Zinc Sulphate–Induced Hepatic Histopathological Alterations in The Freshwater Fish (Bloch) *Channa Punctatus*

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***Abstract*—Aquatic ecosystems are increasingly threatened by contamination from agrochemicals and industrial pollutants, which adversely affect non-target organisms, particularly freshwater fishes. Zinc sulphate, a commonly used inorganic compound, enters aquatic environments through agricultural runoff and industrial effluents, posing potential toxic risks to aquatic fauna.**

The present investigation evaluates the toxic effects of zinc sulphate on the liver histopathology of the freshwater fish Channa punctatus (Bloch) under controlled laboratory conditions. Experimental fishes were divided into control and treatment groups. The lethal concentration (LC_{50}) and sub-lethal concentration of zinc sulphate were determined as 6.6 mg/L and 3.3 mg/L, respectively. Fish were exposed to the sub-lethal concentration for 7, 14, and 21 days. Histopathological examination of liver tissues revealed marked structural alterations in exposed fish compared to the control group. The observed pathological changes included disruption of the normal hepatic cord arrangement, degeneration and shrinkage of hepatocytes, nuclear hypertrophy, and disorganization of hepatic architecture.

The severity of histological damage increased with the duration of exposure. The findings of the present study demonstrate that zinc sulphate induces significant hepatic toxicity in Channa punctatus, emphasizing its potential ecological risk and the need for continuous monitoring of heavy metal contamination in freshwater ecosystems.

***Index Terms*—*Channa punctatus*; Zinc sulphate; Histopathology; Liver; Heavy metal toxicity; Freshwater fish.**

I. INTRODUCTION

Aquatic ecosystems are increasingly subjected to contamination from industrial effluents, agricultural runoff, and domestic waste, *resulting* in the accumulation of toxic substances such as heavy metals in water bodies. Among these pollutants, zinc is an essential trace element required for normal metabolic functions; however, excessive concentrations can be toxic to aquatic organisms. Zinc sulphate is widely used in agriculture, pharmaceuticals, and industrial processes, and its indiscriminate discharge into aquatic environments poses a serious threat to freshwater biota.

Fish are considered excellent bioindicators of aquatic pollution due to their sensitivity to environmental changes and their ability to accumulate toxic substances in vital organs. The liver plays a central role in metabolism, detoxification, and biotransformation of xenobiotics and is therefore one of the primary target organs affected by heavy metal toxicity. Histopathological examination of fish liver provides valuable information on the physiological and pathological effects of pollutants at the tissue level and serves as a reliable biomarker for environmental monitoring.

Channa punctatus (Bloch), a widely distributed freshwater fish in India, is ecologically and economically important and commonly used in toxicological studies due to its hardy nature and adaptability to laboratory conditions. Although several studies have reported the toxic effects of heavy metals on fish, limited information is available regarding the histopathological impact of zinc sulphate on the liver of Channa punctatus. Therefore, the present study was undertaken to evaluate the sub-lethal effects of zinc sulphate on liver histopathology of *Channa punctatus* under controlled laboratory conditions.

Freshwater is vital for various aquatic animals and plants but the quality of freshwater has been abruptly changing due to the introduction of different chemicals including pesticides, industrial discharges, metallic pollutants etc. Aquatic systems are exposed to a number of pollutants that are mainly released from effluents discharged from industries, sewage treatment plants and drainage from urban and agricultural areas. Industrial growth is an important part of the evolution of human civilization and is vital for the development and property of any country. However, industries also often prove hazardous to aquatic life when their toxic effluents are discharged into water, more so when this is done without any pre-treatment. These pollutants cause serious damage to aquatic life (Mishra and Tripati, 2012).

In the recent years, world consumption of fish has increased simultaneously with the growing concern of their nutritional and therapeutic benefits. The American Heart Association recommended eating fish at least twice per week in order to reach the daily intake of omega-3 fatty acids. However, fish are relatively situated at the top of the aquatic food chain; therefore, they normally can accumulate heavy metals from food, water and sediments. The content of toxic heavy metals in fish can counter act their beneficial effects; several adverse effects of heavy metals to human health have been known for long time. This may include serious threats like renal failure, liver damage, cardiovascular diseases and even death.

Therefore, many international monitoring programs have been established in order to assess the quality of fish for human consumption and to monitor the health of the aquatic ecosystem (Moselhy *et al*., 2014). Zinc metal contamination definitely affects the aquatic life of the freshwater fish. In chronically exposed fish internal environment is disturbed gradually at cellular level. This poses the physiological stress inside the various fish tissues due to the progressively Zn metal accumulation in the course of time. (Kumar *et al.* 2015).

Histological study appears to be a very sensitive parameter and is crucial in determining cellular changes that may occur in target organs, such as the liver. Exposure to heavy metals may cause histological changes in the liver. Fish liver histology could therefore serve as a model for studying the interactions between environmental factors and hepatic structures and functions. It has been noted that heavy metals had a negative impact on all relevant parameters and caused histo-pathological changes in fish. Zinc is one of the most important trace elements in the body and participates in the biological function of several proteins and enzymes.

II. MATERIALS AND METHODS

2.1 Collection and Acclimatization of Experimental Fish

Healthy adult specimens of Channa punctatus of approximately uniform size and weight were collected from local freshwater bodies in the Chandur Bazar region, Amravati District, Maharashtra, India. The fishes were transported to the laboratory in aerated containers and acclimatized for 15 days in large glass aquaria containing dechlorinated tap water. During the acclimatization period, fishes were fed commercial fish feed daily, and water was renewed regularly to maintain optimal conditions. Feeding was stopped 24 hours prior to the experiment.

2.2 Determination of Lethal and Sub-Lethal Concentrations

The lethal concentration (LC_{50}) of zinc sulphate for Channa punctatus was determined using standard bioassay methods. Based on preliminary trials, the LC_{50} value was found to be 6.6 mg/L, and the sub-lethal concentration was fixed at 3.3 mg/L (50% of LC_{50}) for experimental exposure.

III. EXPERIMENTAL DESIGN

The fishes were divided into two groups:

Control Group:

Maintained in zinc sulphate–free water

Experimental Group:

Exposed to sub-lethal concentration (3.3 mg/L) of zinc sulphate

The exposure duration was 7, 14, and 21 days. Each group contained an equal number of fishes, and the experiment was conducted under laboratory conditions with continuous aeration.

IV. HISTOPATHOLOGICAL STUDY

At the end of each exposure period (7, 14, and 21 days), fishes were sacrificed, and liver tissues were

carefully dissected out. The tissues were fixed in 10% neutral buffered formalin for 24 hours, dehydrated through graded series of alcohol, cleared in xylene, and embedded in paraffin wax. Thin sections of 5–6 µm thickness were prepared using a rotary microtome and stained with hematoxylin and eosin (H&E). The stained sections were examined under a light microscope, and histopathological changes were recorded and photographed.

V. RESULTS

HISTOPATHOLOGICAL STUDIES:

- After 7, 14, and 21 days the male & female fish of control as well as experimental set were scarified immediately by giving blow on the head & were dissecting it.
- The tissue (Liver) was dissected out & rinsed in a saline to remove cell debris & blood stain.
- Then tissue (Liver) was cut into small pieces of desirable size & fix into aqueous Bovines Fluid. All possible precautions were taken to insure proper fixation of tissue.
- After fixation the tissue (Liver) was washed thoroughly under running water for 3 hours. Then it was dehydrated, clean & embedded in paraffin wax as per regular procedure.
- The section of the tissue (liver) was cut at 5µ thickness & was stained with Hematoxilin-Eosin.

Effect of sub-lethal concentration of zinc sulphate on a liver:

- The liver is the main organ for metal regulation in fish. In zinc sulphate exposed fish, the histopathological observe in liver were duration dependent. After 7 days of exposure the hepatocyte showed disruption of regular cordial arrangement and prominent shrinkage of hepatic cells observed in a structure of liver (Fig.2).
- After 14 days exposure to the zinc sulphate solution, in a liver structure shows the nuclei of hepatocytes became prominent along with disarray of hepatic cords (Fig.3).
- After 21 days exposure of fish to the toxicant, zinc sulphate noticed that the hepatocytes became vacuolated and blood coagulation also observed, shrinkage of blood vessels, clump erythrocytes and widely separated bile canaliculi were observed.
- Several other degenerative changes occurred due to acute toxicity of zinc sulphate includes picnotic nuclei and damaged connective tissue, disorganization of hepatic cells and hepatic cords. The necrosis and acute hemorrhage were also prominent, observed in a transverse section of liver (Fig.4).

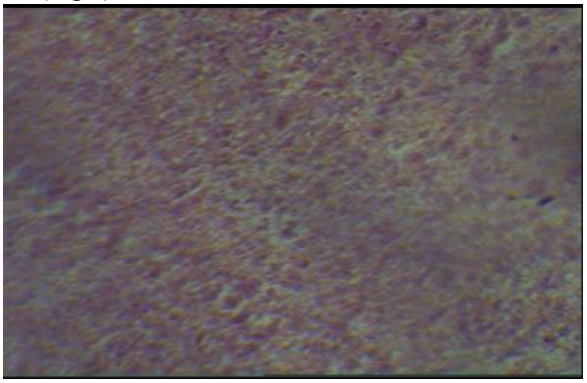

Fig. (1): T.S. Through liver of Fish *Channa punctatus* (Control) (Hematoxyline-Eosin × 630)

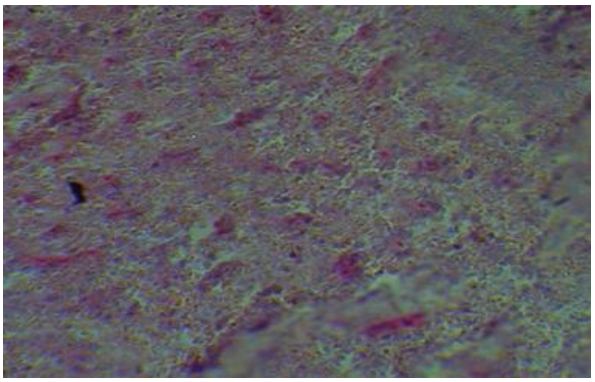

Fig (2): T.S. Through liver of Fish *Channa punctatus* Exposed to Sub-lethal concentration of $ZnSO_4$ for 7 days. (Hematoxylie- Eosine × 630)

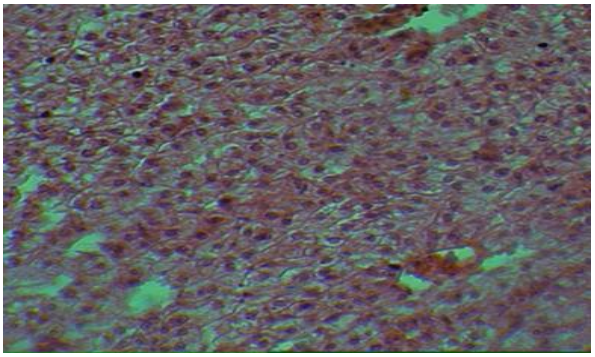

Fig. (3): T.S. Through liver of Fish *Channa pucntatus* Exposed to sub-lethal Concentration of $ZnSO_4$ For 14 days (Hematoxyline-Eosin × 630)

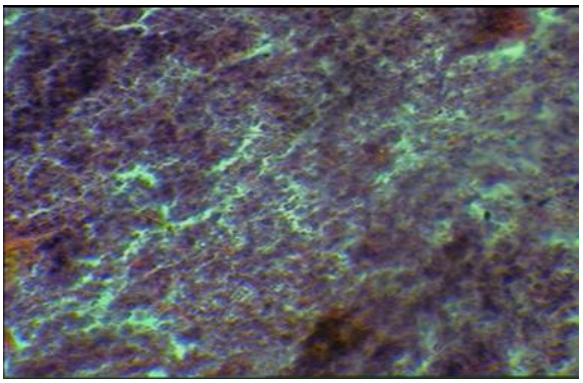

Fig. (4): T.S. Through liver of Fish *Channa punctatus* Exposed to sub-lethal concentration of $ZnSO_4$ For 21 days (Hematoxyline-Eosin × 630)

Histological examination of liver sections from control fishes revealed normal hepatic architecture characterized by well-arranged hepatic cords, polygonal hepatocytes with centrally located nuclei, and clear sinusoids.

In contrast, liver sections from zinc sulphate–exposed fishes showed marked histopathological alterations. After 7 days of exposure, mild disruption of hepatic cords and slight vacuolization of hepatocytes were observed. Prolonged exposure for 14 days resulted in degeneration and shrinkage of hepatocytes, nuclear hypertrophy, and disorganization of hepatic cords. After 21 days of exposure, severe pathological changes were evident, including extensive degeneration of hepatocytes, loss of normal hepatic architecture, prominent nuclei, and disarray of hepatic cords. The severity of histopathological damage increased progressively with the duration of exposure.

VI. DISCUSSION

The present study demonstrates that sub-lethal exposure to zinc sulphate induces significant histopathological alterations in the liver of Channa punctatus. The liver is a primary organ involved in detoxification processes and is highly susceptible to heavy metal accumulation. Disruption of hepatic cords and degeneration of hepatocytes observed in the present study indicate impaired metabolic and detoxification functions of the liver.

Similar histopathological changes in fish liver exposed to heavy metals have been reported by earlier researchers, suggesting that zinc interferes with cellular metabolism and induces oxidative stress, leading to tissue damage. Nuclear hypertrophy and cellular shrinkage observed in the present investigation may be attributed to increased metabolic activity and cellular stress caused by zinc toxicity. The progressive increase in tissue damage with exposure duration indicates a time.

Avinashe and Patil (2012), have reported that the typical change in the size of hepatocytes and their nuclei was displaced to the periphery and cell membrane of some cells were ruptured resulting into fusion between two or more cells, exhibiting binucleate or multinucleate appearance of cells at several places. Hoq and Haque (2014), at higher concentration of zinc sulphate it exerts adverse effect by occurring structural damage, which affect the growth, development and survival of fish.

Figueiredo-Fernandes *et al*. (2007), concluded from their study that the changes induced by chromium in the liver hepatocytes such as vacuolization, necrosis and nuclear condensation were also reported for copper exposure. Bhatkar (2011), the liver of *Labeo rohita*, after exposure of the fish to zinc chloride for ten days revealed swelling of hepatic nuclei, disorganization of hepatic cells with edematous hepatocytes and many cells were devoid of cytoplasmic contents.

Olurin *et al*. (2006), noticed the lesions developed in the liver might be due to the cumulative action of toxicant on blood and ultimately to other cellular structures. There seems to be a definite correlation between tissue damage and certain physiological alterations.

Ojolo *et al*. (2005) and Saxena *et al*. (2008), liver is the major metabolic center and any damage to this organ would subsequently do, so many physiological disturbances leading to subsequent mortality of fish.

VII. CONCLUSION

The present investigation concludes that zinc sulphate exerts pronounced toxic effects on the liver of the freshwater fish Channa punctatus even at sub-lethal concentrations. Histopathological alterations such as degeneration of hepatocytes, disruption of hepatic cords, and nuclear abnormalities serve as reliable

biomarkers of zinc-induced toxicity. These findings highlight the potential ecological risk posed by zinc sulphate contamination in freshwater ecosystems and emphasize the need for strict regulation and monitoring of heavy metal pollution to protect aquatic biodiversity.

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REFERENCES

[1] APHA (1998): In standard method for the examinations of water and waste water. American Public Health Association,874.

[2] Avinashe A.M. And G.P. Patil (2012): Histopathological lesions in liver of the fresh water fish *Channa punctatus* (Bloch), Expose to selenium-bi-oxide. Vidyabharati International Interdisciplinary research journal, Vol.1:11-15.

[3] Bhatkar N.V. (2011): Chromium, Nickel and Zinc Induced Histopathological Alterations in the Liver of Indian Common Carp *Labeo rohita* (Ham.). J. Appl. Sci. Environ. Manage. Vol. 15 (2): 331 – 336.

[4] Figueiredo-Fernandes A., Ferreira-Cardoso J.V., Garcia-Santons S. Monteiro S.M. Carrola J., Matos P. Fontainhas-Fernandes A. (2007): Histopathological changes in liver and gill epithelium of Nile tilapia, Oreachromis niloticus, exposed to waterborne copper. Pesqui.Vet. Bras.27(3):103-109.

[5] Heath, A.G. (1995): Water Pollution and Fish Physiology. CRC Press, USA.

[6] Hinton, D.E., & Lauren, D.J. (1990): Liver structural alterations accompanying chronic toxicity in fishes. Fish Physiology and Biochemistry, 8, 35–45.

[7] Ho E. (2004): Zinc deficiency, DNA damage and cancer risk. J. Nutr. Biochem., Vol.15: 572–578.

[8] Hoq T. and Haque N. (2014): Effects of Sublethal Concentrations of Zinc (II) Sulphate Heptahydrate (Znso4.7H2O) On Blood Glucose Level of Freshwater Fish *Heteropneustes fossilis.* International Journal of Scientific & Technology Research Vol. 3, ISSUE 5: 2277-8616.

[9] Kumar M., Ratna A., Prashad R., Trivedi S.P., Sharma3 Y.K. & Shukla A.K. (2015): Assessment of Zinc Bioaccumulation in Fish *Channa punctatus* Exposed Chronically. Global Journal of Bioscience & Biotechnology. Vol.4 (4): 347-355.

[10] Mishra B.B. and Tripathi S.P. (2012): Impact of Zinc sulphate on biochemical parameters in Reproductive cycle of *Colisa fasciatus*. International Journal of Basic and Applied Sciences Vol.1 No.3: 250-254.

[11] Moselhy K.M.E., Othman A.I., Azem H.A.E., Metwally M.E.A. (2014): Bioaccumulation of heavy metals in some tissues of fish in the Red Sea, Egypt. Egyptian Journal of basic and applied Sciences. Vol. 1: 9 7-1 0 5.

[12] Olojo, E.A.A., Olurin, K.B., Mbaka G. and A.D. Oluwemimo, (2005): Histopathology of gill and liver tissue of the african catfish *Clarias gariepinus* exposed to lead. African J. Biotechnol. Vol.4(1):117-122.

[13] Sharma, G., & Kumar, A. (2018): Heavy metal toxicity in freshwater fish: A histopathological approach. Environmental Toxicology, 33, 123–130.

[14] Shukla V., Dhankhar M., Prakash J. and Sastry K.V. (2007): Bioaccumulation of Zn, Cu and Cd in *Channa punctatus*. Journal of Environmental Biology. Vol.28(2):395-397.