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Studies on the Effect of Cadmium Chloride on the Behavioral and Histopathological Changes in *Cyprinus carpio* : A Short-Term Bioassay

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ABSTRACT

Cadmium (Cd) is a common heavy metal known for its detrimental impact on aquatic organisms. The presence of this non-essential element in the food chain poses a significant threat to human health due to its biomagnifying effects. The present study was undertaken to investigate the effects of cadmium toxicity on the behavioral and histopathological alterations in the gill, liver and kidney tissues of the common carp species, *Cyprinus carpio*. Six groups of experimental fish with three replicates were exposed to different concentrations of cadmium chloride i.e., 0, 60, 70, 80, 90 and 100 mg/L respectively for a period of 96 h. The 96 h LC_{50} value of cadmium chloride for *C. carpio* was determined to be 74.65 mg/L. Treated fishes with higher doses i.e., 80, 90 and 100 mg/L exhibited increased breathing, accelerated ventilation with rapid opercular movement and air gulping, erratic swimming, collision against wall, loss of equilibrium, jumping, restlessness and sluggishness. Histopathological changes were also observed in gill, kidney and liver tissue. The changes in gills were marked by lamellar fusion, epithelial hyperplasia, epithelial lifting, telangiectasia, lamellar aneurism, blood congestion and necrosis of epithelial cells. The trunk kidney of the exposed fish exhibited glomerular distortion, fibrous edema, infiltration of edematous fluid, expansion of Bowman's space, hemorrhage, and damage in uriniferous tubules. The liver hepatocytes showed cytoplasmic vacuolation, pyknotic nucleus, hypertrophy of hepatocytes, erythrocyte infiltration, patchy degeneration, enlargement of sinusoids and loosening of hepatic tissues. The findings demonstrated that acute exposure to cadmium has a significant impact on the essential organs and normal behavior, potentially leading to harmful consequences for fish populations.

Keywords *Cyprinus carpio*, Cadmium, LC_{50}, Behavioral changes, Histopathology.

INTRODUCTION

Heavy metals present in the environment have long biological half-lives, posing a significant risk to aquatic organisms, with fishes being particularly susceptible to their harmful effects. At high concentrations, heavy metals exhibit lethal effects on aquatic organisms, while at sub-acute levels, they progres-

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sively accumulate within diverse aquatic organisms as they reach higher tropic levels of the food chain. Heavy metals are known to be very harmful pollutants due to their ability to undergo bio-magnification within the food chain. The water pollution caused by heavy metals on a global scale is an important environmental concern. The contamination of water sources has become more prevalent with the emergence of the agricultural and industrial revolution (Breida *et al.* 2019). Significant pollution of aquatic ecosystems is caused by industrial discharges of hazardous and toxic substances, including heavy metals (Zaynab *et al.* 2022). Freshwater ecosystems can become contaminated with heavy metals, which subsequently enter the food chain and directly affect the fish, which occupy the highest trophic level of aquatic food chain (Afshan *et al.* 2014, Authman *et al.* 2015). Thus, fish exhibit a higher susceptibility to toxic substances found in aquatic environments. However, fishes play a significant role in the human diet due to their considerable protein content, minimal levels of saturated fat, and adequate presence of omega fatty acids (Mohanty *et al.* 2019). Therefore, it is of the utmost importance to carry out research on the contamination of various species of fish by heavy metals. Cadmium (Cd) is the predominant toxic heavy metal found in aquatic environments, and its presence has become an important worldwide concern due to its potential adverse effects on public health. The determination of the LC_{50} or LD_{50} value is important as it can serve as an indicator of the extent to which a population exhibits resistance to the harmful effects of metals (Lane *et al.* 2023). Fish living in water that has been contaminated may display changes in their morphology and behavioral responses, which can be used as biomarkers that indicate the presence of contamination. The ability of an organism to adapt to a changing environment through behavioral adjustments can lead to significant morphological alterations. It has become more prevalent in recent years to use these abnormalities that have been observed in fish as biomarkers. According to Sabullah *et al.* (2015), these biomarkers have the potential to offer valuable insights regarding the current state of environmental conditions. Behavioral bioassays are more promising than lethality evaluating bioassays, which are currently used for toxicant risk assessment. Behavioral changes serve as early indicators of the health status of a population that are not accounted for by other conventional tests. Additionally, alterations in behavior can serve as indicators of lesions in important organs such as the gills, liver and kidney. Freshwater fish, such as the common carp, have been shown to primarily accumulate cadmium in their gills, liver and kidneys. The gills are regarded as the primary site for the absorption of waterborne cadmium, while the liver and kidney serve as the main organs responsible for storing and detoxifying cadmium in fish (Liu *et al.* 2022). Histopathological examinations have been widely recognized as reliable indicators of stress in fish (Ruiz-Picos *et al.* 2015).

The present study focused on cadmium induced histopathology of vital organs such as gill, liver and kidney. In addition to this, the toxic impact on the modification of the behavioral pattern was also observed.

MATERIALS AND METHODS

Collection of fish and acclimation conditions

For the current study, live and healthy *Cyprinus carpio* of almost uniform size were collected from the Naihati fish farm of the Nadia District, West Bengal along with the water samples. The specimens had a mean weight of 20.84±0.68 g and average length of 9.8 ± 0.52 cm. In order to prevent dermal infections, the specimens were bathed twice in a solution of potassium permanganate containing 0.05% for a period of two minutes. The fish were acclimatized in a semi-static system for one week before being exposed to $CdCl_2.H_2O$. Excrement and other byproducts were regularly siphoned off to minimize the ammonia levels in the water.

Experimental water

The bioassays and chemical analysis of the water were carried out in accordance with the procedures that were outlined in the guidelines provided by the American Public Health Association (APHA 2012). As a diluent medium, deep tube well water was stored in an overhead tank. The mean values for the water quality tests were as follows: Temperature 26±2.05 ◦C, pH 7.3 ± 0.28, free CO_2 6.0 ± 0.21 mg/l, DO 6.12

± 0.45 mg/l, alkalinity 190 ± 8.11 mg/l as $CaCO_3$, total hardness 440 ± 6.0 mg/l, nitrate 2.7 mg/l and ammonia 0.28 mg/l).

Experimental design and LC_{50} analysis of cadmium in *Cyprinus carpio*

Following acclimatization, the fish were subjected to a 24 hrs fasting period prior to the commencement of the experiment, and the fish were provided with a basal diet throughout the acute toxicity tests. For the preparation of stock solutions, cadmium chloride of analytical grade (purity 98%, obtained from Himedia (India) Ltd Mumbai) was utilized, which was then diluted to achieve the desired concentrations. The experiment was performed with a stocking density of 8 fish per aquarium tank and the experiment was carried out in triplicate. Fish were exposed to varying concentrations of cadmium chloride, specifically 60, 70, 80, 90 and 100 mg/L (ppm). The animal (*Cyprinus carpio*) was presumed deceased when it was observed floating immobile on its dorsal side and displaying no response when stimulated with a blunt glass rod. The mortality of the fish was documented at 24, 48, 72 and 96 h intervals following exposure to Cd and deceased fish were instantly taken out from the test media. In addition to the actual experiments, there was a parallel control group. The control group was maintained in experimental water excluding the inclusion of cadmium chloride, while all other conditions were kept constant. The duration of all experiments was 96 h. Finney's Probit analysis method has been used for statistical analysis in order to determine the 96 h LC_{50} (Finney 1971). The remaining fish and the heavy metal-treated water were disposed of carefully in compliance with institutional and national guidelines.

Behavioral studies

The control and Cd exposed fishes were closely monitored throughout the experimental period in order to analyze any changes in their behavior. The observable behaviors of *C. carpio*, such as opercular movement, erratic swimming, air gulping, collision against wall, loss of equilibrium, restlessness and sluggishness were systematically recorded using visual observation for a period of 96 h.

Tissue preparations

After completion of the exposure period, fish from each group were euthanized and the gills, liver and kidneys of both the control and treated fish were carefully removed and placed in a solution of 10% neutral-buffered formalin (NBF) for fixation for a period of 48 hrs. Following fixation, the tissues were dehydrated using a series of ethanol solutions with increasing concentration, followed by clearing with xylene. Then, the tissues were infiltrated with paraffin and ultimately embedded in paraffin.

Histology

Paraffin blocks of gills, liver and kidney of all the experimental groups sectioned into 6 µm thickness and then stretched on the sterilized glass slides. Following the removal of paraffin by using xylene, the sections were subjected to staining with Hematoxylin-Eosin and examined using light microscopy. Histopathological alterations in the tissues of ten randomly selected sections from each fish were examined.

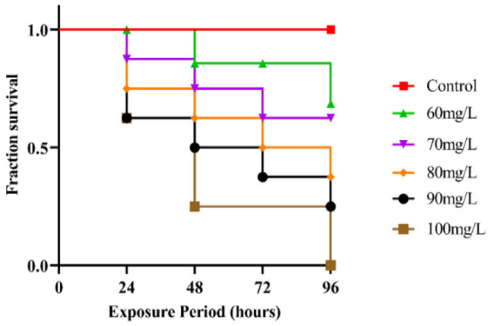
Statistical analysis

Finney's probit analysis was applied to calculate lethal concentrations (LC_{50}) over a 96 hr period with 95% confidence limits. During the exposure period, the Kaplan-Meier estimator was utilized to compute the survival rates of fish. Acute toxicity data was utilized to calculate toxicological end points, specifically the Lowest Observed Effect Concentration (LOEC) and No Observed Effect Concentration (NOEC), at a duration of 96 hrs. The MATC (Maximum Allowed Toxicant Concentration) was determined by multiplying the 96 hr LC_{50} value by the Application Factor 0.1, as described by Gheorghe *et al.* (2017).

RESULTS AND DISCUSSION

Assessment 96 h mortality and determination of LC_{50} value

The Kaplan–Meier curve illustrates that cadmium had a negative effect on the overall survival rates of *Cyprinus carpio*, with the extent of the impact being dependent on the dosage and duration of exposure



Chi Square	12.65
df	5
P value	0.0269
P value Summary	*
Are the Survival curve are Significant?	Yes

Fig. 1. Kaplan–Meier survival curves of *Cyprinus carpio* exposed to different concentrations of cadmium at different exposure periods (24, 48, 72 and 96 h).

(Fig. 1). This effect was observed to be statistically significant when compared to the control group, as determined by the Mantel-Cox log-rank test ($p < 0.05$). There were no deaths recorded in the control group throughout the course of this experiment. The 96 hr NOEC, LOEC and MATC values for *C. carpio* were determined to be 60, 70 and 7.465 mg/l, respectively (Fig. 2). A statistically significant correlation was observed between the mortality rate of *C. carpio* and exposure times (24, 48, 72 and 96 h) across all concentrations of the toxicant ($p < 0.05$).

The current study determined that the 96 hr LC_{50} of cadmium chloride for the freshwater fish, *Cyprinus carpio*, is 74.65 mg/l. The relationship between the mortality rate and the concentration of cadmium chloride has been illustrated in Table 1. Fig. 3 displays the regression line illustrating the relationship between the probit kill of *Cyprinus carpio* and log concentration of cadmium chloride. The control group fishes did not show any mortality within a 96 h period. The data obtained from these toxicity tests were assessed using probit analysis.

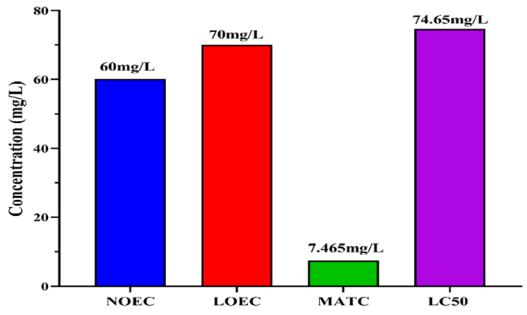


Fig. 2. Magnitude of 96 hrs NOEC, LOEC, MATC and LC_{50} value of cadmium chloride in *Cyprinus carpio*.

The common carp (*Cyprinus carpio*) showed a susceptibility to the lethal impact of cadmium chloride that was dependent on both the concentration and duration of exposure. Mortality rates increased as the concentration of cadmium chloride increased. The LC_{50} value cadmium chloride reveals the suscep-

Table 1. Mortality of *Cyprinus carpio* in different concentration of cadmium chloride at 96 h exposure period.

Concentration of $CdCl_2.H_2O$ (mg/l)	Log 10 concentration	% dead	Probit of kill
60	1.77815125	17	4.05
70	1.84509804	33	4.56
80	1.903089987	58	5.2
90	1.954242509	79	5.81
100	2	96	6.75

Table 2. Effect of acute $CdCl_2$ exposure on behavioral responses of *Cyprinus carpio*. (-) Normal, (+) Nil, (++) Less Change, (+++) Moderate Change and (++++) Prominent Change.

Behavioral changes	Exposure of $CdCl_2$ concentration (mg/L)					
	0 (Control)	60	70	80	90	100
Opercular movement	-	++	++	+++	++++	++++
Erratic swimming	-	+	++	+++	++++	++++
Air gulping	-	++	+++	+++	++++	++++
Collision against wall	-	+	+	++	+++	++++
Loss of equilibrium	-	+	++	+++	+++	++++
Jumping	-	+	+	++	+++	+++
Restlessness	-	++	++	+++	++++	++++
Sluggishness	-	+	++	+++	++++	++++

Table 3. Histopathological changes in the gill, liver and kidney of *C. carpio* exposed to cadmium chloride for 96 hrs at different concentrations. Note: = None (0%), + = mild (<10%), ++ = moderate (10-50%), +++ = severe (>50%).

Organ	Tissue changes	Concentration of cadmium chloride					
		0.0 (Control)	60 mg/L	70 mg/L	80 mg/L	90 mg/L	100 mg/L
Gill	Necrosis of epithelial cells	-	+	-	+	++	+++
	Epithelial lifting	-	+	-	-	++	++
	Hyperplasia	-	+	+	-	++	+++
	Telangiectasia	-	-	+	+	++	+++
	Fusion of secondary lamellae	-	-	+	++	+	++
	Edema	-	-	-	+	+	++
	Rupture of pillar cells	-	-	-	+	+	++
	Lamellar aneurism	-	-	-	-	+	++
	Blood congestion	-	-	-	-	++	+++
Liver	Vacuolation in hepatocytes	-	+	-	++	+	++
	Enlargement of sinusoids	-	+	-	-	-	++
	Loosening of hepatic tissues	-	-	++	+	++	+++
	Degeneration of nuclei	-	-	+	++	+	++
	Erythrocyte infiltration	-	-	+	++	+	++
	Pyknotic nucleus	-	-	-	+	++	+++
	Foamy cells	-	-	-	+	++	++
	Hypertrophy of hepatocyte	-	-	+	+	++	+++
	Patchy degeneration	-	-	-	-	+	+++
Kidney	Damage uriniferous tubule	-	+	+	-	++	+++
	Hemorrhage	-	+	-	-	-	+++
	Glomerular distortion	-	-	+	-	+	++
	Fibrous edematous	-	-	++	++	+	+
	Reduction of Bowman's space	-	-	-	++	+	+
	Vacuolation	-	-	+	++	+	++
	Expansion of Bowman's space	-	-	-	-	+	+
	Necrosis of proximal tubules	-	-	-	+	++	+++
	Atrophy of renal tubules	-	-	-	-	++	+
	Infiltration of edematous fluid	-	-	-	-	+	++

tibility of common carp to lethal concentrations of cadmium, demonstrating that toxicity is influenced by the concentration and duration of exposure. The current study determined that the 96 hr LC_{50} value of cadmium chloride for the freshwater fish species *C. carpio* was 74.65 mg/l. A study conducted by Ali *et al.* (2018) revealed that the 96 hr LC_{50} value for cadmium in *C. carpio* koi was determined to be 925 ppm. According to Mani *et al.* (2013), the LC_{50} of cadmium chloride for *Arius arius* after 96 h was determined to be 56.4 mg/L. Dhara *et al.* (2014) have reported 96 h LC_{50} as 82.66 mg/L for *Clarias batrachus*. Singh and Saxena (2020) determined that the 96 hr LC_{50} value of cadmium chloride for *Channa punctatus* was 80.62 mg/L. Chandra and Verma (2021) reported that 96 h LC_{50} value of *Channa punctata* for cadmium chloride was found to be 9.908 mg/L. The observed variations in the LC_{50} values of cadmium chloride across these studies provide evidence that the lethality of fish subjected to toxic stress is primarily influenced by the specific species and heavy metal involved, as well as the concentration of the toxicant and the duration of exposure (Verep *et al.* 2016), as observed in the current study as well. Furthermore, LC_{50} values are influenced by several other parameters, including

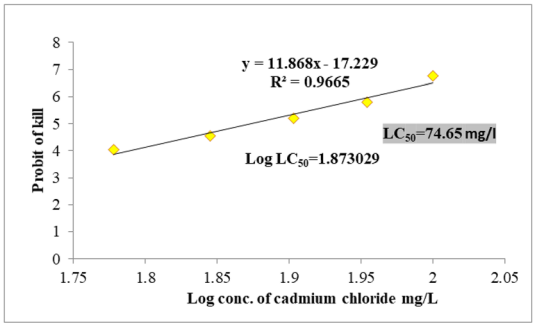


Fig. 3. Regression line between the Probit kill value of *C. carpio* at different log 10 concentrations of cadmium chloride.

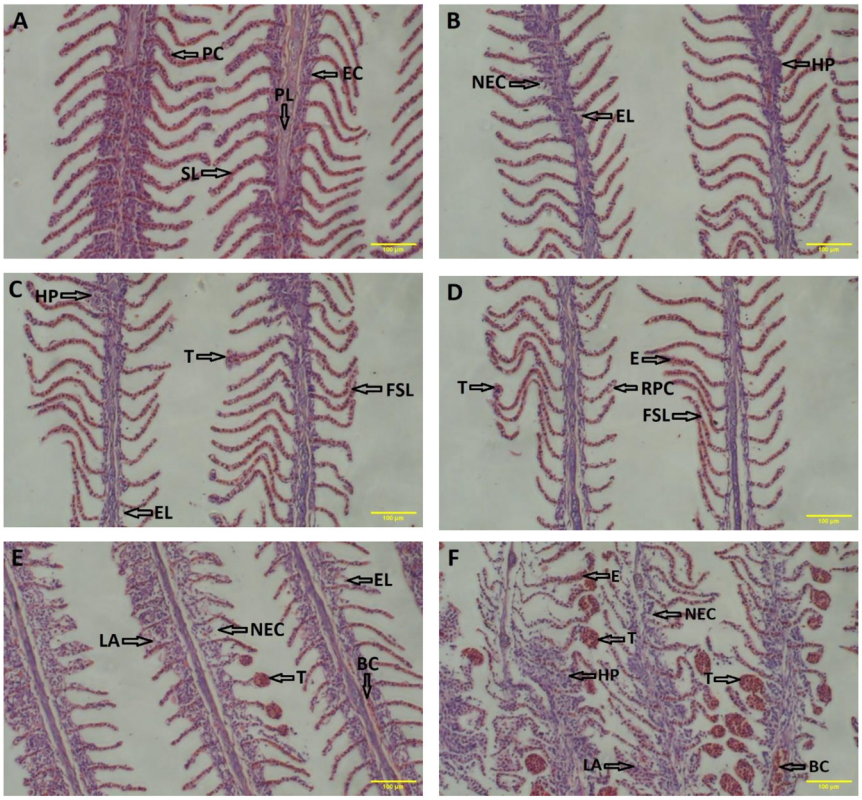


Fig. 4. Photomicrograph showing histological structures through gill of *C. carpio* exposed to different concentrations of Cd. A. Control Group, B. 60 mg/L, C. 70 mg/L, D. 80 mg/L, E. 90 mg/L F. 100 mg/L. PC = Pillar cells, EC = Epithelial cells, PL = Primary lamellae, SL = Secondary lamellae, NEC = Necrosis of epithelial cells, EL = Epithelial lifting, HP = Hyperplasia, T = Telangiectasia, FSL = Fusion of secondary lamellae, E = Edema, RPC = Rupture of pillar cells, LA = Lamellar aneurism, BC = Blood congestion (H and E 10X).

salinity, pH, and temperature of water, as well as the species, sex, age, weight of the fish (Tiwari and Prakash 2021).

Behavioral changes

During the LC_{50} assays, some selective behavioral changes were observed due cadmium toxicity. In the current study, when *Cyprinus carpio* was exposed to various concentrations of cadmium chloride, it was found that higher levels of cadmium exposure led to a higher mortality rate and induced various behavioral alterations (Table 2). The behavior and condition of the fishes were monitored throughout the entire experiment in both control and test aquaria. When fish were subjected to varying concentrations of cadmium chloride, they exhibited significant alterations as the concentrations increased, while the control group did not display any such changes. The cadmium treated fishes of the test aquaria, exhibited symptoms of impaired swimming, including erratic swimming, loss of equilibrium, restlessness, sluggishness, collisions against the walls of the test aquariums and attempts to escape the test aquariums in order to avoid the chemical. At concentrations of 60 and 70 mg/L, there were only slight behavioral changes observed, in contrast to higher concentrations. The behavior changes were most pronounced at both 90 and 100 mg/L. These observed behavioral alterations in the cadmium treated fish are consistent with the previous reports on cadmium (Kawade and Khillare 2014). The findings of the study suggested that a neurological impairment in the central nervous system might be responsible for the loss of balance that was observed

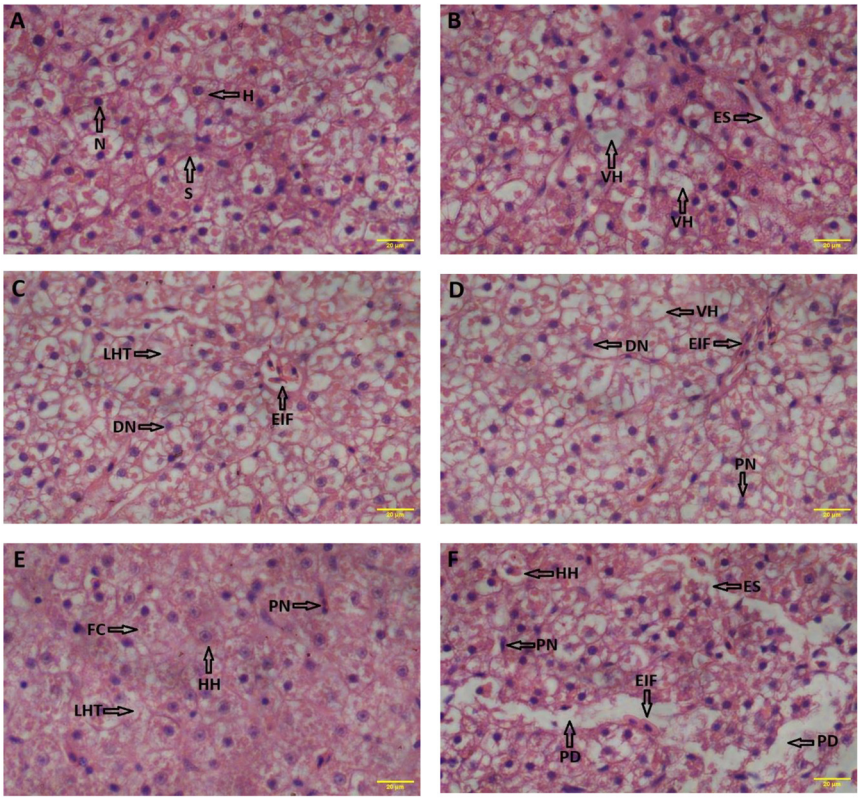


Fig. 5. Histological structure of liver of *C. carpio* showing histopathological alterations due to short-term $CdCl_2$ exposure at different concentrations after 96 hrs. A. Control Group, B. 60 mg/L, C. 70 mg/L, D. 80 mg/L, E. 90 mg/L F. 100 mg/L. H = Hepatocytes, S = Sinusoids, N = Nucleus, VH = Vacuolation in hepatocytes, ES = Enlargement of sinusoids, LHT = Loosening of hepatic tissues, DN = Degeneration of nuclei, EIF = Erythrocyte infiltration, PN = Pyknotic nucleus, FC = Foamy cells, HH = Hypertrophy of hepatocyte, PD = Patchy degeneration (H and E, 40X).

during swimming. The observed sluggishness at the completion of exposure periods could be attributed to a depletion of energy caused by erratic swimming, jumping and restlessness (Singh and Saxena 2020). The observed result suggest that the fish show a high degree of sensitivity in their behavior and any alterations in behavior is associated with toxicity.

Histopathological changes

The exposure of *C. carpio* to various concentrations of cadmium chloride resulted in notable alterations in the gill, liver and kidney tissues, as shown in Table 3. The gills of the control group exhibited a normal distribution of cellular components and an organized pattern of primary and secondary lamellae, pillar cells and blood vessels (Fig. 4 A). The treatments with cadmium (Fig. 4 B, C, D, E and F) resulted in several forms of histopathological changes such as epithelial lifting, necrosis of epithelial cells, hyperplasia of epithelial cells, lamellar aneurism and blood congestion occurs in the primary lamellae. Other observations during the experiment include telangiectasia, fusion of lamellae, rupture of pillar cells and edema occurs in the secondary lamellae. Epithelial lifting was observed in the gills of all treatment groups exposed to concentrations of 60, 70 and 90 mg/L. The rupture of pillar cells presented only in 80 mg/L. With increasing the concentration level of cadmium, the telangiectasis were also increased and maximum telangiectasis observed in gills of *C. carpio* treated with 100 mg/L. Necrosis became evident in the treatment groups receiving concentrations of 90 mg/L and 100 mg/L. The lamellar aneurysm and blood congestion

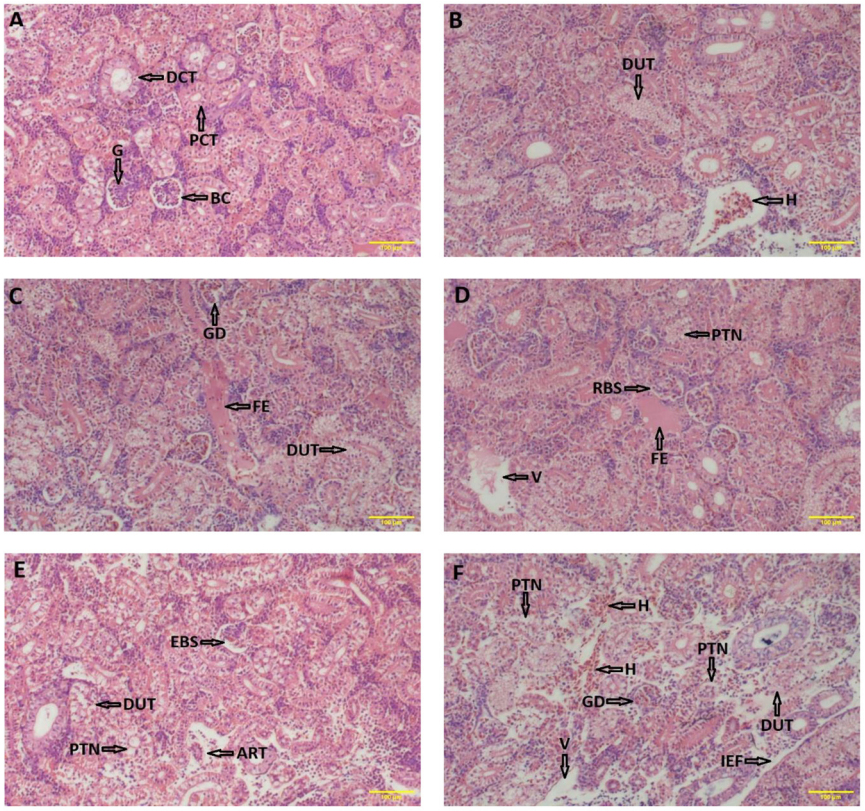


Fig. 6. Histological structure of Kidney of *C. carpio* showing histopathological alterations due to short-term $CdCl_2$ exposure at different concentrations. A. Control Group, B. 60 mg/L, C. 70 mg/L, D. 80 mg/L, E. 90 mg/L, F. 100 mg/L, G = Glomerulus, BC = Bowman's capsule, PCT = Proximal convoluted tubule, DCT = Distal convoluted tubule, DUT = Damage uriniferous tubule, H = Hemorrhage, GD = Glomerular distortion, FE = Fibrous edematous, RBS = Reduction of Bowman's space, V = Vacuolation, EBS = Expansion of Bowman's space, PTN = Necrosis of proximal tubules, ART = Atrophy of renal tubules, N = Necrosis, IEF = Infiltration of edematous fluid (H and E 10X).

observed in both 90 and 100 mg/L treatment. The histopathology of the gills is an appropriate bio-indicator for pollution monitoring (Marinović *et al.* 2021). Our findings corroborate the results reported by Mustafa *et al.* (2017) regarding the effects of lead acetate on gill tissue of *C. carpio*. Gill histopathological damage was also observed after exposure of cadmium chloride in *Anabas testudineus* (Padhy *et al.* 2018), *Cyprinus carpio* (Ghiasi *et al.* 2017), *Clarias batrachus* (Pundir 2020), and *Heteropneustes fossilis* (Jayakumar and Subburaj 2017), *Oreochromis niloticus* (Gulzar *et al.* 2023). The damage observed in the gills of fish treated with cadmium chloride may be related to oxidative damage to the cells in this tissue. The occurrence of severe edema is said to cause lifting (Hassaninezhad *et al.* 2014), as demonstrated in the current experiment. The destruction of capillary pillar cells causes telangiectasia and erythrocyte accumulation in the secondary lamella (Naz *et al.* 2023).

In the present study, the liver of control fish showed typical and well organized histo-architecture which was characterized by normal hepatocytes with granular cytoplasm and nuclei, as well as the presence of sinusoids as depicted in Fig. 5A. Vacuolation in hepatocytes and enlargement of sinusoids was presented in 60 mg/L treatment (Fig. 5B). Degeneration of nuclei and erythrocyte infiltration was observed at both 70 and 80 mg/L treatment (Fig. 5C and D). Fish exposed to 90 and 100 mg/L concentrations of cad-

mium chloride had serious hyperplasia and pyknotic nuclei of the hepatocyte cells of the liver tissue (Fig. 5 E and F). Degeneration of hepatocytes with cytoplasm vacuolation appears as foamy cells and severe patchy degeneration were also noticed in 90 and 100 mg/L treatment group. The liver is the main organ responsible for the biochemical transformation of organic foreign substances in teleosts (Topić Popović *et al.* 2023). The important abnormality noticed in the liver of *Cyprinus carpio* after the exposure of different concentration of cadmium chloride. The similar results were also made by Jayakumar and Subburaj (2017) in *Heteropneustes fossilis* exposed to cadmium chloride at concentration of 4.41 and 17.64 mg/L. Younis *et al.* (2013) has observed the erythrocyte infiltration into blood sinusoids, increased vacuolation in hepatocytes and degenerated nuclei, formation of thrombosis in central veins, expansion and obstruction in blood sinusoids and fibrosis in *Oreochromis niloticus* exposed to 1.68, 3.36 and 5.04 mg/L concentrations of cadmium. Azad *et al.* (2022) have also observed degeneration in liver tissues, necrosis and karyolysis of hepatocytes, remarkable cirrhosis and severe vacuolation in *Labeo rohita* fingerlings exposed to 1 ppm of waterborne Cd.

The kidney showed several tissue changes after 96 hrs of cadmium exposure. No histopathological lesions were observed in the kidney of the control group. The findings of this study show that cadmium chloride concentrations cause several kinds of histopathological alterations to the kidneys of exposed animals (Fig. 6 A- F). Gross changes included damage uriniferous tubule, necrosis of renal tubules, glomerular distortion, both reduction of and expansion of Bowman's space, vacuolation, cellular degeneration, atrophy of renal tubules, in addition to the fibrous edematous, hemorrhage and infiltration of edematous fluid. Damage uriniferous tubules occurs in the 60 and 70 mg/L cadmium exposure group which was more severe in the 100 mg/L group. Incidence of mild necrosis was observed in all treatments ranging from 80 to 100 mg/L, while infiltration of edematous fluid was specifically observed in the 100 mg/L treatment. Glomerulus distortion was observed at 70 mg/L treatment and expansion of Bowman's space and cellular degeneration presented in 90 mg/L. According to these findings, the incidence of tissue alterations became more severe as the concentration of cadmium chloride increased. Fish kidneys are crucial for maintaining osmotic homeostasis. The kidney is a vulnerable organ that can be harmed by toxic substances in water, as it receives blood from the gills and functions as an excretory organ, responsible for eliminating waste products resulting from metabolism and detoxification (Yancheva *et al.* 2016). Yalappa and Asiya Nuzhat (2016) reported detrimental effects in the kidney, such as glomerular expansion, severe damage in the proximal and distal convoluted tubules, renal corpuscle damage, hemorrhage, expansion of Bowman's space, erythrocyte infiltration in interstial fluid of common carp exposed to 2.8 mg/L cadmium chloride. Jayakumar and Subburaj (2017) observed histological changes like degenerated and shrunken glomerulus, melanomacrophage centers, increased peritubular space, increased periglomerular space, loss of cytoplasm, and vacuolation were observed in the kidney of *H. fossilis* exposed to 4.41 and 17.64 mg/L cadmium. Ahmed *et al.* (2014) found degenerative kidney tubules, necrosis of hematopoietic cells hemorrhage and lymphocytic infiltration in the kidney tissue of *A. testudineus* that was exposed to a concentration of 2 mg/L of $PbCl_2$ and $CdCl_2$. The occurrence of tubule degeneration and necrosis in renal tubules in this study suggests that the kidney experienced damage following exposure to cadmium.

CONCLUSION

The present study demonstrates that cadmium can induce significant histopathological changes in crucial organs of *C. carpio*. The histopathologic examination of fish organs is a highly valuable tool for assessing the toxic effects of cadmium on carp. The extent of histopathological alterations is contingent upon the dosage of cadmium to which the fish are exposed. The presence of abnormal behavior and changes in the histopathology of important organs indicate the significant harmful consequences of acute exposure to cadmium chloride in *C. carpio*. Thus, acute cadmium exposure can result in severe physiological problems, which can ultimately result in the death of fish.

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