Original Article

Histological Effects of Cadmium on Hepatopancreas and Gill in *Cyprinus carpio*

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ABSTRACT

Background: Histological studies in organs like hepatopancreas and gill of the freshwater fish, *Cyprinus carpio,* were made to assess tissue damage due to sub-lethal concentration of cadmium after a long exposure period.

Methods: This study was conducted in University of Tehran, in 2013. The fish were divided into two groups in 1000 litre fiberglass tanks supplied with dechlorinated water [hardness 302.6 mg $CaCo_3$/l, pH 7, O_2 7.8 mg/L and temperature 15 ± 2 ºC] and continuous aeration. Group 1 was without any cadmium considered as control group. Group 2 was exposed to 30 ppb $CdCl_2$ (Merck) containing 7.8 ppb cadmium. The tissue samples from: hepatopancreas, gill were collected 30 days post exposure and processed by histological procedures.

Results: The main lesions in cadmium exposed groups were: (a) pancreatitis necrosis in endocrine part of pancreas (b) cholangitis and necrosis in liver parenchyma (c) hypertrophy, fusion and telangiectasia in secondary lamellae

Conclusion: Cadmium chloride at low concentration can induce pathological alterations in hepatopancreas and gill of common carp.

Keywords: Cadmium, Common Carp, Gill, Hepatopancreas, Histological Effects.

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INTRODUCTION

Cadmium is a non-essential element that can have severe toxic effect on aquatic organisms when present in excessive amount. Cadmium is important because of its use in various industrial processes, as by-product of zinc mining, fossil fuel, base metal smelting and combustion [1]. The role of sub-lethal concentration of cadmium in changing haematological parameters in *Hetroclarias* is reported [2] and lysozyme, leukocyte count and phagocytic index in *Cyprinus carpio* [3]. There are many investigations on the histopathological changes in common carp kidney due to cadmium exposure [4, 5]. Randi et al. [1] studied the histopathological effects of cadmium on the gill of *Macropsobrycon uruguayanae.* Pathological damages in the liver of *Oreochromis mossambicus* after short and long time exposure to cadmium and zinc was documented [6]. Histopathological alterations of white sea bass, *Lates calcarifer* in acute and sub-chronic cadmium exposure have been reported [7]. Exposures to low levels of cadmium can cause DNA damage and stress in common carp [8]. Cadmium is a risk factor for pancreatic cancer in human [9].

The goal of the current study was to identify whether cadmium at sub-lethal concentration, can cause histological changes in the hepatopancreas and gill of *C. carpio*.

MATERIALS AND METHODS

The current study was done in University of Tehran in 2013. Common carp obtained from a local fish farm were acclimated in holding tank for 1 week, then 20 apparently healthy fish, mean weight 700 g were randomly divided into two groups (1-2) in 1000 litre indoor fiberglass tanks supplied with dechlorinated water (hardness 302.6 mg/L as $CaCo_3$, dissolved oxygen 7.8 mg/L, pH 7, temprature15 ± 2ºC), without water flow and with continuous aeration. Fish in group 1 was held in water without cadmium and group 2 exposed to 30 ppb cadmium chloride (Merck) for 30 d This

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concentrations were less than 1% of LC50 96 h for this species [10] [11]. Water quality was measured throughout the exposure two times weekly. The fish were fed 1% body weight weekly with commercial food. Five fish from each tank were euthanized and sampled for tissues material containing hepatopancreas, gill 30 d post exposure. Tissue samples were fixed in 10% buffered formalin, embedded in paraffin, Sectioned at 5µ, and stained with hematoxylin and eosin (H&E) for histopathology [12].

All procedures were conducted according to ethical guidelines of animal experiences (CEE 86/609 regulation).

RESULTS

No death occurred either in the control or in the experimental group during the whole period of the experiment. After 30 days post challenge histopathological alterations in group 2 were as follows: Focal necrosis and fatty change (Fig. 1) and mononuclear cholangitis (Fig. 2,3) were seen in the liver.

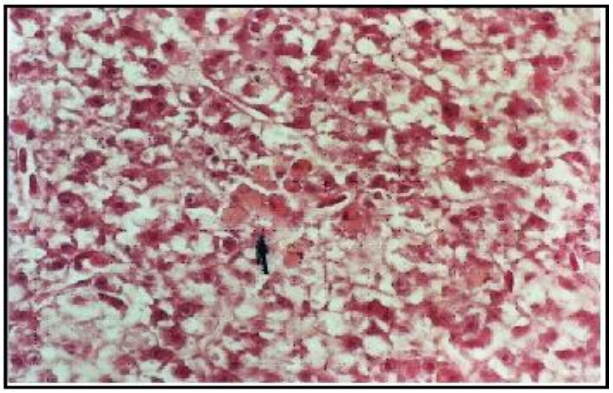

Figure 1. Focal necrosis (arrow) and fatty change in the liver (H&E×400)**.**

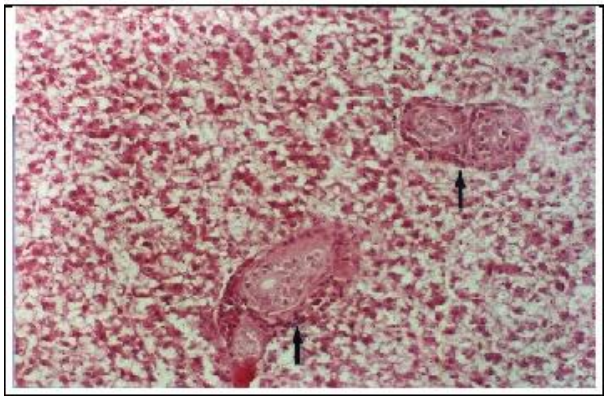

Figure 2. Mononuclear cholangitis in the liver (arrow (H&E×200).

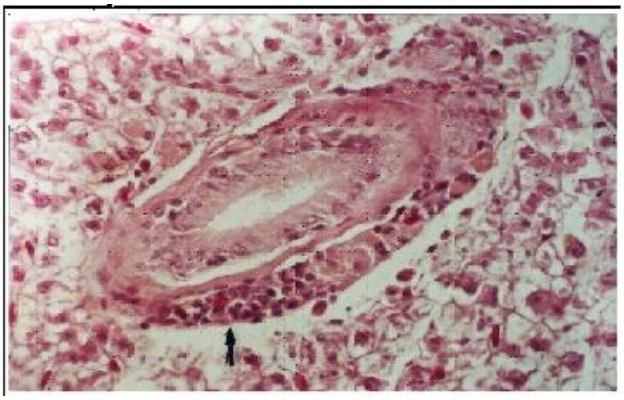

Figure 3. Mononuclear cholangitis in the liver (arrow)) (H&E×400).

Mononuclear (lymphocytic) pancreatitis (Fig. 4) and single cell necrosis were observed in pancreas (Fig. 5). Hyperplasia of secondary lamellae, lamellar hypertrophy and fusion and lamellar clubbing (Fig. 6) and telangiectasia in the secondary gill lamellae (Fig. 7) were seen. No histopathological changes were observed in control group during experiments period.

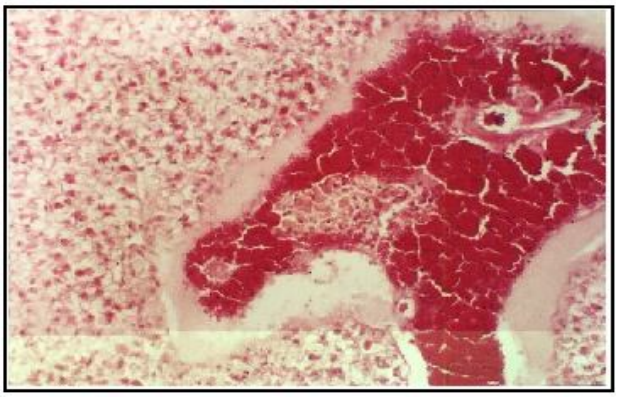

Figure 4. Necrosis in the endocrine portion of pancreas (arrow)(H&E×200).

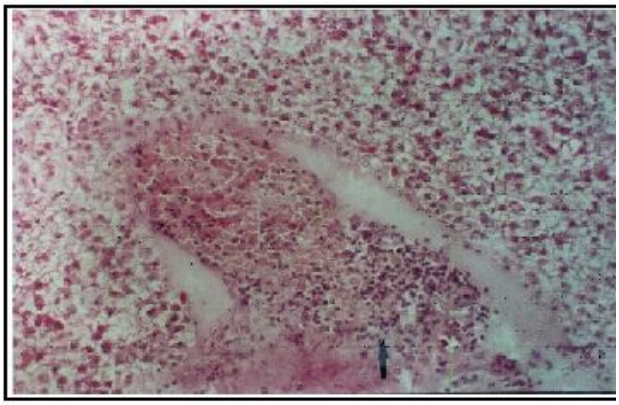

Figure 5. Pancreatitis and necrosis in the endocrine portion of pancreas (arrow) (H&E×200).

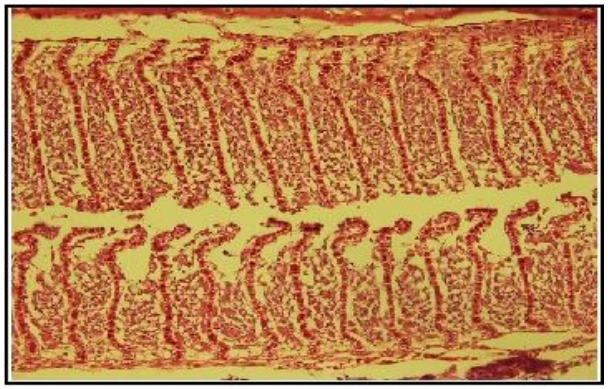

Figure 6. Hyperplasia of secondary lamellae, lamellar hypertrophy and fusion and lamellar clubbing (H&E×200).

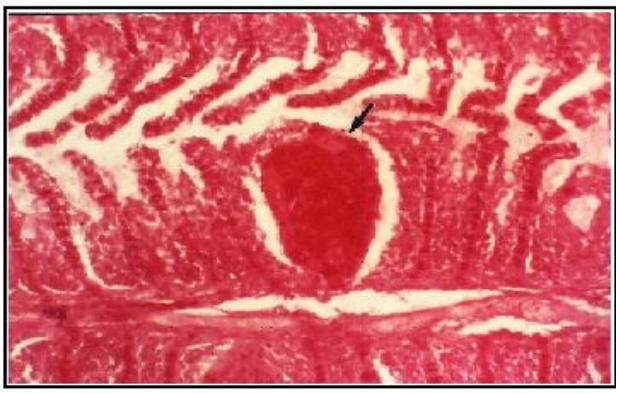

Figure 7. Telangiectasia in the secondary lamellae (arrow) (H&E×200).

DISCUSSION

Histology supplies a fast way to detect effects of irritants, especially chronic ones, in various tissues and organs [13]. In the current study, tissue abnormalities were found in all investigated organs. As our findings after 30 d post challenge telangiectasia in gills, focal necrosis in endocrine part of pancreas, fatty changes and focal necrosis in liver were only observed in cadmium exposure group. The cadmium induced histopathological changes in the gill of *C. carpio* were similar to lesions shown in other fish due to heavy metal toxicity [1]. Our findings are supported by previous finding that showed fusion of adjacent secondary lamellae and lamellar telangiectasia were only induced by exposure to cadmium while hyperplasia of secondary lamellar epithelium produced by either cadmium, or starvation even exposure time [1].

Cadmium chloride at sub-lethal dose can cause hypertrophy of lamellar epithelium, destroy of gill lamellar, blood congestion, hypertrophy and necrosis in liver of fresh water fish *Ophicephalus striatus* [14]. Necrosis in liver induced by cadmium in fresh water fish *Channa striatus* is reported [15]. In tissues such as kidney, spleen, liver and gill a low concentration of cadmium can be enlarged [16]. Seven percent of the human populations have kidney dysfunction due to cadmium exposure [17]. Metallothioneins (MTs) are cysteine rich proteins with low-molecular- weight, which can bind with metal ions [18]. It is critical for protecting human health from cadmium toxicity [17]. It is an important protein in the cellular defense against Cd toxicity and lethality [19]. MT synthesis in fish is related with organs involved in metal uptake, metabolism, and excretion, such as gill, liver, kidney, and intestine [20].

Possible ways by which MT may protect against metal toxicity include reduction of metal transport into cells; sequestration of metal within the cells; and increasing metal export out of cells. The mechanism of MT in cellular defense against Cd toxicity includes removal of free (toxic) cadmium form the inactive Cd-MT complex and alter the intracellular distribution of Cd [19] pathological effects would occur when the rate of metal uptake exceeds the rate of metallothionein. Once the metalothionien pool is saturated, free cadmium initiate toxic damage to the cells.

CONCLUSION

Cadmium chloride at low concentration can cause pathological damage in hepatopancreas and gill of common carp. Tissue abnormalities were found in all investigated organs. Pathological injury and telangiectasia of gill filaments were found. Liver tissue revealed hemorrhages, blood congestion and necrotic cells with mononuclear cell infiltration, but for clear detection of the damages we need to use quantitative histopathology methods. Histopathology could be used as an effective index to monitor toxicological changes induced by polluted environment and fish health status during exposure to pollutants such as heavy metals.

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