HISTOPATHOLOGICAL LESIONS IN LIVER OF THE FRESHWATER FISH, Channa punctatus (BLOCH) EXPOSED TO SELENIUM DIOXIDE

A.M. Avinashe and G. P. Patil*

Department of Zoology, S.S.S.K.R.InnaniMv.Karanja (Lad),Dist. Washim.

*Principal and Head, P.G. Dept. of Zoology, S.S.S.K.R.Innani Mahavidyalaya Karanja (Lad)

ABSTRACT

Environmental pollution is a global issue at the present moment. Many factors are behind the various types of environmental pollution and varied effects revealed by the pollutants. Aquatic pollution has concern with human life, as water is the main component of living organism like fish.

The harmful effect of any toxicant which was moved to water bodies can be assessed by investigating health of fish fauna.In the present study, an attempt has been made to examine the sublethal (concentration 0.5mg/L) toxic effect of selenium dioxide in freshwater fish, Channa punctatus. The histopathological lesions in liver have been observed after 7 days, 14 days, 21 days and 28 days of exposure to selenium dioxide, which were duration dependents.

Key Words: Histopathological alterations, Channa punctatus, selenium dioxide.

Introduction

Pollution of the aquatic environment generally causes changes in the physiological and structural aspects of the inhabitant organism, particularly the fish. Selenium compound can be harmful at when consumed in excess than needed. The seriousness of the effect of excess selenium demands on how much selenium is consumed and how often.Accidental swallowing of the large amount of sodium selenite could be life threatening for the fish.

Selenium after being absorbed into the blood is found to be associated with both erythrocytes and plasma albumin and globulin. Albumin appears to be the immediate receptor and is found to be involved in the transport of selenium to more stable sites in the blood and tissue (Stolman and Stewart, 1960). Absorbed selenium is found to be rapid distributed among the tissue. The distribution of selenium was found to be more in liver. Gunther (1965) reported that the intercellular distribution varied with the tissue and selenium level. In liver, 50 % of total was in the soluble fraction, 25 % in the microsomes and 2% in the nuclei.

Materials and Methods

Live specimens of the freshwater fish, *Channa punctatus* (Bloch) were selected for the present research work. The fish were procured for the experimental purpose from the freshwater “Rishi Lake” of Karanja (lad) Dist. Washim, M.S., India. They were washed with 1%KMnO4 solution for five minutes for dermal disinfection. The fish were acclimatized to the laboratory condition for a period of fortnight before conducting the experiment. Particularly in morning hours, fish fed on small pieces of boiled eggs, once in a day.

The 100 fish including males &females, weighing between 46-50gms were selected for the experimental work. They were maintained in separate aquaria, containing aged tap water. As per standard methods, the physicochemical parameters of aged tap water were determined periodically, (APHA, 1998).

To study the toxic effect of selenium dioxide on various organs, the experiments were conducted in two phases. In the first phase of experiment lethal concentrations & sub lethal concentrations of the experimental toxicant selenium dioxide were studied.96h LC 50 value was calculated by Probit analysis method (Sprague, 1973),

as 2.5mg/L. In second set of experiment the fish were exposed to sublethal concentration of toxicant selenium dioxide, which was 1/5 of the 96h LC 50 i.e.0.5mg /L. The experiments were carried out in sublethal concentration of toxicant selenium dioxide for a period of 7, 14, 21, and 28 days. Parallel sets of control fish were run simultaneously in separate aquaria.

After 7,14,21 and 28 days five males and five females fish of control as well as experimental group were weighed and sacrificed immediately by giving a blow on the head and were dissected. The liver tissue from both the control and experimental fish were removed & rinsed in a saline to remove the cell debris. Then all tissues were cut into small pieces of desirable size and fixed to aqueous bouins fixative separately. The tissues were embedded in a paraffin wax using routine technique & sections were cut at 5µ thickness and stained with haematoxyline-eosin staining method. Histological observations were made by employing light microscopy to assess the toxicant selenium dioxide effects on fish tissues.

Results and Discussion

Histopathology of Liver:

Control

The main hepatic structure includes hepatic parenchyma and blood vessels. The parenchymatus cells forming cords lie irregularly and get separated by blood sinusoids. The polyhedral liver cells bear prominent central nuclei. Each sinusoid consists of an outer peripheral connective tissue and an inner lining of the endothelial cells (fig. 1).

Experimental

In the present study, the histopathological lesions were observed in the liver of freshwater fish, *Channa punctatus after* exposure to sublethal concentration of selenium dioxide unto 28 days. Histopathological lesions began with hypertrophy of hepatocytes and disorientation of liver cords. After 7 days of exposure the hepatocytes Showed disruption of regular hepatic cordal arrangement and prominent shrinkage of hepatocytes (fig. 2).

After 14 days, the nuclei ofhepatocytes became prominent along with disarray ofhepatic cords (fig. 3). After 21 days of exposure the hepatocytes became vacuolated and blood coagulation was also observed. Shrinkage of blood vessels, clumped erythrocytes and widely separated bile canaliculi were noticed. Several other degenerative changes occurred due to acute toxicity of selenium dioxide, includes picnotic nuclei, damaged connective tissue, disorganization of hepatic cells and hepatic cords. Focal necrosis and acute haemorrhage was also prominent (fig.4).

After 28 days of exposure to selenium dioxide there was a typical change in the size of hepatocytes and their nuclei were displaced to the periphery .The cell membranes of some cells were ruptured resulting into fusion between two or more cells, exhibiting binucleate or multinucleate appearance of cells at several places. The vacuolization and clumping of hepatocytes was prominent. The entire liver tissue became necrotic spongy mass and thus a liver as a whole showed cirrhosis (fig. 5).

Cope et al.(1969) reported swollen hepatocytes, disorientation of liver cords, cytoplasmic vacuolization and karyolysis of hepatocytes of *blue gills* after exposures of dichlorobennil.

The liver is the primary organ for detoxification of xenobiotic (Metelev et al.1971). Disturbed hepatocyte with loss of normal palisade arrangement was evident in

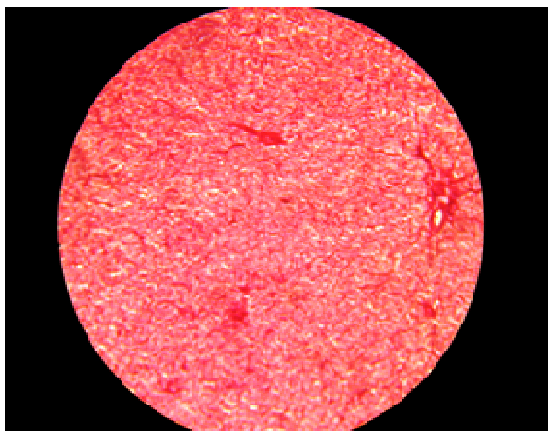

Fig.1: Control – T. S. through the liver of fish ,*Channa punctatus* (Bloch) Haemat.-eosin , x 630.

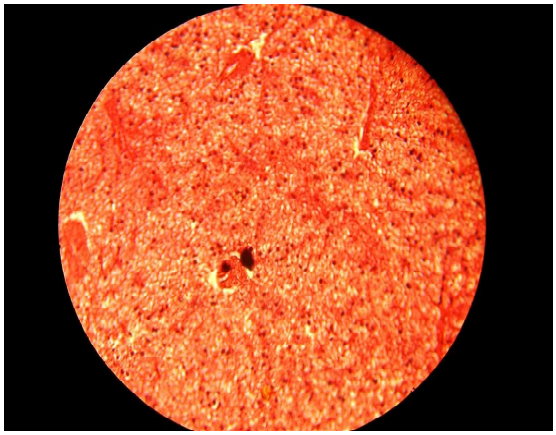

Fig.2: T. S. through the liver of fish ,*Channa punctatus* (Bloch)exposed to sublethalconc. of Selenium dioxide for 7 days Haemat.-eosin , x 630.

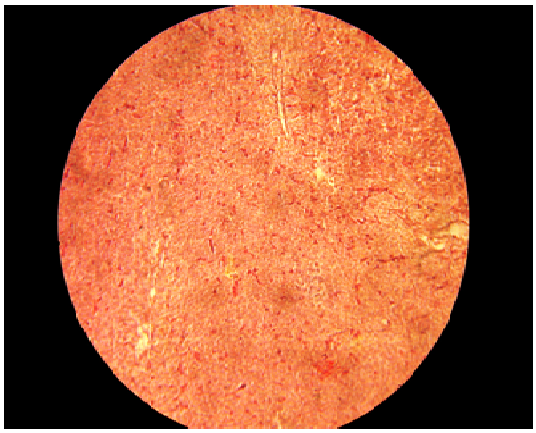

Fig.3: T. S. through the liver of fish ,*Channa punctatus* (Bloch)exposed to sublethalconc. of Selenium dioxide for 14 days Haemat.-eosin , x

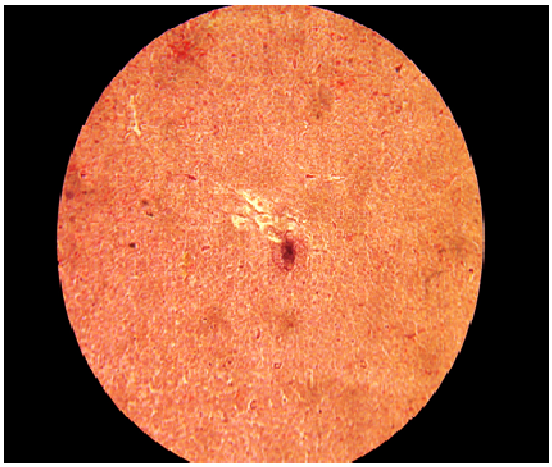

Fig.4: T. S. through the liver of fish *Channa punctatus* (Bloch)exposed to sublethalconc. of Selenium dioxide for 21 days . Hemato-eosin , x 630.

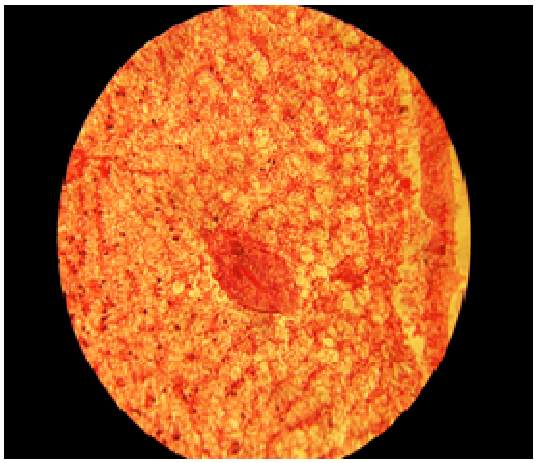

Fig.5: T. S. through the liver of fish ,*Channa punctatus* (Bloch)exposed to sublethalconc. of Selenium dioxide for 28 days . Hemato-eosin , x 630.

the liver of *Labeo rohita* with increasing exposure to the toxicant.

Infiltration of blood cells and formation of blood filled space in the liver along with disarray of cords supports our view that heavy metal cause haemorrhage in internal organs (Kendall, 1977; Nemcsok and Huges, 1988; Thakur and Pandey, 1989; Dutta, 1993; Singh and Bhati, 1994).

Kabirand Begum (1978); Narayan and Singh(1991)observed extensive degeneration of cytoplasm with pyknosis in the liver tissue of *Heteropnustesfossilis*when subjected to acute thiodan toxicity.

Banarjee and Bhattaracharaya (1997) have reported the disrupted orientation as well as vacuolization of hepatic cells in the fish, *Channapunctatus* after 7 days of exposure to toxicant elsen.

The organ most associated with the detoxification and biotransformation process is the liver and due to its function, position and blood supply(Van der Oost et al. 2003)it is also one of the organs most affected by contaminants in the water (Rodrigues and Fanta, 1998).

According to Rajamanickam and Muthuswamy (2008), the fish show various

stress responses comparable to other vertebrates.

Butchiram et al. (2009) reported alachlor induced pathological changes in the liver tissues of the fish, *Channa punctatus*, these changes includes degeneration of cytoplasm in hepatocytes, atrophy, formation of evacuoles', rupturing blood vessels, necrosis and disappearance of hepatocyte wall and dispositioning of hepatic cords.

According to Ajani and Akpoilin (2010), the vacuolization of hepatocytes may be due to inhibition of protein synthesis, energy depletion, disintegration of microtubules and shift in substance utilization caused as a effect of chronicdietary copper exposure on histology of liver of *Common carp*.

Belicheva and Sharova (2011) demonstrated strongly that the fish of Vygozero reservoir, North West Russia were under long term chronic pollution as much as accidental exposure. The pathology life hepatocellular vacuolization is known to be an acute exposure of organism, while the fibrosis of bile duct, necrosis vacuolization and cystic degeneration in the liver indicates that the long term chronic response of the fish

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