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Research Article

A HISTOLOGICAL OBSERVATION ON HEAVY METAL INDUCED IMPLICATIONS ON RESPIRATORY EPITHELIUM AND ITS CAUSATIVE IMPACT ON A CATFISH *HETEROPNEUSTES FOSSILIS* (BLOCH)

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ABSTRACT

Present study is based on lead nitrate induced histological effect on the respiratory organs of *Heteropneustes fossilis* (Bloch). Lead nitrate is a heavy metal compound which used in many industries and indiscriminately drains out in water system and contaminates aquatic medium leads serious hazardous effect on respiratory epithelium of fish. Lead nitrate induced alteration on respiratory epithelium associated with the air breathing organ, suprabranchial chamber and air sacs of *Heteropneustes fossilis* (Bloch) and also affect the oxygen consumption capacity of fish which adversely affect its survival. Heavy metal poisoning produced remarkable histopathological deleterious effect on the accessory respiratory organ decreases the rate of oxygen consumption. Various concentration of lead nitrate showed noticeable histopathological changes which is a sign of serious effect of this heavy metal and its bioaccumulation ultimately dangerous for human consumption of contaminated fish through food chain.

Keywords: Lead Nitrate, Histological changes, Oxygen consumption.

INTRODUCTION

Heteropneustes fossilis (Bloch) is an air breathing catfish capable to use atmospheric oxygen through its accessory respiratory organ known as suprabranchial chamber and air sacs. Diffusion of respiratory gas takes place by its respiratory epithelium of gills and air breathing organs. Gills are always in contact with the aquatic medium which baths it for respiratory need through gaseous exchanges. The fish uses its air breathing organ (suprabranchial chamber and air sacs) to utilize ambient oxygen for certain duration when the water is insufficient in its habitat or it is outside of aquatic environment. The air sacs are present in a pair and extended from anterior to posterior side of body and is a highly vascularised structure permeable for gaseous exchange. Heavy metals are now a day polluted water bodies and produce deleterious effect in the respiratory epithelium of fish inhabiting in it. Bioaccumulation of the Lead nitrate induces alterations in respiratory organs gills and suprabranchial chamber which ultimately minimize capacity of oxygen consumption of the fish. Various industries like paint, ceramics, automobiles, batteries etc, are indiscriminately drain their industrial effluents directly in the water bodies which leads threat for living bodies including fish species. Under experimental condition, various concentrations of Lead nitrate impair respiratory organs and cause primarily asphyxiation as a sign of heavy metal toxicity in the fish. In north India, especially in Bihar, the *H. fossilis*is highly usable fish for human consumption by the people suffering from anaemia because this fish is a good dietary source of iron. So the fish from heavy metal polluted water bodies can affect human population and leads adverse effect in their many vital organs including neural system.

Larsson *et al*., (1981) investigated the histopathological changes due to heavy metals in association with disturbance in osmotic and ionic balance. They also elucidated the chronic exposure to sublethal concentrations of lead nitrate resulting the damage of organs of respiration and excretion like kidney through impaired metabolism of ions like sodium, potassium, calcium, magnesium, chloride and inorganic phosphate in the flounder, *Platichthys flesus*. Pool (1981) proposed that other heavy metals like Cd also possesses strong affinity for sulphur, dithiol and other S-groups in the biological materials. Under laboratory conditions, hyperglycaemia is a typical symptom of cadmium poisoning, as reported in an

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Indian fresh water catfish *H. fossilis* (Sastry and Subhadra - 1982). Histological alteration in liver and intestine of *Sarotherodon mossambicus* in response to Hg toxicity was depicted by Naidu *et al*., (1983). They recorded engorged blood sinusoid, vacuolation, rupture, granular degeneration of hepatocytes, edemafocal necrosis and proliferation in fibroblast in liver. Jhingaran (1985) narrated air breathing fish, *Clarias batrachus* to be more refractive to cadmium toxicity than the non-airbreathing ones, viz., *Labeo rohita*. Dubale and shah (1979) exposed the *Channa punctatus* to different concentration of cadmium nitrate (0.01 to 0.05 ppm) and observed hepatopathic effects such as necrosis of hepatocytes. The reason behind the observation of Dubale and Shah (1979) were envisaged that these might happen due to the effect of cadmium on sulphur. Besides the work as mentioned above as toxic effect of notable heavy metals like Cadmium, Hg, on respiratory organs, the effect of lead nitrate in respiratory epithelium of accessory respiratory organ in an air breathing catfish *H.fossilis* is significantly noticed.

MATERIALS AND METHODS

Equal size (length-20 cm and weight about 30 gm) of test fish, *H.fossilis* bought from the fisherman of the local market and kept for two weeks in laboratory condition for acclimatization in aquarium. The fish were fed with tubifex and chopped goat liver during laboratory acclimatization. No mortality was noticed in these periods. The experimental fish were pre treated with 1%Methylene blue for 15 minutes to avoid disinfection. Dechlorinated tap water is used as test water. Three test glass battery jar used as test container in which first container contained no heavy metal and acted as control. Reagent grade Lead nitrate was obtained from scientific supplier, its calculated amount dissolved in 10 litres of water. Before diluting them, few drops of methanol used in it. Two series of lead nitrate with 30 ppm and 60 ppm concentration were prepared for experiment on present toxicological experiment.

Exposure procedure

Five fishes of almost equal body size were exposed to 30 ppm and 60 ppm of lead nitrate for 72 and 96 hours of duration. During the period of exposure, both the control and experimental fish were fed with 3% chopped goat liver of the total body weight of the fish. Replacement of test water performed every alternate day to prevent any chance of adverse effect of the faecal matters of the fish. After experiment, the desired tissues from the suprabranchial chamber and air sacs were collected from both the control and experimental specimen and processed separately for histological studies. The tissues were fixed in Bouin's fixative for different histopathological studies. Paraffin section of 6 micron was stained using delafields haematoxylin-eosin stain. Some selected slides showed different types of histological effect were photomicro graphed as test result and used for observation.

RESULTS AND DISCUSSION

Behavioural Manifestation Behavioural studies on the experimental fish *H.fossilis* exposed in to the various concentration of Lead nitrate, the fish showed restlessness, respiratory distress and rapid swimming than control. Increased opercular movement and gulping activity was evident. During the early stage of exposure, excessive secretion of mucous into medium was observed. In the 60 ppm of lead nitrate exposure, the fish exhibited lethargy and erratic swimming, suggesting loss of equilibrium. Instead of restlessness and excess mucous secretion, frequent opercular movement recurrent water breaking habit are some noticeable behavioural noticeable effect which commonly showed by heavy metal treated fish with several concentrations of Lead nitrate. On low concentration of toxicant, increased rate of swimming was commonly observed, but on high concentration of lead nitrate, sluggish swimming habit was more common which mark the suggestive adverse effect of toxic heavy metal.

Respiratory surface of suprabranchial chamber and air sac showed remarkable alteration like acute mucous cell hyperplasia after 96 hour exposure at 30 and 60 ppm of test toxicant. At some places respiratory epithelium of suprabranchial chamber was retracted. Blood capillaries dilation (telangiectasis) was observed in the form of swollen epithelium. Breakage of micro blood vessels erosion and hyperplasia of epithelium cells and fusion in the vascular papilla were also observed after 96 hours exposure at 60 ppm. The lining of the suprabranchial chamber showed vacuolation, the epithelial lining of air sac was corrugated. It result in the massive sloughing of epithelial cells, lesions thereby affected the microanatomy and histopathological changes in suprabranchial chamber and air sacs. According to Eisler (1971), the breathing distress in the fishes is due to clogging of the gills by mucous precipitation besides direct damage of the gill and other respiratory surfaces by heavy metal ion leading to anoxia, carbon dioxide retention and collapse of blood vessels. Lloyd (1960) pointed out that it is the cellular damage of the gills that cause respiratory distress in the fish and not the mucous coagulation. In this regard, it is reasonable to assume that the damage in the respiratory epithelium is main cause for the death of the fishes because of the breakdown of the vital functions of the body due to decreased efficiency of gaseus exchange through gills and accessory respiratory organ. Wedemeyer (1976) also pointed out that the reduced oxygen uptake occurs due to damage of gills and respiratory epithelium in the fish exposed to heavy metal salts.

Histopathological alteration in accessory respiratory organ of *Heteropneustes fossilis* (Bloch) clearly revealed that lead nitrate is a potent inhibitor of oxygen diffusion through suprabranchial chamber and air sacs of the fish. Possible reason of lead nitrate toxicity on the respiratory epithelium is because of histological damage and excess mucous secretion as well as the high concentration of toxicant might have produced negative implications on respiratory centre of the brain which ultimately exert respiratory impairment in the test fish through gills and air

breathing organs. Primary site of damage due to toxic contamination is gills through which gaseous exchange takes place. Change in histology of respiratory epithelium of gills and accessory respiratory organs gradually minimize the oxygen consumption through it and cause asphyxiation in fish. High dose of lead nitrate drastically decrease the capacity of oxygen consumption and lead death of the fish.

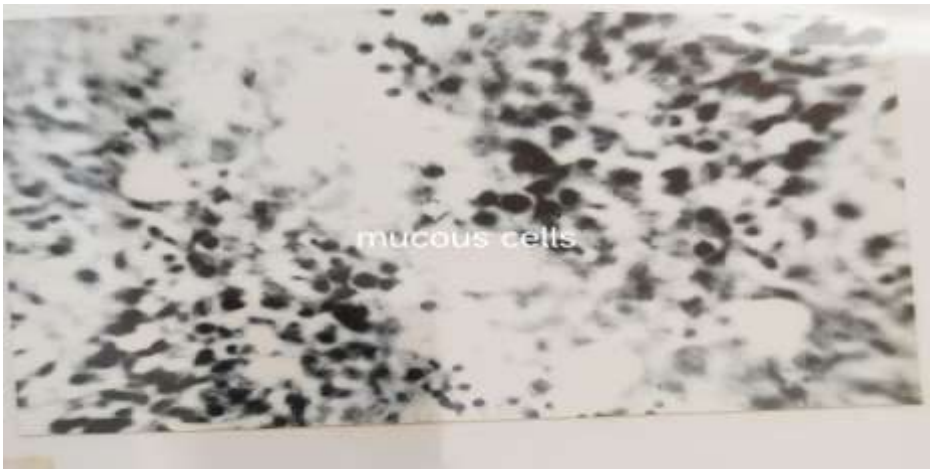

Figure 1. Control-T.S. of the suprabranchial chamber and posterior part of air sac of *H.fossilis* shows numerous folds in the epithelial lining of lumen (H/E × 350).

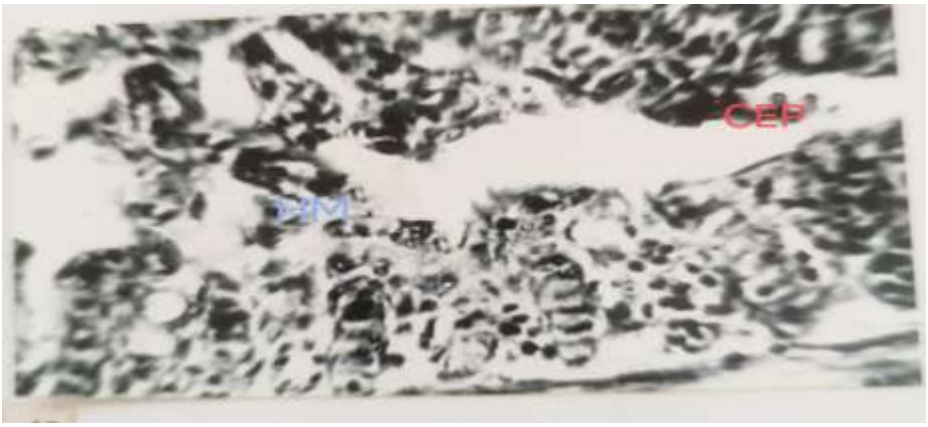

Figure 2. Treated- T.S. of suprabranchial chamber treated with 30 ppm of lead nitrate for 72 hours shows blood capillary dilation (H/E × 350) HM-Hyperplasia of mucous cells, CEP- Corrugated epithelium.

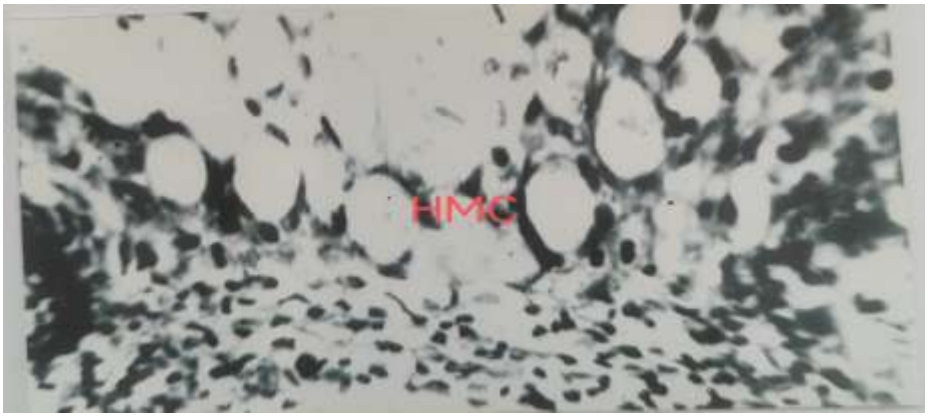

Figure 3. Treated-T.S. shows retracted respiratory epithelium of suprabranchial chamber (H/E × 850) HMC -Hyperplasia of mucous cells. Treated with 60 ppm lead nitrate for 96 hours.

CONCLUSION

Lead nitrate toxicity not only cause adverse effect in but may also affect human population through food chain by consumption of heavy metal contaminated fish. High concentration of lead nitrate cause irreversible changes in the respiratory organs result in mucous cell hyperplasia, rupturing of blood vessels and ultimate oxygen distress with asphyxiation. Asphyxiated air breathing fish *H. fossilis* vigorously start to gulp atmospheric air respiration through its accessory respiratory organs, suprabranchial chamber and air sacs because of the affected diffusion and gaseous exchange by its gills due to severe adversity of lead nitrate. These conditions completely hamper the fish to take oxygen for respiration and thus lead nitrate induced damage of respiratory organs created fatal conditions and increase its death toll.

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