SUMMARY OF THE FINDINGS

1. The median lethal concentration of cadmium for 24 and 96 h were found to be 35.97 mg L\(^{-1}\) and 22.38 mg L\(^{-1}\), respectively. LC50 concentration of cadmium for 24 h was taken for acute toxicity test. 1/10\(^{th}\) (as Treatment I) and 1/5\(^{th}\) (as Treatment II) of 24 h LC50 value was taken for sublethal toxicity test.

2. During acute treatment, fish exhibited significant behavioral changes like erratic swimming, jerky movements, increased opercular movements, restlessness, abnormal swimming behavior, floating upside down, loss of balance, and excessive mucous secretions.

3. In the present study, the accumulation of cadmium during acute and sublethal treatment in gill may be due to the element complexing with the mucus, which is impossible to remove completely from between the lamellae; accumulation of cadmium in liver may be attributed to the affinity or strong coordination of metallothionein protein with cadmium; accumulation of cadmium in kidney may be associated with the excretory/ionoregulatory function of the kidney in fish.

4. The inhibition of thyroxine (T4) and triiodothyronine (T3) during acute and sublethal study, may be due to cadmium might have inhibited the iodide uptake via competitive binding with the sodium /iodide symporter in thyroid follicles. This results in reduced uptake of iodide and diminished thyroid hormone production, which may lead to reduced serum concentrations of thyroxine (T4) and triiodothyronine (T3).

5. The decreased activities of GSH antioxidant enzymes observed in the present study at acute and sublethal treatment may be due to over production of ROS especially O\(_2^{-}\) by Cd or leading to accumulation of oxidative substances.

6. GST activity was inhibited at both acute and sublethal study may be due to excess production of ROS cum unable to contribute detoxification mechanism or severe oxidative stress may suppress GST activity due to the exhaustion of GSH and/or disruption of its synthesis.
7. The present result indicates that decreased activities of GPx at both acute and sublethal treatments. These changes may be due metal binding of SH groups at the active sites of enzyme molecules or impairment of GSH-Px may reflect the inability of mitochondria to maintain a normal functional state, thus favouring the development of peroxidative damage.

8. In the present study, increased LPO activity observed at acute and sublethal exposure may be due to oxidative stress and shown that Cd can compete with essential metals in protein-binding sites, triggering a release of Fe$^{2+}$ and Cu$^{2+}$ ions and causing increased generation of ROS.

9. Plasma glucose exhibits hyperglycaemic condition in both acute as well as sublethal exposure, may be due to alteration in the carbohydrate metabolism due to cadmium induced stress. Whereas, decreased glucose content observed at the end of 28$^{th}$ day of sublethal exposure (Treatment I), may be attributed to hypoglycaemia resulted from utilization of stored glycogen in order to meet the energy requirements of the fish.

10. Decreased protein level during acute and sublethal study may be due to cadmium induced dysfunction in tissues or to compensate for an increased demand for energy or due to the disruption of protein synthesis or denaturation of protein or interruption in the amino acid synthesis.

11. In the present study the significant increase in plasma bilirubin during acute and sublethal treatment may be due to hemolysis or irregularities in the uptake and conjugation of bilirubin by the liver cells or may be a cause of severe cellular damage leading to impaired cellular function or inability of the liver to eliminate the toxicant.

12. Decrease in ACP activity during acute and sublethal treatment may be due to alterations in the structure and integrity of cell organelles due to high accumulation of cadmium.
13. In the present study, the significant increase in plasma ACP activity during sublethal treatment (Treatment II) is possibly related to increase in lysosomal activity in the injured cells occurring as part of prenecrotic changes.

14. Decreased plasma ALP activities observed during acute treatment may be due to hepatic parenchymal damage or hepatocytic necrosis or alterations in protein synthesis and uncoupling of oxidative phosphorylation.

15. In the present study, the elevation in ALP activity during sublethal treatment may be due to cell necrosis in liver or related to the changes in the histological structure of the hepatic and extra-hepatic tissues.

16. During acute and sublethal treatment the gills showed thickening of the primary lamellar epithelium, secondary lamellae exhibits hyperplasia of epithelial cells, epithelial lifting, epithelial necrosis and desquamation, aneurism as well as curling of secondary lamellae and hypertrophy. These changes may be due to severe impairment in the gill structure or collapse of the pillar cell system due to cadmium toxicity. Further, the inflammatory responses may indicate the defence mechanism of the fish and cell proliferation, mucus secretion may indicate the compensatory responses of fish to cadmium toxicity. Both responses help to prevent damages caused by the direct effects of cadmium.

17. In the present study at acute and sublethal treatment, the hepatocytes began to swell, and contained lipid vacuoles, congestion, cellular oedema, necrotic hepatocytes, hyaline droplets were found in the cytoplasm may be result of disruption of metabolic pathways induced by cadmium. Further, sudden withdrawal and utilization of stored glycogen from the liver cells meet the energy demand caused by cadmium may be another possible reason for the observed changes in the liver.

18. Kidney exhibited distinct morphological changes at acute and sublethal study were hyaline droplet degeneration, in some cells of the tubular epithelium and occlusion of the lumen of some tubules, necrosis of the proximal and distal tubules, hypertrophy of nuclei and cells, rupture and disorganization of cells and
tubular degeneration may be result of fibrosis or increased permeability of the glomerular filter or cadmium induced alterations in process of reabsorption and secretion in the tubules.

19. In conclusion, cadmium has induced bioaccumulation and alterations in biological biomarkers such as changes in hormonal changes, antioxidant defenses, biochemical constituents and histopathological variations in fish, *Cirrhinus mrigala* during acute and sublethal concentrations. The bioaccumulation markers and biological biomarkers could be of vital importance and provides a means for the complex evaluation of the health status of aquatic organisms and environmental monitoring programme.


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