Universal Journal of Environmental Research and Technology
 eISSN 2249 0256
Available Online at: **www.environmentaljournal.org**
Volume 3, Issue 2: 173-180

Open Access **Research Article**

Effects of Cadmium on Digestive Organs of Teleost Fish Ophiocephalus (Channa)

R. Ananda Babu

Department of Zoology, Presidency College, Chennai, Tamil Nadu – 600 005

Corresponding author: anandbabu9351@gmail.com

Abstract:

Heavy metals and their salts constitute a very important group of environment pollutants. Since they are potent metabolic inhibitors of both terrestrial and aquatic plants and animals. The various heavy metals Zn, Cd and Cu are widely distributed and important as regard to their deleterious effects. Cadmium is a heavy metal and well known as a toxicant that could have an adverse effect on fish. The sources responsible for cadmium contamination are mainly of the anthropogenic origin, food; cigarette smokings are the most important sources of cadmium apart from water. The fish showed pathological changes in cadmium. Cadmium has induced marked pathological changes in fish gills, liver tissue of *Channa punctatus* exposed to cadmium toxicity evidenced marked pathological changes. Discrete pathological changes were noticed in the intestine of the fishes exposed to cadmium at different duration with the same concentration of 172 ppm. This paper explained about histopathological changes induced by cadmium on digestive organs of *Channa punctatus.*

Keywords: cadmium, channa, digestive organs, heavy metals, histopathalogy

1.0. Introduction:

Heavy metal pollution has created due to mining agricultural and forestral activities, waste disposal and fuel combustion. These have led to contamination of the aquatic environment. Heavy metals and their salts constitute a very important group of environment pollutants, since they are potent metabolic inhibitors of both terrestrial and aquatic plants and animals. They exert toxic effects in the organisms at tissue, cellular, sub cellular and molecular levels the impact of heavy metals, such as cd, cu, Hg and Zn, on aquatic organisms, it is important to understand the chemical and physiological processes that control their uptake, accumulation, storage and elimination. Acute metal toxicity fish is often characterized by gill damage and hypersecretion of muco ensuring mortalities are related to secondary physiological respiratory disturbance resulting in ion regulatory and acid base balance disturbance. The extent of physiological disturbance depends upon uptake and bio accumulation of metals.

The various heavy metals Zn, Cd, and Cu are widely distributed and important as regard to their deleterious effects. According to an estimate approximately 500 tonnes of cadmium enters the environment annually as a result of natural weathering and about 2000 tonnes is released annually as a result of human activities. Cadmium is a heavy metal and well known as a toxicant that could have an adverse effect on fish (Srivastava, 1982; Nriagu and Pachana, 1988). Cadmium is always found in association with zinc generally at levels around 0.5 percent of the zinc levels. The sources responsible for cadmium contamination are mainly of anthropogenic origin, food; cigarette smoking are the most important sources of cadmium apart from water. Daily dietary intake of cadmium ranges from 40 to 50 mg / day (WHO, 1987). Therefore in the present study the tissue uptake of Cd, individually by the locally available poor men's fish *Channa punctatus* has been examined. (Sultana and Rao (1998), studied the bioaccumulation pattern of Zn, Zu, Pb and Cd in grey mullet, mugil cephalus from harbour waters of visakhapatanum. Cadmium enters surface water with the discharge of industrial wastes or by leaching of soil to which sewage sludge is added. It is biologically very reactive and therefore gives rise to both acute and chronic poisoning.

The present study aimed at understanding the physiological responses of fish following exposure to heavy metal containing Cd and includes the bioaccumulation and tissue distribution of individual metal Cd in gill, liver, intestine in the fresh water fish *Channa punctatus*. In this study to observe the possible histopathological changes in

certain vital tissues like gill, liver and intestine of the teleost fish the *Channa punctatus* exposed to sub lethal concentrations of cadmium chloride for 15 days.

2.0 Materials and Methods:

2.1.Collection and maintenance of fishes:

Fresh water fish *Channa punctatus* were collected from Chambarmabakkam lake. These fishes were transported to the laboratory in oxygenated polythene bag. The healthy adult specimens of *Channa Punctatus* ranging in length 10-12 cm and weighing about 12-13g were used for the experiment. Fishes were acclimated for 2-3 weeks in large plastic tubs containing plain tap water and small aerator were used for the aeration of water.The physio-chemical characteristics of water were analyzed as per methods given in APHA et al., (1989).

2.2. Physiochemical Characteristics of water:

Temperature	25^0C to 27^0C
PH	7 - 7.2
Dissolved Content	7 -7.5 mg/ l
Hardness	210-215 mg / l
Chloride Content	35-38 mg / l
Alkalinity	67-69 mg / l

During acclimatization fishes were regularly fed with minced goat liver on alternate days and the water was renewed after every feed.

2.3. Biology of the fish:

Channa punctatus is an teleost fish belonging to the class : Telestomi, sub class – Actinoptergii, Family – Channide order – Ophiocephali formes (Fins without Spine, accessory respiratory organs are present). Channa punctatus is commonly called as (snake headed) murrels, in Tamil it is called koravai. It is medium sized murrel, body dark brown or greenish brown above and yellowish below. However colour varies according to the surrounding water. It may attain a length of 30cm in plains but on hills it is only about 12cm. It is carnivorous feeds on worms, insects, small fishes, tadpoles and frogs. Accessory respiratory organs present in the folded linings in the paired cavities on the roof of the pharynx.

2.4. Bioassay tests:

According to Cairns (1980) bioassay is merely a dose response evaluation', i.e., living organisms are used to determine their response to a series of different toxicant / chemical concentration (i.e., doses).

2.5. Determination of lethal concentration:

Bioassay or toxicity tests were carried out for the determination of LC_{50} value by following FAO procedure for short term bioassays (Reish and oshida, 1987). The duration of the test is 96 hours. All the experiment were conducted in a 20 litre plastic trough.

2.6. Preparation of stock solution:

Stock solution of cadmium was prepared by diluting 2.74 mg in 100ml of distilled water. From the stock solution different concentrations of cadmium nitrate namely 780ppm, 820ppm, 860ppm, 900ppm, 940ppm were prepared by diluting the respective milligram of the stock solution in one litre of test water.

2.7. Experiment:

Healthy fishes of uniform weight and length were maintained in each test solution for 96 hours to determine the LC_{50} values. Feeding was stopped one day prior to the experiment and also during the experimented period. The LC_{50} value was calculated by arithmetic graph method. Each data point is plotted and connected to form a graph. A horizontal line was drawn from the 50% mortality point to intersect the plot. A vertical line from the intersection was then dropped to the abscissa. The intersection point on the abscissa. Corresponds to the 96 hours LC_{50} (Reish and Oshida, 1987).

The LC_{50} value for 96 hours exposure was taken from these values $1/5^{th}$ of the concentration (172 ppm) was taken for the experiment. In each set of experiment about 10 fishes were introduced in a 20 litre plastic trough. The duration of the experiment was 24hrs, 48hrs, 96hrs, 168hrs (7 days) 240hrs (10 days), 364 hrs (15 days). Feeding was stopped during the experimental period. The control was maintained simultaneously.

2.8. Histological Techniques:

Both the control and the experimental fishes were dissected out and the gill, liver and the intestine were removed from the sets of fishes. They were fixed in FAA (Formalin, Acetic acid, alcohol). The fixed tissues were processed using alcohol dehydration and Tetrahydrofuran for clearing. They were embedded in paraffin wax (Congealing point 58-60°C) and longitudinal sections and transverse sections of serial sections of 5-8µ thickness were taken (Ashwani & Kumar, 2006).The sections of these tissues were stained in Haemotoxylin and Eosin (HE). The sections were deparaffinised through two changes of xylene each of ten minutes duration. The hydrated sections

were then stained in Delafields Haemotoxylin for five minutes and differentiated in acid alcohol by just dipping and then washed in running water for five minutes. After dehydration the sections were counter stained in Eosin by just dipping and the excess stain removed by placing in 90% alcohol, for 30 seconds and absolute alcohol for five minutes. The dehydrated sections were blotted once again and cleared in two changes of xylene with the first change in the ten minutes duration and second change in fifteen minutes duration and further in blotted and mounted in DPX (Diestrene plasticizer xylene). The tissues were examined under light microscope and then microphotographed.

3.0 Results and Discussions:

3.1. Behavioral responses of fishes exposed to various sub lethal concentration of Cadmium

The entire body responses of the fish such as frequent surfacing opercular moments were observed in the control and also the experimental fishes of *Channa punctatus*.

3.2. Effects of Cadmium on histology of gills

Cadmium has induced marked pathological changes in fish gills. When exposed to 172 ppm concentration of cadmium for 4 days showed less affected gill structure primary gill Lemellae (PGL), and curling of secondary gill lamellae (SGL).

The gills of the fish exposed to 7 days sub lethal exposure at 172 ppm concentration of Cadmium noticed necrosis damages in the secondary Gill Lamellae (SGL).

The damages of gills of the exposed to the same concentration at longer duration of 10 days shows fusion of secondary gill lamellae (FSGL) and damages in secondary gill lamellae were well marked. Besides these changes shortening of secondary gill lamellae. Damages in secondary gill lamellae was significant when exposed to cadmium for 16days duration at 172 ppm concentration. (Fig1-5)

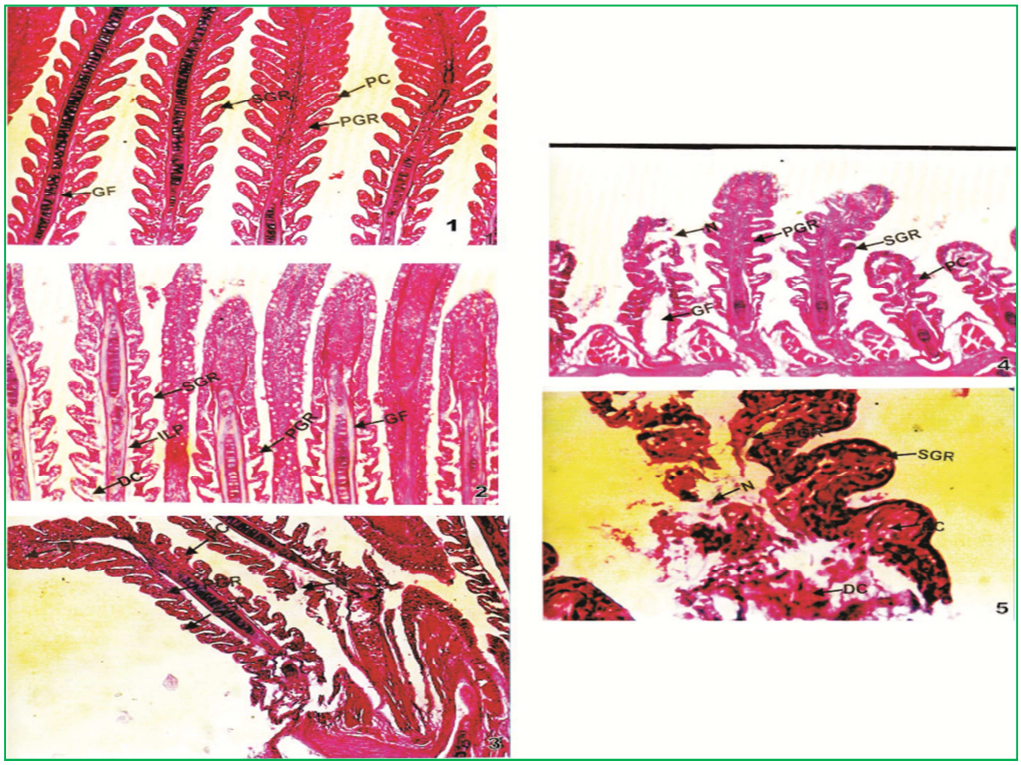


Figure: 1 Longitudinal section showing the normal organization of the gill structure of control fish *Channa punctatus* Primary Gill Lamellae (PGL), Secondary Gill Lamellae (SGL), Central Axis (CA), Pillar Cells (PC), Formalin Acetic acid Acetone (FAA), 6µ, Haematoxylin Eosin (HE). X45

Figure: 2 Longitudinal section of the gill of the fish *Channa punctatus* exposed to 24 hours sub lethal exposure of cadmium at 172 ppm concentration showing less affected structure and Dilation of Primary Gill Lamellae (DPGL). FAA, 6µ, Haenmatoxylin Eosin (HE). X45

Figure: 3 Longitudinal section of the gill of the fish *Channa punctatus* exposed to 96 hours sub lethal exposure of cadmium at 172 ppm concentration showing lifting up of epithelium, fusion of secondary gill lamellae and necrosis of the primary and secondary gill lamellae. FAA, 6µ, Haematcxylin Eosin (HE). X45

Figure: 4 Longitudinal section of the gill of the fish *Channa punctatus* exposed to 168 hours (7 days) sub lethal exposure of cadmium at 172 ppm concentration showing necrosis of epithelial cells and slight bulging of the tip of the lamellae. FAA, 6µ, Haematoxylin Eosin (HE).
Figure: 5 Longitudinal section of the gill of the fish *Channa punctatus* exposed to 240 hours (10 days) sub lethal exposure of cadmium at 172 ppm concentration showing severe necrosis, degeneration of Primary and Secondary gill lamellae and disintegration of pillar cells. FAA, 6µ, Haematoxylin Eosin (HE). X45

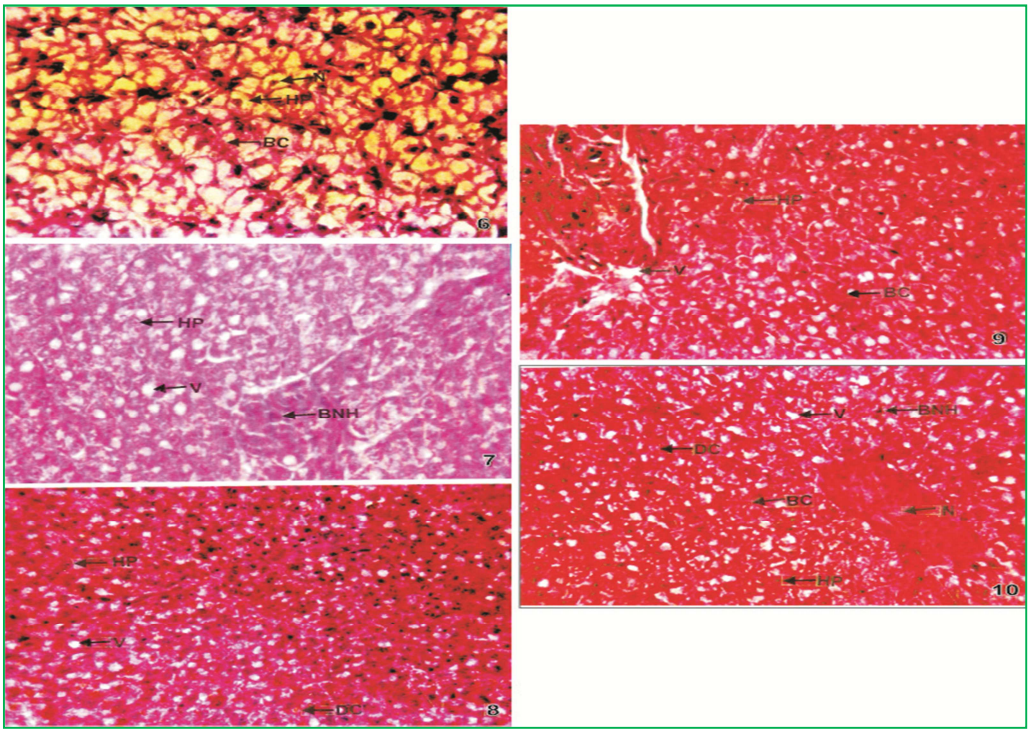


Figure: 6 Transverse section of the liver of the control fish *Channa punctatus* showing normal organization of the liver tissue with Hepatic Cell (HC), Blood Capillaries (BC) and Nucleus (N). FAA, 6µ, HAematoxylin Eosin (HE). X45
Figure: 7 Transverse section of the liver of the *Channa punctatus* exposed to 24 hours sub lethal exposure of cadmium at 172 ppm concentration showing degeneration of cytoplasm, [Binucleated Hepatic Cells (BHC) and showing partially vacuolated hepatocytes]. FAA, 6µ, Hamatoxylin Eosin (HE). X45.
Figure: 8 Transverse section of the liver of the *Channa punctatus* exposed to 96 hours sub lethal exposure of cadmium at 172 ppm concentration showing complete degeneration of cytoplasm and disintegration of hepatic cells. FAA, 6µ, Haematoxylin Eosin (HE). X45
Figure: 9 Transverse section of the liver of the *Channa punctatus* exposed to 168 hours (7 days) sub lethal exposure of cadmium at 172 ppm concentration showing rupture in blood capillaries, disappearance of hepatocytic cell wall and also more number of vacuoles. FAA, 6µ, Haematoxylin Eosin (HE). X45
Figure: 10 Transverse section of the liver of the *Channa punctatus* exposed to 240 hours (10 days) sub lethal exposure of cadmium at 172 ppm concentration showing hepatic cords were disorganized, severe necrosis, remarkable disintegrated cells and blood capillaries also affected. FAA, 6µ, Haematoxylin Eosin (HE). X45

3.3. Effect of cadmium on histology of Liver:
Liver tissue of *Channa punctatus* exposed to cadmium toxicity evidenced marked pathological changes. When exposed to 4 days sub lethal exposure of cadmium at 172 ppm concentration, shows a few hepatocytes with completely vacuolated nuclei. Fish exposed to 7 days sub lethal exposure of cadmium at 172 ppm, The liver showed swelling and pyknosis of hepatocyte nuclei. The liver showed extensive changes in hepatocytes. The liver as days prolonged showed advanced stage of hepatocyte damage deplection glycogen. The liver was severely damaged showing biliary proliferative hyperplasia peribiliary cirrhosis was manifested by fibrosis of hepatic tissue.

The liver bile ducts had thickened external and generalized swelling and pyknosis of hapatocytes nuclei. Effect on 30 days cadmium treated fish.
The fish showed pathological changes. The liver parenchyma was affected showing necrosis. Area around bile ducts showed shrinkage in hepatocytes and extensive cytoplasmic vacuolization. The hepatocytes were damaged. (Fig. 6-10).

3.4. Effect of cadmium on histology of intestine

Discrete pathological changes were noticed in the intestine of the fishes exposed to cadmium at different duration with the same concentration of 172 ppm. When the fish exposed for 4 days showed less affected structure. Necrosis in columnar epithelial cells was noticed when exposed to 7 days.

Fish exposed to 10 days shows appearance of goblet cells and slight breakage of columnar epithelial cells were seen. The fish exposed to 16 days sub lethal exposure at 172 ppm concentration shows more breakage of columnar epithelial cells. (Fig. 11-15).

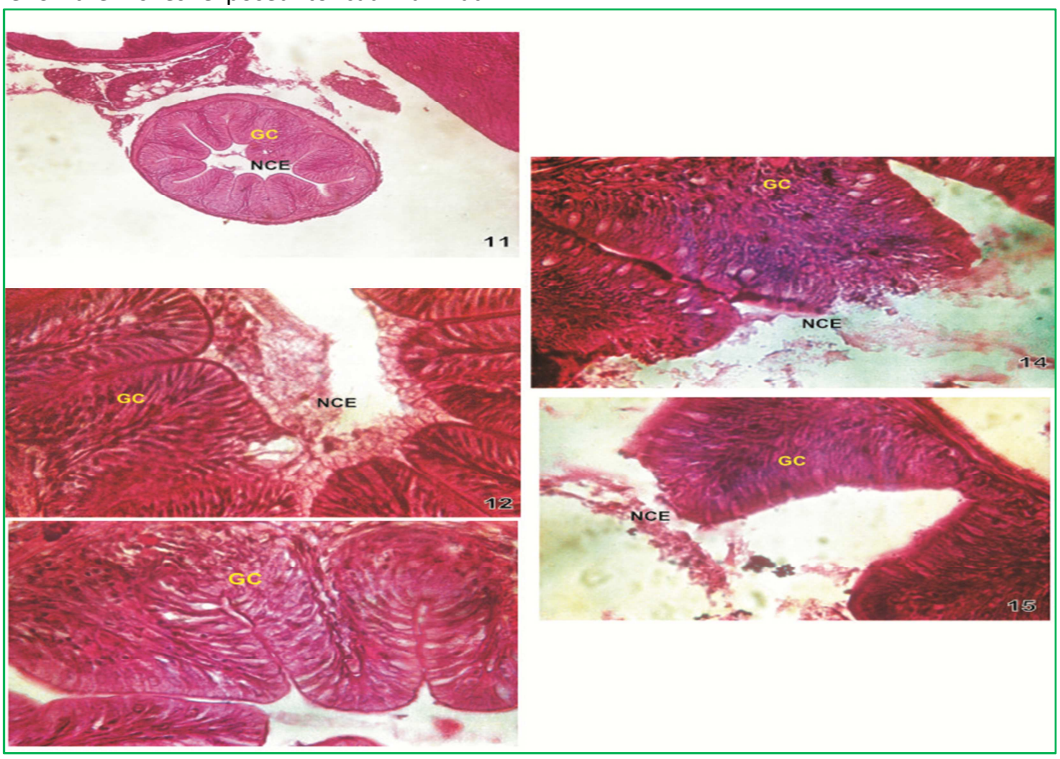


Figure: 11 Transverse section of the intestine of the control fish *Channa punctatus* showing the normal organization of intestine, Columnar Epithelium (CE), sub mucosa and serosa. FAA, 6µ, Haematoxylin Eosin (HE). X45

Figure: 12 Transverse section of the intestine of the *Channa punctatus* exposed to 24 hours sub lethal exposure of cadmium at 172 ppm concentration showing less affected in normal structure compare to control fish columnar epithelial cells with prominent nuclei (indicated in arrows) FAA, 6µ, Hamatoxylin Eosin (HE). 45

Figure: 13 Transverse section of the intestine of the *Channa punctatus* exposed to 96 hours sub lethal exposure of cadmium at 172 concentration showing rupture of muscular layer and flattening of intestinal folds slight shrinkage and appearance of few goblet cells (indicating in arrows). FAA, 6µ, Haematoxylin Eosin (HE). X45

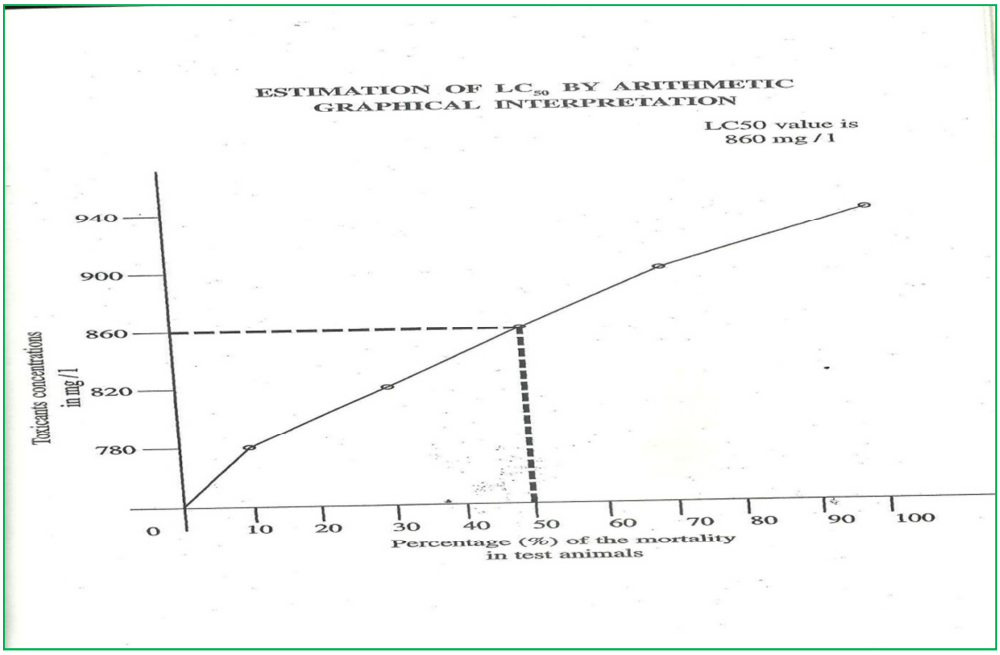
Figure: 14 Transverse section of the intestine of the *Channa punctatus* exposed to 168 hours (7 days) sub lethal exposure of cadmium at 172 ppm concentration showing shrinkage in the columnar epithelial cells with a few goblet cells. FAA, 6µ, Haematoxylin Eosin (HE). X45

Figure: 15 Transverse section of the intestine of the *Channa punctatus* exposed to 240 hours (10 days) sub lethal exposure of cadmium at 172 ppm concentration showing more goblet cells and disintegration of cell wall, necrosis of columnar epithelial cells. FAA, 6µ, Haematoxylin Eosin (HE). X45

Table 2: Percentage (%) of the Mortality in Test Animals

S. No	No. Of Fishes	Toxicant Concentration in Ppm	Mortality In Test Animals(Fish)	
			96 Hrs	%
1	10	780	1	5
2	10	820	3	25
3	10	860	5	50
4	10	900	7	75
5	10	940	10	100

3.5. LC50 Value:



In the fish Channa punctatus the 96 hrs LC50 value for Cadmium was found to be 860 ppm. The arithmetic graph for LC50 for cadmium in Channa punctatus was plotted (Graph-1, table no.2). *Channa punctatus* were exposed to 780 ppm, 820 ppm, 860 ppm, 990 ppm, and 940 ppm of Cadmium, During these exposure tests the impact toxins was observed in the from of changes in the behavioral pattern of the fish. Many workers reported the behavioral changes in terms of toxic effects of cadmium. Yilmaz *et al* (2004), Reported on the effect of cadmium (cd) metal salt on behavior of the guppy (Poecilia reticulate). The fish *Channa Punctatus* is exposed for 96 hours and the Lc 50 value for cadmium was found to be 860 ppm. Similarly the Lc50 value for Cadmium was determined for the guppy (Poecilia reticulates). It was found to be 30.4 mg/L (Yilmaz et al, 2004). The acute toxicity of freshwater species was found to vary from 0.0018 ppm to 126 ppm (Abbasic *et al*, 1998). Castano *et al* (1998) determined value to be 1mg/L when exposed for 72 hours.

The fish gill is a highly specialized organ with a number of vital functions, It offers a suitable material for studies of the effects of toxic substances because toxicants enter the fish body mainly through the gill surface several investigators have reported accumulation of cadmium in fish gills offer exposure to compounds of cadmium corresponding damage, a significant loss of gill protein, with impaired oxidative metabolism of the gill were observed in Cadmium exposed fish reflecting the potent toxicity of the metal to gills. Usha Rani (1999), reported histopathological changes in gills exposed to cadmium of *Oreschromis mossambicus*. Gills serve as sensitive bioindicator of metal pollution in aquatic medium and pathological studies in turn reveal the extent of pollution as heavy metals selectively damages a particular organ.

After the treatments of cadmium gills of *Channa punctatus* showed damages like blackening of gill filaments was noticed. It can be assumed that the blackening of gill filaments was due to toxic effect of this metal on exposure to different concentration of heavy metals. Swelling of Secondary gill lamellae was observed. Cyst formation was also noticed. Mucous secreted after exposure form a protective layer over gill filaments and skin, resulted in the reduction of gill filaments and skin, resulted in the reduction of gaseous exchange between blood and water. In the present study, cadmium has induced marked pathological changes in Fish gills, Necrosis, Curling, Fusion and Shortening of secondary gill lamella was noticed when exposed to 156ppm of Cadmium. The same effect was noted by Pandey *et al* (1970) in the gills of liza parsia exposed to lead.

Liver is the target organ attacked by any foreign molecule through portal circulation and then subjected to damage (Jayantaha Rao, 1992). Liver is an important organ of detoxification which breads down toxic substances and metabolites of administered substances. This breakdown is carried by endoplasmic reticulum of hepatocytes. Several changes were observed in the hepatic tissue including hyperplasia, fatty infiltration at certain places (Nutan Kumari et al, 1989). In the present investigation, the effect of cadmium on the histology of liver of *Channa Punctatus,* showed the damage to a few hepatocytes with completely vacuolated nuclei, necrosis in blood cells, pyknosis, more vacuoles and necrosis of hepatocytes.

The intestine of Channa changes when exposed to 156 ppm of cadmium at different periods. It showed necrosis in columnar epithelial cells, appearance of goblet cells and breakage of columnar epithelial cells. Rupturing of mucosa of the stomach, degeneration of gastric glands and reduction in the pepsinogen granules, were all disowned. The degeneration of the mucosal epithelium of the intestine and hyperactivity of the goblet cells is issued cells. In the present study the action of Cadmium, a well-known environmental pollutant, the intestine was evaluated by estimating the rate of net Cl absorption expressed as short circuit current.Toxic metals can alter the concentration of electrolytes in blood. Changes in the Levels of plasma ions in present study were due to gill damage and inhibition of enzyme activity.

4.0 Conclusions:

1. Acute metal toxicity fish is often characterized by gill damage and hypersecretion of muco ensuring mortalities are related to secondary physiological respiratory disturbance resulting in ion regulatory and acid base balance disturbance. The extent of physiological
2. The damages of gills of the exposed to the 172ppm concentration at longer duration of 10 days shows fusion of secondary gill lamellae (FSGL) and damages in secondary gill lamellae were well marked.
3. The liver showed swelling and pyknosis of hepatocyte nuclei. The liver showed extensive changes in hepatocytes
4. Discrete pathological changes were noticed in the intestine of the fishes exposed to cadmium at different duration with the same concentration of 172 ppm. When the fish exposed for 4 days showed less affected structure
5. In the present investigation, the effect of cadmium on the histology of liver of *Channa Punctatus,* showed the damage to a few hepatocytes

5.0 Acknowledgement:

The author is grateful thanks to Dr.S.Kalavathy for his guidance and encouragement.

References:

1) Abbaji, S.A., Abbasi, N., Soni, R**.,** (1998) **:** Heavy metals in the environment. Mittal Publications, New delhi-110059 (India). Cadmiun pp: 89-105.
2) Alpha, (1995): Standard methods for the examination of water and waste water 9th ed. American Public Health Association Washington.
3) Castano, A., Carbonell, G., Carballo, M., Fernandez, C., Boleas, S., Tarazona, J.V., (1998): Sublethal effects of repeated interperitoneal cadmium injection on rainbow trout (*Oncorhynchus mykiss*). *Ecotoxicol Environ Sat*. Sep, 41(1): 29-35.
4) Chandra, P. Khuda-Bukhsh, A.R., (2004): Geneotoxic effects of cadmium chloride and azadirachtin treated singly and in combination in fish. Ecotoxicol Environ Saf. 58(2):194-201.
5) Kapila Manoj and G. Raghothaman**. (**1999): Mercury, copper and cadmium induced changes in the total protein level muscle tissue of an edible estuarine fish *Boleophthalmus dessumieri.* Cuv. J. Envi. Biol. 20 (3),

6) Kapila Manoj, Ragothaman**,** (1999): Effect of sublethal concenterations of cadmium of the gills of an estuarine edible fish Boleophthalmus dussumieri(cur) Environmedia p.ll:Res.18(2):145-148.
7) Coy, MC., C.P., O' Hara, T.M., Benneth, L.W., Boyle, C.R., Lynn, B.C., (1995): Liver and Kidney concentrations of Zinc, Copper and Cadmium in channel Cat Fish (Icatalurus Punctatus): Variation due to size, Season and health status, vet. Human Toxicol. 37(1), 11-14..
8) Nariagu, O. Jerome and John. B.S. Sprangu**.** (1983): Cadmium on the aquatic environment. Vol. 9, A. Wietly Interscience Publication.
9) Nutan Kumari, Rekha Kumari, B.N., Yadav and R.P. Singh. (1989): Effect of cadmium chloride on certain tissues and mortality trend in channa punctatus; Advances in Zoology: edited by Dr. A.K. Sharma, (Bulleteins Zoological Series, No:1).
10) Pandey, A.K., George, K.C., Per Mohammad, M., (1997): Histopathological altractions in the gill and kidney of an estuarine Mullet, Liza Parsia (Hamilton-Buchanan) caused by sublethal exposure to lead (pb). Indian J. Fish, 44(2); 171-180.
11) Rao, L.M., and Manjula Sree Patnaik, R., (1997): Acute toxicity of Zn, ph, cd. In the freshwater catfish mystus vittatus (Bloch)-Indian J.Fish., 44(4): 405-408. rational approach, Ecotoxicol. Environ sat. 13: 208-215.
12) Reish, D.L., and Oshida, P.S., (1987): Manual of methods in aquatic Environmental Research, Part – 10 Short term bioassays. FAO. Tech 247: 1-62.
13) Sabiha Khan, **(** 2006): Effect of Sublethal concentration of cadmium chloride on the Liver of Guppy (Lebistes reticulates) at 40'C. Environ. Media: 25(1): 207-208.
14) Sastry, K.V., and Malik, P.V**.,** (1978) Studies on the effect of dimecron on the digestive system channa punctatus (Abstract) IV all India Congress of zoological society of India, BodhGaya.
15) Sastyarathi Chinni, Prabhakare Rao Yallapratada, **(**2000) Toxicity of copper, Cadmium, Zinc and lead to penaeus Indicus postlarvae: Effects of individual metals. J. Environ. Niol., 21(3), 255-258.
16) Sesha Srinivas, V., and Bala Parameswara Rao, M**.,** (1999): Chromium induced histological alteration in the gill of the freshwater teleust fish Labeo rohita.
17) Spehar, R.L., (1976): Cadmium and Zinc toxicity to Flag Fish Lordanella Floridae. Journal of Fisheries board of Canada. 33: 1939-1945.
18) Srivastava D.K., (1982): Comparative effects of copper Cadmium and mercury in tissue glycogen of the Catfish, Heteropneutes Fossilis. Tosicol Lett 11: 135-139.
19) Usha Rani**,** (1999): Pathological observations in gills of the freshwater teolost oreochromis mossambicus (Tilapia) with reference to Cadmium toxicity-*Indian Journal of comparative Animal Physiology*: Vol 17.1, p: 18-22

.