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ORIGINAL RESEARCH ARTICLE

Studies on the Effect of Cadmium Compound on the Biochemical Parameters of Fresh Water Fish in *Cirrhinus mrigala*

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ABSTRACT

In the present investigation the effect of cadmium sulphate on the histology of gill, liver and kidney were investigated in the edible freshwater fish *Cirrhinus mrigala*. Further its effects on glycogen and protein contents in the gills, liver and kidney were analysed. A significant decrease of glycogen content was registered in liver, gills and kidney of metal treated fishes when compared to their controls. Similarly, the protein levels were decreased in gills, liver and kidney of cadmium sulphate treated fishes when compared to their controls. The significance of biomarkers was discussed in this present study.

Key words: *Cirrhinus mrigala*, glycogen, protein, gills, liver and kidney.

1. INTRODUCTION

The heavy metal Cadmium gets bioaccumulated in freshwater biota and affects severely. Normally a good correlation exists between total cadmium concentration in water and plant tissues. A positive correlation between residues of cadmium levels in benthic organisms and sediments are found. Mclntosh *et al*. (1978)[1] found that population of *Potamogeton crispus* inhabitant near industrially contaminated lake had absorbed 1.5 kg of cadmium and that release of cadmium from dead plants could rise water concentration by a maximum of 1.0 ppm. Its importance in environmental toxicity lies with the fact that it can be toxic at very low concentration to aquatic organisms. Cadmium is extremely toxic to aquatic organisms.

The toxicity is quite variable and depends upon the type of test organism, its life stage, time of exposure and environmental parameters such as temperature, pH, dissolved oxygen, hardness, concentration of organic substance. The factors such as hardness and pH may affect amount of biologically active forms of cadmium, while others like dissolved oxygen and temperature may influence the tolerance capacity of the organisms. Silverberg (1976)[2] reported that exposure of species of green algae to $CdCl_2$ resulted in the formation of intra mitochondrial cadmium granules. Cadmium also caused swelling, vacuolization and degeneration of mitochondria. Cadmium concentrations of 0.01 - 0.1 ppm also reduce the ATP and chlorophyll in many species and decrease oxygen production. Cadmium has been reported to cause depression in oxygen consumption of *Carcinus maenas* and *Cancer irroratus* at 0.5 to 4.0 ppm and 0.12 to 1.0 ppm respectively during 48 hours of exposure [3]. In tests conducted for 30 days with cadmium concentration of 0.003 to 0.006 ppm. Thurberg *et al*. (1973)[3] found that the oxygen consumption was significantly increased. Lake *et al*. (1979)[4] investigated that cadmium may induce a number of biochemical changes at acute and subacute levels; these include changes in enzyme activities, modification of carbohydrate metabolism, hematological changes, and changes in ability of blood proteins to bind cadmium. Berglind (1986)[5] exposed *Daphnia magna* at 0.2 and 2.0 ppm cadmium for 8 days and found that the metal increased ALA-D activities at both the concentrations, but at 2.0 ppm haemoglobin content decreased.

Acute toxicity of Cd to fish is mainly caused due to gill damage, the fish has to obtain oxygen from water and results anoxia. Similarly it has also been stated that Cd inhibits the action of acetyl cholinesterase, causing death through paralysis of the respiratory muscles and depression of respiratory system. However, Cearley and Coleman (1974)[6] suggested that damage to the ion regulating mechanism in fish rather than

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respiratory impairment or damage to the nervous system, is more likely to be the cause of death. Part and Svanberg (1981)[7] studied the transfer of cadmium through per fused gills from rainbow trout and found that a ten fold increase in Cd concentration resulted in a hundred fold increase in cadmium transfer. The cadmium transfer through the gills was reduced by addition of a strong complexing agent to the solution which suggests that the transfer is related to the concentration of the free cadmium ions.

Several studies have been carried on impact of cadmium on biochemical, histological haematological and behavioural responses of fish. Kumada *et al*. (1972)[8] reported decrease in liver and kidney enzyme activities in rainbow trout following a chronic exposure. (Abbasi *et al*., 1991)[9] The toxicity of the metal to freshwater fish depends upon the species, age weight, water quality and presence of other toxicants. Gill and Pant (1985)[10] reported morphological aberrations in mature erythrocytes of *P. conchonius* exposed to 0.63 or 0.84 ppm Cd for 30 to 90 days. Muramato (1981)[11] reported the occurrence of deformed vertebrae in *Cyprinus carpio* exposed to 0.01 and 0.1 ppm cadmium. Singh and Singh (1979)[12] found that 0.0034 ppm Cadmium inhibited the oxygen consumption of *Mystus vittatus* by 50 per cent exposure for 12 hours. Cearley and Coleman (1973)[13] noted erratic movements, muscular spasms, and convulsions in bluegills after 13 weeks exposure to 0.85 ppm Cd in hard water. Ellagard *et al*. (1978)[14] showed that bluegills exposed to 0.1 and 0.25 ppm Cadmium for 14 days swam two to eight times faster than the controls. These fishes treated for the same test period with 0.5 ppm Cadmium exhibited depressed swimming activity and 30 per cent mortality.

Ingestion of large amounts of cadmium (i.e., 13 to 15 µg/g cadmium) leads to nausea, diarrhoea, abdominal pains, muscular rheumatism and weakness [15]. The acute lethal dose by ingestion has been estimated to be between 5 and 50 µg/gm of body weight. Chronic cadmium poisoning was first recognised in industrial workers and mostly involved kidney damage, obstructive lung disease and tubular proteinuria. The initial symptom of the diseases was lumber pain and muscular rheumatism in the legs and later skeletal deformation took place as a result of softening of the bones.

2. METRIALS AND METHODS

Cirrhinus mrigala were procured from Maheswari fish farm, Tharangampadi. They were transported to the laboratory and reared. The fishes and the aquarium tanks are disinfected with potassium permanganate solution [16]. The fishes are maintained in the aquarium tanks and acclimatized in the laboratory conditions for a period of 3 months. The medium was renewed alternative days. The fishes are fed with boiled egg and commercial fish feeds. The weight of the fishes ranges from 50 to 75 g. The biochemical analysis of Kidney, liver, gills and muscles were removed from the control and experimental fishes. Estimation of glycogen was carried out by the method of Carroll *et al*. (1956)[17]. Estimation of protein was carried out by the method of Lowry *et al*. (1951)[18].

3. RESULTS

3.1. Kidney

The kidney is composed of many nephrons and intestinal lymphoidal tissue. Nephron consists of malpighian capsule and urinary tuble. Each malpighian capsule consists of glomerulus and Bowman's capsule, and it is made up of epithelial cells. Renal tubules consist of proximal and distal convoluted tubules. The proximal convoluted tubule is lined by columnar epithelial cells and the distal convoluted tubule is lined by dome shaped cells, as well as basophilic cells.

The experimental fishes showed some ruptured tubular cells and nuclei show sign of shrinkage and clumping of blood cells. The shrinkage of nuclei and tubular cells was observed. The vacuole formation was severe in cell components. Accumulation of melanomacrophage resulted in the formation of brown colour pigments.

The protein content in the liver tissue of control and metal treated fish was given in the Table – 1. The protein content in the control fish was 10.20 ± 0.50 mg/g wet weight of tissue. In the fish treated with sub-lethal concentration of cadmium sulphate the protein contents were 9.40 ± 0.40, 7.30 ± 0.35, 5.20 ± 0.20 and 3.10 ± 0.10 mg/g wet weight of liver tissue for 24, 48, 72 and 96 hours experimental periods respectively.

The protein content in the kidney tissues of control fish and heavy metal treated fish was given in the (**Table 1**). In the control fish the protein content in the kidney tissue was 8.95 ± 0.31 mg/g wet weight of tissue. In the sub-lethal concentration treated fish the protein contents were 7.60 ± 0.19, 5.51 ± 0.18, 4.29 ± 0.11 and 3.09 ± 0.05 mg/g wet weight of kidney tissues for

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24, 48, 72 and 96 hours experimental periods respectively.

The protein content in the gills of control and experimental fish was given in (Table 1). The protein content in the control fish was 6.40 ± 0.17 mg/g wet weight of tissues. In the fish treated with sub-lethal concentration of cadmium sulphate the protein contents were 5.90 ± 0.10, 4.08 ± 0.08, 3.08 ± 0.05 and 2.10 ± 0.01 mg/g wet weight of tissue for 24, 48, 72 and 96 hours experimental periods respectively.

3.2. Glycogen

The glycogen content in the liver tissue of control and metal treated fish was given in the (**Table 2**). The glycogen in the control fish was 6.02 ± 0.03 mg/g wet weight of tissue. In the fish treated with sub-lethal concentration of cadmium sulphate the protein contents were 5.77 ± 0.08, 4.60 ± 0.30, 3.30 ± 0.40 and 2.90 ± 0.09 mg/g wet weight of liver tissue for 24, 48, 72 and 96 hours experimental periods respectively.

The glycogen content in the kidney tissue of control fish and heavy metal treated fish was given in the (Table 2).

In the control fish the protein content in the kidney tissue was 5.10 ± 0.13 mg/g wet weight of tissue. In the sub-lethal concentration treated fish the protein contents were 4.80 ± 0.20, 3.63 ± 0.15, 3.01 ± 0.03 and 2.47 ± 0.08 mg/g wet weight of kidney tissues for 24, 48, 72 and 96 hours experimental periods respectively.

The glycogen content in the gill of control and experimental fish was given in (Table 2). The glycogen content in the control fish was 4.20 ± 0.17 mg/g wet weight of tissue. In the fish treated with sub-lethal concentration of cadmium sulphate the glycogen contents were 3.93 ± 0.27, 3.11 ± 0.06, 2.55 ± 0.19 and 2.07 ± 0.05 mg/g wet weight of tissue for 24, 48, 72 and 96 hours experimental periods respectively.

Table 1: Effect of sub-lethal concentration of cadmium sulphate on the protein content in the selected organs of *Cirrhinus mrigala*

Organs	Exposure period (Hours)				
	Control	24	48	72	96
Liver	10.20±0.50	9.40±0.40	7.30±0.35	5.20±0.20	3.10±0.10
Kidney	8.95±0.31	7.60±0.19	5.51±0.18	4.29±0.11	3.09±0.05
Gills	6.40±0.17	5.90±0.10	4.08±0.08	3.08±0.05	2.10±0.01

Values expressed in mg/g wet weight of tissue
Values of mean ± SE of six observations

Table 2: Effect of sub-lethal concentration of cadmium sulphate on the glycogen content in the selected organs of *Cirrhinus mrigala*.

Organs	Exposure period (Hours)				
	Control	24	48	72	96
Liver	6.02±0.03	5.77±0.08	4.60±0.30	3.30±0.40	2.90±0.09
Kidney	5.10±0.13	4.80±0.20	3.63±0.15	3.01±0.03	2.47±0.08
Gills	4.20±0.17	3.93±0.27	3.11±0.06	2.55±0.19	2.07±0.05

Values expressed in mg/g wet weight of tissue
Values of mean ± SE of six observations

4. DISCUSSION

Evidences reveal that exposure of lead (poisoning) brings the changes in the biochemical profiles of the fish. The observations indicate that alteration in the normal behaviour and biochemical parameters serve as an index of the toxic effects on different tissues in fishes. The glycogen content was depleted by 22.5% and 59% in the liver of *Barbus conchonius* after 30 and 60 days respectively when it was exposed to lead.. Similar results were also reported in the fish *Cyprinus caprio* [19] and *Channa punctatus*.

The results of the present study revealed a significant depletion of glycogen content in liver of *Cirrhinus mrigala* when exposed to cadmium sulphate. Al-Ekel (1994)[20] studied the toxic effect of lead in *Cyprinus carpio* and reported that the glycogen content of liver has been decreased. Exposure of carbamate pesticide in *Channa punctatus* caused decrease of glycogen content in liver and muscle. Muscle glycogen was decreased when the fish *Nephrops norvegicus* exposed to copper and manganese. Radhakrishnaiah *et al*. (1992)[21] reported that the muscle and liver glycogen contents were decreased when *Labeo rohita* exposed to copper which may be due to the utilization of glycogen through anaerobic glycolysis to meet extra requirement under hypoxia caused by chemical stress and physiological dysfunctions.

Bash (2002)[22] reported that the glycogen content was decreased in *Clarius gariepinus* due to exposure to lead. Likewise, glycogen content was decreased when *Anabas testudineus* exposed to lead nitrate [23]. The metabolic pathways of fish can be altered by a variety of biological, chemical

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and physiological factors, which could be assessed throughout several biochemical procedures. The influence of toxicant on total protein content in the selected tissues of fish has been taken into account in evaluating response of the fish against stressors.

Proteins are the most important organic constituents of fish tissues, as they play an important role in spare energy [24]. It has been shown that the protein content in the control fish of *Cirrhinus mrigala* was maximum in the liver tissue followed by kidney and gill and blood. The highest protein content in the liver tissue might be due to greater concentration of enzymes in the liver, since the liver is the major site for protein synthesis and metabolism.

In the sub-lethal concentration of heavy metal cadmium, treated fish, the protein contents were progressively decreased in all the exposure periods. Though the liver does not come into contact with the pollutants directly, it was affected indirectly by pollutants carried through the body fluid. The liver tissue also acts as sensitive index to toxicants. The depletion of tissue protein contents in fish exposed to various toxicants were observed by many workers. Baskaran and Palanisamy (1990)[25] have observed the depletion of tissue protein in *Oreochromis mossambicus* when exposed to ammonium chloride. They suggested that the dietary protein consumed by the fish is not stored in the body tissues under toxic stress. Verma and Tonk (1983)[26] observed a decrease in protein content of *Notopterus notopterus* exposed to mercury and they suggest that the reduction in tissue protein may be attributed to the great energy demands and cellular damage of toxicated fish. Singh and Srivastava (1992)[27] have also observed tissues of *Heteropneustes fossilis* exposed to toxicant and pointed out that the depletion might be due to decreased protein synthesis and decreased DNA and RNA contents during toxic stress.

Vincent *et al*. (1995)[28] have reported that depletion of protein content in the liver, gill kidney, intestine and brain tissues of *Catla catla* exposed to chromium. They suggested that the depletion of tissue protein might be due to diversification of energy to meet the impending energy demand when fish was under toxic stress. A marked fall in protein content under stress of pollution might also be due to altered enzyme activities. In conformity with the above observation, in the present study also the protein content in the fish *Cirrhinus mrigala* exposed to sub-lethal concentration of cadmium, the reduction in protein content reached the maximum at 96 hrs. It showed the cumulative effect of the cadmium stress on fish. Further the decline in the protein content in all the tissues might be due to intensive proteolysis in the respective tissues or inhibiting protein synthesis due to respective tissues of cadmium toxicated fish.

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