Analysis of the Biochemical and Histopathological Impact of Polystyrene Microplastic on *Channa punctata* (Bloch, 1793) Fish.

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Abstract

Background: Polystyrene microplastics (PS-MS) are emerging contaminants in aquatic ecosystems and a serious threat to aquatic organisms. The toxicological effects of PS-MS on *Channa punctata* were evaluated by biochemical, histopathological, and physiological parameters.

Methods: Four groups (Control, (1 mg/l) Dose 1, (5 mg/l Dose 2, (10 mg/l) Dose 3) of fish were exposed to PS-MS for 28 days. Fulton's Condition Factor (K), Hepatosomatic Index (HSI), and Kidney Somatic Index (KSI) were measured as biometric indices. Serum biochemical markers of liver (ALT, AST) and kidney (Creatinine, BUN) function were measured. Structural abnormalities in liver and kidney tissues were observed by histopathological analysis.

Results: Significant dose-dependent reductions in biometric indices were observed, consistent with physiological stress. Dose-dependent increases in liver and kidney function markers confirmed hepatic and renal toxicity. Liver tissues and kidney tissues showed severe vacuolization, necrosis sinusoidal congestion in liver tissues and tubular degeneration, glomerular atrophy, and necrosis in kidney tissues, particularly at higher doses.

Conclusion: *Channa punctata* exposed to PS-MS experiences significant dose-dependent physiological and cellular disruptions. The findings underscore the need for immediate action to mitigate microplastic pollution and protect aquatic biodiversity.

Keywords: Polystyrene microplastics, *Channa punctata*, Biochemical markers, Histopathology, Liver toxicity

Introduction

Plastic particles less than 5 millimeters in diameter have become ubiquitous contaminants of aquatic ecosystems, known as microplastics. Microbeads are the degradation products of larger plastic debris and the direct release of microbeads from consumer products (Andrady, 2011). They are persistent because they do not biodegrade, resulting in long-term environmental impacts. Microplastics have been confirmed to be widespread in marine ecosystems, including surface waters, the water column, and sediments (Barnes et al., 2009). Just like freshwater systems, reports of microplastic contamination in rivers, lakes, and wetlands have been documented, especially in heavily populated areas (Wagner et al., 2014). This pollution has ecological consequences at such a vast scale that microplastics are already being ingested by aquatic organisms at all trophic levels, from zooplankton to large fish, frequently at production times and in some instances, even mistaking microplastics for food (Cole et al., 2011).

Physical blockages, reduced feeding efficiency, and impaired energy reserves can follow from the ingestion of microplastics. Also, microplastics serve as vectors for toxic chemicals including persistent organic pollutants (POPs) and heavy metals that adhere to them. Once ingested, these toxins desorb in the gastrointestinal tracts of organisms and therefore bioaccumulate and potentially bio-magnify in the food web (Rochman et al., 2013). The emerging concern of human exposure to microplastics comes from seafood consumption and drinking water (Cox et al., 2019), and with implications for human health.

Marine environments have been the main focus of microplastic research, but freshwater systems are increasingly recognized as important reservoirs and pathways for microplastic pollution. Microplastics are conveyed from their terrestrial source to marine environments via rivers (Lechner et al., 2014). However, Lakes and Reservoirs are sinks, classic ways to accumulate microplastics over time.

Consequences in freshwater systems are similar to those in marine environments. Microplastics have been shown to affect freshwater biodiversity by changing the behavior, physiology, and reproduction of aquatic organisms (de Sá et al., 2018). For example, exposure to microplastics has been associated with reduced growth rates, disrupted endocrine balance, and increased mortality in fish and invertebrates (Batel et al., 2016).

Microplastics ingestion by aquatic organisms is a physical and chemical risk. Microplastics have the potential to physically block the digestive tract, reducing nutrient absorption and causing starvation (Wright et al., 2013). Microplastics are chemically used as carriers for hydrophobic pollutants, such as polycyclic aromatic hydrocarbons (PAHs) and bisphenol A (BPA), that desorb upon ingestion and interact with biological systems (Koelmans et al., 2016). The effects of ingesting microplastics in fish include altered feeding behavior, oxidative stress, and inflammation of tissues (Cortés-Gómez et al., 2018). The histopathological studies have revealed structural damage in the liver, kidneys,

and intestines and, thus, the microplastics appear to interfere with normal physiological functions. Microplastics have also been shown to impact the reproductive systems of aquatic organisms reducing fecundity and developmental abnormalities (Lonnstedt & Eklov, 2016).

Considering the disaster of microplastics spreading into freshwater ecosystems, a comprehensive understanding of their impact on aquatic organisms is very important. The freshwater fish *Channa punctata* is a readily accessible model organism to assess the toxicological effects of microplastics. The research fills critical knowledge gaps by linking dose-dependent microplastic exposure to measurable changes in organ function and structure. The results are expected to help fill in the broader picture of microplastic toxicity and guide efforts to reduce their effects on freshwater ecosystems.

Objective

The objective of this study is to assess the biochemical, histopathological, and physiological responses of *Channa punctata* to different concentrations of polystyrene microplastics.

Methodology

Fish Samples

Channa punctata fish were chosen for the experiment as a test organism for toxicological studies. Standard laboratory conditions were maintained for 28 days before the experimental procedures were initiated. While the fish were out of the water tank, they were kept in well-aerated water tanks, and the temperature and light conditions were controlled to avoid it becoming stressed. The fish were plump and healthy, fed a standard diet, and water quality parameters—pH, temperature, and dissolved oxygen—were regularly measured to maintain optimal conditions.

Test Chemical

In this study, polystyrene microplastic beads of 100 nm size were used. Sigma Aldrich product number 43302 batch number BCCH1665 was used to procure these beads. The beads were supplied as an aqueous solution with a 10% solid concentration and a density of 1.05 g/cm³. The polystyrene microplastics were chosen because they are a relevant pollutant in environmental studies.

Preparation of Stock Solution

The 10% w/v aqueous solution of polystyrene microplastic as provided by the manufacturer was used to prepare the stock solution. The supplied solution was diluted 1000 times with Milli-Q to create a parent stock stock of 1000 mg/L concentration. Ultrasonication was used to rigorously homogenize the stock solution to achieve uniform particle distribution. Working concentrations for the experiment were prepared by serial dilutions. The stock solution was stored in a refrigerator at 2–8°C, as recommended by the manufacturer, to ensure the stability of the stock solution during the study.

Experimental Design

Prior literature was used to formulate the experimental design to ensure relevance and validity. Groups of fish were exposed to different doses of polystyrene microplastics for a predetermined experimental period. To serve as a baseline for comparison, control groups were maintained without exposure to the microplastics. According to the designed protocol, specific doses of polystyrene microplastics were given to the experimental groups, which were prepared from the stock solution. Optimization of exposure duration and doses was made to reflect realistic environmental conditions.

Biometric Assay and Organo somatic Indices (HSI, KSI)

The biometric parameters of the fish were measured to assess their general health condition. The fork length (cm) and body weight (g) of each fish were recorded accurately. These measurements were used to calculate Fulton's condition factor (K) using the formula:

$$K = \frac{W \times 100}{L^3}$$

Where W represented the weight of the fish in grams and L denoted the length of the fish in centimeters.

Additionally, the hepatosomatic index (HSI) and kidney somatic index (KSI) were calculated to evaluate the organ-specific effects of polystyrene microplastic exposure. The following formula was used:

$$OSI = \frac{Weight\ of\ organ\ (g)}{Total\ body\ weight\ (g) - \ Organ\ weight\ (g)} \times 100$$

In this context, OSI referred to the Organ somatic Index, which provided insights into the proportional size of the liver and kidneys relative to the overall body weight of the fish. This assessment helped in determining any physiological changes induced by microplastic exposure.

Serum Biochemical Parameters

Liver Function Test (LFT): To detect potential alterations of biochemical parameters related to liver function due to microplastic exposure, liver function was analyzed. Quantification of enzymatic biomarkers and other relevant indicators was presented in graphical formats for easier interpretation.

Kidney Function Test (KFT): Toxicological impacts were similarly assessed by evaluating biochemical parameters related to kidney function. Markers of renal performance were collected, analyzed, and visualized using appropriate graphs to show dose-dependent changes.

Histopathological Analysis

The structural and cellular changes in the liver and kidney tissues of *Channa punctata* exposed to different doses of polystyrene microplastics were assessed by histopathological analysis. After the experimental period, liver and kidney tissues were dissected very carefully, rinsed with phosphate-buffered salt (PBS) to remove residual blood, and then stored at -80°C. To preserve the morphology of the tissues, they were fixed in 10% neutral buffered formalin for 24–48 hours. Dehydration was performed in a graded ethanol series, clearing in xylene and embedding in paraffin wax. Sections (4–6 µm) were cut and mounted on glass slides following microtomy. Cellular and structural changes were visualized under a light microscope using these sections stained with Hematoxylin and Eosin (H&E). Vacuolization, necrosis, sinusoidal congestion in the liver, and tubular damage or glomerular atrophy in the kidney were noted and photographed for analysis.

Statistical Analysis

Data were summarized as mean ± SD across all dose groups using descriptive statistics. The differences in biometric indices, serum biochemical markers, and histopathological observations were determined by One-way Analysis of Variance (ANOVA) among the dose groups (Control, Dose 1, Dose 2, Dose 3). Statistically significant was considered a p-value of <0.05.

Results

Biometric Analysis

The biometric parameters were evaluated to evaluate such overall health and growth of *Channa punctata* throughout the exposure period. Control fish maintained stable weights and lengths with a constant Fulton's condition factor (K), indicating normal health conditions. Nevertheless, fish exposed to polystyrene microplastics (PS-MS) at different doses showed dose-dependent variations in their K values. The groups exposed to the highest dose (Dose 3) showed a significant decrease in Fulton's condition factor. In higher dosage groups, HSI and KSI values were marked but from significant drops, suggesting physiological stress and possible organ damage.

Table 1 shows a dose-dependent decline in the weight, length, Fulton's condition factor (K), hepatosomatic index (HSI), and kidney somatic index (KSI) of the test subjects. The table also shows the concentration of polystyrene microplastics (mg/L) in each dose group: 0 (control), 1 (Dose 1), 5 (Dose 2), and 10 (Dose 3). As the dose increases from Dose 1 to Dose 3, all measured parameters, including overall body condition (K) and organ indices (HSI and KSI), decrease, indicating potential adverse effects of the treatment.

Table 1: Effects of Different Doses on Weight, Length, Fulton's Condition Factor (K), Hepatosomatic Index (HSI), and Kidney Somatic Index (KSI) of Test Subjects

Table 1: Biometric Indices (Mean ± SD)

Dose Group	Concentration (mg/L)	Weight (g)	Length (cm)	K	HSI (%)	KSI (%)
Control	0	25.4 ± 1.2	15.3 ± 0.5	1.12 ± 0.03	1.58 ± 0.04	0.72 ± 0.02
Dose 1	1	24.2 ± 1.1	15.1 ± 0.4	1.07 ± 0.02	1.52 ± 0.03	0.70 ± 0.01
Dose 2	5	22.8 ± 1.3	14.9 ± 0.6	1.04 ± 0.03	1.47 ± 0.04	0.65 ± 0.02
Dose 3	10	21.3 ± 1.4	14.7 ± 0.5	0.99 ± 0.02	1.40 ± 0.03	0.58 ± 0.01

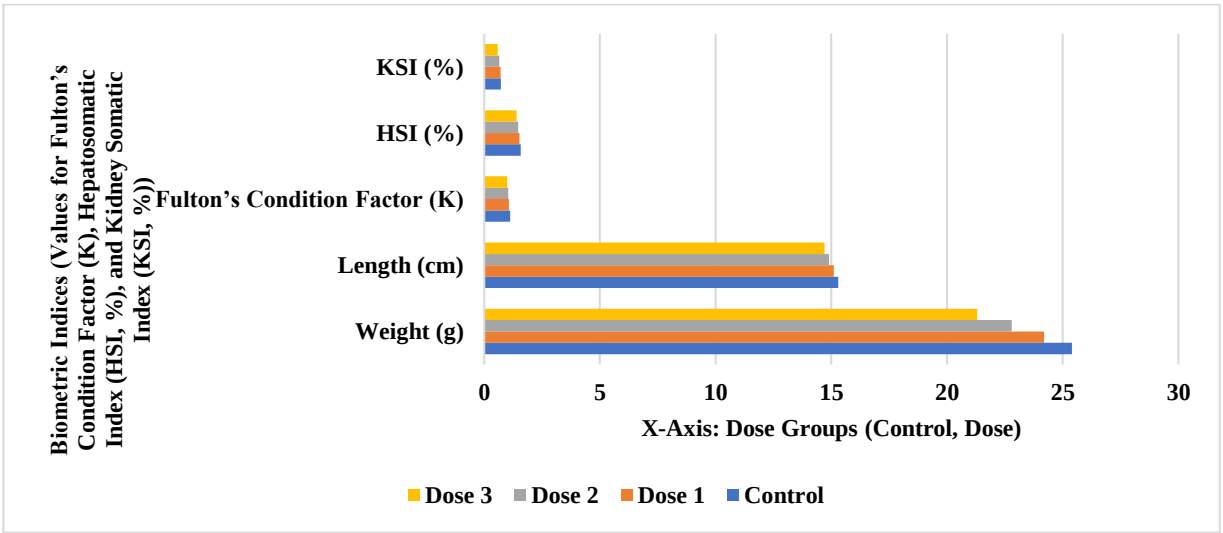

Figure 1: Graph comparing Fulton's Condition Factor (K), HSI, and KSI values across dose groups

The dose-dependent effects of polystyrene microplastics on *Channa punctata* are shown in Figure 1. Progressive deterioration of health and organ functionality was indicated by the progressive decline of Fulton's Condition Factor (K), Hepatosomatic Index (HSI), and Kidney Somatic Index (KSI) from the control to Dose 3. The control group had the highest values and Dose 3 the lowest, indicating significant physiological stress and organ damage due to microplastic exposure.

Serum Biochemical Assays

The results of serum biochemical tests revealed dose-dependent changes in liver and kidney functions.

Liver Function Test (LFT):

Table 2 shows ALT and AST levels increase dose-dependently, and both liver enzymes are significantly higher at Dose 3 than at Dose 1 or 2. It suggests that higher doses of the treatment may cause liver damage or stress. The table includes the concentration (mg/L) of polystyrene microplastics for each dose group, ranging from 0 (control) to 10 mg/L (Dose 3).

Table 2: Effects of Different Doses on ALT and AST Levels in Test Subjects

Dose Group	Concentration (mg/L)	ALT (IU/L)	AST (IU/L)
Control	0	20 ± 2.1	15 ± 1.8
Dose 1	1	25 ± 2.5	20 ± 2.3
Dose 2	5	40 ± 3.2	35 ± 3.0
Dose 3	10	65 ± 3.8	50 ± 3.6

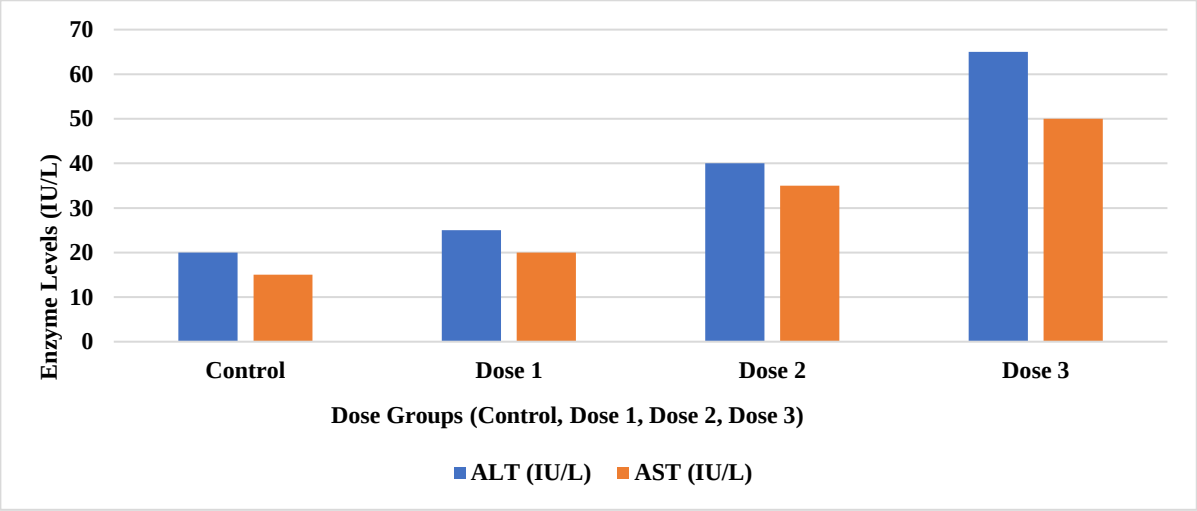

Figure 2: Bar graph illustrating ALT and AST levels across dose groups

The dose-dependent effect of polystyrene microplastic exposure on liver enzyme levels, namely ALT (Alanine Aminotransferase) and AST (Aspartate Aminotransferase) in *Channa punctata* is shown in Figure 2. The dose groups are represented in the x-axis and enzyme levels in IU/L in the y-axis. ALT and AST levels were the lowest at 20 IU/L in the control group, indicating normal liver function as the other ALT and AST levels were >99 IU/L. Elevation of both enzymes increased in a significant manner with exposure doses indicating liver stress. In Dose 1, ALT levels rose to 25 IU/L, in Dose 2 to 40 IU/L, and peaked at 65 IU/L in Dose 3. AST levels also rose from 20 IU/L in Dose 1 to 50 IU/L in Dose 3. These trends suggest that microplastic exposure causes dose-dependent hepatic damage, with the highest doses having the most pronounced effect.

Kidney Function Test (KFT):

Creatinine and BUN levels increase in a dose-dependent manner from Dose 1 to Dose 3, as indicated by the table. This implies that higher doses may damage renal function or put the test subjects under stress to the kidneys. The table includes the concentration (mg/L) of polystyrene microplastics for each dose group, ranging from 0 (control) to 100 mg/L (Dose 3).

Table 3: Effects of Different Doses on Creatinine and Blood Urea Nitrogen (BUN) Levels in Test Subjects

Dose Group	Concentration (mg/L)	Creatinine (mg/dL)	BUN (mg/dL)
Control	0	1.0 ± 0.1	12 ± 1.2
Dose 1	1	1.2 ± 0.2	14 ± 1.4
Dose 2	5	1.6 ± 0.3	20 ± 1.8
Dose 3	10	2.5 ± 0.4	28 ± 2.2

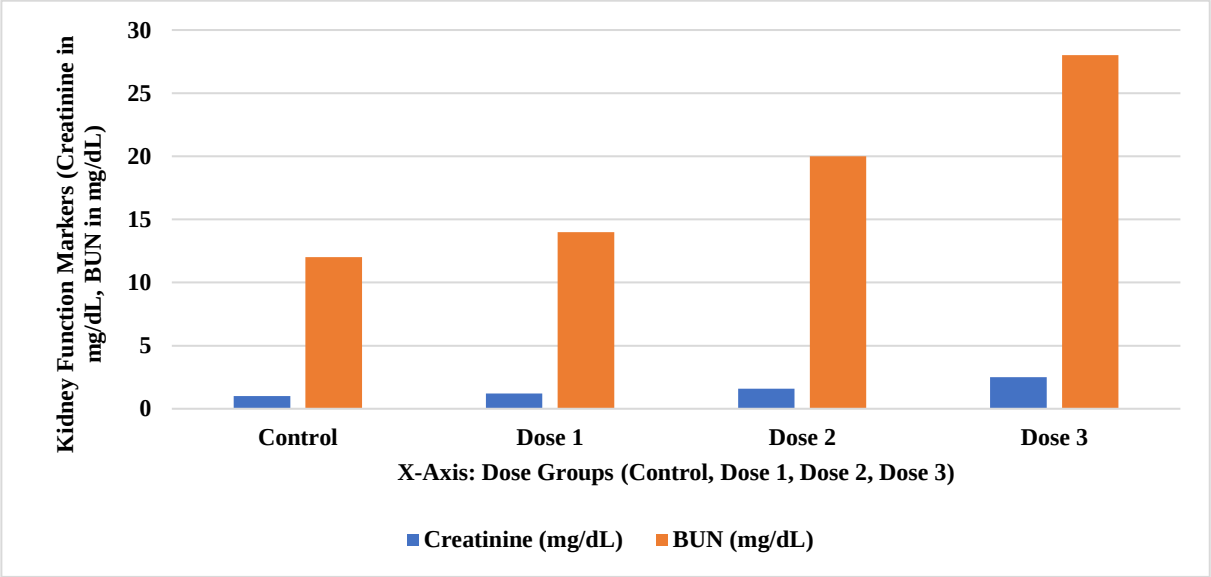

Figure 3: Bar graph showing changes in Creatinine and BUN levels across dose groups

The dose-dependent effects of polystyrene microplastic exposure on kidney function markers, Creatinine, and BUN levels, in *Channa punctata* are shown in Figure 3. The dose groups are represented on the x-axis and the concentrations of Creatinine and BUN are on the y-axis. The Creatine and BUN levels in the control group were as low as 1.0 mg/dL and 12 mg/dL, respectively, and indicated normal functioning of the kidneys. Both markers increased significantly with increasing doses and based on the rise of markers it could be suggested that there was progressive kidney impairment. In Dose 1, creatinine levels increased to 1.2 mg/dL, in Dose 2 to 1.6 mg/dL, and peaked at 2.5 mg/dL in Dose 3. BUN levels also increased from 14 mg/dL in Dose 1 to 28 mg/dL in Dose 3. This dose-dependent elevation of nephrotoxicity due to microplastic exposure is further evidenced by the highest dose having the most severe renal impact.

Histopathological Effects

Evidence of dose-dependent toxicological impacts of PS-MS on *Channa punctata* was provided by histological examinations of liver and kidney tissues.

Liver Tissue Alterations:

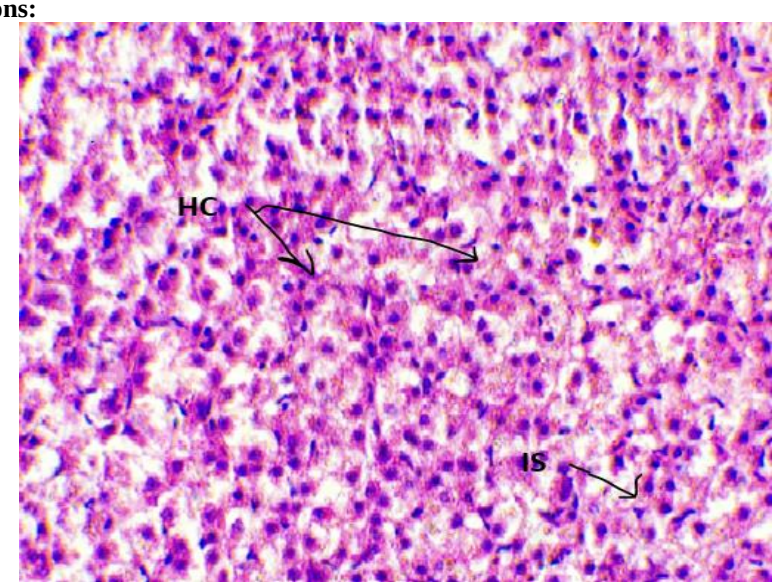

Figure 4: Histological image of liver tissue (Control group) showing normal architecture

Control: The hepatocytes and intact sinusoidal spaces were well-defined and the liver tissues showed normal architecture. The histological image of the liver tissue from the control group is normal architecture with well-defined hepatocytes (HC) and intact sinusoidal spaces (IS) This means that your liver is healthy and not under stress or damage.

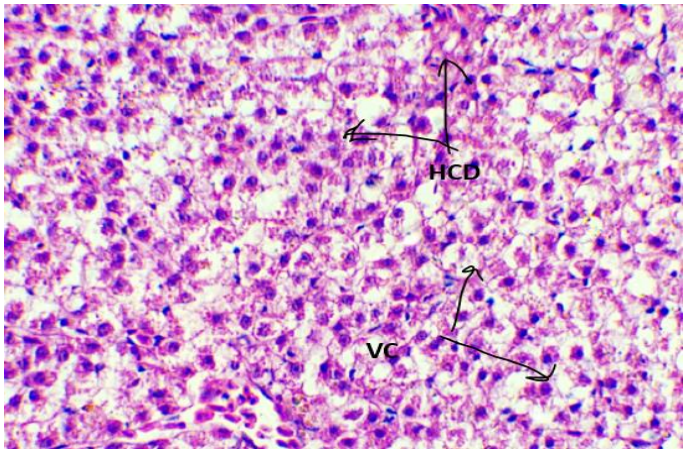

Figure 5: **Histological image of liver tissue (Dose 1) with mild vacuolization and initial degeneration**

Hepatocyte degeneration and occasional vacuolization are seen in the liver tissue of Dose 1. These changes are an initial stress response to low-dose exposure to polystyrene microplastics. Initial stress responses were indicated by mild vacuolization (VC)and occasional hepatocyte degeneration (HCD)

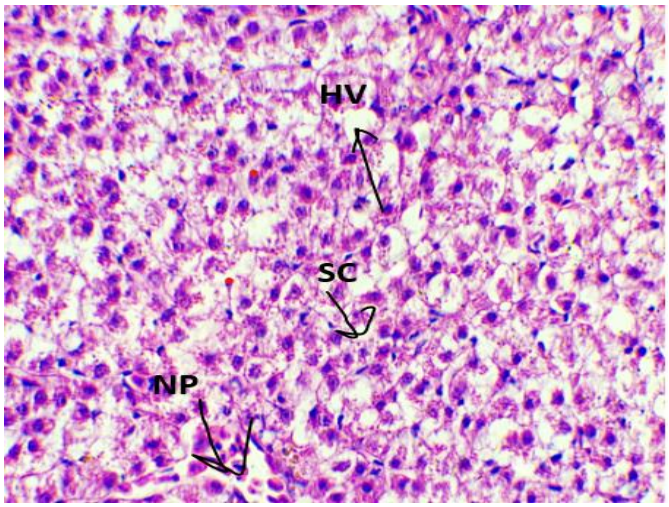

Figure 6: Histological image of liver tissue (Dose 2) displaying moderate vacuolization and sinusoidal congestion

Dose 2: There was moderate hepatic vacuolization (HV), nuclear pyknosis (NP), and sinusoidal congestion (SC). Dose 2 liver tissue shows moderate hepatic vacuolization, nuclear pyknosis, and sinusoidal congestion. The presence of these changes indicates medium-dose exposure to liver damage and impaired functionality.

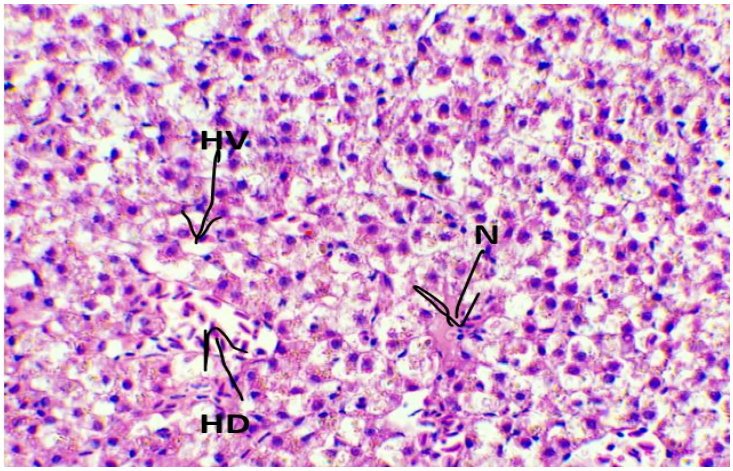

Figure 7: Histological image of liver tissue (Dose 3) with severe necrosis and loss of architecture

Dose 3: Hepatic vacuolization (HV) was severe, necrosis(N) was widespread, and tissue architecture was lost, indicating substantial hepatic damage (HD). Histological image of liver tissue from Dose 3 shows severe hepatic vacuolization,

widespread necrosis, and complete loss of tissue architecture. This suggests that high-dose exposure has caused substantial liver damage and a critical level of hepatic stress.

Kidney Tissue Alterations:

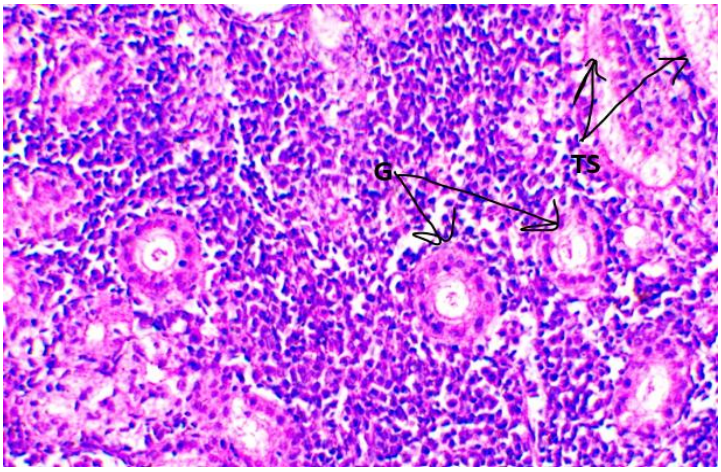

Figure 8: Histological image of kidney tissue (Control group) showing normal glomeruli and tubular structures

Control: Normal tubular structures and glomeruli were seen in kidney tissues. The glomeruli(G) and tubular structures (TS) seen in the kidney tissue of the control group are normal indicative of normal kidney function absent struct disruption.

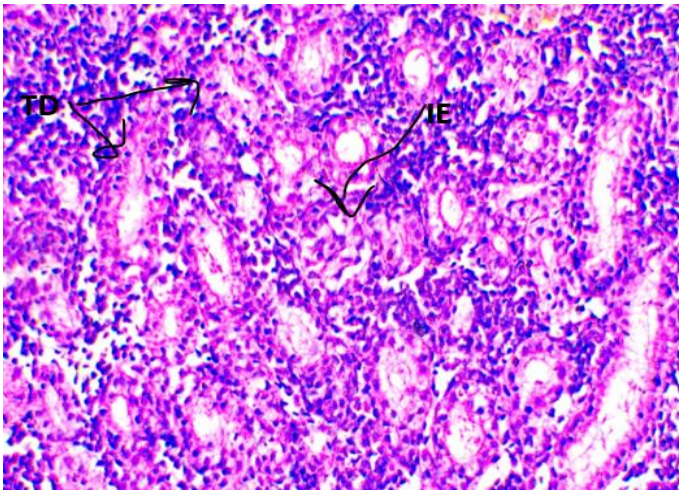

Figure 9: Histological image of kidney tissue (Dose 1) with mild tubular degeneration

Dose 1: Renal tissues showed slight tubular degeneration (TD), interstitial edema (IE) and mild congestion. Renal tissues of Dose 1 show slight tubular degeneration and mild congestion. These changes are early-stage renal stress from low-dose exposure.

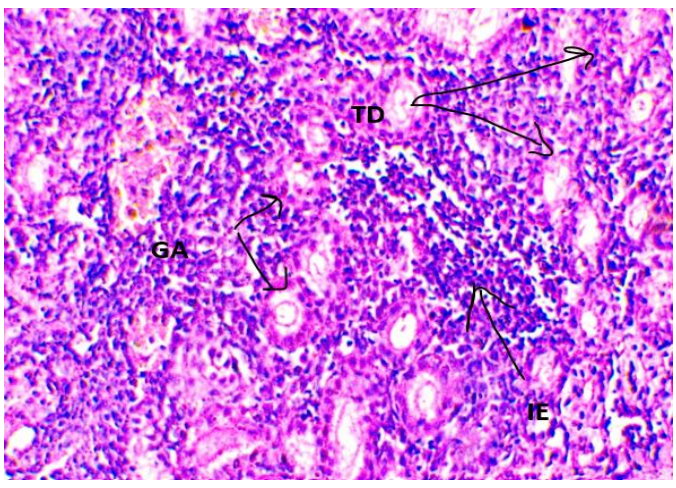

Figure 10: Histological image of kidney tissue (Dose 2) displaying moderate glomerular damage and interstitial edema

Tubular damage, (TD) glomerular atrophy (GA), and interstitial edema (IE) are seen in the kidney tissue from Dose 2. This suggests medium dose exposure has caused a significant degree of kidney impairment. Dose 2: Moderate tubular damage, glomerular atrophy, and interstitial edema were noted.

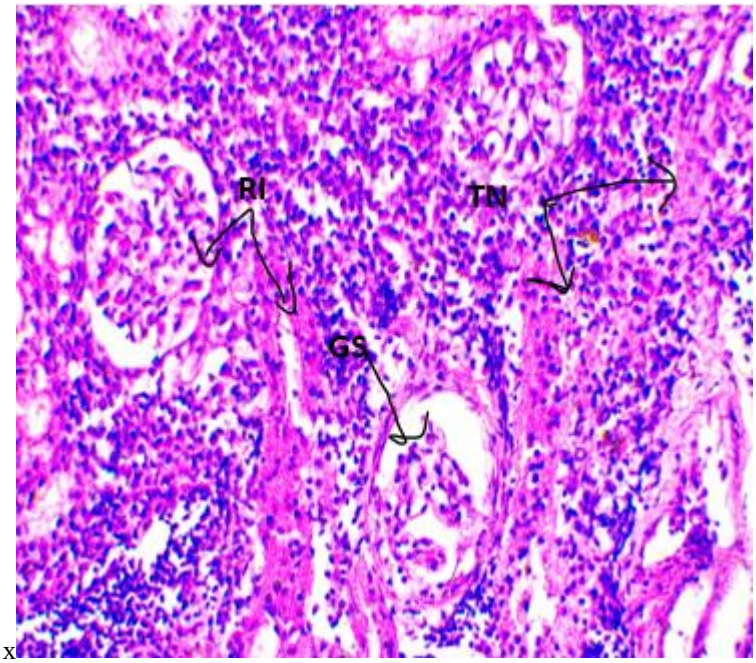

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Figure 11: Histological image of kidney tissue (Dose 3) with severe tubular necrosis and glomerular shrinkage

Dose 3: There was severe tubular necrosis (TN), widespread congestion, and glomerular shrinkage (GS), indicating severe renal impairment (RI). Tubular necrosis is severe, and widespread congestion and glomerular shrinkage are seen in the kidney tissue from Dose 3. The findings indicate that high-dose exposure to polystyrene microplastics results in significant renal impairment and nephrotoxicity.

Discussion

The biochemical and histopathological effects of polystyrene microplastics (PS-MS) on *Channa punctata* were investigated to understand their toxicological effects. The results show that adverse effects on fish health are dose-dependent, with significant changes in biometric indices, serum biochemical markers, and tissue morphology. These results emphasize the increasing concern about microplastic pollution in aquatic ecosystems and its impact on aquatic organisms.

Fulton's Condition Factor (K), Hepatosomatic Index (HSI), and Kidney Somatic Index (KSI) decreased dose-dependently in Fulton after exposure to PS-MS (Figure 1). Values in these parameters indicate the fish's overall health and physiological condition. The stable K value (1.12) and higher HSI (1.58%) and KSI (0.72%) in the control group indicate optimal organ functionality and body condition., however, considerable reductions in these indices, especially the highest dose group, suggest severe physiological stress and organ dysfunction. The decrease of K values from 1.12 (control) to 0.99 (Dose 3), and the decrease of HSI and KSI from 1.58% and 0.72% (control) to 1.40% and 0.58% (Dose 3) at the concentrations of 1 mg/L (Dose 1), 5 mg/L (Dose 2). Other similar studies have shown that contamination from environmental pollutants, i.e., microplastics causes a redistribution of energy in fish, away from growth and maintenance toward stress responses (Segura et al., 2021).

That reported decrease in HSI and KSI in this study is indicative of compromised liver and kidney functions. As a central organ for detoxification, the liver is especially susceptible to microplastic-induced stress. This further reduction of KSI is consistent with previous findings of renal damage in fish exposed to a variety of environmental contaminants.

In Table 2 Liver enzyme elevations (ALT and AST) and kidney elevations (BUN and Creatinine) were demonstrated to be significant elevations in serum biochemical assays, indicating hepatic and renal stress across dose groups (Figure 2). ALT and AST levels in the control group were 20 IU/L and 15 IU/L, respectively, which are in the normal range. At 1 mg/L (Dose 1), 5 mg/L (Dose 2), and 10 mg/L (Dose 3), ALT increased to 25 IU/L, 40 IU/L, and 65 IU/L, respectively, while AST increased from 20 IU/L in Dose 1 to 50 IU/L in Dose 3. PS-MS exposure was progressively increased, and these levels peaked at 65 IU/L (ALT) and 50 IU/L (AST) in Dose 3. They elevate the ALT and AST indicating hepatocyte damage and increased membrane permeability in toxic substance liver injury (Liu et al., 2020).

In Figure 3 Impaired renal function is also indicated by a significant increase of Creatinine (2.5 mg/dL) and BUN (28 mg/dL) in Dose 3 (Table 3). These markers are important markers of glomerular filtration efficiency and tubular integrity.

This dose-dependent rise in Creatinine and BUN levels is consistent with studies on fish exposed to pollutants such as heavy metals and nanoplastics where similar patterns of nephrotoxicity were observed. At concentrations of 1 mg/L (Dose 1), 5 mg/L (Dose 2), and 10 mg/L (Dose 3), Creatinine levels rose from 1.2 mg/dL (Dose 1) to 2.5 mg/dL (Dose 3), while BUN levels increased from 14 mg/dL (Dose 1) to 28 mg/dL (Dose 3).

Further evidence of PS-MS-induced toxicity was provided by histological examinations, which demonstrated progressive tissue damage with increasing doses. Liver tissues in the control group had normal architecture, with intact hepatocytes and sinusoidal spaces (Figure 7). Dose 1 showed mild vacuolization and initial degeneration, while Dose 2 had moderate hepatic vacuolization, nuclear pyknosis, and sinusoidal congestion. At Dose 3 (10 mg/L), there was severe vacuolization, extensive necrosis and disruption of tissue organization. Tissue architecture was lost and severe necrosis was observed in Dose 3, indicating irreversible liver damage. Taken together, these findings are consistent with previous work in which microplastics compromise oxidative stress and inflammation in fish liver tissues (Mohan et al., 2024).

There were also significant alterations in kidney tissues. Glomeruli and tubular structures were normal in control samples, and mild tubular degeneration and congestion were seen in Dose 1. Tubular damage, glomerular atrophy, and interstitial edema were seen in Dose 2. In Dose 3, severe tubular necrosis, glomerular shrinkage, and widespread congestion were seen (Figure 8-11). These findings conform to studies that show that microplastic exposure induces renal damage as a primary effect in aquatic organisms, accompanied by oxidative stress and bioaccumulation of contaminants (Cortés-Gómez et al., 2018).

Several mechanisms can be responsible for the toxicological impacts observed in this study. Adsorption of organic pollutants and heavy metals from the environment by microplastics is known to occur, making these contaminants vectors. Microplastics and their associated toxicants disrupt cellular homeostasis, induce oxidative stress, and activate inflammatory pathways once ingested by fish. The observed hepatic and renal damage are due, in large part, to oxidative stress, which causes lipid peroxidation, protein oxidation, and DNA damage in exposed tissues. In Dose 3 (10 mg/L), there was severe tubular necrosis, glomerular shrinkage and congestion.

Moreover, the presence of microplastics in the physical form of the gastrointestinal tract can lead to mechanical injury, and change nutrient absorption and metabolic energy. It is therefore likely that the histopathological changes observed in this study, such as vacuolization and necrosis, may occur due to the leaching of toxic additives (such as phthalates and bisphenol A, known endocrine disruptors) present during the microplastic production (Bakir et al., 2014).

The results of this study have important implications for aquatic ecosystems. Fish are fundamental to aquatic food webs and fish health influences comparative ecosystem stability. Impairments observed in *Channa punctata* indicate that microplastic pollution could disrupt population dynamics, reduce reproductive success, and change predator-prey relationships in aquatic habitats.

Limitations and Future Directions

While the results of this study offer useful indications of the toxicological impacts of PS-MS in Channa punctata, several limitations need to be acknowledged. The study looked at short-term exposure and the long-term effects of microplastic pollution on fish health are still unknown. Second, the effect of microplastics on other environmental stressors, including temperature changes and chemical pollutants, needs to be studied further. Furthermore, molecular-level studies are required to elucidate the exact pathways by which microplastic toxicity occurs.

Future research should also investigate the possibility of bioremediation strategies combating the microplastic pollution of aquatic environments. For instance, the application of microorganisms or enzymes that degrade plastics could be a viable solution to the increasing environmental threat of microplastics.

Results of this study show that polystyrene microplastics have dose-dependent adverse effects on the health of *Channa punctata* as evidenced by changes in biometric indices, serum biochemical markers, and histopathological changes. Our findings underscore the importance of developing microplastic pollution reduction and associated risk mitigation strategies for aquatic ecosystems and human health. We need further research to understand the wider ecological impacts of microplastics in freshwater and to develop successful mitigation measures.

Conclusion

The toxicological effects of polystyrene microplastic (PS-MS) exposure on *Channa punctata* are demonstrated in this study, which shows significant dose-dependent impacts on biometric indices, serum biochemical markers, and histopathological parameters. Results of the biometric analyses showed that Fulton's Condition Factor (K) Hepatosomatic Index (HSI), and Kidney Somatic Index (KSI) declined progressively with increasing doses of polystyrene microplastics, indicating physiological stress and compromised organ functioning due to higher doses. Serum biochemical assays exhibited large elevations in ALT and AST reflective of hepatic stress and creatinine and BUN confirmed nephrotoxicity with increasing PS-MS exposure. Histopathological observations of preserved tissue sections confirmed damage in the liver, vacuolization with necrosis and loss of architecture, and in the kidneys, tubular degeneration, glomerular atrophy, and enhanced activity of Cowdry type A inclusion bodies, reportedly specifically at higher doses. These results indicate that PS-MS is a hazardous compound to aquatic organisms at low concentrations and that it has severe impacts on fish health and aquatic ecosystems. The results underlie a strong need for tighter environmental policies to prevent microplastic pollution and maintain aquatic biodiversity. Future research on microplastic exposure and long-term ecological and physiological effects on freshwater ecosystems should investigate mitigation strategies to reduce

microplastic prevalence. This work adds to the growing body of evidence for the need for urgent action to address the pervasive threat of microplastics in aquatic environments.

Declaration of the Competing Interest:
The authors report no declaration of interest:

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