

PESTICIDE POLLUTION AND AQUATIC HEALTH: CYHALOTHRIN TOXICITY IN CYPRINUS CARPIO

Dr Umme Habeeba^{1*}, Dr M David²

^{1*}Associate Professor, Government First Grade College, Rajnagar, Hubli, Karnataka
habeeba0402@gmail.com

²Professor, Department of studies in Zoology, karnatak University, Dharwad, Karnataka

ABSTRACT

Synthetic pyrethroid pesticides, with lambda-cyhalothrin (LCH) being the most frequently used, have been applied extensively in agriculture, but there is much concern about their possible ecotoxicological impacts on aquatic ecosystems. This investigates the sublethal paraphernalia of LCH on the bio-chemical, enzymatic, haematological, and histopathological parameters of *Cyprinus carpio*, a sentinel freshwater species. Under semi-static conditions, fish were exposed to a LCH effective concentration (1.6 µg/L) for 1, 5, 10, 15, and 20 days. Physiological response was assessed in multiple tissues, including the liver, gill, kidney, muscle, intestine, ovary, testis, and blood. Subsequently, it was found that there was time time-dependent decline in soluble, structural, and total protein concentrations with increased free amino acids, protease activity, indicating disturbed protein metabolism as determined by biochemical assays. Elevated acetylcholine levels and inhibited acetylcholinesterase activity were evidenced as neurotoxic effects. Osmoregulatory dysfunction was manifested as ionic imbalance and suppressed activities of Na⁺/K⁺, Ca²⁺, and Mg²⁺ ATPases. It was found that antioxidant enzymes activities (catalase, SOD, GPx, GST) were reduced while malondialdehydes levels were increased, which indicates oxidative stress. There were signs of hepatic injury in the serum AST, ALT, and ALP. Haematological analysis showed marked decreases in RBC and hemoglobin as well as increased WBC counts consistent with anemia and immune stress. Biochemical findings were validated by histopathological changes of necrosis and cellular degeneration, not only in the kidneys, but also in other organs. However, these results underscore the fact that even sublethal exposure to LCH can result in systemic toxicity in fish and thus present serious ecological risks. Ensuring environmentally friendly 'preparedness' and scrutinised pesticide regulation is vital to protecting aquatic life from pesticide exposure, the study concluded.

Keywords: *Cyprinus carpio*, lambda-cyhalothrin, oxidative stress, neurotoxicity, histopathology

1. INTRODUCTION

An unprecedented increase in the use of synthetic pesticides has been triggered by the ever-growing need for the intensification of agriculture. Pyrethroid insecticides may be among these, as they are operative against a extensive range of agricultural pests (Andem et al. 2016). Nevertheless, the advantages of these chemicals are offset by their negative effects on nontarget organisms, especially aquatic species. Similarly with agricultural runoff significantly affects the degradation of the quality of water and its biodiversity in aquatic ecosystems (Ensibi et al., 2013). Lambda-cyhalothrin, a widely applied insecticide (type II), has drawn attention as one finest potent and environmentally persistent type II pyrethroids. Although it is preferred over older pesticides due to its low mammalian toxicity, it has been demonstrated to have severe physiological and cellular impact on water-dwelling creatures, especially freshwater fish (Jaffer et al., 2017). Especially at risk of pesticide contamination, the *Cyprinus carpio* is an ecologically and commercially vital creatures. It represents a suitable model for toxicological assessment because it is widely distributed and sensitive to environmental stressors. Many studies have shown that aquatic organisms exposed to pyrethroids have biochemical, hematological, enzymatic, and histopathological changes. Similarly, these changes are not only bad for the health and survival of individual fish but will also lead to continued threats to the dynamics of the population and the ability of aquatic ecosystems to be self-sustaining (Banaee, 2013; Srivastava et al., 2016). High priority presents the need for identification of ecotoxicological impacts of pesticides in water bodies, particularly chronic and sublethal exposures (Korkmaz et al., 2009).

Lambda-cyhalothrin appears to cause significant biochemical and histological damage among certain fish variety. Richterová et al. (2014) showed that cyhalothrin based pesticides negatively harm the developmental stage of *Cyprinus carpio*, causing growth retardation, deformities and increased mortality. In another study, Velmurugan et al. (2007) reported extensive tissue damage in the gills, hepatic, kidney, and intestine of *Cirrhinus mrigala* bare to lambda-cyhalothrin, which indicates its systemic toxicity (Orun et al., 2014). Consistently, histopathological studies have revealed that lambda cyhalothrin damages the integrity of cells in various organ systems. For example, El-Bendary et al. (2010) reported degeneration and necrosis in hepatic and renal tissues in pesticide-exposed models. In addition, there are alterations in enzyme activities and oxidative stress responses as visualized in biochemical assays. Bifenthrin, another pyrethroid, was stated by Velisek et al. (2009) to affect both hematological and bio-chemical indices in common carp and to be an early biomarker of toxicity. Pyrethroids are particularly well documented for their neurotoxic effects. These compounds inhibit acetylcholinesterase (AChE) action and cause accumulation of acetylcholine (ACh) in neural tissues and disrupt normal synaptic transmission (Neelima et al., 2015). Moreover, studies have demonstrated that pesticide exposure induces an increase in oxidative stress markers, namely lipid peroxidation and a deterioration in antioxidative enzymes like catalase and glutathione peroxidase (Altun et al., 2017; Yasser, 2012). For instance, studies done by Kumar et al. (2012) and Yekeen et al. (2013) indicate that pyrethroid toxicity also affects haematological indices, as well as reproductive physiology (changing RBC and WBC counts, and gonadal tissues structure). The fact that sublethal pesticide exposure can have such a huge impact on fish health at multiple biological levels at concentrations below the LC₅₀ threshold (Stoyanova et al., 2016) further highlights these results.

Although lambda-cyhalothrin has been well characterized for its acute toxicity, research on its sublethal effects, particularly after prolonged exposure, is lacking. To date, most of the existing studies have been in terms of acute histopathological changes or singular biochemical markers. Few comprehensive evaluations combine histological, biochemical, enzymatic, neurophysiological, and reproductive parameters under sublethal exposures. The existence of such a knowledge gap is worrying because chronic, low-level pesticide exposure is more ecologically meaningful and reflects the kinds of things that occur in the atmosphere. Thus, it is essential to examine the integrated paraphernalia of sublethal lambda-cyhalothrin exposure on *Cyprinus carpio* using a multiparametric approach. Such studies are essential to aid in a better understanding of the risks from agricultural pollutants to aquatic life and to use as the basis of environmental regulatory policy. This study is designed with the following objectives:

1. To evaluate the biochemical, enzymatic, reproductive, and histopathological alterations in *Cyprinus carpio* subjected to sublethal exposure to lambda-cyhalothrin. This includes measuring protein metabolism, oxidative stress markers, ionic regulation, enzyme activity, and tissue-level changes.
2. To assess the potential ecotoxicological risks posed by prolonged, sublethal pesticide exposure in freshwater environments. This will help to identify early biomarkers of toxicity and provide scientific insights for the management of pesticide pollution.

2. METHODOLOGY

2.1 Study Design

An experimental toxicological bioassay approach was adopted in the current study, to study the sublethal impact of lambda-cyhalothrin on *Cyprinus carpio*. The exposure system was semi-static, following the guidelines of the “Organization for Economic Cooperation and Development” (OECD) for fish acute harmfulness testing. Daily water and toxicant concentrations could be renewed in order to maintain chemical stability and provide consistent exposure throughout the experimental period. The persistence of the investigate was to determine the physiological, biochemical, and histopathological responses of common carp to a known sublethal concentration of lambda-cyhalothrin under environmentally realistic, chronic exposure conditions.

2.2 Study Location and Population

The experiment was carried out in the Post Graduate Department of Zoology, Karnatak Science College, Dharwad, Karnataka, India. It was a controlled laboratory environment that was suitable for ecotoxicological studies. Healthy

fingerlings and mature adult specimens of *Cyprinus carpio* were obtained from the State Fisheries Department hatchery, Karnataka. Turned the fish acclimatized in big fiberglass tanks comprising dechlorinated regular water (tap water) under optimal physicochemical conditions (temperature: $25 \pm 2^\circ\text{C}$, pH: 7.1 ± 0.3 , dissolved oxygen: 6.8 ± 0.2 mg/L, 12:12 light: dark cycle) at least 15 days before experimentation. Test organisms were required to be visibly healthy, active, and free from external or internal signs of disease, and the inclusion criteria for test organisms were. The fingerlings used for the study weighed about 6 ± 2 grams and had a body length of 5.5 ± 0.3 cm, while adult fish had an average body weight of 70 ± 4 grams. Fish exhibiting abnormal behavior, signs of infection, injury, or those unable to acclimate during the preliminary conditioning phase were excluded from the study.

2.3 Sample Size

The study utilized 1000 fish in total, with 500 in each of the control and experimental groups. To provide statistical robustness and reproducibility of the data, the groups were further subdivided into six replicates. Six individuals per replicate were randomly sampled at each of six designated exposure durations (i.e., 1, 5, 10, 15, and 20 days) and then subjected to a variety of biochemical, histological, and enzymatic assessments. Mortality and behavioral anomalies were daily monitored.

2.4 Data Collection

Preliminary LC_{50} trials were used to determine a sublethal concentration of lambda-cyhalothrin ($1.6 \mu\text{g/L}$) to which fish in the handling assembly were showed. Five intervals of exposure durations were defined: 1, 5, 10, 15, and 20 days. Fish were euthanized post-exposure using a buffered solution of MS-222, dissected, and specific tissues (liver, gill, kidney, intestine, muscle, ovary, and testis) were collected. Caudal vein puncture was also used to extract blood samples for serum preparation. The physiological and biochemical parameters analyzed were a broad range. Soluble, structural, and total protein concentrations were measured using the Lowry method, and protease activity was measured through casein digestion. Liver tissues were quantified to evaluate neurophysiological activity by acetylcholine (ACh) and acetylcholinesterase (AChE) levels. The concentrations of Na^+ , K^+ , and Ca^{2+} in various tissues were determined to monitor ion regulation as well as activities of ion-regulating enzymes, including Na^+/K^+ ATPase, Ca^{2+} ATPase, and Mg^{2+} ATPase.

The key enzymatic biomarkers to assess oxidative stress were determined by liver homogenates with: catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx), glutathione S-transferase (GST), and malondialdehyde (MDA). Also, stages of hepatic functioning enzymes including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were determined in the serum. The Gonadosomatic Index (GSI) and histology of the gonads variable were assessed to evaluate reproductive impact. Blood samples were used to assess hematological parameters, including RBC count, WBC count, hemoglobin (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). The gill, liver, kidney, intestine, and gonadal tissues were subjected to histopathological analysis. The segments were entrenched in paraffin wax and were sliced to $5 \mu\text{m}$ -thick slices employing a microtome, performed in Bouin's solution, and were then marked with hematoxylin and eosin (H&E). The morphological fluctuations observed were necrosis, cellular degeneration, and tissue disintegration were then examined using a compound light microscope to observe the slides.

2.5 Data Analysis

Biochemical assays were presented as mean \pm standard error (SE). All the parameters were plotted with time dependence to see trends with respect to different exposure durations. The 96-hour LC_{50} of lambda-cyhalothrin was calculated using probit analysis from preliminary acute toxicity testing mortality data. Dose-response relationships and confidence intervals were obtained using the plot of the probit kill values versus the logarithmic concentrations.

2.6 Statistical Analysis

ANOVA was employed to do a statistic-based comparison between the control and treated groups. Duncan's Multiple Range Test (DMRT) was further used to find significant differences, with a probability value (p-value) less than 0.05 contemplated statistically significant. To ensure data reliability and reproducibility, all statistical procedures were done using SPSS software (Version 20.0).

2.7 Ethical Consideration

All experimental procedures, as well as animal welfare and ethics regulations, were applied according to institutional guidelines. The study abided by ethical standards in handling and anesthesia of fish and euthanizing them to avoid suffering and distress. The "Institutional Animal Ethics Committee" (IAEC) of Karnatak Science College, Dharwad, approved the experimental procedures.

3. RESULTS

3.1 Alterations in Protein Metabolism

The soluble, structural, and total protein concentrations in the hepatic tissues of Common carp were progressively and significantly reduced by sublethal exposure to lambda-cyhalothrin. This trend is observed and increasing metabolic suppression and hepatic damage with time (day 5 onward to day 20).

Table 1. Protein Profile in Liver Tissue of *Cyprinus carpio* Exposed to Cyhalothrin

Exposure Duration (days)	Soluble Protein (mg/g)	Structural Protein (mg/g)	Total Protein (mg/g)
0 (Control)	40.07 ± 0.09	72.42 ± 0.05	112.49 ± 0.11
1	41.28 ± 0.08	70.33 ± 0.07	111.61 ± 0.10
5	34.17 ± 0.11	68.11 ± 0.06	102.28 ± 0.09
10	29.34 ± 0.13	56.21 ± 0.12	85.55 ± 0.08
15	27.26 ± 0.14	50.16 ± 0.09	82.42 ± 0.07
20	28.11 ± 0.15	40.35 ± 0.23	68.46 ± 0.16

Table 1 shows that cyhalothrin reduces the soluble, structural, and total protein concentrations of the liver of *Cyprinus carpio*. These reductions, particularly after 10 days, represent hepatic stress and metabolic disruption and indicate the negative biochemical effect of sublethal pesticide exposure on protein synthesis and integrity.

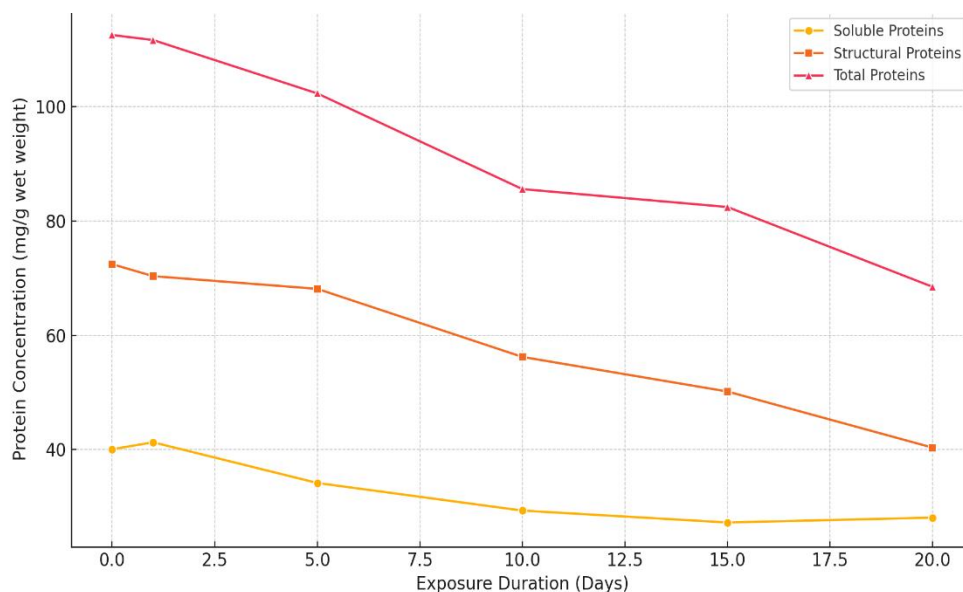
**Figure 1. Levels of Soluble, Structural, and Total Proteins in Liver Tissue of *Cyprinus carpio***

Figure 1 demonstrates that the range of soluble, structural, and total protein in the hepatic tissue of *Cyprinus carpio* bare to lambda-cyhalothrin decline progressively. The data from this reduction show hepatic dysfunction and metabolic stress in freshwater fish exposed for prolonged periods to sublethal toxic conditions, leading to the conclusion of hepatocyte protein synthesis inhibition and proteolysis promoted by this exposure to pesticides.

3.2 Neurotoxicity Markers

Cholinergic regulation was disrupted in liver tissue by exposure to lambda cyhalothrin: acetylcholine (ACh) content was increased, and acetylcholinesterase (AChE) activity was suppressed. The reciprocal effects indicate the onset of neurotoxicity and neural transmission inhibition.

Table 2. ACh and AChE Activity in Liver Tissue of *Cyprinus carpio*

Exposure Duration (days)	ACh ($\mu\text{M/g}$ wet wt)	AChE ($\mu\text{M ACh hydrolyzed/mg protein/h}$)
0 (Control)	14.39 ± 0.54	3.77 ± 0.06
1	14.22 ± 0.34	3.80 ± 0.04
5	14.45 ± 0.44	3.28 ± 0.05
10	14.92 ± 0.36	2.99 ± 0.08
15	15.47 ± 0.26	2.41 ± 0.10
20	16.01 ± 0.18	2.03 ± 0.47

Table 2 shows that exposure to lambda-cyhalothrin resulted in a progressive increase in acetylcholine and a highly significant decrease in acetylcholinesterase activity in *Cyprinus carpio*. This inverse relationship confirms the inhibitory effect of the pesticide on cholinergic regulation during the 20-day exposure period.

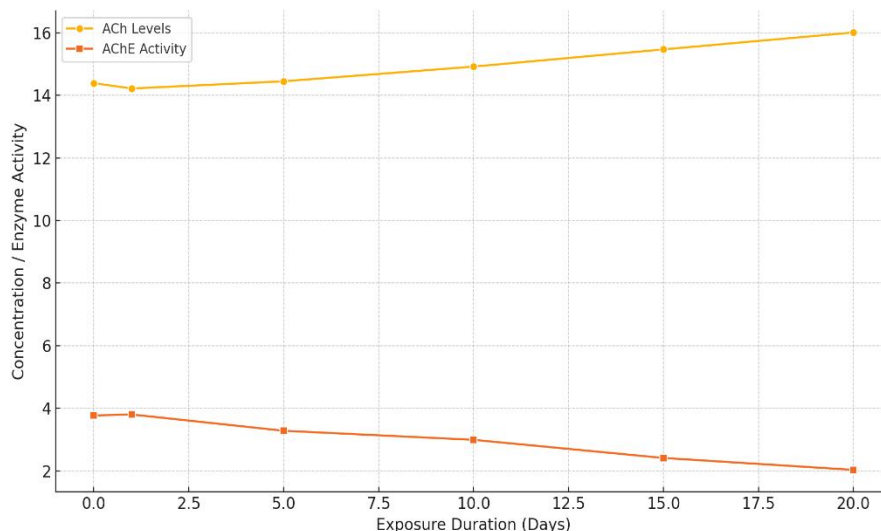


Figure 2. Levels of Acetylcholine (ACh) and Acetylcholinesterase (AChE) in Liver Tissue

Figure 2 shows that the levels of acetylcholine ACh increased and the AChE activity decreased in the liver tissue of *Cyprinus carpio* exposed to lambda-cyhalothrin. This is due to the pattern of inhibited AChE function that enables ACh to accumulate, disrupting neural communication and signaling, and neurotoxicity onset.

3.3 Oxidative Stress Response

Exposed fish had a state of oxidative stress confirmed by biochemical markers. All exposure durations caused a significant decrease in catalase (CAT) activity and an rise in malondialdehyde (MDA), a product of lipid peroxidation.

Table 3. Catalase and MDA Levels in Liver Tissue of *Cyprinus carpio*

Exposure Duration (days)	CAT (U/mg protein)	MDA (nmol/mg protein)
0 (Control)	10.35 ± 0.03	2.26 ± 0.05
1	10.38 ± 0.07	2.48 ± 0.08
5	9.37 ± 0.06	2.92 ± 0.04
10	8.82 ± 0.05	3.44 ± 0.06
15	8.32 ± 0.08	3.97 ± 0.03
20	7.44 ± 0.08	5.83 ± 0.01

Table 3 highlights the oxidative stress response in *Cyprinus carpio* under lambda-cyhalothrin exposure. Significant decrease in the catalase activity with time implied a degraded antioxidant defense, whereas a continuous intensification of the malondialdehyde (MDA) level, reflected the enhancement of lipid peroxidation. This confirms that prolonged pesticide stress results in cellular oxidative damage and redox imbalance in liver tissue.

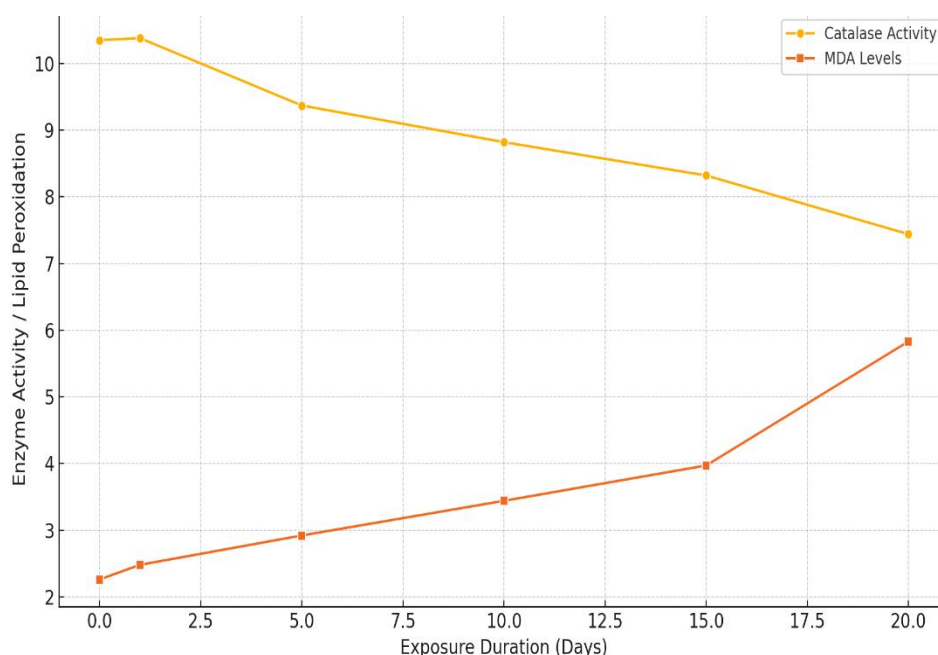


Figure 3. Catalase Activity and MDA Levels Indicating Oxidative Stress

Figure 3 shows that cyhalothrin substantially reduced catalase activity while simultaneously increasing malondialdehyde (MDA) in the liver of *Cyprinus carpio*. These confirmatory parameters of the inverse trend with these two parameters, together with a reduced antioxidant defense and elevated lipid peroxidation, indicate oxidative stress associated with cellular damage associated with prolonged exposure duration.

3.4 Ionic Imbalance and ATPase Activity

The ionic regulation and enzyme-mediated transport were affected by exposure to cyhalothrin. Parts of the blood were depleted of sodium (Na^+), potassium (K^+), and calcium (Ca^{2+}). In addition, ATPase enzymes responsible for ion homeostasis were also inhibited.

Table 4. Ionic and ATPase Changes in Liver Tissue

Days	Na^+ ($\mu\text{g/g}$)	K^+ ($\mu\text{g/g}$)	Ca^{2+} ($\mu\text{g/g}$)	Na^+/K^+ ATPase	Ca^{2+} ATPase	Mg^{2+} ATPase
0	52.27	51.72	70.93	6.94	2.69	2.82
20	50.03	45.58	69.77	6.48	2.53	2.40

Table 4 shows the following disruption of ionic homeostasis and suppression of ATPase actions in the liver tissue of Common carp after cyhalothrin treatment. Under pesticide stress, Na^+ , K^+ , and Ca^{2+} concentrations and Na^+ , K^+ , Ca^{2+} , and Mg^{2+} ATPase activity are found to decrease consistently, suggesting impaired ion transport and osmoregulation.

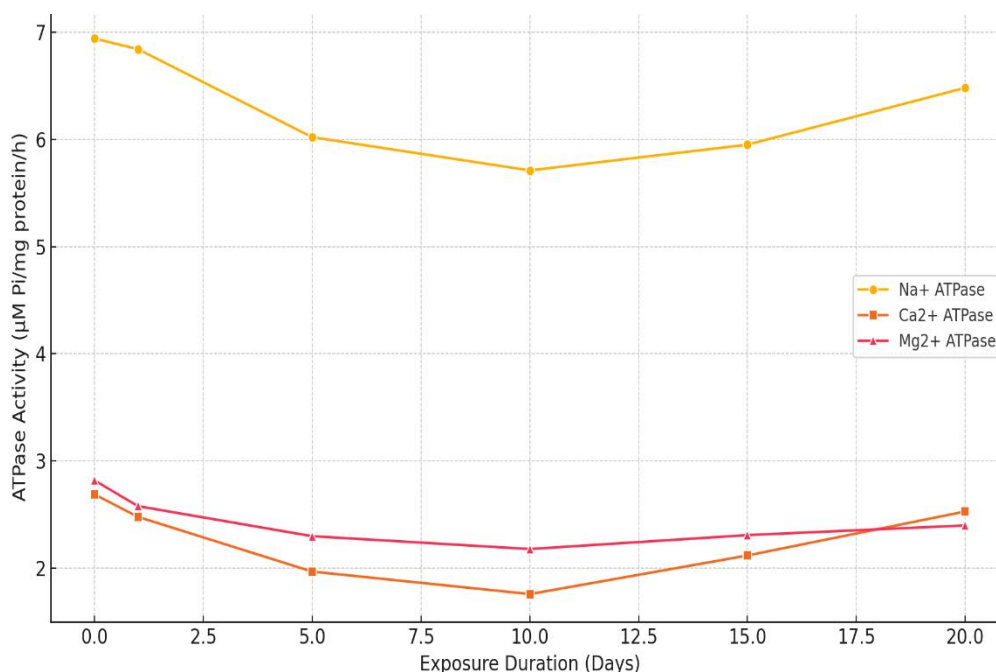


Figure 4. Activities of Na^+/K^+ , Ca^{2+} , and Mg^{2+} ATPase Enzymes

Figure 4 shows the declining trend in the hepatic tissue of Common carp exposed to lambda-cyhalothrin, in Na^+/K^+ , Ca^{2+} , and Mg^{2+} ATPase enzyme activities. This reduction implies a disrupted ion.

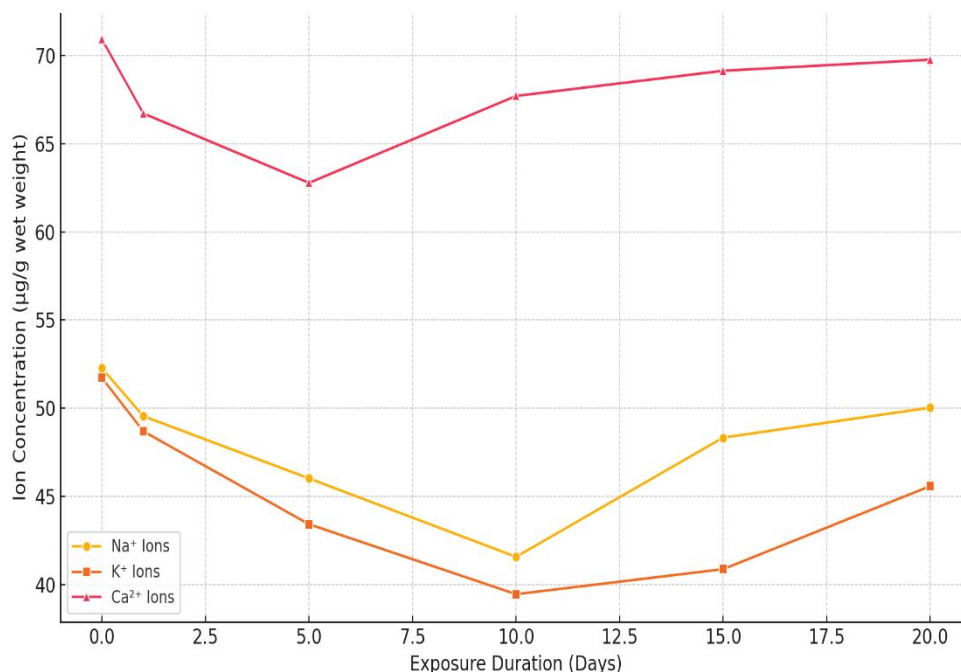


Figure 5. Ionic Concentrations in Liver Tissue (Na⁺, K⁺, Ca²⁺)

Figure 5 shows decreases in sodium (Na⁺), potassium (K⁺), and calcium (Ca²⁺) ion concentrations in the *Cyprinus carpio* liver tissue after cyhalothrin exposure. On exposure days, there is progressive reduction, which indicates ionic imbalance and disrupted osmoregulation, with the pesticide interfering with membrane transport and electrolyte homeostasis in hepatic cells.

3.5 Liver Enzyme and Hematological Changes

The hepatocellular damage was indicated by significant elevations in serum AST, chem. ALT, and chem. ALP. There were also haematological disruptions with decreases in RBC count and hemoglobin and increases in WBC count.

Table 5. Liver Enzyme and Blood Parameters in *Cyprinus carpio*

Days	AST (U/L)	ALT (U/L)	ALP (U/L)	RBC (×10 ⁶ /µL)	WBC (×10 ³ /µL)	Hb (g/dL)
0	32	28	48	2.3	5.4	8.5
20	72	68	92	1.3	8.1	5.8

Table 5 provides significant physiological disturbance caused by lambda-cyhalothrin exposure in *Cyprinus carpio*. Hepatic stress is indicated by elevated liver enzymes (AST, ALT, ALP) and anemia by decreased RBC and hemoglobin levels. The more WBCs in the blood, the more immune activation. Together, these markers confirm that systemic toxicity and fail to support metabolic function in response to pesticide stress.

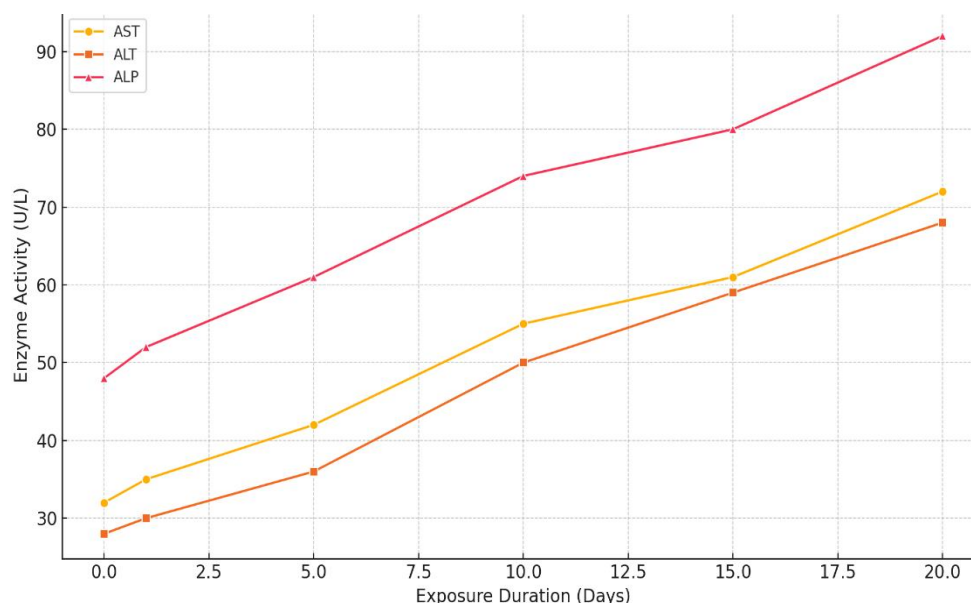


Figure 6. Serum AST, ALT, and ALP Activity

Figure 6 shows a progressive intensification in serum AST, ALT, and ALP activities in *Cyprinus carpio* exposed to lambda-cyhalothrin. It shows this elevation to indicate liver dysfunction and hepatocellular injury, which is cellular membrane damage and leakage of enzymes into the bloodstream. The trend confirms that cyhalothrin causes hepatic stress even at sublethal concentrations.

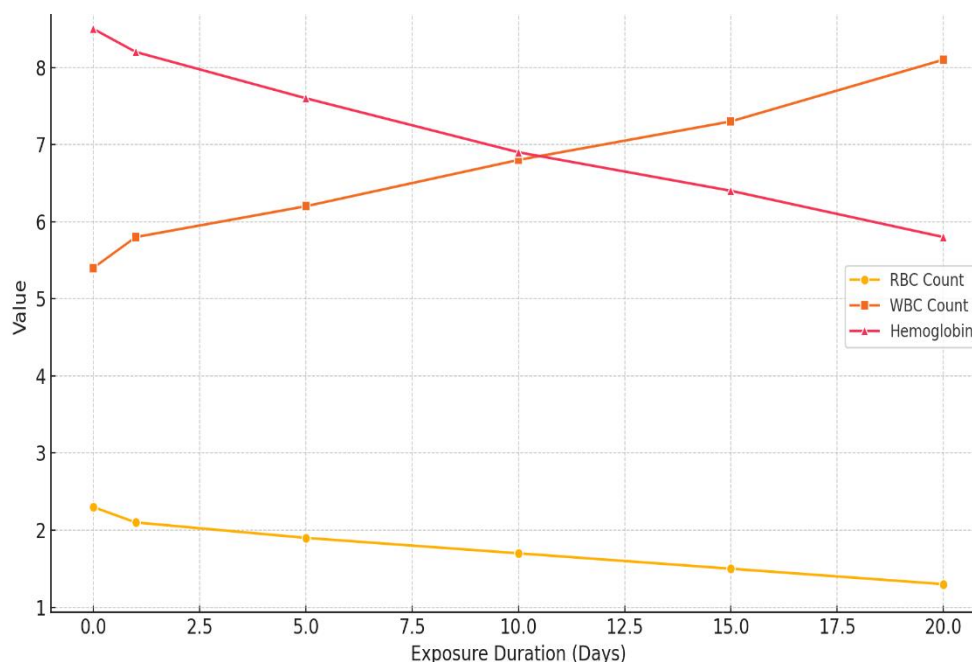


Figure 7. Hematological Changes (RBC ↓, WBC ↑, Hb ↓)

Figure 7 illustrates hematological modifications in *Cyprinus carpio* due to cyhalothrin exposure. If RBC and Hb decrease in a progressive fashion, or WBC increase if the immune response is activated, it indicates anemia. However, analogous changes are systemic physiological stress, confirmed as a toxicological impact of lambda-cyhalothrin on fish health and blood homeostasis.

4. DISCUSSION

This study shows that sublethal exposure to lambda-cyhalothrin (LCH) causes progressive physiological, biochemical, and histopathological impairments in *Cyprinus carpio*. Close to almost all of the effects we saw were on protein metabolism, with large reductions in soluble, structural, and total protein in hepatic tissue. This is likely a result of increased proteolysis, less than synthesis, or damage to protein-synthesizing organelles, suggesting metabolic stress. Consistent increases in ACh and concomitant AChE inhibition, therefore, indicated neurophysiological changes and a synaptically dysregulated cholinergic system. These are typical neurochemical changes of pyrethroid neurotoxicity, where excessive ACh accumulation interferes with normal nerve impulse transmission. Further, ionic imbalance became a critical effect as there was a noticeable declining in tissue amount of both sodium, potassium, and calcium with inhibition of Na^+/K^+ , Ca^{2+} , and Mg^{2+} ATPase activity. Membrane destabilization or direct hindering of ion-regulating environment enzymes may account for the compromised ionic homeostasis. Nevertheless, partial recovery of ionic levels and enzyme activity after a long exposure indicates that the fish may have activated compensatory mechanisms or adaptive responses. The generation of “reactive oxygen species” (ROS) caused an increased MDA in the plasma, and decreased antioxidant enzymes, like catalase and glutathione peroxidase, were evidenced. Histological degeneration in the hepatic, gill, kidney, intestine, and gonadal tissues was due to lipid peroxidation of cellular membranes under oxidative stress. Collectively, these results demonstrate the systemic toxicity of LCH and its capacity to broadly impair multiple biological pathways, even at sublethal concentrations.

The outcomes reported in this work are consistent with the reports of other earlier studies on pyrethroid-induced toxicity in freshwater fish. Parallel declines in total protein levels and hematological disruption following pesticide exposure were also reported by Murthy (1987), and this decline was interpreted as metabolic and systemic stress responses. The neurochemical trends observed in the current study are consistent with the neurotoxic effects of pyrethroids documented by Bradbury and Coats (1989) through AChE inhibition. Other researchers have also observed comparable histopathological lesions such as hepatic necrosis, gill lamellar fusion, and renal tubular degeneration in fish exposed to cypermethrin and deltamethrin. The conserved mode of action of pyrethroids to disrupt structural and functional integrity is evidenced through these lesions. The findings of this study, therefore, not only validate but also extend existing knowledge by integrating biochemical, histological, and physiological endpoints under a unified sublethal exposure model. What is lacking, however, is knowledge of whether or not environmentally realistic, sublethal concentrations of LCH can induce cascading physiological disruptions in freshwater fish. While not immediately lethal, these effects compromise the fish’s overall health, behavior, reproductive capacity, and survival probability. Such sublethal toxicity in the ecological setting would lead to reduced population fitness and changed community structures,

with a possible consequence of long-term ecological imbalance. Use of *Cyprinus carpio* as a model species also highlights the possibility of aquaculture being subject to periodic water quality breakdown resulting from agricultural runoff. The observed haematological and enzymatic biomarkers can be used as early warning indicators of pesticide contamination and can be used in environmental risk assessment and the development of monitoring frameworks.

The study successfully links LCH exposure to systemic toxicity in *C. carpio*, but its results are limited to controlled laboratory conditions. Natural ecosystems are subjected to multiple interacting stressors, namely temperature fluctuations, mixed pollutants, and predation risk, which may either dampen or accentuate the effects observed here. Therefore, extrapolation of results to field conditions should be done with caution. The most important limitation, however, lies in being based upon biochemical and histological parameters due to its exclusive focus. No molecular-level changes, such as gene expression profiling, proteomics, or transcriptomic alterations, were assessed. The results from these could give more mechanistic insights into the cellular pathways that the stress of pyrethroid has on the cell. The effects of sublethal LCH exposure should be expanded in the future to include its multi-generational and chronic effects in natural settings. Those include monitoring reproductive success, developmental deformities, and transgenerational epigenetic impacts. Such studies would give us a broader picture of pesticide toxicity and its ecological connotations. In addition, bioremediation strategies against pesticide contamination in such environments should be urgently developed. The microbial consortia, phytoremediation, and biofiltration systems could give eco-friendly solutions to shield aquatic diversity from synthetic chemical invasion.

5. CONCLUSION

The outcome of this investigation conclusively demonstrated that sublethal revelation to lambda-cyhalothrin has substantial biochemical, physiological, and histopathological effects on *Cyprinus carpio*. Decreased levels of soluble, structural, and total protein demonstrate my disruption of hepatic function and indicate impaired protein metabolism. Increased acetylcholine levels and decreased acetylcholinesterase activity seen in lambda-cyhalothrin neurophysiological disturbances are characteristic of pyrethroid toxicity. By the same token, reduced levels of sodium, potassium, and calcium ions indicate ionic imbalance, together with suppression of ion-regulating ATPase enzymes involved in membrane transport and osmoregulatory failure. This is accompanied by the rise in the level of malondialdehyde, consistent with the commencement of oxidative stress and reactive oxygen species accumulation, accompanied by a reduction of antioxidant enzyme activities. These biochemical disturbances resulted in visible histological injury in the liver, gill, kidney, intestine, and gonadal tissues. Further haematological parameters supported the evidence of systemic toxicity with anemia-like conditions and immunological stress. The elevation of blood liver enzymes, comprising ALT, ALP, and AST, emphasizes hepatocellular dysfunction. Results were taken together and confirm that even sublethal concentrations of lambda-cyhalothrin represent a significant risk to fish health. These findings give us a critical reference for environmental risk assessment and highlight the need for strict regulation of pesticide use in aquatic systems. Such toxicological impacts should be mitigated, and aquatic biodiversity preserved, and preventive strategies and monitoring frameworks are required.

REFERENCES:

- Altun, S., Özdemir, S., & Arslan, H. (2017). Histopathological effects, responses of oxidative stress, inflammation, apoptosis biomarkers, and alteration of gene expressions related to apoptosis, oxidative stress, and the reproductive system in chlorpyrifos-exposed common carp (*Cyprinus carpio* L.). *Environmental Pollution*, 230, 432-443.
- Andem, A. B., Ibor, O. R., Joseph, A. P., Eyo, V. O., & Edet, A. A. (2016). Toxicological evaluation and histopathological changes of synthetic pyrethroid pesticide (Cypermethrin) exposed to African Clariid mud catfish (*Clarias gariepinus*) fingerlings. *Journal of Toxicological and Pharmacological Research*. 2016b, 8(5), 360-367.
- Banaee, M. (2013). Physiological dysfunction in fish after insecticide exposure. In *Insecticides-Development of safer and more effective technologies*. IntechOpen.
- El-Bendary, H. M., Negm, S. E., Saleh, A. A., Kady, M. M., & Hosam Eldeen, F. A. (2010). HISTOPATHOLOGICAL CHANGES ASSOCIATED WITH EXPOSURE OF MALE MICE TO PYRETHROID PESTICIDE (LAMBDA-CYHALOTHRIN). *Journal of Plant Protection and Pathology*, 1(9), 697-710.
- Ensibi, C., Pérez-López, M., Rodríguez, F. S., Míguez-Santiyán, M. P., Yahya, M. D., & Hernández-Moreno, D. (2013). Effects of deltamethrin on biometric parameters and liver biomarkers in common carp (*Cyprinus carpio* L.). *Environmental Toxicology and Pharmacology*, 36(2), 384-391.
- Jaffer, N. S., Rabee, A. M., & Al-Chalabi, S. M. (2017). Biochemical and hematological parameters and histological alterations in fish *Cyprinus carpio* L. as biomarkers for water pollution with chlorpyrifos. *Human and Ecological Risk Assessment: An International Journal*, 23(3), 605-616.
- Korkmaz, N., Cengiz, E. I., Unlu, E., Uysal, E., & Yanar, M. A. H. M. U. T. (2009). Cypermethrin-induced histopathological and biochemical changes in Nile tilapia (*Oreochromis niloticus*), and the protective and recuperative effect of ascorbic acid. *Environmental toxicology and pharmacology*, 28(2), 198-205.
- Kumar, A., Sharma, B., & Pandey, R. S. (2012). Assessment of stress in response to pyrethroid insecticides, Î»-cyhalothrin and cypermethrin, in a freshwater fish, *Channa punctatus* (BLOCH). *Cellular and Molecular Biology*, 58(1), 153-159.
- Neelima, P., Rao, K. G., Rao, N. G., & Jammu, C. S. R. (2015). Enzymatic alterations as biomarkers of cypermethrin (25% EC) toxicity in a freshwater fish, *Cyprinus carpio* (Linn.). *Int J Fish Aquat Stud*, 3(1), 149-158.
- Orun, I., Selamoglu, Z., Gulhan, M. F., & Erdogan, K. (2014). Role of propolis on biochemical and hematological parameters of *Oncorhynchus mykiss* exposed to cypermethrin. *Journal of Survey in Fisheries Sciences*, 1(1), 21-35.

11. Richterová, Z., Máchová, J., Stará, A., Tumová, J., Velíšek, J., Ševčíková, M., & Svobodová, Z. (2014). Effects of Cyhalothrin-Based Pesticide on Early Life Stages of Common Carp (*Cyprinus carpio* L.). *BioMed Research International*, 2014(1), 107373.
12. Srivastava, P., Singh, A., & Pandey, A. K. (2016). Pesticides toxicity in fishes: biochemical, physiological and genotoxic aspects. *Biochemical and cellular archives*, 16(2), 199-218.
13. Stoyanova, S., Yancheva, V., Iliev, I., Vasileva, T., Bivolarski, V., Velcheva, I., & Georgieva, E. (2016). Biochemical, histological, and histochemical changes in *Aristichthys nobilis* Rich. liver exposed to thiamethoxam. *Periodicum biologorum*, 118(1).
14. Velisek, J., Svobodova, Z., & Machova, J. (2009). Effects of bifenthrin on some haematological, biochemical and histopathological parameters of common carp (*Cyprinus carpio* L.). *Fish physiology and biochemistry*, 35, 583-590.
15. Velmurugan, B., Selvanayagam, M., Cengiz, E. I., & Unlu, E. (2007). Histopathology of lambda-cyhalothrin on tissues (gill, kidney, liver, and intestine) of *Cirrhinus mrigala*. *Environmental Toxicology and Pharmacology*, 24(3), 286-291.
16. Yasser, A. G. (2012). Haematological and micronuclei alterations in common carp *Cyprinus carpio* exposed to cypermethrin. *J Thi-Qar Sci*, 3(3), 37-46.
17. Yekeen, T. A., Fawole, O. O., & Bakare, A. A. (2013). Evaluation of toxic effects of lambdacyhalothrin on the haematology and selected biochemical parameters of African catfish *Clarias gariepinus*. *Zoology and Ecology*, 23(1), 45-52.