



Histological Changes in Gills of Fish *Channa Punctatus* (Bloch) Exposed To Sub-Lethal Concentrations Of Zinc

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Abstract

The present study is aimed to assess, histological changes in the gills of the freshwater fish *Channa punctatus* (Bloch), caused by zinc toxicity. Fish *Channa punctatus* were exposed to three sub-lethal concentrations of zinc for 15 days. The effects of this exposure have been studied on histology of gills at intervals of 8, 10 and 15 days. Severe histopathological changes were observed in gills of all exposed groups. Severity of damage in the gills were time and dose dependent. Result suggests that zinc treatment probably dysfunction the detoxification mechanism of the organ. Present investigation indicate that histological damage caused to the gill tissue may result in hypoxia (respiratory distress) followed by other deleterious changes culminating in death of animal.

Keywords: *Channa punctatus*, histopathology, gills, zinc.

INTRODUCTION

Pollution of aquatic ecosystem by chemicals used in industry and agriculture is increasing day by day. Heavy metals are known to cause extensive pollution [Sultana and Uma Devi, 1995; Singh and Gaur, 1997; Maruti and Subba Rao, 2000 ; Shekhawat , 2001; Shukla et al. 2002]. Among these heavy metals, zinc is essential to sustain biological processes [Sultana and Uma Devi, 1995; Shukla et al. 2002; Srivastava et al., 2002 , Srivastava ,2004; Gupta and Srivastava, 2005]. However, when zinc occurs at levels higher than normal, it produces a wide range of harmful effects on the ichthyofauna [Sultana and Uma Devi, 1995; Singh and Gaur, 1997; Banerjee, 1998; Agarwal and Srivastava , 2003 ; Tyagi , et al. , 2004] .

Excessive zinc from the environment may enter fish body through nutrients, general surface of the body and gills. Handy and Eddy (1990) studied zinc accumulation in gills, skin, body mucus, and blood plasma of rainbow trout and observed that gills and body mucus were the primary sites of zinc accumulation in fish.

Histopathological changes in gills have been reported by Llyod (1960) in rainbow trout, Mukhopadhyay and Konar (1984) in *Tilapia mossambicus* and Crespo and Sala (1986), in *Scyliorhinus canicular* following zinc toxicity.

As fish are good biological indicators of pollution in aquatic system (Janardana Reddy et al., 1998; Srivastava and Kaushik, 2001) and *Channa Punctatus* is an edible species the present day study has been undertaken to correlate gill pathology to the amount of zinc present in the medium.

Materials and Methods

Live specimens of *Channa Punctatus* (Bloch) were obtained from local water bodies. They were acclimatized for 15 days to laboratory conditions. During acclimatization the fish were fed chopped goat liver. Physico-chemical conditions were analyzed as per methods given in APHA et al.(1998) before and during the experiment (Table 1). Acclimatized fish were distributed into four groups of forty fish each. Group I served as control whereas groups II,III and IV were exposed to sub-lethal concentrations of zinc i.e. 10 mg Zn/l, 15 mg Zn/l and 25 mg Zn/l respectively for 15 days. These doses are below 50% LC₅₀ value (56.52 mg/l). Fish were autopsied at day 8,10 and 15.

At each autopsy interval, gills from the control and experimental fish were excised very carefully, washed in saline solution to eliminate mucus and blood deposits and fixed in Bouin's fixative. Gill filaments from middle region were taken for histological studies. The tissues were embedded in paraffin wax using routine techniques and sectioned at 5 μ thickness. These were stained with HARRIS haematoxylin and eosin and observed for histopathological changes.

Results and Discussion

Results of present study showed histopathological changes in all exposed groups and continue till the termination of the experiment. Gills show vacuolization, fusion, hyperplasia and telangiectasis in secondary gill lamellae (Fig. 2). Blood cell accumulation is observed at the base of secondary gill lamellae (Fig 3). Detachment of basement membrane from pillar cell system can also be seen (Fig 4). Vacuolization, hyperplasia, telangiectasis, fusion of secondary gill lamellae and blood cell accumulation is prominent in groups III and IV (Fig 5,6). Bud formation in cartilaginous core is also visible (Fig 6). These damages become severe by day 15 and is more pronounced in group III and IV.

Pathological condition in the gills have been recorded in fish following exposure to various heavy metals and other pollutants (Mukhopadhyay, 1985; Roy and Dutta Munshi, 1991; Anitha Kumari and Shree ram Kumar, 1997; Dhanapakiam et al., 1998).

Fusion, vacuolization, necrosis, hypertrophy of secondary gill lamellae and accumulation of blood cells in the blood capillaries and intercellular lymphoid spaces of the secondary gill lamellae have been reported in the gills of *Heteropneustes fossilis* after exposure to lethal and sub-lethal concentrations of cadmium (Gupta and Rajbanshi, 1982). Mukhopadhyay (1985) observed haemorrhage in the gills of *Tilapia mosambicus* treated with lethal concentrations of Cu, Zn and Fe individually and in a mixture for 96 hrs. Similarly, haemorrhagic conditions are seen in present study

It can be suggested that these histopathological changes reduce the respiratory area thereby greatly reducing respiratory and osmoregulatory potential of the fish. Present results on *C. punctatus* further suggest that blocking of the spaces between adjacent secondary gill lamellae (due to hyperplasia and fusion of adjacent secondary gill lamellae) affect blood circulation in gill capillaries which in turn reduces oxygen supply and produces hypoxic conditions. A characteristic pathological condition to the gill "lamellar telangiectasis" is observed in the present study, this also results in impaired respiratory function. Similar detrimental changes were noted by Anitha Kumari and Shree Ram Kumari (1997). They suggested that

hyperplasia of interlamellar cells and necrosis of secondary gill lamellae reduces the respiratory surface. Severe histopathological damages and a decrease in oxygen consumption by fish has been reported by Ikedayagoi et al. (1996) on treatment with cadmium.

Venkataramana and Radhakrishnaiah (1987) have suggested that high concentration of metals in the gills may cause severe structural damage and impaired physiological function. In the present study, histopathological changes in gills may also be caused due to high zinc concentration in the gills (observed by author in the same experiment). High zinc accumulation probably dysfunction the detoxification mechanism within the organs hence retarding metal elimination and enhancing its accumulation (Geetha et al., 1996). Severity of damages increase as the concentration of zinc increases in the tissue.

From the present investigation it emerges that histopathological changes in gills adversely affect the functioning of this organ and cause tissue hypoxia (respiratory disorder) followed by other deleterious influences on body functioning. It is obvious that vital processes like respiration and osmoregulation are in turn hampered and can cause death of animal

Table 1

Physico-Chemical Characteristics of Water.

Temperature	25°C to 27°C
pH	7 – 7.2
Dissolve oxygen	7 – 7.5 mg/l
Hardness	210 – 215 mg/l
Chloride content	35 – 38 mg/l
Alkalinity	67 – 69 mg/l

LEGANDS

Fig 1: Photomicrograph of the gill of *C. punctatus*(group-I) showing normal structure (Sgl-secondary gill lamellae, pcs-pillar cell system, cc-cartilaginous core, Bm-basement membrane, E-epithelium, Bc-blood capillary.)

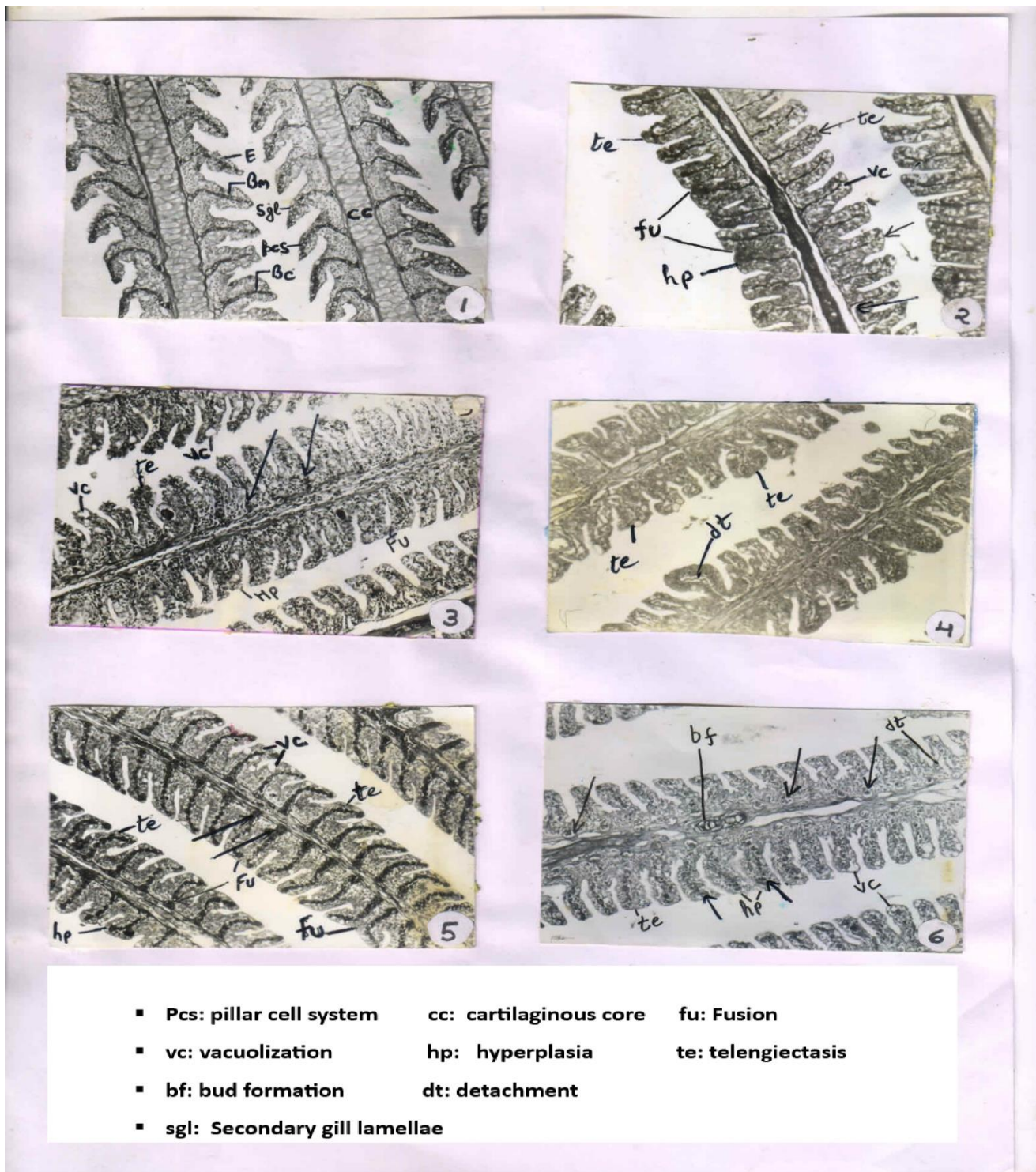
Fig 2: Photomicrograph of the gill *C. punctatus* showing vacuolization (vc), hyperplasia(hp), fusion (fu), telangiectasis (te) in secondary gill lamellae at day 8 interval. X 100.

Fig 3: Photomicrograph of the gill of *C. punctatus* showing prominent vacuolization (vc), hyperplasia (hp), fusion (fu) and telangiectasis (te) in secondary gill lamellae. Note blood cell accumulation at the basal region of secondary gill lamellae (arrow) at day 10 interval. X 100

Fig 4: Photomicrograph of the gill *C. punctatus* showing Telangiectasis (te). Note detachment of basement membrane from pillar cell system (dt) at day 10 interval. X 100.

Fig 5: Photomicrograph of the gill of *C. punctatus* showing prominent vacuolization (vc), hyperplasia (hp), telangiectasis (te), fusion (fu) of secondary gill lamellae. Note enlarged basal blood capillaries of secondary gill lamellae (arrow) at day 15. X 100.

Fig 6: Photomicrograph of the gill *C. punctatus* showing blood cell accumulation at basal region as well as at the tip of secondary gill lamellae (arrow). Note detachment of basement membrane (dt) and bud formation in cartilaginous core (bf) at day 15 interval. X 100



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