Research Article

Journal of Fish and Fisheries

Neurohepatic Biochemical Alterations: Protein Profile Shifts in Cirrhinus Mrigala Under Emamectin Benzoate Toxicity

Madhav Pralhad Bhilave*

Division of Fisheries Science, Department of Zoology, Shivaji University, India

***Corresponding Author**
Madhav Pralhad Bhilave, Division of Fisheries Science, Department of Zoology, Shivaji University, India.

Submitted: 2025, Sep 02; **Accepted:** 2025, Oct 06; **Published:** 2025, Oct 14

Citation: Bhilave, M. P. (2025). Neurohepatic Biochemical Alterations: Protein Profile Shifts in Cirrhinus Mrigala Under Emamectin Benzoate Toxicity. *J Fish Fisheries, 2*(1), 01-04.

Abstract

Emamectin Benzoate (EB) is a broad-spectrum insecticide primarily formulated for the management of lepidopteran pests. However, due to agricultural runoff and leaching, considerable amounts of EB may reach aquatic ecosystems, where it can exert toxic effects on non-target organisms, particularly fish. Biochemical alterations in fish serve as sensitive biomarkers and provide the earliest indications of stress under pesticide exposure. The present investigation was undertaken to evaluate the toxic impact of EB on protein metabolism in fingerlings of the freshwater major carp Cirrhinus mrigala. Fingerlings were exposed to predetermined sub-lethal (LC_0) and median lethal (LC_{50}) concentrations of EB, and acute toxicity responses were assessed. Brain and liver tissues two metabolically active and physiologically vital organs were chosen for analysis to determine tissue-specific biochemical responses. The experimental design comprised three groups: a control group, an LC_0 group, and an LC_{50} group. After 96 hours of exposure, protein content was estimated using the Lowry method. The findings demonstrated a marked decrease in protein content in both brain and liver tissues of the exposed groups compared to controls. The decline was more pronounced at LC_{50} concentrations, indicating concentration-dependent stress. These results suggest that EB exposure disrupts protein metabolism in Cirrhinus mrigala, possibly due to enhanced proteolysis, impaired protein synthesis, or increased energy demands under toxic stress.

Keywords: Emamectin Benzoate, *Cirrhinus Mrigala*, Protein Content, Biochemical Alterations, Insecticide Toxicity

1. Introduction

The natural environment is increasingly threatened by diverse pollutants that arise from industrial, agricultural, and urban activities. Effluents from distilleries, cotton mills, tanneries, paper mills, jute mills, and mining operations, as well as agricultural runoffs, are major contributors to water pollution worldwide. Among these, the widespread and often indiscriminate application of insecticides in agriculture has become a serious ecological concern. While these chemicals play an important role in crop protection, they also pose detrimental risks to non-target terrestrial and aquatic organisms. Fish, being in constant interaction with their aquatic environment, are highly susceptible to toxic pollutants.

They serve as an important source of high-quality protein for humans and play a vital role in aquatic food webs. However, bioaccumulation of pesticides in fish tissues can not only impair fish health but may also transfer toxic residues to humans through the food chain, leading to significant health hazards. Moreover, fish are recognized as reliable bio-indicators of aquatic ecosystem health, since toxic compounds entering their bodies induce alterations in physiological and biochemical processes. One such agrochemical of rising concern is Emamectin Benzoate (EB), a semi-synthetic derivative of avermectin. EB is extensively employed to control lepidopteran pests in a wide range of crops including tea, coffee, chilli, cabbage, brinjal, okra, and grapes.

Despite its effectiveness in agriculture, there is a growing need to understand its ecotoxicological impact on non-target aquatic organisms. Current knowledge on the biochemical responses of freshwater fish to EB exposure is limited, leaving a critical gap in our understanding of its potential risks. Considering the economic and nutritional importance of the freshwater major carp *Cirrhinus mrigala*, the present study was undertaken to assess the toxic effects of EB on protein metabolism. Since proteins are essential biomolecules involved in growth, repair, and energy metabolism, alterations in their levels can serve as early biomarkers of toxic stress. Therefore, the present investigation aimed to evaluate protein content in the brain and liver tissues of *Cirrhinus mrigala* fingerlings following acute exposure to sub-lethal (LC_0) and median lethal (LC_{50}) concentrations of EB.

2. Materials and Methods

Fingerlings of *Cirrhinus mrigala* were collected and transported to the laboratory under well-aerated conditions. Upon arrival, the fingerlings were treated with 0.1% potassium permanganate $KMnO_4$ solution for a short duration to eliminate external infections and ectoparasites. Thereafter, the fish were acclimatized for 15 days in large plastic containers filled with dechlorinated tap water under controlled laboratory conditions. During acclimatization, they were fed daily with commercially available floating fish feed. The physico-chemical parameters of the water, including temperature, pH, dissolved oxygen, free carbon dioxide, alkalinity, hardness, and conductivity, were routinely monitored throughout the experimental period.All analyses were carried out following the standard protocols of APHA (1998) to ensure a stable aquatic environment. Water was renewed on alternate days to maintain hygiene and minimize stress to the fish.

The test pesticide Emamectin Benzoate (EB) was obtained. Stock solutions were freshly prepared in dechlorinated tap water before use to maintain uniformity and prevent degradation.Healthy fingerlings with an average body length of 6 ± 2 cm and body weight of 6 ± 2 g were selected for the experiment. The study was designed with three treatment groups: Control group- fish unexposed to EB; LC_0 group- fish exposed to a sub-lethal concentration of 0.583 ppm EB and LC_{50} group- fish exposed to a median lethal concentration of 0.833 ppm EB. For each group, ten fish were maintained in containers holding 20 litres of dechlorinated tap water under static conditions with continuous aeration.

Fish were exposed to the respective concentrations for 96 hours. The experiment was carried out in triplicate to ensure reproducibility. At the end of the exposure period, fish from each group were sacrificed, and tissues were excised for biochemical analysis. The total protein content in tissues was estimated by the method, using bovine serum albumin (BSA) as a standard. The data obtained from biochemical estimations were expressed as mean ± standard error SE. Statistical significance between control and experimental groups was determined using one-way analysis of variance (ANOVA). A significance level of $p < 0.05$ was considered to indicate statistically significant differences.

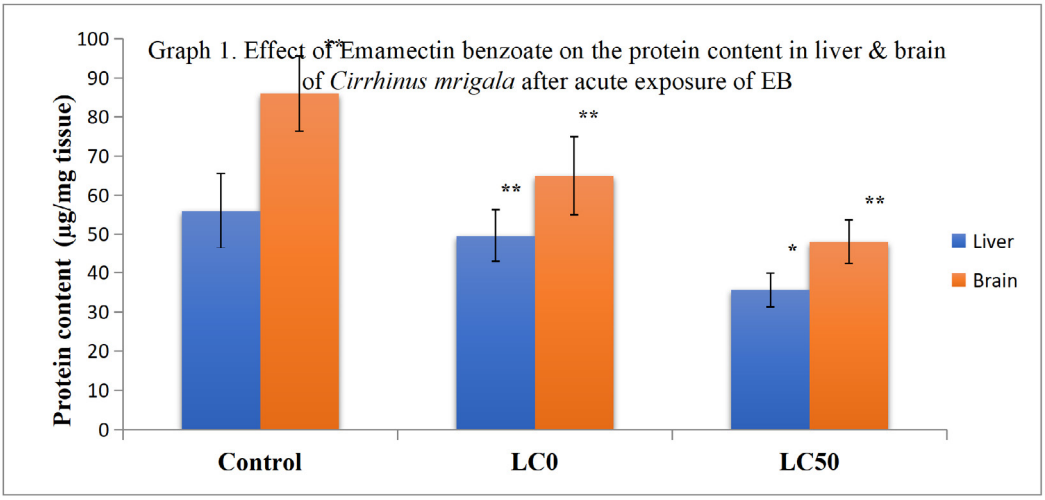
3. Results

The findings presented in the table clearly demonstrate a marked reduction in protein content in both the LC_0 and LC_{50} exposure groups when compared with the control group. The decline in protein levels indicates that *Cirrhinus mrigala* experienced significant biochemical stress upon exposure to Emamectin Benzoate (EB). Such reduction may be attributed to disruption in the protein synthesis machinery or enhancement of catabolic processes leading to the breakdown of proteins into free amino acids, which are then utilized as an alternate energy source under toxic stress conditions [1]. Similar observations were made by, who reported that toxic stress in fish causes a decrease in protein content due to altered metabolic processes [2]. Also documented a reduction in protein levels in the liver of freshwater fish *Catla catla* exposed to cadmium chloride, suggesting that heavy metal toxicity impairs protein metabolism [3].

Furthermore, emphasized that the decline in protein concentration may be associated with a reduction in the rate of anabolic processes, reflecting inhibited biosynthesis of proteins [4] .The decreased protein content observed in the present study can therefore be attributed to several interlinked mechanisms like structural damage or necrosis of cells, impairment of protein biosynthetic pathways, and enhanced degradation of proteins into amino acids to meet the elevated energy demand under stress conditions. Such alterations in protein metabolism not only serve as sensitive biomarkers of xenobiotic toxicity but also highlight the compromised physiological status of fish under pesticide stress.

Sr. No.	Protein(ug/mg wet wt. of tissue)	Experimental Group		
		Control	LC_0	LC_{50}
1.	Liver	56± 9.6	49.6±6.7**	35.6± 4.3*
2.	Brain	86±9.6**	67± 6.7**	48±5.7**
(Values expressed as Arithmetic mean of (n=5); ±SD) *=P<0.05; **=P<0.01; ***P<0.0001; NS=>0.05)				

Table 1: Effect of insecticide Emamectin Benzoate (EB) On Protein Content in Liver and Brain Tissue of *Cirrhinus mrigala*



The observed significant decline in total protein content in the LC_0 and LC_{50} groups compared to the control group indicates that exposure to Emamectin Benzoate imposes biochemical stress on *Cirrhinus mrigala*. This aligns with previous reports, and recent studies provide further insights into the possible mechanisms by which chemical stressors reduce protein levels in fish.

4. Discussion

Pesticides and organic pollutants commonly generate reactive oxygen species ROS in exposed fish tissues. ROS can damage cellular proteins directly by oxidizing amino acid side chains, or indirectly via lipid peroxidation products which then react with proteins. These processes both degrade existing proteins and interfere with protein function e.g., enzyme inactivation. In particular, the review Mechanisms of Pesticide Toxicity in Fish (2025) notes that pesticide exposure leads to protein oxidation, enzyme inhibition, and damage to physiological systems, including disruption to protein synthesis machinery. Toxicants like EB might impair transcription and/or translation processes. Damage to nucleic acids, interference with ribosomal function or regulatory pathways e.g., via disruption of transcription factors or signalling can reduce the rate of protein synthesis. Moreover, stress responses often involve allocation of metabolic resources away from growth and biosynthesis, diverting energy toward detoxification, repair, and maintaining homeostasis. Under stress, fish often catabolize break down proteins to meet elevated energy demands, especially when other energy sources lipids, carbohydrates are depleted or disrupted.

Amino acids from protein breakdown may feed into gluconeogenesis or other metabolic pathways. Chronic stress elevates stress hormones e.g., cortisol that can enhance proteolytic pathways e.g., ubiquitin-proteasome, autophagy and suppress protein synthesis. Even social stress studies in rainbow trout showed such patterns elevated protein degradation in muscle, shifts in protein synthesis in liver. High toxicant concentrations can cause cell damage, necrosis or apoptosis in organs crucial for metabolism liver, kidney, gill, etc.

Loss of viable functional cells leads to loss of protein storage, enzyme capacity, and general metabolic decline. Histological changes often accompany such biochemical changes. The comprehensive review Assessing the Effects of Pesticides on Aquaculture Fish and Ecosystems reports that exposure to pesticides in many fish species leads to decreased total protein content particularly in liver, muscles, gills and kidneys. These decreases are consistent across different classes of pesticides, exposure durations, and fish species.

Another recent piece, pesticide pollution detrimental outcomes and possible mechanisms of fish exposure to common organophosphates and triazines highlights that increased oxidative damage protein carbonyls, etc. is a common mechanism by which proteins are damaged under pesticide stress. Furthermore, mechanisms of pesticide toxicity in fish insights into the ameliorative role of plant-derived compounds underscores that oxidative stress and enzyme inhibition including those necessary for protein synthesis are central to how pesticide exposure reduces protein content.

It also details that phytochemicals may partially mitigate these effects. Such reduction may be attributed to disruption in the protein synthesis machinery or enhancement of catabolic processes leading to the breakdown of proteins into free amino acids, which are then utilized as an alternate energy source under toxic stress conditions [1]. Similar observations were made by, who reported that toxic stress in fish causes a decrease in protein content due to altered metabolic processes [2].

Also documented a reduction in protein levels in the liver of freshwater fish *Catla catla* exposed to cadmium chloride, suggesting that heavy metal toxicity impairs protein metabolism [3]. Emphasized that the decline in protein concentration may be associated with a reduction in the rate of anabolic processes, reflecting inhibited biosynthesis of proteins [4].

5. Summary

Exposure of *Cirrhinus mrigala* fingerlings to Emamectin Benzoate resulted in a significant decrease in total protein content, even at

sub-lethal concentrations. This decline is consistent with earlier and recent studies on pesticide toxicity in fish and can be attributed to multiple mechanisms, including oxidative stress–induced protein oxidation, suppression of protein synthesis, enhanced protein catabolism for energy, and tissue necrosis. Recent literature highlights that pesticides broadly disrupt protein metabolism in fish by damaging biosynthetic pathways, increasing proteolytic activity, and impairing cellular integrity.

Such alterations compromise growth, immunity, and overall physiological functions, making protein content a sensitive biomarker of xenobiotic stress. The findings emphasize the need for detailed molecular, biochemical, and histological investigations, as well as long-term and mitigation studies, to better understand and address the toxic impacts of Emamectin Benzoate on fish health and aquaculture sustainability.

6. Conclusion

The present study demonstrates that exposure to Emamectin Benzoate significantly reduces protein content in *Cirrhinus mrigala*, reflecting biochemical stress even at sub-lethal concentrations. This decrease likely results from impaired protein synthesis, increased proteolysis, and tissue damage, highlighting protein as a sensitive biomarker of xenobiotic toxicity. These findings underscore the need for careful monitoring of pesticide exposure in aquatic environments to safeguard fish health and aquaculture productivity.

Future Avenues

Future studies should focus on long-term and chronic exposure effects of Emamectin Benzoate on *Cirrhinus mrigala*, including organ and tissue- pecific protein analyses, histopathological examinations and molecular assessment of protein synthesis and degradation pathways. Investigating oxidative stress markers, stress hormone levels, and potential mitigation strategies such as dietary antioxidants or plant-derived compounds could provide deeper insights into protective mechanisms. Additionally, establishing precise dose-response relationships will help define safe exposure limits and inform sustainable aquaculture practices.

References

1. Ganeshwade, R. M. (2011). Biochemical changes induced by dimethoate (Rogor 30% EC) in the gills of freshwater fish Puntius ticto (Hamilton). Journal of Environmental Biology, 32(5), 553–556.
2. Borah, S., & Yadav, R. N. S. (1995). Alteration in the protein, free amino acid, nucleic acid and carbohydrate content of muscle and gills in rogor exposed freshwater fish. *Environmental Toxicology and Pharmacology, 1*(2), 113–118.
3. Sobha, K., Poornima, A., Harini, P., & Veeraiah, K. (2007). A study on biochemical changes in the fresh water fish, Catla catla (Hamilton) exposed to the heavy metal toxicant cadmium chloride. *Kathmandu University Journal of Science Engineering and Technology, 3*(2).
4. Khare, S. K., & Singh, S. (2002). Toxicity of pesticides to fish: A review. Environmental Toxicology and Pharmacology, 12(3), 151–157.