WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 7.523
Volume 2, Issue 2, 505-510. Research Article ISSN 2277– 7105

EFFECT OF CHROMIUM ON THE GILL AND LIVER OF FRESH WATER SOUTH INDIAN FISH LABEO ROHITA

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Article Received on
18 Jan. 2013,
Revised on 08 Feb. 2013,
Accepted on 28 Feb. 2013
DOI: 10.20959/wjpr20132-8620

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ABSTRACT

The toxic effect of chromium on the histology of gill and liver of Labeo rohita fingerlings was studied. The fingerlings were exposed for 10 and 20 days in 10% sub lethal concentration of 96 h LC50 of chromium (3.5 ppm). The gills exposed to sub lethal concentration of chromium showed mild histological alterations during 10 days of exposure. However after 20 days, fusion of gill lamellae, hypertrophy and degeneration of epithelium were prominent. Liver lesions consisted of vacuolation, degeneration of hepatocytes and disintegration of cell boundaries of hepatocytes. These changes occurred predominantly in the 20 days exposure.

KEYWORDS: chromium, gill lamellae, hypertrophy, hepatocytes.

INTRODUCTION

Fish is an excellent source of protein in human diet. The unique feature which differentiates fish food other animal protein sources is the presence of omega-3 fatty acids such as Linolenic acid, Decosa hexaenoic acid (DHA) and Eicosa pentaenoic acid (EPA).[1] DHA promotes learning ability in children and improved memory in adults. DHA is also essential for the foetal growth and development. Omega fatty acid is also good for heart and helps to control diabetics by improving insulin action (Lee and Reasner, 2000 and Lemos et al., 2005).[2-3] That's why it must be included in human diet at least 1.3 kg per week (FAO, 1989). However, the fish habitates are being contaminated alarmingly through a number of aquatic pollutants. Among these pollutants heavy metals are most injurious to fish. These pollutants have not only depleted the fish stock but also have threatened the human health by incorporating into food chain (Pip, 1995).[4-5] Heavy metal chromium is widely used in metallurgic, refractory, chemical and tannery industries. Tanneries are the major industries

that use chromium for the treatment of leather and nearly 40% of the chromium used is released into the environment. In the present work, an attempt was made to evaluate the long term exposure effect of chromium on the histology of gill and liver of the freshwater fish Labeo rohita.

MATERIALS AND METHOD

The fish, L. rohita fingerlings (Wt:8 0.5g; Length 7.1 cm) were collected from the Katherasan Aqua Farm near Thanjavur, Tamil Nadu. They were acclimatized for 15 days in large cement tanks (Temperature 24.2°C, pH 7.2 ±0.3 and Dissolved Oxygen 7.9±0.7 mg/l) Previously washed with 1% potassium permanganate. The water as renewed every 24 h. The LC50 of potassium dichromate for 96h was found out by using Probit method (Finney, 1971). Stock solution of chromium was prepared and the toxicity tests were conducted to be 3.5 ppm. For histological studies L. rohita were reared in sublethal concentration (10% of 96 hours LC50) for a period of 10, 20 and 30 days. The gill arches and liver were dissected out and fixed in Bouin's fluid. The tissues were embedded in paraffin (58°C) and sectioned at 6µ thickness. The sections were stained with haemotoxylin and eosin (Gurr, 1959). The stained slides were examined for histopathological changes and were photomicrographed.

RESULTS 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 (Figure 1). The overall observed results in the present investigation indicates that marked histopathological changes have been found in the gill and liver of fish L. rohita under sublethal concentrations of chromium in chronic exposure. Fusion and shortening lamellae, hypertrophy, degeneration of epithelium and necrosis were found in the gills of chromium treated L. rohita (Figures 2 & 3).[6-8] Higher degree of hypertrophy and fusion of gill lamellae were prominent in the gills of fish exposed to 30 days. Hemalatha and Banerjee (1977) 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)[9-10] observed severe hyperplasia in secondary gill lamellae which lead to complete embedding in adjacent lamellae in copper, cadmium, lead and mercury treated Oreochromis niloticus. 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)[11-13] also noted severe gill lesions in copper treated Catla catla. In the present investigation the gill epithelium of chromium treated fish was completely desquamated, fuion and shapeless secondary lamellae and were broken at several places (Figure 4). Daoust et al. (1984) also observed similar pathological lesions in the gill of copper treated rainbow trout, Salmo gairdneri. Further, Hemalatha and Banerjee (1997) and Al-Attar (2007)[14-15] also observed such gill damages in zinc chloride and nickel treated Heteropneustes fossilis and Oreochromis niloticus. Liver of fish is responsible for digestion, filtration and storage of glycogen. The liver also produces many enzymes that stored in the gall bladder. The liver functions to store food energy. The normal liver is made up of continuous mass of hepatocytes with large number of blood sinusoids (Figure 5). The fish exposed to the sublethal concentrations of chromium showed the vacuolation, loose arrangement of hepatic cells, histolysis and disintegration of cell boundries (Figures 4-5). The damage as more severe and progressive after 30 days exposure. Histological changes in the liver of fishes have been extensively reported. Athikesavan et al. (2006) reported the histological lesions in the liver of Hypophthalamichthys molitrix exposed to nickel. They observed marked changes like degeneration of blood vessels hypertrophy, vacuolisation necrosis and pyknotic nuclei of the exposed fish. The histological lesions due to cadmium and zinc poisoning were reported by Van Dyk et al. (2007)[16] in Oreochromis mossambicus. Loganathan et al. (2006) and Radhakrishnan and Hemalatha (2010) have also observed the histological changes in the liver of zinc treated L. rohita and cadmium chloride treated Channa striatus. They observed that the cytoplasmic vacuolization of hepatocytes, congestion of blood vessel, leucocytic infiltration and necrosis. The results of the present observations in L. rohita exposed to chromium were in agreement with those of the earlier workers especially in the vacuolization and necrosis in hepatic tissue. Intracellular vacuolation, necrosis and shrinkage of nuclei were also apparent in the present study in chromium treated L. rohita. Similar changes are observed by Loganathan et al. (2006) in zinc treated L. rohita. The development of necrosis congestion of hepatic blood vessels and vacuolization in chromium treated L. rohita were mainly due to large scale accumulation of these metals in liver. Liver is the vital organ for detoxificationof unwanted and toxic substances. Accumulation and elimination processes of metals ions in the liver may lead to hepatic lesions.[17]

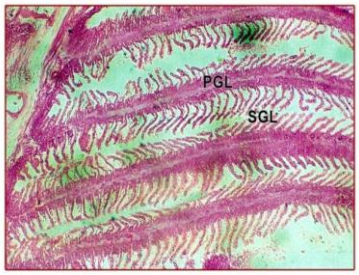

Fig 1

Fig 2

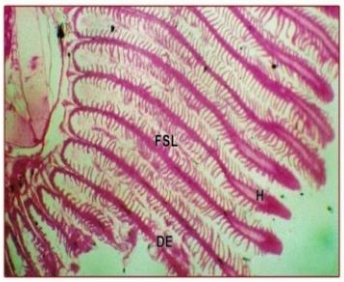

Fig 3

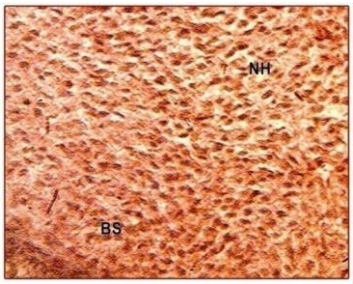

Fig 4

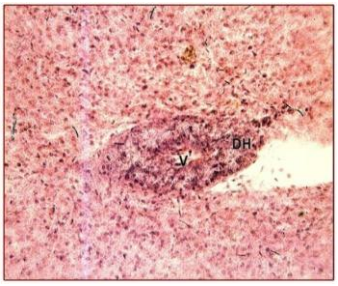

Fig 5

Figure 1. Control fish gill. PGL - Primary Gill Lamellae SGL - Secondary Gill Lamellae Figure 2. 10% SLC of Chromium treated fish gill 10 days. EGL - Erosion of Secondary Lamellae FSL - Fusion of Secondary Lamellae Figure 3. 10% SLC of Chromium treated fish gill after 20 days. DE - Degeneration of Epithelium, H – Hypertrophy FSL - Fusion of Secondary Lamellae Figure 4. Control fish liver. NH - Normal hepatocytes, BS - Blood Sinus. Figure 5. 10 % SLC of Chromium treated fish Liver after 10 days. DH - Degeneration of hepatocytes, V – Vacuolization.

CONCLUSION

The results in the present study showed that the exposure of rohu to chromium caused pathology in their organs such as gill and liver and they were associated with the exposure period. Histological alterations in rohu under the toxicity of heavy metal chromium can be used as a sensitive model to monitor the aquatic pollution.

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