

ORIGINAL RESEARCH ARTICLE

Effect of Cadmium Compound on the Histological Changes of Various Vital Organs of the Fresh Water Fish *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. Histopathological studies on gills, liver and kidney were carried out. The primary and secondary gill lamellae were affected, Pillar cells, epithelial cells and cartilagenous cells were fully damaged; adjacent gill lamellae were fused; necrosis and bulging of tip gill lamellae observed. Ruptured hepatocytes with loss of its polygonal shape, dilated and coagulated blood cells, large vacuoles in the hepatocytes with displaced nucleus observed in the liver of treated fishes. The kidney tissue of cadmium treated fishes showed ruptured tubular cells, vacuoles between the cells, malanomacrophages and brown coloured pigment.

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

1. INTRODUCTION

In India, owing to the unprecedented and exponentially growing human population, increasing pace of urbanization and rapid industrialization of the country has become inevitable^[1]. At present, even from the past, more attention has been focused on the fate of the heavy metals and their derivatives in the aquatic environment. The impact of heavy metals play important role in the environmental pollution. Therefore, a thorough understanding about the toxic effects of metals on living animals is needed nowadays. Though some metals are essential nutrients, they also serve as industrial and environmental hazards. The increasing industrialisation leads to the continual addition of pollutants to the environment. Recently, the accumulations of air-borne metals in plants and soils have received increasing attention. The pollutants from industries change the chemistry of water, thereby, damaging the biotic life. The heavy metals act either synergistically or antagonistically on the aquatic biota and in some cases may cause a decrease in biotic diversity.

Histological examinations of gills of fingerlings of *Salmo gairdineri* exposed to methyl mercury indicated necrosis of gill epithelium at 16.0 and 24.0 ppm during 105 days of exposure. Mercury

at 0.003 ppm also caused significant increase in cough frequency of fish. Dutta and Haghghi (1986)^[2] found that exposure of *Lepomis macrochirus* to 0.087 ppm as methyl mercuric chloride for 24, 48 and 72 hours resulted in mean serum cholesterol levels of 0.5, 0.08 and 0.2 mg/ml compared to a mean control value of 0.3 mg/ml. Fletcher and White (1986)^[3] exposed *Pleuronectes platessa* in 0.3 ppm mercury for 4 and 7 days and resulted in 47 and 72 per cent reduction in blood haematocrit on 4th and 7th day respectively due to erythrocytolysis.

Gill and Pant (1985)^[4] reported that exposure of 0.036, 0.060 and 0.181 ppm mercuric chloride caused various changes in erythrocytes of freshwater teleost *Barbus conchoniis* Sastry and Rao (1984)^[5] experimenting exposure of *Barbus conchoniis* for 120 days to 0.003 ppm mercury observed that mercury treated fish were hypoglycemic and hypolactaemic, the glycogen content of liver and muscle were unaltered but muscle lactic acid was decreased and the rate of intestinal glucose absorption was reduced and further reported alterations in several enzyme activities of gills, brain and kidney. Ram and Sathyanesan (1983)^[6] reported that exposure of 0.01 ppm mercuric chloride for 6 months resulted

in inhibition of growth of gonads in fish. Menezes and Qasim (1984)^[7] noted significant reduction in growth of *Tilapia mossambica* at 0.04 ppm mercury. In humans, methyl mercury is more toxic than inorganic mercury, because of its greater lipid solubility which permits the metal to cross biological membranes more easily into the brain, spinal cord and peripheral nerves and across the placenta. Inorganic mercury is concentrated in the human kidney and exerts its major effect there. Rana and Sudhir Raizada (1999)^[8] studied the histo-pathological changes induced by tannery and textile dyeing effluents in the kidney of *Labeo rohita* and observed marked alterations in renal interstitial tubules and inflammatory reaction in kidney. Dhanapakiam and Ramasamy (2001)^[9] studied the toxic effects of copper and zinc mixtures on some haematological and biochemical parameters in common carp *Cyprinus carpio* and after 30 days exposure of the copper and zinc mixture showed decreased protein in serum. Tilak *et al.* (2001)^[10] studied the histopathological changes in the gill tissues of the fish *Labeo rohita* exposed to chloropyrifos. Keeping in mind the above mentioned references, an attempt has been made in the present study to understand the effect of heavy metal, Cadmium sulphate on the histology of liver, kidney and gills and biochemical changes especially protein and glycogen in the selected tissues in the fresh water fish *Cirrhinus mrigala*.

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^[11]. 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. To study the histopathological changes, the tissue samples like gill, liver and kidney were carefully removed from both control and experimental group at 0th, 10th and 20th day. The tissues were immediately washed in 0.9% NaOH solution to remove the adherence of mucous and blood. It was kept on the blotting paper to drain the moisture. The tissue samples were processed for histopathological observation. The gill, liver, kidney, and muscle of male and female sexes of both the groups were fixed in physiological saline solution for 24 hrs,

using tetrahydrofuran as a dehydrating and clearing agent. The section of 6 μ thickness were selected to observe the changes in the gill, liver and the kidney by adding haematoxylin and Eosin counter stain^[12].

3. RESULTS

The organs used for histological studies are gills, liver and kidney. These tissues showed damages when exposed to sub-lethal concentration of cadmium sulphate.

3.1. Gill tissue

The normal fish has three sets of four holobranchs forming the sides of the pharynx. Each holobranch consists of two hemibranchs projecting from the bronchial arch, which consist of a row of primary gill lamellae which in turn exhibit the formation of secondary gill lamellae. The primary gill lamellae were supported by cartilagenous gill bar containing cartilaginous cells. The surface of the secondary gill lamellae are covered with simple squamous epithelial cells which forms an interface between the fish blood and the surrounding water. The epithelial layers of two sides are separated by pillar cells. The pillar cells are arranged in rows occupying the whole of the secondary gill lamellae. On the basement membrane, the neighbouring pillar cells enclosed the afferent and efferent capillaries (**vide plate 1**).

The experimental fishes shows that the tip of the gill lamellae was bulged and the arrangement of piller cells are disturbed. Further, the fishes showed the shrinkage of epithelial cells, and the blood capilleries of the primary gill lamellae were found to be collapsed. The pillar cells were disintegrated. Adjacent secondary gill lamellae were fused and disintegrated. Atropy of secondary gill lamellae and cell necrosis were observed (**vide plate 1c**).

3.2. Liver

The liver has a palisade arrangement around the central vein. The surface of liver was covered with serous membrane and some connective tissue extended inward into the parenchymal cells, hepatic cells and lattice fibers, which support the former. Hepatic cells are polygonal in shape containing clear spherical nucleus. They were located among sinusoids forming cord like structures known as hepatic cell cards. Bile canaliculus was centrally located in each cord. The walls separating the neighbouring cords were found to be two cells in thickness (**vide plate 1**). The experimental fishes showed the hepatic cells have lost their polygonal shape and partially

vacuolated. The pygnotic nuclei migrated towards the periphery of the cell due to vacuolation of cytoplasm. Sinusoids appeared to be disintegrated. Necroses of hepatic cells were observed.

The histopathology of liver in experimental fish (96 hours) was given in (Plate 2 (Fig 2c)). The 96 hours treatment of sub-lethal concentration the liver cells showed severe damage and marked proliferation. The liver tissue was converted into sponge mass and the cells were showed scattered nature. The pancreatic tissue was broken and large vacuoles were seen.

3.3. Kidney

The kidney is composed of many nephrons and intestinal lymphoidal tissue. Nephron consists of malpighian capsule and urinary tube. 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.

Fig 1:

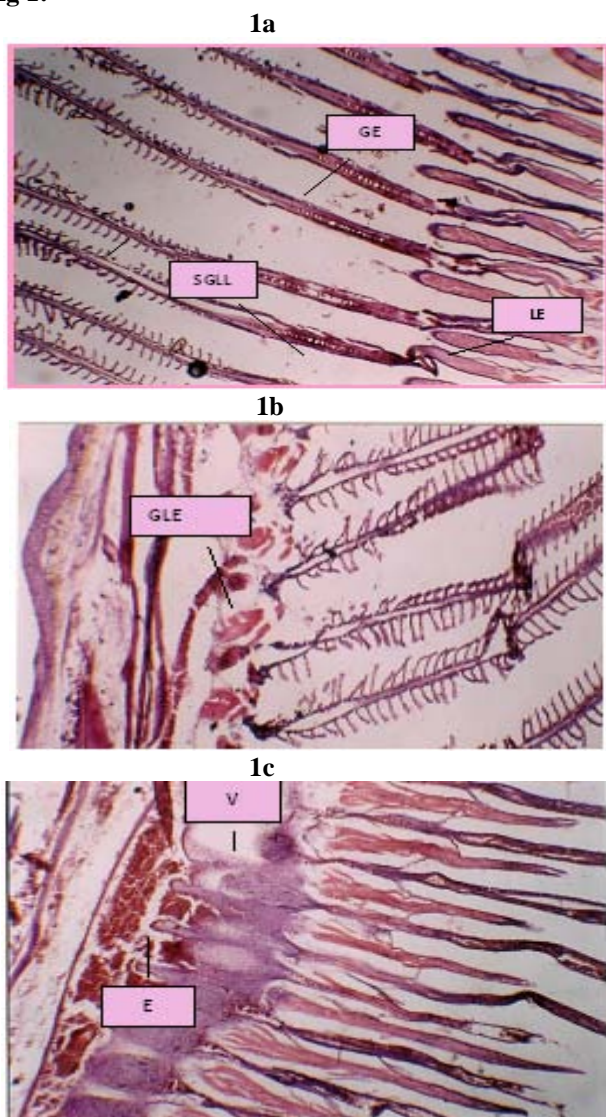
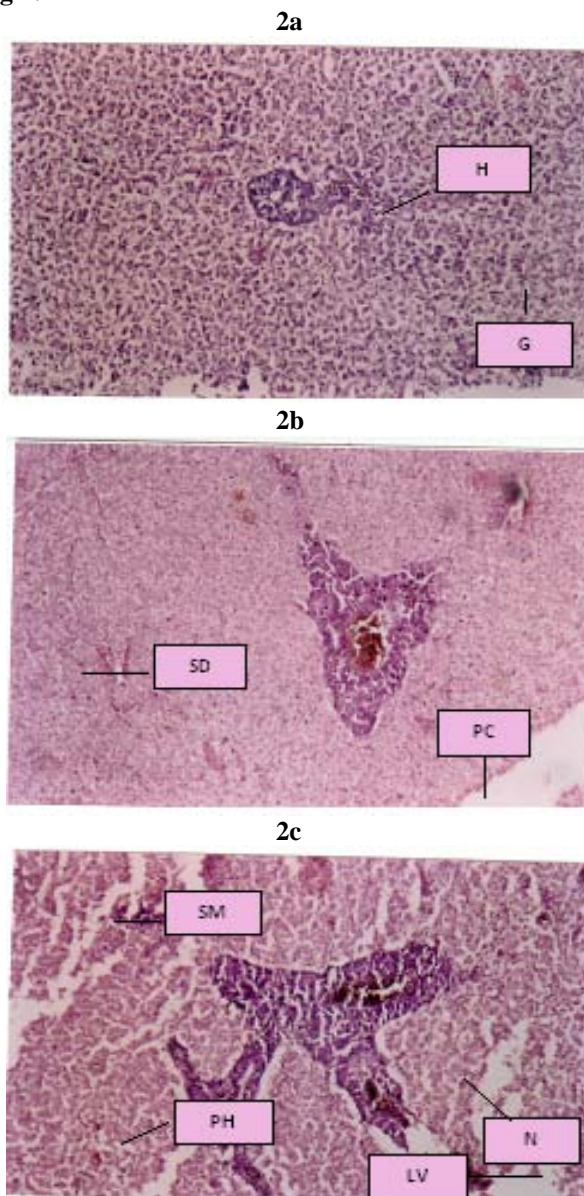


Fig 2:



4. DISCUSSION

The gills of a fish are a specialized organ having a number of vital functions. It seems a suitable material for studies on the effects of toxic substances because toxicants can enter into the body through the gill surface. Aardt *et al.* (2004)^[13] reported that exposure of lead at 96 hours in fresh water fish *Tilapia sparrmanii* reduced the ability of oxygen uptake by the gill epithelium.

When *Paralichthys olivaceus* exposed to sub-lethal concentrations of copper, lead, and cadmium has resulted in accumulation of these heavy metals in the gills, visceral mass and muscles. They further suggested that the order of heavy metal accumulation in fish, as visceral mass > gills > muscles. Usha Rani (1999)^[14] observed hypertrophy, hyperplasia, bulging of secondary gill lamellae and separation of epithelial layer when *Oreochromis mossambicus* exposed to cadmium. Singh (1993)^[15] reported fusion and

loss of secondary lamellae due to exposure of pesticides and the total respiratory surface area decreased with lowered oxygen uptake. The rate of oxygen consumption of *Clarias gariepinus* decreased when the fish exposed to heavy metals. Geoffrey Smart (1976)^[16] investigated the effect of ammonia on gill lamellae in *Salmo gairdneri*. Haemorrhage and necrosis observed in the secondary gill lamellae. When *Liza parsia* exposed to lead. In the present study, sub-lethal concentration of cadmium sulphate in *Cirrhinus mrigala* has been shown to affect the epithelial cells of the primary and secondary gill lamellae. The pillar cells of the secondary gill lamellae are disintegrated. The adjacent lamellae were fused together, and bulging of secondary gill lamellae. Atrophy and necrosis of also observed as reported by earlier workers^[17].

4.1. Liver tissue

Liver is the largest organ of the body doing several physiological functions. It has no direct contact with pollutants dissolved in water. But due to its contact with blood it is indirectly affected. The liver is susceptible to a number of toxic and metallic compounds and serves as a suitable index to toxicity of surrounding water^[18]. The present work has been carried out to study the toxic effects of cadmium sulphate in liver of *Cirrhinus mrigala* by histological methods. The hepatocytes of liver tissue have lost their polygonal shape after exposure. Vacuoles formed in the cytoplasm of cells and the nucleus becomes disintegrated. The blood cells dilated and coagulated in some places. Similar changes have been observed in the liver of *Oreochromis mossambicus* after exposure to copper sulphate. Similar changes have also been reported in *Siniperca chuatsis*, when the fish exposed to lead^[19]. Salim Sultan *et al.* (1983)^[20] studied the toxic effects of copper sulphate in the liver of *Carassius auratus* and reported the destruction of cytoplasmic material and partial vacuolation of hepatocytes and degranulation of blood sinusoids. By sub-lethal concentration of cadmium in *Heteropneustus fossilis* the liver cells showed of large vacuoles, nucleus displaced to eccentric position and blood cells dilated and coagulated at some places.

4.2. Kidney tissue

Kidney is an important organ of excretion and osmoregulation, and is highly susceptible to toxic substance because of its high blood supply. In the present study, the effect of cadmium on the histology of kidney in *Cirrhinus mrigala*, was

observed. Rupture of tubule boundary cells, formation of melanomacrophage, congregation of nuclei, damage of epithelial cells and coagulated mass of blood cells were observed. Nutan kumai *et al.* (1989)^[21] studied the effect of cadmium chloride in *Channa punctatus* on kidney tissues where shrinkage of glomerulus was observed. Dubale and Punita Shah (1981)^[22], observed that the proximal tubules of the kidney were fully damaged when *Channa punctatus* exposed to cadmium chloride. Severe necrosis, vacuoles around renal tubules and haemorrhage were observed in the kidney of fish *Cirrhinus mrigala* when it was exposed to Fenvalerate (Anita Susan and Tilak, 2003)^[23].

In fresh water cyprinoid fishes, hyperplasia of renal tubules, rupture of boundary cells of tubules, break down of outer walls of glomerulus clumping of RBC and haemorrhage in glomerulus occurred when exposed to squin. Loss of haemopoietic tissue, degeneration of glomerulus, widening of lumen and rupture of tubules with vacuolated glomerular cells observed by Mandel and Kulshrestha (1980)^[24] when *Clarias batrachus* exposed to sumithion. The same results observed when the fish *Letalurus punctatus* exposed to heavy metal (Mc-Coy *et al.*, 1995)^[25].

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