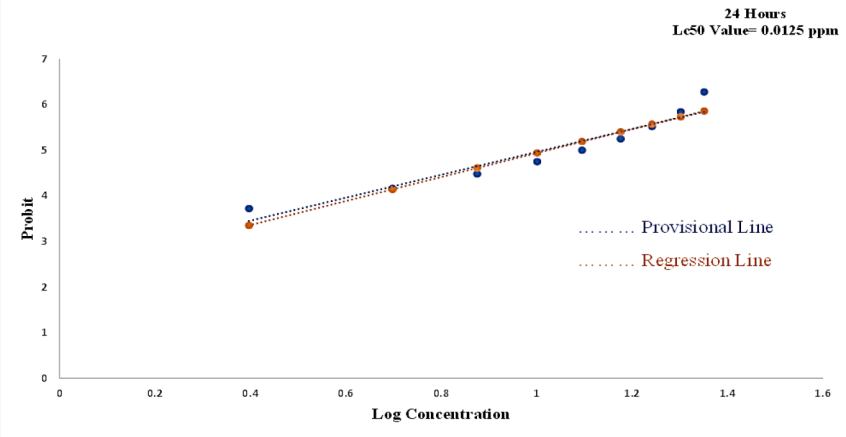


Figure 1. Calculation of 24-hour LC_{50} value and regression line.

- 20-day exposure group
- 30-day exposure group

Experimental animals were exposed to 0.00075 ppm deltamethrin, corresponding to one-tenth of the 48-h LC_{50} value. Crabs were fed freshly cut earthworm pieces *ad libitum* except during the 24 h preceding tissue collection. Water quality parameters were maintained within the ranges described above throughout the experiment.

2.5 Tissue Collection and Histopathological Analysis

At the end of each exposure period, crabs were anaesthetised by immersion in ice-chilled water (0–4°C) for approximately 15–20 min until complete immobilisation was achieved. Animals were subsequently dissected, and the hepatopancreas was carefully excised, rinsed in physiological saline (0.75% NaCl), and immediately fixed in Bouin's solution for 24 h. Fixed tissues were dehydrated through a graded ethanol series, cleared in xylene, and embedded in paraffin wax. Serial sections with a thickness of 9 µm were cut with a rotary microtome, mounted on clean glass slides and dried overnight at 37°C. Sections were stained with Hematoxylin and Eosin (H and E) following standard histological procedures. Histological preparations were examined using a Cilika research microscope, and representative photomicrographs were captured at 40× magnification. Scale bars (600µm) were incorporated into all photomicrographs using the microscope imaging software.

2.6 Semi-Quantitative Histopathological Scoring

Histopathological alterations were assessed using a semi-quantitative lesion scoring system adapted from de Melo *et al.*[11]. Four pathological parameters were evaluated:

- Vacuolation
- Tubular disintegration
- Nuclear pyknosis
- Lumen alteration

Lesions Were Scored According to the Following Criteria

Score	Histopathological Severity
0	No observable alteration
1	Mild alteration (<25% tissue affected)
2	Moderate alteration (25–50% tissue affected)
3	Severe alteration (>50% tissue affected)

Histopathological scoring was performed independently by two observers without knowledge of treatment groups. Mean lesion scores were calculated for each experimental group.

2.7 Statistical Analysis

Histopathological observations were evaluated qualitatively through microscopic examination and quantitatively using semi-quantitative lesion scores. Data are presented as mean ± Standard Deviation (SD). The normality of data distribution was assessed using the Shapiro–Wilk test. Since lesion scores represented ordinal data, differences among treatment groups were analysed using the Kruskal–Wallis non-parametric test. Statistical analyses were performed using GraphPad Prism version 10.0 (GraphPad Software, San Diego, CA, USA). Statistical significance was accepted at $p < 0.0001$.

3. Results

3.1 Histological Structure of the Control Hepatopancreas

The hepatopancreas of control *B. cunicularis* exhibited a well-organised architecture consisting of numerous oval to circular hepatopancreatic tubules separated by connective tissue (Figure 1). A separate layer of epithelium surrounded the lumen in the centre of each tubule. Two main types of cells were found in the tubules: absorptive cells and secretory cells. Absorptive cells were columnar-shaped with basally positioned nuclei and characteristic cytoplasmic vacuolation associated with nutrient storage, whereas secretory cells were larger, rounded and distributed throughout the tubular epithelium. The control hepatopancreas showed no structural abnormalities.

3.2 Effects of Acute Deltamethrin Exposure

Progressive histopathological changes in the hepatopancreas were observed following acute deltamethrin exposure, with the severity of lesions being related to the time and concentration of exposure (Table 2).

24-Hour Exposure (0.0125 ppm)

Mild histopathological changes were observed after 24 h of exposure (Figure 2). The hepatopancreatic tubules showed a slight decrease in the number of absorptive cells and an apparent increase in secretory cells. The tubular architecture was generally preserved.

48-Hour Exposure (0.0075 ppm)

Moderate structural alterations were evident after 48 h of exposure (Figure 3). Tubules exhibited constricted lumens, and epithelial cells exhibited darkly stained pyknotic nuclei, indicating the initial stage of cellular degeneration. Mild disorganisation of the epithelium was also seen.

72-Hour Exposure (0.0060 ppm)

Further deterioration of hepatopancreatic structure was observed after 72 h exposure (Figure 4). Tubules showed a significant reduction in lumen diameter, increased frequency of pyknotic nuclei and disruption of epithelial organisation. Cytoplasmic vacuolation was more pronounced than during the earlier exposure periods.

96-Hour Exposure (0.0049 ppm)

The most significant changes were observed after 96 h of exposure (Figure 5). Hepatopancreatic tubules showed severe disorganisation, extensive vacuolation, loss of normal epithelial degeneration and arrangement of connective tissue between neighbouring tubules. These changes demonstrated considerable disruption of the normal hepatopancreatic architecture.

3.3 Effects of Chronic Deltamethrin Exposure

Chronic exposure to a sublethal concentration of deltamethrin (0.00075 ppm) caused progressive histopathological alterations to the hepatopancreas, which increased with the duration of exposure (Table 2).

10-Day Exposure

After 10 days of exposure, hepatopancreas tubules showed mild vacuolation and moderate expansion of the tubular lumen (Figure 6). Absorptive cells could still be recognised, but there were signs of cellular alteration.

20-Day Exposure

After 20 days of exposure, more marked structural changes were seen (Figure 7). Tubules showed increased cytoplasmic vacuolation, shrinkage of absorptive cells and enlargement of the lumen. In some areas, the epithelial lining was irregular.

30-Day Exposure

The highest tissue damage was observed after 30 days of exposure (Figure 8).

The hepatopancreatic tubules showed severe vacuolation, marked disruption of the tubular organisation, degeneration of epithelial cells and extensive distortion of the architecture. Cellular degeneration was markedly more severe than that observed at earlier chronic exposure intervals.

3.4 Semi-Quantitative Histopathological Assessment

Semi-quantitative lesion scoring revealed a dose-dependent increase in tissue damage with increasing

Table 2. Semi-quantitative scoring of histopathological alterations in the hepatopancreas of *B. cunicularis* following acute and chronic exposure to deltamethrin. Scores represent mean ± standard deviation (n = 10 per group) for vacuolation, tubular disintegration, nuclear pyknosis, and lumen alteration, graded on a scale from 0 (no alteration) to 3 (severe alteration). The Kruskal–Wallis test indicated significant differences among treatment groups for all parameters ($p < 0.0001$).

Group	Vacuolation (mean ± SD)	Tubular Disintegration (mean ± SD)	Nuclear Pyknosis (mean ± SD)	Lumen Alteration (mean ± SD)
Control	0.12 ± 0.23	0.05 ± 0.09	0.16 ± 0.27	0.24 ± 0.35
Acute 24 h	0.94 ± 0.37	0.99 ± 0.50	0.92 ± 0.43	1.12 ± 0.58
Acute 48 h	0.84 ± 0.27	2.05 ± 0.42	1.13 ± 0.56	1.97 ± 0.37
Acute 72 h	2.09 ± 0.43	2.19 ± 0.43	1.76 ± 0.53	1.88 ± 0.48
Acute 96 h	2.84 ± 0.20	2.78 ± 0.30	2.87 ± 0.20	2.86 ± 0.23
Chronic 10d	0.94 ± 0.62	1.17 ± 0.76	1.14 ± 0.45	2.13 ± 0.46
Chronic 20d	2.14 ± 0.59	1.93 ± 0.32	1.83 ± 0.74	2.73 ± 0.29
Chronic 30d	2.93 ± 0.15	2.82 ± 0.30	2.97 ± 0.05	2.84 ± 0.21

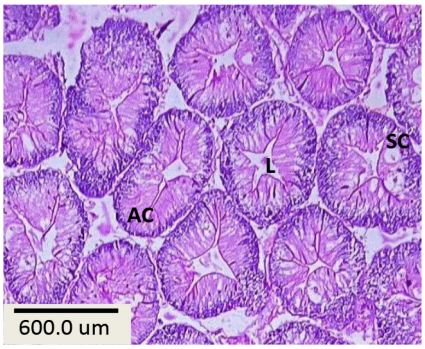


Figure 1. Histological section of the hepatopancreas of control *B. cunicularis* showing normal hepatopancreatic tubules with intact epithelial lining, Absorptive Cells (AC), Secretory Cells (SC), and Central Lumen (L). (H and E stain, Scale bar =600µm).

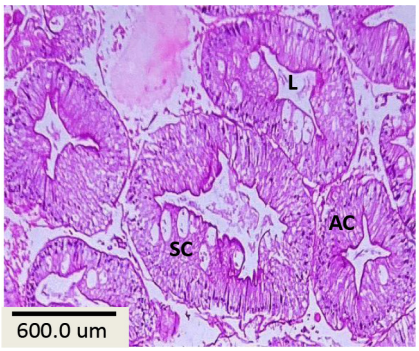


Figure 2. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.0125 ppm) for 24 h showing a slight reduction in absorptive cells and mild alteration of tubular organisation. (H and E stain, Scale bar = 600µm).

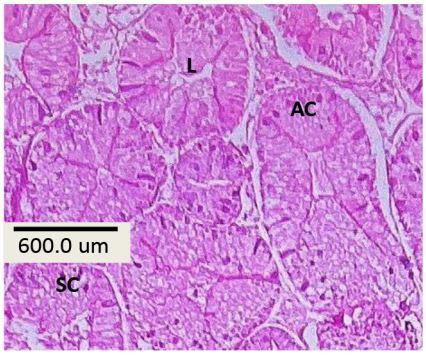


Figure 3. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.0075 ppm) for 48 h, showing lumen constriction, epithelial disorganisation, and pyknotic nuclei. (H and E stain, Scale bar = 600µm).

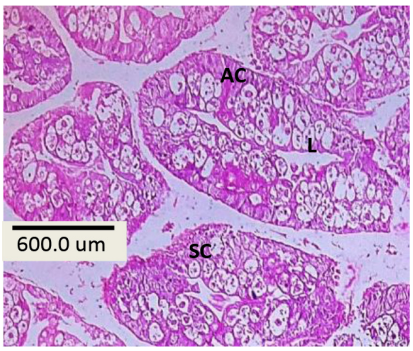


Figure 4. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.0060 ppm) for 72 h, showing increased vacuolation, reduced lumen diameter, and epithelial disruption. (H and E stain, Scale bar = 600 µm).



Figure 5. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.0049 ppm) for 96 h, showing severe vacuolation, tubular disorganisation, epithelial degeneration, and connective tissue disruption. (H and E stain, Scale bar = 600µm).



Figure 6. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.00075 ppm) for 10 days, showing mild vacuolation and enlargement of the tubular lumen. (H and E stain, Scale bar = 600µm).

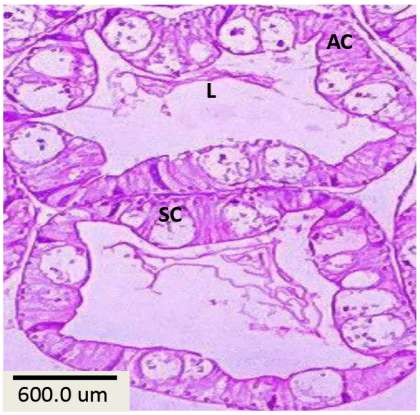


Figure 7. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.00075 ppm) for 20 days, showing increased vacuolation, shrinkage of absorptive cells, and lumen enlargement. (H and E stain, Scale bar = 600µm).
Abbreviations: AC = absorptive cells; SC = secretory cells; L = lumen; V = vacuolation; PN = nuclear pyknosis; TD = tubular disorganisation; ED = epithelial degeneration; LA = lumen alteration.

exposure duration under both acute and chronic treatment conditions. There were significant increases

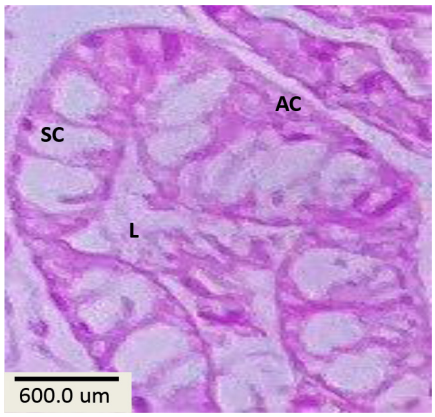


Figure 8. Histological section of the hepatopancreas of *B. cunicularis* exposed to deltamethrin (0.00075 ppm) for 30 days, showing severe vacuolation, tubular disorganisation, epithelial degeneration, and marked architectural distortion. (H and E stain, Scale bar = 600µm).

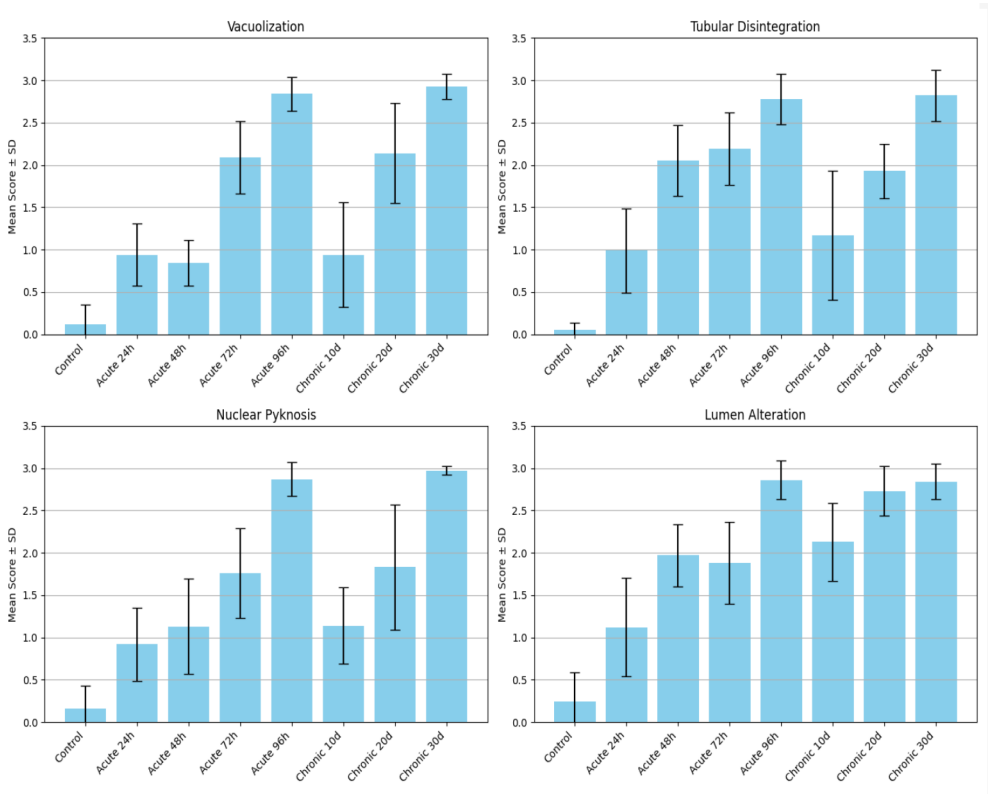


Figure 9. Mean semi-quantitative lesion scores (mean ± SD) for vacuolation, tubular disintegration, nuclear pyknosis, and lumen alteration in the hepatopancreas of *B. cunicularis* following acute (24, 48, 72, and 96 h) and chronic (10, 20, and 30 d) exposure to deltamethrin. Scores were assigned on a scale of 0 (no alteration) to 3 (severe alteration). Statistical analysis using the Kruskal–Wallis test revealed significant differences among treatment groups ($p < 0.0001$), indicating progressive time- and exposure-dependent histopathological damage.

in vacuolation, tubular disorganisation, lumen alteration and nuclear pyknosis in exposed groups compared to controls (Kruskal–Wallis test, $p < 0.0001$). The highest lesion scores were recorded in the 96-h acute exposure group and the 30-day chronic exposure group, indicating a clear time-dependent progression of hepatopancreatic injury following deltamethrin exposure.

4. Discussion

The present study reveals deltamethrin-induced progressive and severe histopathological alterations in the hepatopancreas of *B. cunicularis* under both the conditions of acute and chronic exposure. The observed lesions were vacuolation, tubular disintegration, nuclear pyknosis, lumen alteration and epithelial degeneration. All these lesions increased with the duration and concentration of exposure. The semi-quantitative lesion scoring demonstrated a distinct time-dependent progression of tissue injury, with the highest lesion scores in the Acute 96 h and Chronic 30 d exposure groups. The findings revealed that the hepatopancreas was the main target organ for deltamethrin toxicity in freshwater crabs.

The hepatopancreas is a multifunctional organ that plays major roles in digestion, absorption of nutrients, storage of reserve materials and detoxification of xenobiotics in decapod crustaceans[9,10]. This organ is directly involved in metabolic regulation and detoxification and is therefore especially susceptible to environmental contaminants. The structural damage to the hepatopancreatic tubules may lead to a decrease in digestive efficiency, nutrient assimilation and detoxification capacity and thus to an impairment of the physiological fitness of exposed organisms[9]. The progressive loss of hepatopancreatic architecture observed in the present study indicates major disruption of these vital biological processes.

The extensive vacuolation and epithelial degeneration observed in the present study are similar to alterations reported by Negro *et al.*[12] in the freshwater crab *Zilchiopsis collastinensis* exposed to endosulfan. Similarly, Negro and Collins[15] reported persistent hepatopancreatic lesions after exposure to chlorpyrifos, such as epithelial degeneration and disorganisation of the tubular structure. The studies were carried out with different classes of pesticides, but the repeated occurrence of hepatopancreatic lesions suggests that this organ is sensitive to chemical stressors and confirms its importance as a biomarker in aquatic ecotoxicological evaluations. However, the severity of lesions seen after chronic exposure to deltamethrin in *B. cunicularis* suggests that long-term exposure to pyrethroids could lead to cumulative tissue damage even at sublethal concentrations.

Continuous vacuolation of hepatopancreatic cells was among the most prominent pathological features observed in the present study. Vacuoles are often associated with cellular stress, disruption of intracellular transport mechanisms, lysosomal dysfunction, and accumulation of damaged cellular components. Increased vacuolation may therefore be an adaptive response to toxicant exposure during early stages of injury that progresses to cellular degeneration under conditions of prolonged exposure. The significant increase in vacuolation scores during both acute and chronic exposure suggests a progressive disruption of cellular homeostasis within the hepatopancreas.

There is also tubular disintegration and nuclear pyknosis, indicating severe cellular injury. Pyknosis is characterised by chromatin condensation and nuclear shrinkage, and is often linked to irreversible cellular damage and cell death. The high pyknosis scores observed after 96 h acute exposure and 30 d chronic exposure indicate advanced degeneration of the epithelial cells of the hepatopancreas. These histopathological manifestations have been reported in crustaceans exposed to environmental pollutants and are generally considered as indicators of substantial toxic stress[13,16].

The mechanisms responsible for these histopathological changes are likely to be related to the known mode of action of pyrethroid insecticides. The main effect of deltamethrin is binding to voltage-gated sodium channels, prolonging neuronal depolarisation and causing repetitive nerve firing and sustained neuronal excitation[6]. While pyrethroids mainly affect the nervous system, extended neurophysiological stress can interfere with metabolic processes, increase cellular energy demands, and consequently affect other organs involved in detoxification and homeostasis. This can lead to secondary tissue damage in metabolically active tissues such as the hepatopancreas.

Besides neurotoxicity, oxidative stress is thought to be an important mechanism contributing to pyrethroid-induced toxicity. Exposure to deltamethrin causes excessive production of ROS, leading to lipid peroxidation, mitochondrial dysfunction, destabilisation of membranes, and oxidative damage of proteins and nucleic acids[6,13]. ROS cannot be effectively neutralised by antioxidant

defence systems, which may lead to cellular injury and tissue degeneration. The present study reveals extensive vacuolation, epithelial degeneration, tubular disruption and nuclear pyknosis that are consistent with oxidative stress-mediated damage previously reported in aquatic organisms exposed to pyrethroid pesticides. Biochemical markers of oxidative stress were not measured in the present study. However, the histopathological changes observed strongly indicate the involvement of oxidative injury pathways.

An important observation in this study is that the lesions become more severe with chronic exposure than with acute exposure. Acute exposure resulted in marked tissue injury, whereas prolonged exposure to a sublethal concentration caused a progressive deterioration of the hepatopancreatic architecture with consistently high lesion scores. This result indicates that the long-term environmental exposure to low concentrations of deltamethrin can represent a serious ecological risk without apparent mortality. Such cumulative tissue damage may affect feeding efficiency, growth, reproduction and overall survival of populations of freshwater crabs.

The semi-quantitative scoring method used in this study was helpful in following the progress of tissue injury. de Melo *et al.* [11] suggested histopathological indices as sensitive indicators of contaminant-induced cytotoxicity in crustacean hepatopancreas. The marked rise in lesion scores recorded in the present study further corroborates the use of semi-quantitative histopathological evaluation as an effective biomonitoring tool for the assessment of pesticide pollution in freshwater ecosystems.

The present study was limited to histopathological endpoints and did not assess biochemical biomarkers, antioxidant enzyme activities or molecular responses associated with deltamethrin toxicity. Additional studies employing a combination of histopathological, biochemical and molecular approaches would provide a more comprehensive understanding of mechanisms underlying pesticide-induced tissue injury in freshwater crustaceans.

In conclusion, the results show that deltamethrin causes progressive damage in the hepatopancreas with vacuolation, tubular disintegration, nuclear pyknosis, lumen modification and epithelial degeneration. Lesion severity increased with exposure duration and concentration, suggesting the ecological risks of pyrethroid contamination of freshwater environments and supporting the use of hepatopancreatic histopathology as a sensitive biomarker of pesticide exposure.

The present study was limited to histopathological examination and did not explore biochemical biomarkers such as antioxidant enzymes, lipid peroxidation, AChE activity or molecular markers of oxidative stress. Future studies with combined histological, biochemical and molecular endpoints would give a more complete picture of the deltamethrin toxicity in freshwater crustaceans.

5. Conclusion

The present study shows the effects of acute and chronic exposure to deltamethrin on the histopathology of the hepatopancreas of the freshwater crab *B. cunicularis*. The progressive tissue damage resulted in vacuolation, tubular disintegration, nuclear pyknosis, lumen alteration and epithelial degeneration. The severity of the lesions was found to increase with time and concentration of exposure. Semi-quantitative histopathological evaluation showed a clear time-dependent progression of tissue injury, with the most pronounced alterations in the Acute 96 h and Chronic 30 d exposure groups.

The results show that the hepatopancreas is a very sensitive target organ of deltamethrin toxicity in crustaceans from freshwater. The observed structural changes suggest significant cellular stress and disorganisation of the normal tissue organisation after exposure to pesticides. The results highlight the utility of hepatopancreatic histopathology as a sensitive biomarker to assess the toxicity of pesticides in aquatic organisms.

In conclusion, this study provides baseline data on acute and chronic effects of deltamethrin in *B. cunicularis* and contributes to a better understanding of the ecological risks associated with pyrethroid contamination in freshwater ecosystems. Future studies combining histopathological, biochemical and molecular biomarkers are suggested to further understand the mechanisms underlying deltamethrin-induced toxicity in freshwater crustaceans.

6. Data Access and Responsibility

All data supporting the findings of this study are included within the article. The authors had complete access to all data and accept responsibility for the integrity and accuracy of the data analysis.

7. Ethical Considerations

Freshwater crabs (*B. cunicularis*) are invertebrate organisms and did not require Institutional Animal Ethics Committee approval under the applicable guidelines. Specimens were collected with authorisation from the Maharashtra State Biodiversity Board (MSBB), Nagpur, Maharashtra, India. All procedures were conducted in accordance with accepted laboratory practices.

8. Acknowledgements

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Authors' Contributions

Rohan Yadav: Conducted the experimental procedures and prepared the manuscript. N.G. Shinde: Assisted in drafting and revising the manuscript.

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