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Research Article

EFFECT OF ALUMINIUM CHLORIDE TOXICITY AGAINST HISTOPATHOLOGY OF GILL OF FRESH WATER FISH *Labeo rohita* (Ham)

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A B S T R A C T

The toxicity of Aluminium chloride was investigated with emphasis on histopathological effects of fingerlings of *Labeo rohita*. The fishes were exposed Aluminium chloride solution at 24, 48, 72 and 96 hrs of intervals. The lethal concentration (LC_{50}) value of Aluminium chloride was 33ppm for 96 hr of exposure. Exposure of Aluminium chloride**:** Fishes were grouped into control and experimental which were exposed to sub lethal concentration of Aluminium chloride (3.3 ppm), over a period of 96 hr. Histopathology of the organ gills after 96 hr exposure revealed cell proliferation, lamellar fusion, lamellar cell hyperplasia, and epithelial lifting. The changes in these gill tissues occur predominantly in the 96 hr exposure. Which varied with the concentration of the toxicant. Aluminium chloride is highly toxic to *Labeo rohita*., therefore its high concentration of Aluminium chloride in areas close to aquatic bodies should not be encouraged.

INTRODUCTION

Heavy metal contamination of the aquatic environment has drawn increasing attention as it may have devastating effects on the ecological balance of the recipient environment and a diversity of aquatic organisms. Now a day the fresh water ecosystem has been polluted by continuous discharge of waste water from industries, human dwelling and agricultural practices (Maharajan 2012). Waste water contains various amounts of chemical substances and produces the fatal effect on biota including fishes. Poisonous substances accumulate in organisms through food chain and harmful to human beings. Heavy metals pose a serious threat to the aquatic environment because of their greater toxicity and persistence to accumulate in organisms through food chains amplification (Weis and Weis, 1977).

Urban streams are one of the ecosystems most hit by the contamination resulting from human activity (Paul and Meyer, 2001). Agricultural, industrial and domestic effluents generally contain a wide variety of organic and inorganic pollutants, such as solvents, oils, heavy metals, pesticides, fertilizers and suspended solids (Pandey *et al*., 2003) and are, invariably, discharged into rivers and streams, without proper treatment.The contamination of fresh water with a wide range of pollutants has become a matter of concern over the last few decades (Dirilgen, 2001 and Vutukuru, 2005).

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The natural aquatic system is getting extensively contaminated with heavy metals released from domestic, industrial and other anthropogenic activities (Velez and Montoro, 1998). 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). Different industries like distilleries, cotton mills, tanneries, paper mills, jute mills, and fertilizers pass out their effluents in adjoining rivers, ponds, ditches and other water resources. Tissue changes in test organisms exposed to a sub lethal concentration of a toxicant are a functional response of organisms that provides information on the nature of the toxicant **(**Mathur and Gupta 2008). All these chemicals threaten the existence of flora and fauna and adversely affect the ecological balance leading to unwanted mortality of aquatic biota including fishes (Alam 2002).

Fish are continued to be an extremely reliable component of an aquatic monitoring system because they integrate the effect of detrimental environmental changes as consumers which are relatively high in the aquatic food chain. The fish as a bioindicator species plays an increasingly important role in the monitoring of water pollution because it responds with great sensitivity to changes in the aquatic environment. The sudden death of a fish indicates heavy pollution; the effects of exposure to sub lethal levels of pollutants can be measured in terms of biochemical, physiological or behavioural responses of the fish. Fish are very good biosensors of aquatic contaminants (Kanchan Kumari *et al*., 2011). Aluminium toxicity has been recognised in many conditions where exposure is heavy or prolonged, where renal function is limited or where a previously accumulated bone burden is released in

stress of illness. Toxicity may include encephalopathy, osteomalacia or aplasitc bone disease, proximal myopathy, increased risk of infection, increased left ventricular mass and decreased myocardial function, microcytic anemia with very high levels, sudden death. Histopathological changes have also been widely used as biomarkers in the evaluation of the health of fish exposed to contaminants, both in laboratory and in field studies (Das and Mukherjee, 2000). One of the advantages of using histopathological biomarkers in environmental monitoring is that this category of biomarkers allows examining target organs gills are responsible for vital functions, such as respiration.

MATERIALS AND METHODS

Collection of Experimental Fish

The finger lings of the fresh water fish, *labeo rohita* measuring 6 ± 0.5 cm in length and 7±0.5 gm weight were procured from the B.S.P Aquarium, Puthur, near Chidambaram, Tamil Nadu. They were brought to the laboratory in polythene bags filled with aerated water and stocked in a tank of 50 litres capacity filled with tap water and fishes were acclimatized in the laboratory condition for about two weeks. Significant sign of stress or unusual behavioural criteria were not observed in the control fishes throughout the acclimation and test period. During the acclimatization the fishes were fed with pellet feed daily in the evening uneaten feed was removed next day morning followed by 100% water exchange.

Preparation of stock Solution for Aluminium Chloride ($alcl_3$) toxicity test

One gram of Aluminium chloride (Merck, Germany) was dissolved in 1L of double distilled water and used as the stock solution for preparing different concentrations of Aluminium chloride in rearing water. It was stored in a clean standard flask at room temperature in the laboratory.

Sublethal Toxicity Test

Sub lethal studies are helpful to assess the response of the test organism under augumented stress caused by heavy metal. Hence 3.3ppm of the 96 hrs LC_{50} values of aluminium were selected as sublethal concentration for the present investigation.

The experimental fish were exposed to sub lethal concentrations of aluminium for a period of 28 days, at weekly intervals (7, 14, 21 and 28 days). The rate of opercular movement was measured both in control and treated fish. The control and experimental fish were dissected at the end of each period of exposure and the selected tissues viz., gill were collected for tissues were later processed for histopathological observation. After the treatment five fish each from the respective experimental as well as control groups were sacrificed

Histopathology Studies

The organs were removed and prepared for histhopathological observation. They were fixed in bouin's fluid for 24 hours, washed with 70 percent ethanol and dehydrated through a graded series of ethanol (Schalm *et al,* 1975, Kelly, 1979). They were embedded in paraffin, sectioned at 4-5 um thickness stained with haematoxylin and eosin and examined using light microscope and photomicrography (Keneko, 1989).

RESULT AND DISCUSSION

In control fish, the secondary gill lamellae (SGL) appeared as finger-like structures. The SGL was thin, slender and attached on either side of the primary gill lamellae (PGL). The secondary gill lamellae are highly vascularised and surrounded by a thin layer of epithelial cells fig.1.

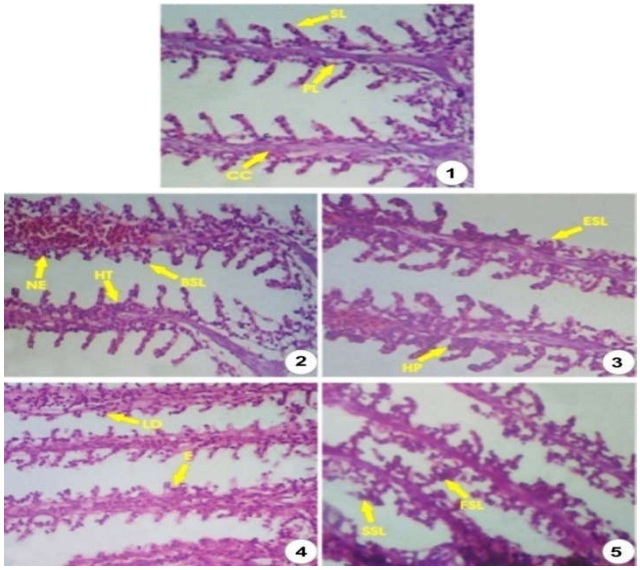

Figure Histological sections of gills, *Labeo rohita*

Gills sections (H and E stained, X100), 1-control, 2,3,4,and 5 are aluminium chloride exposed for 24, 48, 72 and 96 hr, respectively.; primary lamellae(PL), secondary lamellae (SL), center core(CC), necrosis(NE), broken of secondary lamellae(BSL), hypertrophy (HT) hyperplasia (HP), erosion of secondary lamellae(ESL), edema (E), Lamellar disorganization(LD), fusion of secondary lamellae (FSL) and swelling of secondary lamellae(SSL),

The overall observed results in the present investigation indicates that marked histopathological changes have been found in the gill of fish *L. rohita* under sublethal concentrations of aluminium chloride in chronic exposure. Fusion and shortening lamellae, hypertrophy, degeneration of epithelium and necrosis were found in the gill of aluminium chloride treated *L.rohita* (fig.2&3). Higher degree of hypertrophy and fusion of gill lamellae were prominent in the gills of fish exposed to 28 days. Hemalatha and Banerjee (1997) and Gupta and Kumar (2006) noted similar types of gill lesions in zinc treated *Heteropneustes fossilis* and mercury treated *Cirrhinus mrigala* respectively. Kaoud and El-Dahshan (2010) observed severe hyperplasia in secondary gill lamellae which lead to complete embedding in adjacent lamellae in copper, cadmium, lead and mercury treated *Oreochromis niloticus*. In the present study, hypertrophy and degeneration of secondary lamellae were apparent in *L. rohita* exposed to aluminium chloride (fig. 4). These observations are quite comparable to pathological lesions induced in gills by mercuric chloride in *Acipenser persicus* fry (Khoshnood *et al.,* 2011), by lead and cadmium treatment in *Cyprinus carpio* (Patnaik *et al.,* 2011), *Lates calcarifer* (Thophon *et al.,* 2003), *Brachydanio rerio* and *Salmo gairdneri* (Karlson-Norgren *et al.*, 1985). Patel and Bahadur (2010) also noted severe gill lesions in copper treated *Catla Catla.* In the present investigation the gill epithelium of aluminium chloride treated fish was completely desquamated, fusion and shapeless secondary lamellae and were broken at several places (fig.5). The stressful behaviour of respiratory impairment due to the toxic effect of zinc, one of the main components of tannery wastewater on *Labeo rohita* (Loganathan *et al*., 2006), adds

support to this finding. The infiltration of components of tannery wastewater through the gills might have caused these abnormalities. Daoust *et al.,* (1984) also observed similar pathological lesions in the gill of copper treated rainbow trout, *Salmo gairdneri.* Further, Hemalatha and Banergee (1997) and Al-Atter (2007) also observed such gill damages in zinc chloride and nickel treated *Heteropneustes fossilis* and *Oreochromis niloticus.*

CONCLUSION

The present investigation concludes that the heavy metal, Aluminium chloride induced acute toxicity and histopathological alteration and caused significant metabolic effect on the physiological consequences. These histopathological offer a rapid and sensitive means of monitoring towards the impact of Aluminium chloride on *Labeo rohita*. The Aluminium chloride generated from industries, agricultural runoff and domestic sewage pollute the freshwater ecosystem to an injuries level. Fish from such water bodies is not safe for human consumption because of the possibility of toxic material present in the fish. Fishes as well as water due to such pollution are toxic to human beings.

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