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Zinc induced histological changes in brain and liver of *Labeo rohita* (Ham.)

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Abstract: *Labeo rohita fingerlings were exposed to zinc metal toxicity (5 and 10 ppm) for duration of 5 and 15 days. The histological changes were studied in brain and liver of the treated fish. The brain tissues showed enlarged pyramidal cells with extensive vacoulation while severe necrosis, haemorrhage and degeneration of hepatocytes were witnessed in the liver tissues.*

Key words: *Labeo rohita, Zinc, Histological changes.*

Introduction

Metals and numerous other chemicals from industrial operations are toxic to animals and may cause sublethal pathology of the liver, kidney, reproductive system, respiratory system, nervous system (Wilbur, 1969). Environmental stress is an inescapable component of the life of fish, compounded by the effects of adverse environmental conditions, including pollutants and land or water project developments within an area (Wedemeyer *et al.,* 1984).

Zinc has an extensive industrial use in alloys, galvanizing, pigments and electrical equipments. On a relative basis, surface drainage and atmospheric fallout are the most important inputs of zinc to aquatic environments (Spear, 1981). Though zinc is an essential trace element for organisms and plays a vital role in the physiology of living systems, higher concentrations can be toxic to organisms (Williams and Mount, 1965; Ambrose *et al.,* 1994). The present investigation was an attempt to study the histological alterations in the brain and liver of *Labeo rohita* induced by zinc toxicity.

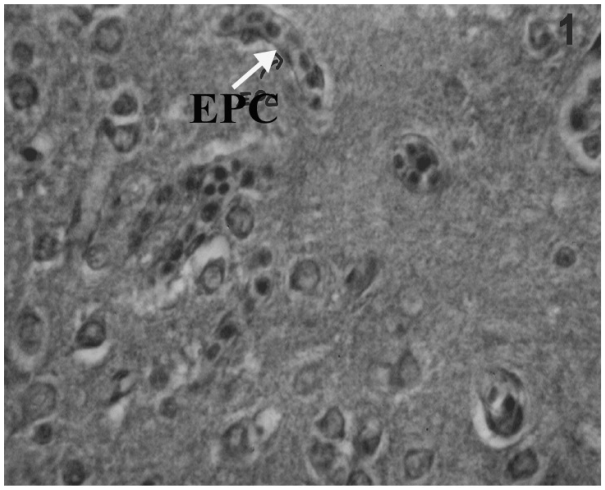

Fig. 1: Histology of zinc treated brain of *Labeo rohita* (Ham.). 5 ppm, 5th day. EPC: Enlarged pyramidal cells (Microphotograph)

Materials and Methods

Labeo rohita fingerlings were obtained from the Tamilnadu fish seed farm at Poondi reservoir and healthy fish were acclimatized in the laboratory for 10 days in glass aquarium of 50l capacity containing well-aerated unchlorinated tap water. The feeding and maintenance of the fish and the physico-chemical characteristics of water used for acclimation, control and experimentation were as per the procedure of APHA (1995).

The 96 hr LC_{50} value for zinc on *Labeo rohita* fingerlings was found to be 156 ppm. For histological studies sublethal concentrations of 5 and 10 ppm were chosen. Fish were sacrificed at the end of 5 and 15 days interval. The whole brain and liver tissues were dissected out and fixed in Bouin's fluid. The sectioning and staining were performed according to the protocol of Culling (1974).

Results and Discussion

Histological changes in brain at 5 ppm exposure showed swelling of pyramidal cells with binucleated nuclei,

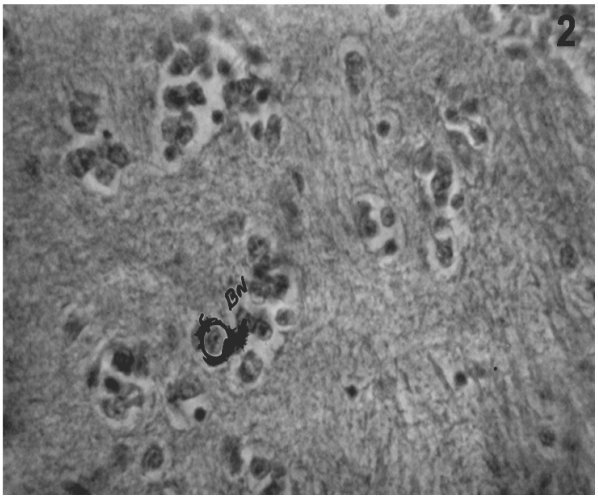

Fig. 2: Histology of zinc treated brain of *Labeo rohita* (Ham.). 5 ppm, 15th day. BN: Binucleated nuclei (Microphotograph).

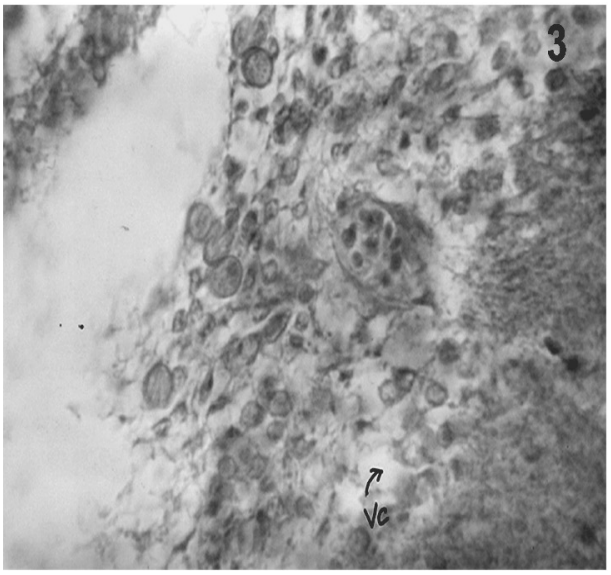

Fig. 3: Histology of zinc treated brain of *Labeo rohita* (Ham.). 10 ppm, 5th day. Vc: Vacoulation (Microphotograph).

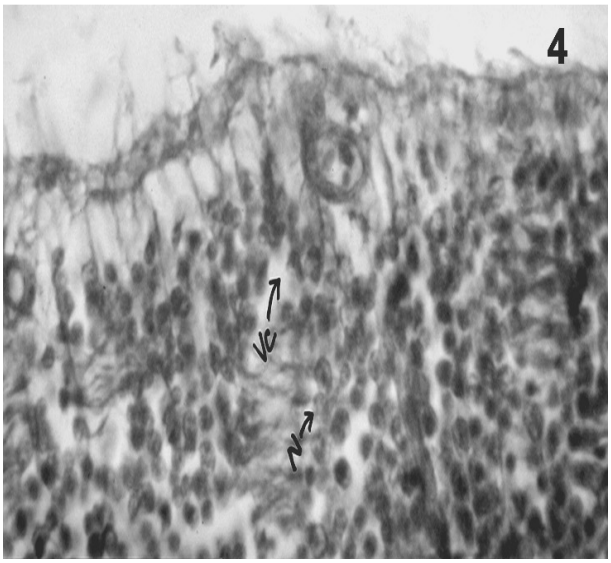

Fig. 4: Histology of zinc treated brain of *Labeo rohita* (Ham.). 10ppm, 15th day. Vc: Vacoulation, N: Necrosis (Microphotograph).

whereas at 10 ppm exposure severe necrosis of neuronal cells of cerebrum was evident, indicating loss of nissl substances. Mild vacuolar changes with empty spaces appeared due to increased concentration and duration of zinc toxicity to *Labeo rohita* (Figs. 1-4).

The histological alterations in the liver of *Labeo rohita* at 5 ppm exposure showed severe necrosis, hemorrhage, distended sinusoids with minor vacoulation. At 10 ppm exposure pyknotic nuclei were much prominent with indistinct single cells (Figs. 6-10).

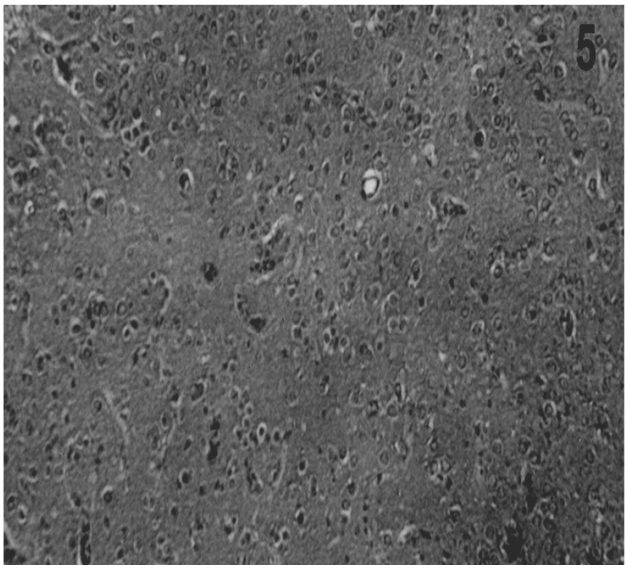

Fig. 5: Histology of control brain of *Labeo rohita* (Ham.). (Microphotograph).

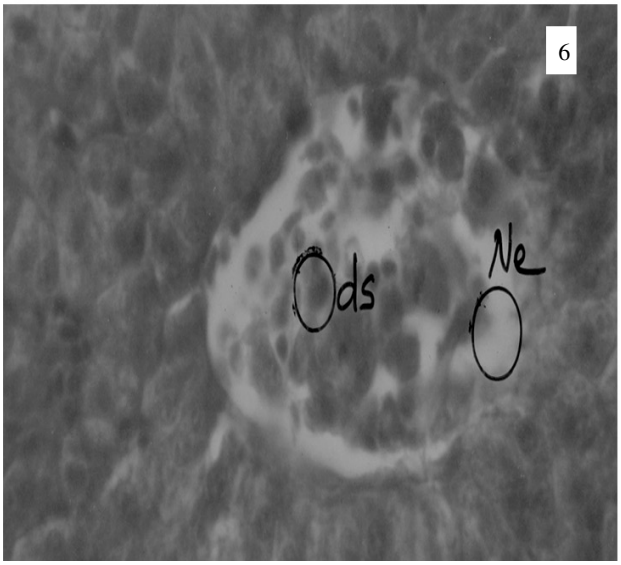

Fig. 6: Histology of zinc treated liver of *Labeo rohita* (Ham.). 5ppm, 5th day. ds: distended sinusoids, Ne: necrosis (Microphotograph)

Changes associated with the brain to sublethal exposure of zinc are not reported in the available literature. The present experimental trials revealed that zinc may also be neurotoxic, evidenced by the histological changes. Similar changes were observed by Das and Mukherjee (2000) in *Labeo rohita* exposed to pesticides and chemicals like hexachlorocyclohexane.

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

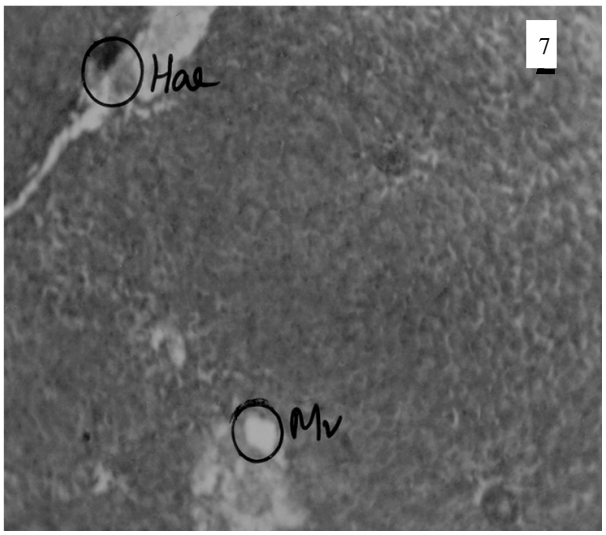

Fig. 7: Histology of zinc treated liver of *Labeo rohita*: 5ppm, 15th day. Hae. hemorrhage, Mv: minor vacoulation (Microphotograph).

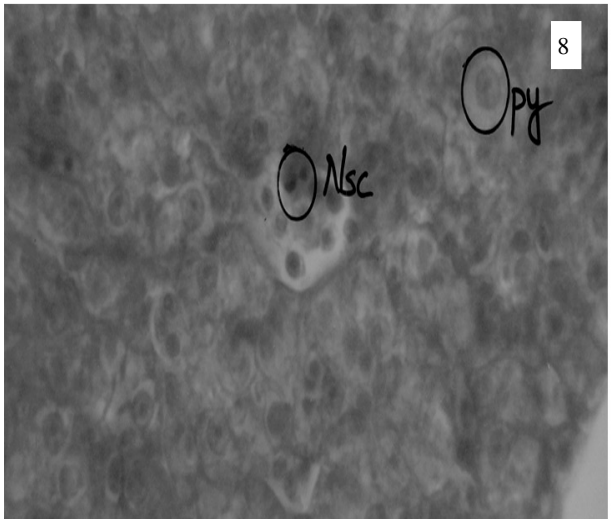

Fig. 8: Histology of zinc treated liver of *Labeo rohita* (Ham.). 10ppm, 5th day. Nsc: Necrotic single cells, Py: Pyknotic stage (Microphotograph).

Labeo rohita with increasing exposure to the toxicant. King (1962) found many small vacuoles in hepatic cells of brown trout fry and adult guppies exposed to 0.0032-3.2 ppm DDT. It appears to be a general feature of the liver of intoxicated fish that the degree of structural heterogeneity is enhanced with increasing concentration of the toxicant (Hawkes, 1980). Kabir and Begum (1978) and Narayan and Singh (1991) observed extensive degeneration of cytoplasm, with pyknosis in the liver tissue of *Heteropneustes fossilis* when subjected to acute thiodan toxicity. Similar changes were recorded in the present study with zinc at higher concentrations.

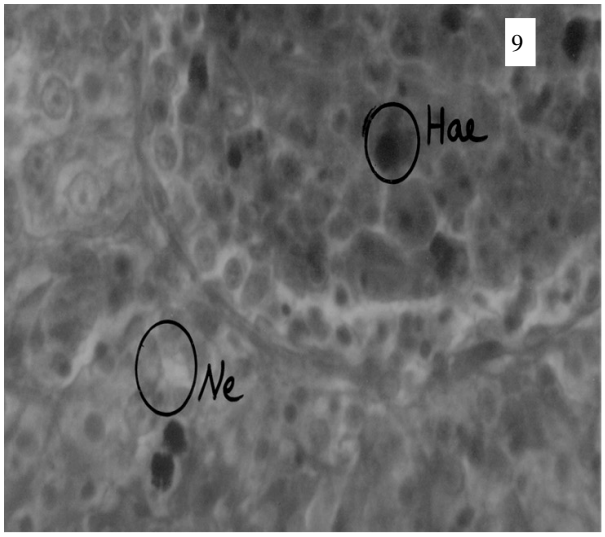

Fig. 9: Histology of zinc treated liver of *Labeo rohita* (Ham.). l0 ppm, 15th day. Hae: hemorrhage, Ne: necrosis. (Microphotograph)

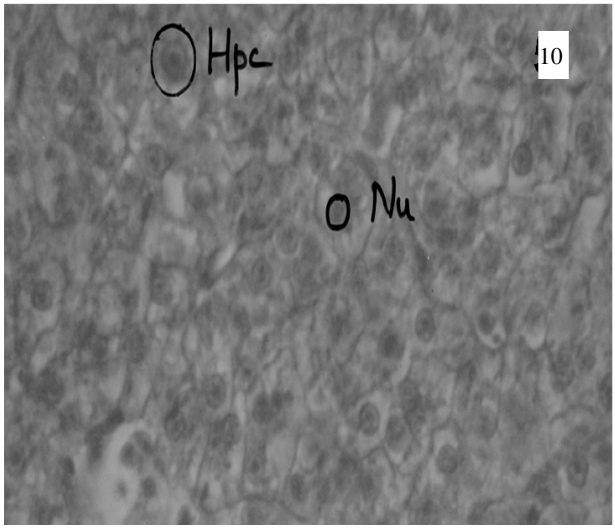

Fig. 10: Histology of control liver of *Labeo rohita*. Hpc: hepatocytes, Nu: nucleus (Microphotograph).

In conclusion sub lethal exposure of zinc led to severe damages in liver compared to the brain tissue of *Labeo rohita*.

This conforms the possibility of zinc to be a major hepatotoxicant and a minor neurotoxic agent.

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