Histopathological alterations in the liver of freshwater fish, *Channa gachua* (Ham.) on acute exposure to Nickel

Sujata Kawade

Assistant Professor,
Department of Zoology,
Shri Shivaji Science College, Amravati, Maharashtra, India.

Abstract: Industrial effluent containing heavy metals, on entering aquatic environment causes histopathological disturbances in the fish. The present study deals with the toxic effect of heavy metal - Nickel (Ni) as $NiSO_4$ on the liver of fresh water fish, *Channa gachua*. Liver was examined in the 96 hours LC_{50} acute test. Histopathological examination of liver revealed marked pathological changes like shrinkage of central vein, accumulation of blood cells in the central vein, rupture of sinusoids, degeneration of hepatic tissue due to necrosis and hemorrhage in the hepatocytes and connective tissue.

Key words: Nickel, Liver, histopathology, *Channa gachua*

INTRODUCTION:

Water pollution is recognised as a potential threat to the aquatic organisms. Population explosion and rapid industrialization are the reasons for this type of pollution. The major sources of aquatic pollution are discharge of industrial effluents, fertilizers, pesticides, domestic sewage, etc. into the water bodies.

Effect of various pollutants on aquatic organisms have been studied by many workers. **Cengiz, (2006)** reported histological alterations in the gills and kidney of freshwater fish *Cyprinus carpio* after acute exposure to deltamethrin**. Santhakumar et al (2001**), reported gill lesions in the perch, *Anabas testudineus*, exposed to **monocrotophos**. **Rao, et al (2005),** reported sublethal effects of **monocrotophos** on locomotor behavior and gill architecture of the mosquito fish, *Gambusia affinis*. **Rana et al, 2015** reported histopathological study of liver and kidney in common carp (*Cyprinus carpio)* exposed to different doses of potassium dichromate.

Among the various pollutants, heavy metals have become a matter of great concern. Heavy metals are natural trace components of the aquatic environment but as they are non-biodegradable, their higher concentration may cause harmful effect on the aquatic organisms. Once discharged in the water bodies, these heavy metals bioaccumulate causing harmful effect on the organisms exposed to them (**Hollis et al, 1999**).

Many workers have reported the harmful effects of heavy metals on the aquatic environment. Effect of cadmium chloride on the histoarchitecture of kidney of freshwater Catfish, *Channa punctatus* was reported by **Amin et al, 2013.** D**rishya et al 2016** reported histopathological changes in the gills of fresh water fish, *Catla catla* on exposure to electroplating effluent. Changes in the biochemical profile including glucose, protein and cholesterol in the fresh water

fish, Cyprinus carpio heave been reported by **Kumar et al, 2011**. **Vutukuru, 2003** reported changes in the biochemical profiles of Labeo rohita on Chromium exposure. Histopathological alterations were observed in the gills and kidney of Cirrhinus mrigala fingerlings on exposure to mercury were reported by **Gupta and Kumar, 2006. Arellano et al, 2000** reported accumulation and histopathological effects of copper on the gills and liver of Sanegales sole and toad fish. **Authman et al, 2013** reported Heavy metal pollution and their effects on gills and liver of the Nile catfish Clarias gariepinus inhabiting ElRahawy drain, Egypt. **Gupta and Srivastava, 2006** reported Effects of sub-lethal concentrations of zinc on histological changes and bioaccumulation of zinc by kidney of fish Channa punctatus (Bloch).

Among heavy metals Nickel is blacklisted by the European community. It is highly toxic and nonessential. It is released into the aquatic environment by industrial sources such as Ni-Cd batteries, plating processes, refining ores, etc. Industrial effluents from these industries are a source of Nickel pollution. Nickle is considered as an important xenobiotic and a nonbiodegradable chemical pollutant of the aquatic environment. Fishes serve as biomarkers of aquatic pollution (**Adams SM. (Ed). 2002**)

Histopathology is an essential tool to study the action and fate of toxicants in the aquatic environment. **Bernet, et al (1999),** Histopathological changes also helps to access the various abnormalities caused due to the administration of pollutant or chemical and determine the order of its lethality. Study of histopathological changes due to the effect of toxicants in fishes is best suitable method to check the aquatic pollution.

MATERIALS AND METHODS:

Adult and live *Channa gachua* were collected from the local market and brought to the laboratory. Only healthy fishes, (Length-12-15 cms; Weight 50-56 gms) were taken for the experiment. Fishes were acclimatized in the glass aquaria for 15 days and were fed with fish food. Water in the aquarium was replaced after every 24 hours.

Stock solution of Nickel Sulphate (NiSO4) was prepared by dissolving appropriate amount of NiSO4 as Ni salt in distilled water. Fishes were exposed to Nickel for a period of 96 hours. Simultaneously the control group were also maintained. At the end of the exposure period, the fishes that survived, were sacrificed, dissected carefully to isolate liver and were fixed in the Bouins' fluid. The tissues were then embedded in paraffin wax, sectioned and proceeded for the Haematoxylin & Eosin (H & E) staining. The sections were examined under the light microscope (400X) and photographed using a digital camera.

RESULTS:

Figure 1 shows the normal histology of the Liver consisting of hepatic cells (hepatocytes) with a distinct central nucleus and a prominent nucleolus. The polygonal cells of liver are tightly packed. Blood capillaries known as sinusoids are irregularly distributed between the hepatocytes. The sinusoids are lined by endothelial cells with prominent nuclei.(Fig 1)

In contrast to this, the Liver of treated fish with lethal concentration of Nickel for an acute period of 96 hrs at 150 ppm (LC50) exhibited marked pathological changes as shrinkage of central vein, accumulation of blood cells in the central vein, rupture of sinusoids, loosening of hepatic tissue due to degeneration and necrosis in the hepatocytes and connective tissue, degeneration and haemorrhage in hepatic tissue. (Fig 2)

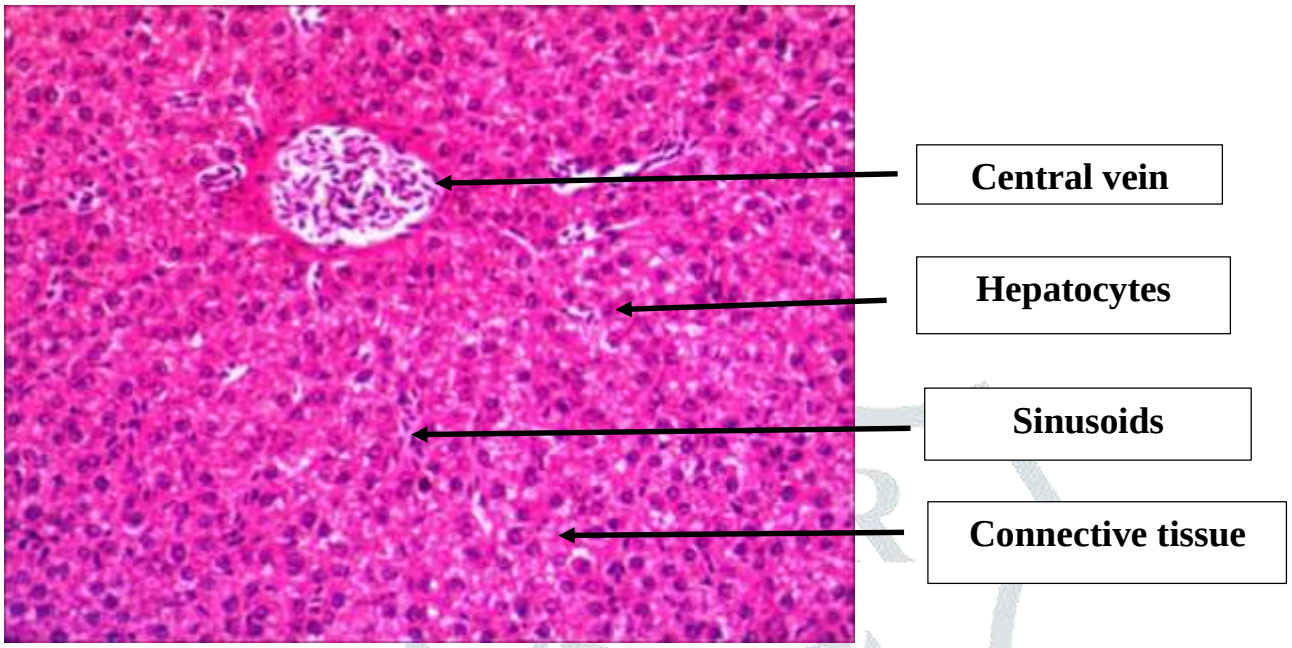

Fig 1: T. S. of Liver of *Channa gachua* (Normal) *(H/E) (400X)*

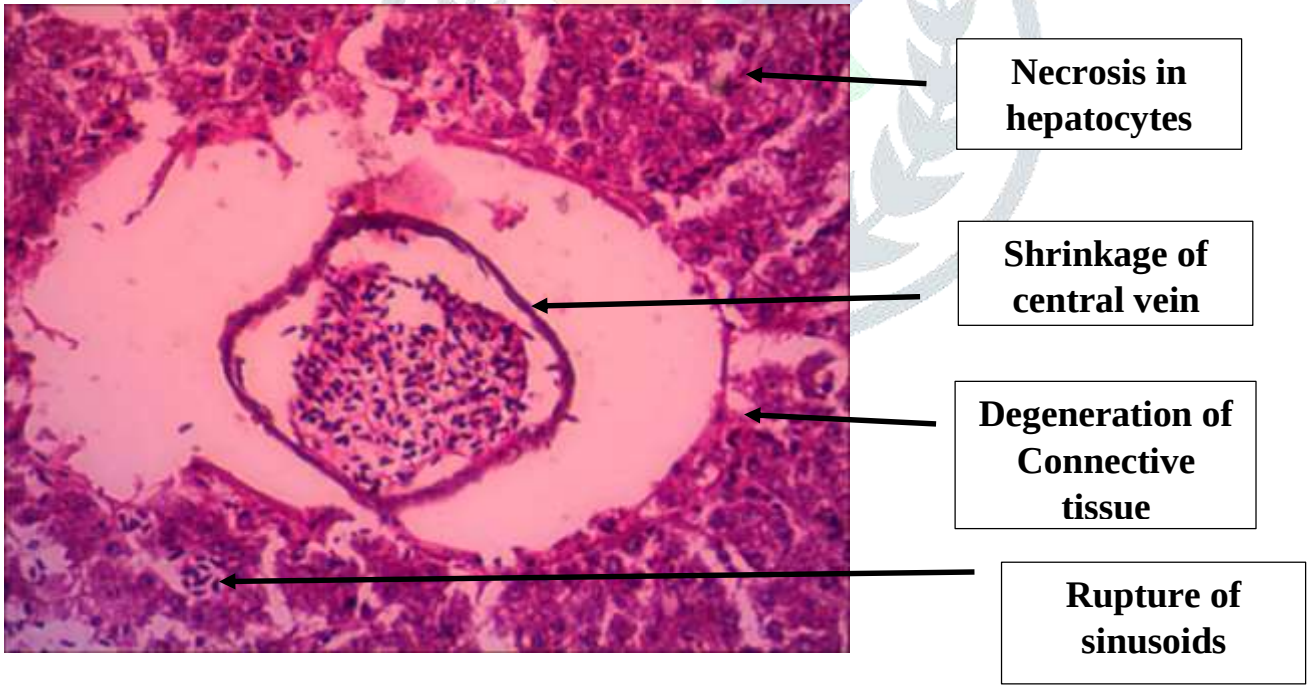

Fig 2.: T.S. of Liver of *Channa gachua* on exposure to heavy metal Nickel as NiSO4 at 150 ppm for a period of 96 hours (H/E) (400X)

Aquatic pollution due to heavy metals has become a worldwide problem due to continuous accumulation and persistency in the aquatic environment. This environmental contamination is due to a drastic increase in the urbanization and industrialization. Among the various pollutants, heavy metals have become a major constituent of the industrial effluent. Results indicate that NiSO4 poses toxic effect on fish, *Channa gachua* which is evident from the findings of the present investigations.

In the present study, histopathological changes were observed in the liver of fish, *Channa gachua*, on exposure to heavy metal Nickel as (NiSO4) for a period of 96 hrs at 150 ppm. The changes observed were marked pathological changes as shrinkage of central vein, accumulation of blood cells in the central vein, rupture of sinusoids, degeneration and necrosis in the hepatocytes and connective tissue. These investigations are in accordance with the histological alterations in the liver of Nile tilapia (*Oreochromis niloticus*) exposed to heavy metals nickel and lead reported by **Shahid et al, 2020.**

The toxicity effects of heavy metals on liver has been studied by many workers. Histological alterations like shrinkage in liver cells, degenerated nuclei of hepatocytes in the liver of *Channa punctatus* on exposure to lead were reported by **Sastry and Gupta, (1978). Shahid et al, (2020)** reported degenerative changes in the liver of fresh water fish, *Oreochromis mossambicus* on exposure to heavy metals.

These histopathological changes in the liver are due to metal ion-liver tissue interaction which lead to degenerative changes resulting in altered metabolic activity **Hinton and Lauren, (1990); Nikalje et al, (2012).** The cellular disintegration in liver is due to accumulation of heavy metal nickel which in turn affect the process of digestion, protein synthesis as well as detoxification mechanism in fish (**Sorenson et al, 1980).** These interactions lead to an unstable internal environment in fishes (**Jalaludeen et al 2012).**

Hence a scientific method of detoxification is essential to improve the health of these economic fishes in any stressed environmental conditions. It could be suggested that precautionary measures should be taken against the discharge or the treatment of this effluent before releasing it in the fresh water bodies.

CONCLUSION:

From the present study, it can be concluded that there is a need for control of this type of pollution, which can be best achieved by reduction or prevention at the source. As a conclusion, the findings of the present histological investigations demonstrated a direct correlation between heavy metal exposure and histopathological disorders observed in the target tissue.

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