J. Sci. Innov. Nat. Earth: Vol. 5, Issue 4, 2025, Page 66-67 ISSN (Online) 2583-2093

OPEN ACCESS

Journal of Science Innovations and Nature of Earth

Journal homepage : https://jsiane.com/index.php/files/index

International, Double-Blind, Quarterly, Peer-Reviewed, Refereed Journal, Edited and Open Access Research Journal

Assessment of Kidney Function Biomarkers in *Mystus seenghala* Following Malathion Exposure

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DOI: https://doi.org/10.59436/jsiane.472.2583-2093

Abstract

The increased reliance on organophosphate pesticide use in agriculture has created concerns regarding how these products may affect freshwater ecosystems. One example would be malathion, as it is one of the most widely used organophosphate insecticides and has high-levels of toxicity toward aquatic organisms (especially fishes). The goal of this study was to determine sub-lethal effects of malathion on the function of kidney biomarker function in the freshwater catfish (*Mystus seenghala*). The study used healthy fish that were held in acclimation conditions within a laboratory prior to treatment, so they would understand the conditions of being held under normal circumstances. All of the fish used in this project were treated with malathion at 1/10th (one-tenth) of their respective 96-hour LC_{50} concentration (malathion), for a total of 6 durations (1, 7, 15, 30, 45 and 60 days). Serum samples of the fish were analyzed for levels of urea, creatinine and ammonium (as dissolved) in accordance with standard biochemical methods. Compared with the control, the results of the trial showed the three markers were significantly elevated, with a duration-dependent effect on all markers over time. The elevation in urea and creatinine was indicative of glomerular function impairment, while the increase in dissolved ammonia indicated a disruption of nitrogen metabolism and poor elimination efficiency. Although there was no evidence of mortality, the trend (progressive) of increased biochemical values supports nephrotoxicity from chronic exposure. These results indicate that kidney biomarkers are sensitive early indicators of pesticide stress on freshwater fish; therefore, ongoing kidney biomarkers biomonitoring is needed to protect freshwater fish and promote/intervene in the ecological balance.

Keywords: Malathion, Renal biomarkers, Nephrotoxicity, *Mystus seenghala*, Organophosphate pesticide

Received 06.08.2025 Revised 16.09.2025 Accepted 18.11.2025 Online Available 01.12.2025

Introduction

The increasing application of pesticides in agriculture and public health has led to serious contamination issues in our aquatic ecosystems (Anees, 1978). Among the various pesticide categories, organophosphate compounds are heavily utilized because they are effective against insect pests. However, their unchecked use has raised significant environmental concerns, particularly about their toxic effects on non-target aquatic species (Kumar & Pant, 1984). Malathion, a commonly used organophosphate insecticide, often makes its way into freshwater systems through agricultural runoff, spray drift, and household discharges, thereby threatening aquatic life (Tripathi & Verma, 2004). Fish are very sensitive to changes in water quality, which is why they are often used as bioindicators for aquatic pollution (Dutta & Arends, 2003). When they are continuously exposed to pesticides, it can disrupt their physiological, biochemical, and metabolic processes, leading to organ damage and a drop in survival rates (Begum, 2004). The kidney is a vital organ that plays a key role in detoxifying and excreting metabolic waste, regulating osmotic balance, and maintaining internal homeostasis. Due to its rich blood supply and filtration function, the kidney is particularly vulnerable to toxins that may be present in the bloodstream (Svoboda *et al*., 2001). When it comes to assessing renal toxicity in fish, scientists often look at changes in kidney function biomarkers like urea, creatinine, and ammonia. Urea, a key nitrogenous waste product that forms during protein metabolism, can signal problems when blood levels are elevated, indicating either impaired renal excretion or increased protein breakdown (Tripathi & Verma, 2004). Creatinine, which comes from the metabolism of muscle creatine, is a trusted marker of kidney function since it's filtered by the glomeruli and not reabsorbed much. Higher creatinine levels usually point to a reduced glomerular filtration rate and potential kidney damage (Begum, 2004). Similarly, ammonia is a toxic nitrogenous waste, and accumulation in the blood, it suggests that the renal excretory systems are failing (Anees, 1978). studies indicate that exposure to organophosphate pesticides can trigger significant biochemical and histopathological changes in the kidneys of fish (Dutta & Arends, 2003). For instance, malathion exposure has been associated with tubular degeneration, glomerular damage, and decreased filtration efficiency, which results in changes to renal biomarkers (Svoboda *et al*., 2001). These biochemical alterations not only suggest nephrotoxicity but also reflect the overall physiological stress that fish face when exposed to pesticides. Extended exposure can lead to cumulative toxic effects, heightening the risk of chronic kidney dysfunction (Kumar & Pant, 1984). *Mystus seenghala* is a freshwater catfish that holds significant economic and ecological value, found throughout the river systems of India. Because of its bottom-dwelling habits and preferred habitats, this species often faces pesticide contamination (Tripathi & Verma, 2004). It's a popular choice for consumption among locals and plays a vital role in regional fisheries. However, there's a surprising lack of information regarding the effects of malathion on kidney toxicity in *Mystus seenghala*, particularly when it comes to kidney function biomarkers. The current study is focused on examining how sub-lethal exposure to malathion affects kidney function biomarkers in *Mystus seenghala*. By measuring levels of urea, creatinine, and ammonia, we aim to gain a better understanding of the renal dysfunction that malathion may cause. This research will also highlight the importance of using renal biomarkers as reliable tools for environmental monitoring and assessing ecological risks (Begum, 2004; Svoboda *et al*., 2001).

Materials and Methods

1.Experimental Fish- We collected healthy freshwater catfish, specifically *Mystus seenghala*, all of uniform size and weight, from local freshwater sources and transported them to the lab in aerated containers. To ensure they were comfortable, we acclimatized the fish to the lab conditions for 10 to 14 days before starting any experiments. During this time, they lived in glass aquariums filled with dechlorinated tap water and were fed a standard commercial diet each day. Water quality parameters such as temperature, pH, and dissolved oxygen were regularly monitored and maintained within suitable limits throughout the experimental period.

2.Chemical Used- Malathion (organophosphate pesticide of analytical grade) served as the toxicant in our study. We prepared a stock solution with distilled water and made the right dilutions to get the sub-lethal concentration needed for our experimental exposure.

3.Experimental Design-After acclimatization, Fish were separated into two groups: a control group and a malathion-treated group. The control group thrived in pesticide-free water, while the experimental group exposed a sub-lethal dose of malathion. fish were observed at various intervals—1, 7, 15, 30, 45, and 60 days. At each of these times, a few fish were randomly selected for biochemical analysis. Throughout the experiment, water was renewed regularly to maintain the desired pesticide concentration and to prevent accumulation of metabolic wastes. No mortality was observed during the exposure period, indicating the sub-lethal nature of the malathion concentration used.

4. Collection of Blood Samples-At each exposure interval, we anesthetized the fish before sampling to keep their stress levels down. We drew blood from the caudal vein with sterilized syringes. After collecting the blood, we let it clot at room temperature, then centrifuged it at 3000 rpm for 10 minutes to separate out the serum. We stored the serum samples low temperature until further biochemical tests.

5.Kidney Function Biomarkers-We assessed kidney function by assessing a few important markers in the blood: urea, creatinine, and dissolved ammonia.

•Urea estimation, we used a standard Colorimetric test which depends on enzymes.
•Creatinine levels were determined by the Alkaline Picrate Method.
•Dissolved ammonia, we used standard Biochemical Procedures suitable for fish serum.
All estimations were performed in line with established protocols to ensure that we achieve both accuracy and reproducibility.

Results

Kidney Function Biomarkers in *Mystus seenghala* Following Malathion Exposure Significant and progressive changes in renal marker proteins resulted from sub-lethal Malathion exposure. At the end of 1, 7, 15, 30, 45, and 60 days post-exposure for urea, creatinine, and dissolved ammonia were significantly elevated. The degree of elevation of these substances was related to duration of exposure allowing for maximum values on the 60th day. While mortality occurred, as expected at sub-lethal doses, biochemical results indicate very strong evidence of renal impairment. Elevated serum urea levels may indicate an increased level of protein catabolism, as well as impaired renal function and subsequent renal excretion. Increased creatinine indicates a decreased GFR and potential nephron damage. Likewise, increased dissolved ammonia indicates a reduced ability of the kidneys to eliminate ammonia and metabolic disturbance.

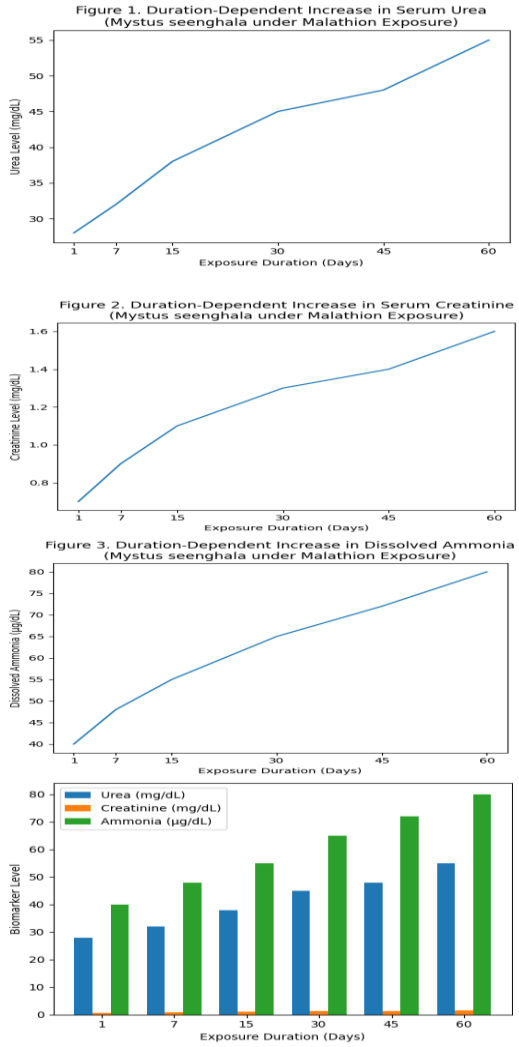

Figure: 4

Exposure Duration (Days)	Urea	Creatinine	Dissolved Ammonia
Control	Normal	Normal	Normal
1 Day	↑ Significant	↑	↑
7 Days	↑↑	↑↑	↑↑
15 Days	↑↑	↑↑	↑↑
30 Days	↑↑↑	↑↑↑	↑↑↑
45 Days	↑↑↑	↑↑↑	↑↑↑
60 Days	↑↑↑ (Maximum)	↑↑↑ (Maximum)	↑↑↑ (Maximum)

Table:1

Discussion

The current research indicates that chronic exposure to malathion at low dosages causes kidney dysfunction (i.e., significant increases in serum urea, serum creatinine, and dissolved ammonia). Although *Mystus seenghala* exposed to sub-lethal levels of malathion do not die, there is clear evidence of nephrotoxicity based on biochemistry data. The most common nitrogenous waste is urea. When it is elevated in the body, it generally indicates both a decline in kidney filter function (glomeral filtration) and an increase in breakdown of protein in the body (catabolism). Therefore, an increase in blood (or serum) urea level is indicative of a reduction in how well the kidneys are excreting waste products as a result of malathion. Of note, some studies have observed increased blood urea levels in aquarium fish exposed to organophosphate (OP) pesticides, which they have linked to damage of kidney tissue as well as metabolic disorder in the fish exposed (Chitra & Abdu 2013; Singh *et al*. 2019). Creatinine is an important marker of kidney function, and elevated creatinine concentrations indicate a slow glomerular filtration rate (GFR). In addition, the increased creatinine concentrations associated with time of exposure support the theory that renal nephrons are structurally and functionally impaired. Elevated serum creatinine concentrations in fish exposed to pesticide further supports the nephrotoxic potential of organophosphates (Van der Oost *et al*., 2003; Ramesh *et al*., 2017). Increased levels of ammonia in aqueous solution give additional evidence for renal functional impairment. Impaired ammonia excretion is attributed to tubular injury and disturbance of nitrogen metabolism. Organophosphate-induced oxidative stress causes injury to renal tissue leading to the accumulation of nitrogenous waste materials (Ahmad *et al*., 2012). The increase in renal biomarkers in M. seenghala was directly dependent on time, which indicates the sensitivity of these kidney parameters to be early indicators of possible malathion toxicity. The data generated by this study demonstrate the potential ecological risks resulting from chronic organophosphate contamination within freshwater ecosystems. Moreover, they underline the need for biomonitoring programs aimed at protecting the environment.

Conclusion

This study shows very clearly that M. seenghala will experience acute renal failure as a result of chronic low-level malathion exposure even though no deaths were noted through out the length of this study. However, all of the chemical markers of renal function measured were progressively altered. Significant increases in serum urea and creatinine are indicative of impaired glomerular function as well as potential structural damage to the kidneys. Furthermore, the elevated levels of total ammonia may be indicative of impaired nitrogen metabolism and the reduced ability to excrete nitrogenous wastes suggests decreased tubular function. Although low levels of malathion may have cumulative harmful effects on aquatic organisms undergoing long-term exposure, there is evidence that urea, creatinine, and ammonia can be used as indicators of physiological disturbance, providing early warning of potential health consequences prior to the onset of visible pathological changes or death. Given the economic and ecological value of M. seenghala as a component of freshwater fisheries, continued exposure to organophosphate pesticides is likely to negatively impact both the health and growth performance of fish, as well as their ability to be sustained at the population level. The findings highlight the importance of stringent regulating and stringent use of organophosphate pesticide and continuing monitoring of the environment to prevent injury from pesticide application to the environment. Incorporating biochemical biomarker analyses into routine assessments of aquatic health may provide early warnings and contribute toward sustainable management and protection of freshwater ecosystems.

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