

A STUDY ON THE HISTOPATHOLOGICAL EFFECTS OF NITRITE (NO<sub>2</sub>-N)  
ON JUVENILE COMMON CARP *Cyprinus carpio*

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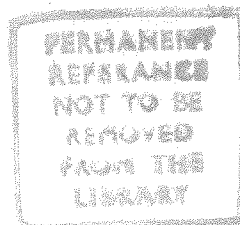
Deepthi Devika Wickramasinghe



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the Department of Zoology, University of Colombo.

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## ABSTRACT

Various chemicals originating from natural and man made sources continue to pollute the aquatic environment, some of which are harmful to fish. Although less attention has been paid to pollution of water bodies by nitrite ( $\text{NO}_2\text{-N}$ ), it is reported to be very toxic to fish. Despite the fact that no studies have been carried out in Sri Lanka, the effects of  $\text{NO}_2\text{-N}$  can not be neglected.

Natural nitrite concentrations in water bodies can be enhanced by the discharge of effluents containing nitrite and by the partial oxidation of ammonial discharges. Nitrite is believed to enter into fish body via gills. One of the major effects of it is reported to be the conversion of haemoglobin to methemoglobin which reduces its oxygen carrying capacity. Thus the present experiments were carried out to study the effects of lethal and sublethal effects of  $\text{NO}_2\text{-N}$  on fish in organs and tissues such as , gills, skin, kidney, liver and spleen.

Common carp (*Cyprinus carpio*) was used as the test species. Fish were acclimatized for seven days. Well aerated, dechlorinated tap water was used as test water. To prepare the exposure medium  $\text{NaNO}_2$  was used. During all tests temperature, oxygen concentration, pH, water hardness and ammonia concentration of the water were determined in regular intervals. The LC50 (96 hours) was determined by exposing the fish to seven  $\text{NO}_2\text{-N}$  concentrations (0.50, 1.00, 5.00, 15.00, 50.00, 100.00 and 200.00 mg/l). Eight fish were exposed to 20.3 mg/l of  $\text{NO}_2\text{-N}$  (LC50, 96hr) and the sampling was done after 24, 48, 72 and 96 hours. Sublethal effects were studied by exposing eight fish to 5.00 mg/l of  $\text{NO}_2\text{-N}$  (25% of LC50) for eight weeks. Sampling was done weekly. All tests were conducted in triplicate. Fish tissues were removed immediately after the fish was sacrificed and were preserved in 10% buffered formalin. The tissues were then processed and sectioned at 4-5  $\mu\text{m}$ . Using standard histological techniques the sections were stained by haematoxylin and eosin. The sections were studied under light microscope and photomicrographs were taken in which the histopathological alternations were observed.

Common behavioural changes due to stress were observed in fish who were exposed to the lethal level of toxicant. No significant behavioural changes were observed for sublethal exposure. Gills, skin, liver, kidney and spleen have undergone changes in the histostructure in varying degrees. These changes were apparently not much different for lethal and sublethal exposure. Nevertheless the longer the exposure more severe the changes. Gills could be stated as the most sensitive organ to the toxicant. The changes of it began form the hyperplasia of the lamellar cell and at both the lethal and sublethal exposure severe necrosis and the loss of gill architecture were observed. There is evidence to show that the gill lesions are not specific to  $\text{NO}_2\text{-N}$  toxicity. Increased number of chloride and goblet cells were evident in the skin, post exposure to toxicant. Lethal exposure to the toxicant seem to have not caused remarkable changes in the kidney. Disintegration of renal tubules and increase in the number of Melano macrophage centers were however observed post exposure to sublethal levels. In comparison, the changes in the spleen due to the lethal exposure were absent.

Sublethal exposure caused loss of tissue architecture and haemorrhage. Liver undergoes vacuolation and necrosis as a result of lethal exposure. Sublethal exposure caused pyknosis.

Nitrite induced methemoglobinemia and hypoxia may be the causes for tissue damage.

Thus,  $\text{NO}_2\text{-N}$  can be regarded as a potential toxicant which causes damage to fish tissues which sometimes may lead to death. Therefore steps should be taken to minimize the pollution of water bodies by nitrite.