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CADMIUM TOXICITY AND ITS EFFECT ON THE HISTOLOPATHOLOGY OF LIVER AND KIDNEY OF THE FRESH WATER TELEOST, *HETEROPNEUSTES FOSSILIS* (BLOCH)

Zoology

Sachin Rastogi Deptt. Of Zoology,Maharishi University of Information,Lucknow(India)226013

Pushpa Yadav Deptt. Of Zoology ,NSCB,Govt. College,Lucknow(India)22601

Madhulika Singh Deptt. Of Zoology,Maharishi University of Information,Lucknow(India)226013

ABSTRACT

In this study, cadmium chloride ($CdCl_2$) toxicity was studied in an experimental aquatic exposure. *Heteropneustes fossilis* (an air-breathing catfish) were chronically exposed to different concentration of $CdCl_2$ (25, 50 and 75 mg/L) for 30 and 60 days. Morphological and histological changes in *H. fossilis* following the doses of $CdCl_2$ were studied. Histopathological investigations revealed greater changes in liver and kidney tissues after chronic exposure. Necrosis of epithelial cells of renal tubules, Glomerular contraction and reduction of Bowman's space were observed in the kidney tissue of exposed fish. Changes observed in the liver tissue post exposure included necrosis, degradation of hepatocytes, degeneration of blood vessels, distended sinusoids with pyknotic nuclei and vacuolation of cells. The degree of damage to the liver and kidney tissue was proportional to the concentrations and duration of the $CdCl_2$ used.

KEYWORDS

Cadmium Chloride, histology, liver, kidney, *Heteropnuestes fossilis.*

INTRODUCTION

Heavy metals have long been recognized as serious pollutant of the aquatic life. Pollution by heavy metals has becomes a serious environmental and public health hazard because the construction related into the environment from industrial processes often exceed permissible levels. Due to their bio accumulative and non biodegradable properties heavy metals constitute a care group of aquatic pollutants.Cadmium, which is the most venomous and non-essential heavy metal has wide distribution in the earth's Cadmium, crust and aquatic environments. In the list of heavy metals such as lead, mercury and cadmium are considered to cause public health hazards (ATSDR, 2003). Burning of fossil fuels or incineration of municipal waste materials are known to be largest sources of cadmium release to the general environment (such as coal or oil). Exposure of Cd can unfavorably influence living organisms and leads to pathological conditions (Cope et al., 1994; Annabi et al., 2011). As a persistent aquatic environment pollutant, freshwater fish are particularly susceptible to cadmium exposure (Sorensen, 1991).

Histological methods represent a practical tool to evaluate the effects of toxicants on living organisms (Cengiz and Unlu, 2005). It has also been extensively used in the evaluation of cadmium toxicity to fish exposed population (Thophon et al.,2003; Au,2004).In fish accumulations of cadmium in the liver, gills, kidney and gastrointestinal tract is reported (Norey et al., 1990).While kidney is suggested as one of the main targets for cadmium accumulation (Annabi et al 2011).Thus, in this paper we aim to investigate cadmium induced histopathological changes in liver and kidney of *H. fossilis*.

MATERIAL AND METHODS

Maintenance of fishes: Air-breathing fish *H. fossiles* were kept in glass aquarium containing cadmium chloride doses ($CdCl_2$) at room temperature (28-30°C). The control groups were maintained in identical condition without $CdCl_2$. During the experimental period (after 30 and 60 days) the fishes were fed *ad libitum* with a complete test diet (Halver and Coates, 1957). For each bioassay test, a series of three test concentrations (25 mg/l, 50mg/l and 75mg/l) of $CdCl_2$ and a control were used.

The tissue samples were taken from the fishes exposed to the first three concentrations (25 mg/l, 50mg/l and 75mg/l). At the end of the experiment (30 and 60 days), live fish samples were collected from the above-mentioned three concentrations, sacrificed and their liver and kidney were excised out and fixed in Bouins fixative (for 24 hrs.) and prepared for histological analysis according to standard procedures dehydrated in successive grades of ethanol series and embedded in paraffin. Serial longitudinal sections (thickness 4–5 µm) were stained with haematoxylin and eosin (H/E) for histological examination under a light microscope Also, light photomicrographs were taken. The morphological changes of the liver and kidney sections noted in the experimental fish were compared with those of control group fish.

RESULT

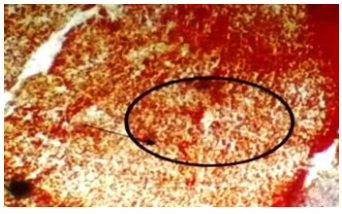
The liver of control *H. fossilis* appears dark brownish red coloured, bilobed gland composed of hepatocytes which are the parts of mass and forms a typical architecture (plate 1). The hepatocytes are arranged in a radial manner around hepatic veins to form hepatic cords. The liver cells are polygonal in shape and contain a prominent nucleus which possesses densely stained nucleoli. Following the exposure of $CdCl_2$ liver of fish *H. fossilis* becomes more fragile and darker in color but no tumour like out growth was seen anywhere in the liver at all tested doses. Histopathologically hypertrophy of hepatic cells has been observed (plate 1). Polygonal shape of the hepatic cells was completely lost at various places. Hepatocytes are found scattered in the hepatic tissue, vacuolization and pycnotic changes have been observed. Dilation of extra cellular spaces, bile canaticuli has also been noticed. In fishes exposed to 25 mg/l. $CdCl_2$ changes included ruptured nucleus, increased kupffer cell, ruptured hepatic tissue, cellular necrosis and increased pycnotic nucleus. Very distinct marked changes such as cellular necrosis, ruptured hepatic tissue, ruptured nucleus and focal necrosis were observed in the liver of fishes exposed to 50 mg/l $CdCl_2$. Further 75 mg/l dose of $CdCl_2$ pronounced the changes including focal necrosis, increased pycnotic nucleus, cellular necrosis and ruptured hepatic tissue (plate 1). All these effects were more pronounced in 60 days groups.

Kidney of control group showed normal histoarchiteceture however in $CdCl_2$ exposed fishes concentration and duration dependant changes were noted. In 30 days $CdCl_2$ (75mg/l) treated group kidney exhibited the degeneration of tubules and necrotic condition. The cells of renal tubules in the central region were fused and cytoplasm of these cells was condensed. Loss of original appearance and degeneration of cytoplasm renal tubules was also noted. The hypertrophy of glomerulus demonstrated. Glomeruli in Bowman's capsule revealed shrunken condition thereby wide space in the capsule was noted. The pyknotic nuclei in haemopoietic tissue were visible and cytoplasm was unevenly distributed. Eccentric and pyknotic condition of nuclei exhibiting in almost all cells (plate 2). In 60 days duration $CdCl_2$ (75mg/l) treatment the renal tubules undergo further degeneration and the changes were more pronounced than 30 days exposure. The cellular structure becomes more hypertrophied, the cells of renal tubules exhibited vacuolated condition and the cell membrane becomes indistinct. Glomeruli in the Bowman's capsule were had thick mass like appearance. The haemopoietic cells were highly vacuolated and severe degenerative changes was seen in haemopoietic tissue (plate 2).

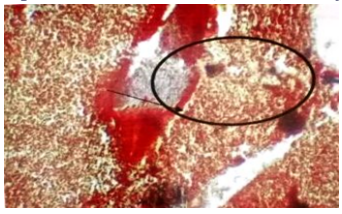
DISCUSSION

The toxicity effect of heavy metals on fishes pathology been studied by several workers. In this study toxic effect of $CdCl_2$ on the external morphology, and histopathology of liver and kidney of *H. fossilis* is in conformity to other similar kind of studies.

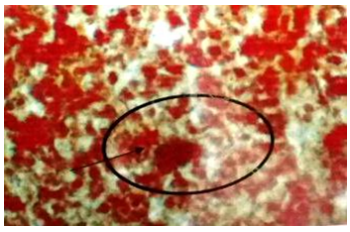
Histological alterations in liver tissues like degeneration of hepatocytes, vacuolization, congestion of hepatic tissues, subcapsular vacuolization, necrosis, indistinct cell boundaries and pyknotic nuclei were observed in the liver of the catfish, *Clarias batrachus* exposed to Cd by Selvanathan et al (2013). Initial lesion in the liver during the present study might be due to physiological changes that took place in the liver tissue in the process of trying to homeostatistically regulating and detoxifying the Cd metal during continuous exposure as suggested by Naigaga (2002).



Photomicrograph of section of Liver (control)of *H. fossilis*

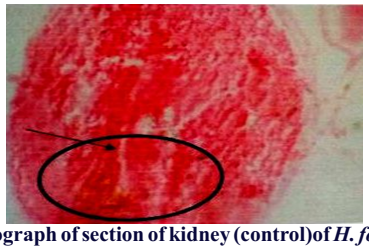


Photomicrograph of section of Liver (30 days exposure of CdCl2)of *H. fossilis*

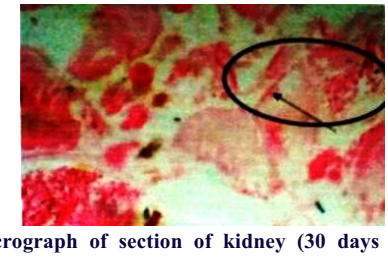


Photomicrograph of section of Liver (60 days exposure of CdCl2control)of *H.fossilis*

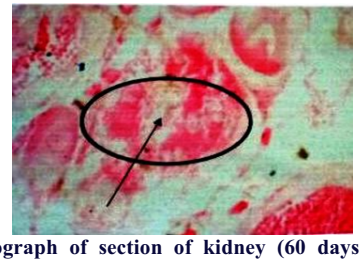
Plate-1



Photomicrograph of section of kidney (control)of *H. fossilis*



Photomicrograph of section of kidney (30 days exposure of $CdCl_2$)of *H. fossilis*



Photomicrograph of section of kidney (60 days exposure of CdCl2)of *H. fossilis Plate-2*

Our present observation on histological alterations on liver is in conformity with observations made in similar work carried out with different toxicants on various fish species too (Ikram and Malik 2009; Pantung et al., 2008) In hepatic tissue, the histological alterations noted during the chronic exposure (30 days and 60 days), focal necrosis, increased pycnotic nucleus, cellular necrosis and ruptured hepatic tissue in Cd concentration dependant manner. These findings are consistent with cadmium inducing greater hepatic alteration in fish after chronic exposure (Van Dyk et al., 2007). Further, in this study identified alterations of liver cells may be the result of diverse biochemical alteration in liver following the Cd toxicity and act as a signal of degenerative processes that suggests metabolic damage also (Pacheco and Santos, 2002). In addition to above chances vacuolation of hepatocytes is also noted which is suggested to be associated with the inhibition of protein synthesis, energy depletion or a shift in substrate utilization (Hinton and Lauren, 1990).

The fish kidney is one of the first organs to be affected by water pollutants (Thophon *et al.,* 2003) and is suggested as preferential site for Cd toxicity in fish (Brown *et al.,* 1984; Allen, 1995). In this study alterations of kidney tissue during the $CdCl_2$ (25, 50 and 75 mg/l) exposure were rigorous and was both concentration and time dependant manner. Following the chronic exposure, severe glomerular alteration was noted in kidney tissue of fishes. These findings are in confirmation of several previous studies on fishes (Olsson et al 1996; Thophon et al., 2003). Upon acute exposure of CdCl2 renal tubule necrosis and degeneration in fish (*Leiostomus xanthorus)* was noted (Hawkins et al., 1980). Hypertrophy of Bowman's capsule cells in fishes exposed to heavy metals was noted (Handy and Penrice, 1993). We also noted presence of pyknotic and fragmented nuclei in kidney epithelial cells is suggestive of apoptotic and necrotic cell death (Weber et al., 2003). Moreover, the presence of dilated tubules is to be a result of dead and dying epithelial cells, while a thickening of Bowman's capsule is due fibrosis (Weber et al., 2003).

CONCLUSION

In conclusion histological alterations in the catfish, *H. fossilis* under the influence of $CdCl_2$ toxicity can be used a sensitive method to monitor the aquatic pollution. The present result suggested that exposure to $CdCl_2$ concentrations leads to alterations in morphology and damages in the tissues of liver and kidney of fresh water fishes.

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