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ARTICLE

Histopathological Study of Grey mullet (*Mugil cephalus*: Linn.) in Veli Lake

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Abstract

Metal contamination has long been recognized as a serious pollution problem of aquatic ecosystems. When fish are exposed to elevated levels of water-borne metals in a polluted aquatic ecosystem, they tend to take these metals directly. Since the Veli Lake is polluted with Mercury, fishes were collected from there to study the toxicity. The partially treated sewage and the completely untreated industrial effluents are the main sources that brought undesirable changes in the lake. Mercury is one of the major components of industrial effluents. To study the effect of mercury toxicity and its impact on histology and histopathology in *Mugil cephalus*, samples of brain, kidney, liver and intestine of fish exposed to 2 sub-lethal concentrations of Mercury after 96 hour exposure period were taken and analyzed using light microscope.

Keywords: Mercury, *Mugil cephalus*, histology and histopathology

Introduction

Metal contamination has long been recognized as a serious problem of aquatic ecosystems. When fishes were exposed to a polluted aquatic ecosystem having elevated levels of metals, they tend to take these metals directly from the environment (Seymore 1994). Heavy metal contamination may have devastating effects on the ecological balance of the recipient environment and a diversity of aquatic organisms (Farombi *et al*. 2007). In the body of fish metals are transported through the blood where these ions are usually bound to proteins. Heavy metals are non-biodegradable and once they are present in an environment, bioaccumulation occurs in the fish tissue by means of metabolic and absorption processes (Wicklund-Glynn 1991). Heavy metals such as cadmium, lead, copper and especially mercury are harmful to most organisms even in very low concentrations and have been reported as hazardous to the environment. The toxicity of these elements is due to their ability to cause, oxidative damage to living tissues. Like many environmental contaminants, mercury undergoes bioaccumulation. Bioaccumulation is the process by which organisms (including humans) can take up contaminants more rapidly than their bodies can eliminate them.

When fishes are cultured in controlled condition in polluted water especially for commercial purpose, mercury enters the food chain while fish consumes plankton or from the sediment. This mercury grows in concentration within the bodies of fish and can be found in the tissues (Cheng and Zhang, 2011). Heavy metals which discharged into the aquatic environment as by product of industrial processing of ores and metals, enters into fish body and leads to severe metabolic disturbances and ultimately result in gradual elimination of the effected species (Nagabhushanam *et al*, 1981).

Materials and Methods

Live specimens of *Mugil cephalus* were collected from Veli Lake. The Veli Lake is the smallest among the back waters of Kerala. It is situated 5km from North-West of Thiruvananthapuram city.

Experimental protocol

Mugil cephalus ranging in size from 14 to 18cm and 50 to 75g were brought to the laboratory and stocked in aquaria. They were fed regularly with artificial feed. The injured and abnormal fishes were discarded. Mercury in the form of Mercuric chloride was used for the experiment. LC 50 was calculated. In this study 70 fishes were grouped in to 7 of ten each and kept in 100L trough and were exposed to varied concentrations of mercuric chloride (0.5, 1, 2, 3, 4 and 5 ppm) for 96hrs. One group was kept as control.The highest concentration at which maximum mortality occurred was noticed. After 96hr samples of brain, kidney, liver and intestine were excised and prepared for histological studies. The slides were prepared by the procedure described by Lamberg and Rothstein (1978).

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Results

Histological observations of the brain showed neuron degeneration. Vacuolization in brain tissue was also noticed. This may be the result of glycolysis leading to microsomal and mitochondrial dysfunctions.

Several histological alterations were noticed in the liver of M.cephalus. The hepatocytes were extremely swollen and their cytoplasm showed a degree of granular degeneration with numerous clear vacuoles. The liver sections showed severe degeneration or necrotic hepatic cells with fibrosis and edema. The hepatic parenchymal cells were highly vacuolated. Dilated vein with severe congestion was also noticed. The blood vessels congested with haemolysis. Lose of surrounding parenchymal cells and increase of kupffer cells were also observed.

Most common alterations found in the kidney of fishes exposed mercury were tubule degeneration (cloudy swelling and hyaline droplets) and changes in the corpuscle, such as dilation of capillaries in the glomerulus and reduction of Bowman´s space.

In the intestine, the changes occurred were vacuolar degeneration in the serosa, severe atrophy in the muscularis. Severe degeneration and necrotic changes in different intestinal layers (serosa, muscularis, submucosa, and mucosa) with aggregations of inflammatory cells. Necrotized cells aggregated in the intestinal lumen. In addition to this, hemorrhage was observed in submucosa. Separation of the serosa and increase goblet cell population was noticed. The

Discussion

The effect of different chemicals and heavy metals on aquatic ecosystems has been a subject of great interest and were studied in detail by various workers. Most of the heavy metals such as Zinc, Mercury, Arsenic and Lead cause serious effects on aquatic animals (Jernlor and Lann 1971). Brain damage in experimental fish may be the result of catabolism of glucose leading to microsomal and mitochondrial dysfunctions. Loganathan *et al.* (2006) reported that when exposed to 10ppm zinc severe necrosis of neuronal cells in the cerebrum occurred. In the present study neuron showed severe necrosis on mercury exposure. Meyer (1958) and Pentschow (1958) observed that, lead acts directly on the cerebral vasculatures including blood-brain barrier and causes cerebral edema.

Bioaccumulation of sub lethal concentrations of metals like mercury and their subsequent tissue damage led to impaired physiology and behavior of the stressed organism. The presence of tubule degeneration, coupled with the necrosis in the kidney indicates that the kidney suffered damage after exposure. In the present study, kidney of the fish often showed cloudy swelling in tubule cells. This alteration can be identified by the hypertrophy of the cells and the presence of small granules in the cytoplasm. This is in agreement with study of Takashima & Hibiya (1995).

Thophon *et al.* (2003) reported that exposure to metals frequently causes alterations in the tubules and glomerulus in the perch (Lates calcarifer) when exposed to cadmium. Handy & Penrice (1993) found swollen Bowman´s capsule cells and melanomacrophages in the kidney of trout (Salmotrutta) and tilapia (Oreochromis mossambicus) when exposed to mercuric chloride. Similar alterations were found in fishes exposed to organic contaminants (Veiga *et al.*, 2002) and mixed environmental contaminants (Schwaiger *et al.*, 1997; Pacheco & Santos, 2002).This initial stage in the degeneration process can progress to hyaline degeneration, characterized by the presence of large eosinophilic granules inside the cells. These granules may be formed inside the cells or by the reabsorption of plasma proteins lost in the urine, indicating damage in the corpuscle (Hinton & Laurén, 1990; Takashima & Hibiya, 1995). Several histological alterations were observed in the intestine of the *Mugil cephalus*. These alterations include severe degeneration and necrotic changes in the different intestinal layers with necrotized cells aggregated in the intestinal lumen, atrophy in the muscularis, edema between the intestinal layers, and haemorrhage in the submucosa.

According to Desai *et al.*, (1984) and Mohammed (2004), the degeneration and necrotic changes observed in the different intestinal layers may be due to a direct effect of the detected pollutants on the cells, to an accumulation of acetylcholine in the tissues or to a reduction in oxygen supply. The present results are in agreement with those observed by many investigators about the effects of different heavy metals on fish intestine (El-Elaimy *et al.*, 1990; Sakr, 1993; Braunbeck and Appelbaum, 1999; Mohammed, 2006 and Dezfuli *et al.*, 2006).

Moreover, Cengiz *et al.*, (2001) observed edema, degeneration, accumulation of lymphocytes and disintegration of villi in the intestine of M.cephalus subjected to pollutants. The present study shows that the histological changes in the liver cause metabolic problems as well. Evidence for this is the bile stagnation in liver of most of the fish studied. This lesion, characterized by the remains of the bile in the form of brownish-yellow granules in the cytoplasm of the hepatocytes (Pacheco & Santos, 2002), indicates that the bile is not being released from the liver. This accumulation of bile indicates possible damage to the hepatic metabolism (Fanta *et al.*, 2003).

Conclusion

Histopathology is an important tool in the field of diagnosis, because organs and tissues can respond to chemicals easily. It is easy to understand whether the tissue is contaminated with pollutants or not by comparing with the normal histology. Since fish are relatively sensitive to changes in their surroundings and environment, their health may therefore reflect and give a good indication of the health status of a specific aquatic ecosystem. The present study focuses on the toxic effects of mercury and how it affects the vital organs.

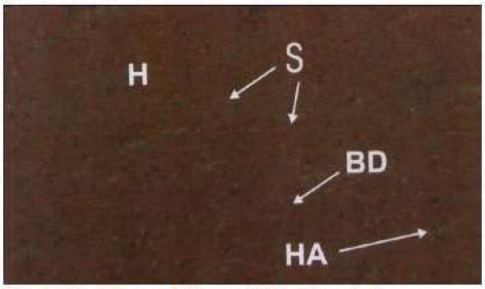

Fig. 1 Histology of liver of *Mugil cephalus* – Control (15 X) (15X)HA-hepatic artery CV-central vein S-sinusoid H-hepatocyte BD-bile duct

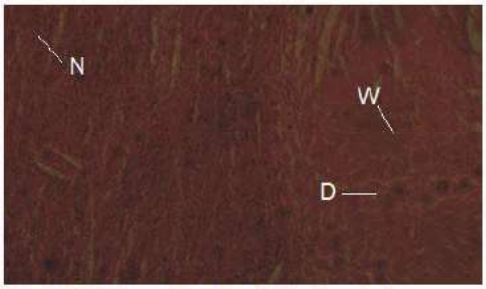

Fig. 5 Histology of brain of *Mugil cephalus* – Control 15 X) N-neuroglia W –whitematter D-Dendrite

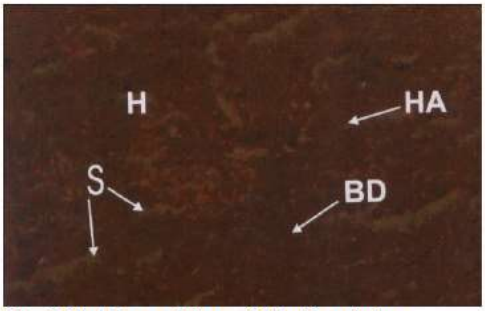

Fig. 2 Histology of liver of *Mugil cephalus* – Experimental (15 X) HA-hepatic artery CV-central vein S-sinusoid H-hepatocyte BD-bile duct

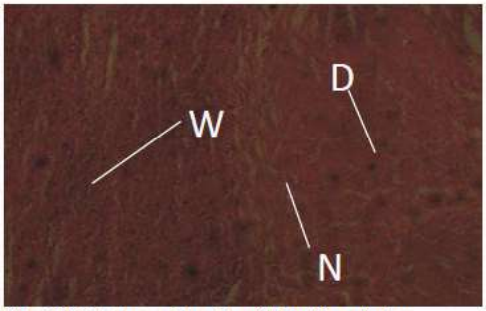

Fig. 6 Histology of brain of *Mugil cephalus* – Experimental (15 X) N-neuroglia W – whitematter D-Dendrite

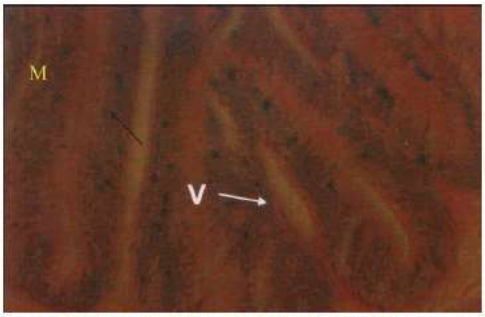

Fig. 4 Histology of intestine of *Mugil cephalus* – Experimental (15 X) MV –microvilli V-villi

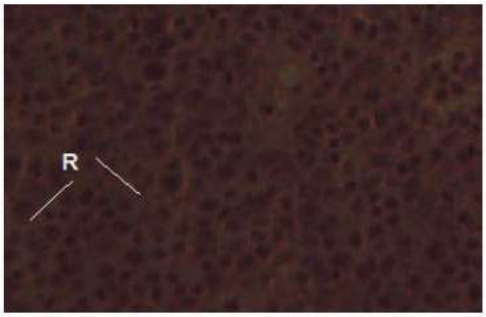

Fig.7 Histology of kidney of *Mugil cephalus* – Experimental (15 X) R-renal tubules

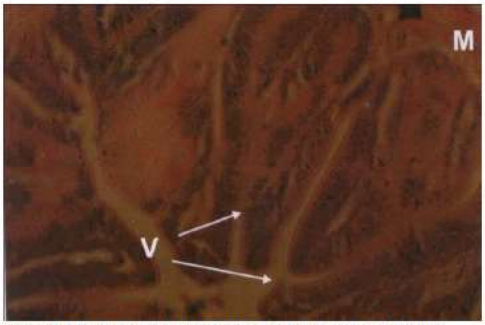

Fig. 3 Histology of intestine of *Mugil cephalus* – Control (15 15 X) MV –microvilli V-villi

Fig.8 Histology of kidney of *Mugil cephalus* – Experimental (15 X) R-renal tubules

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