

TOXICITY EVALUATION OF CHLORDECONE AND ITS EFFECT ON OXIDATIVE IMBALANCE IN THE CICHLID FISH, *ETROPLUS MACULATUS* (BLOCH)

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ABSTRACT

The aim of the present study was to evaluate the toxicity effect of chlordecone on the reactive oxygen species generation in the Cichlid fish, *Etroplus maculatus*. Chlordecone (0.35 µg/L) was used as the test dose and the fishes were treated for 24, 48 and 96 h maintaining a control group without adding the test chemical. Body weight of the treated fishes remained unchanged throughout the experiments. However, mucous deposition was significantly increased in time-dependent manner which states the defensive mechanism of the exposed fishes to chlordecone. Antioxidant parameters such as superoxide dismutase, catalase, glutathione reductase and the levels of hydrogen peroxide and lipid peroxidation were evaluated in both gill and liver tissues. Statistical analysis reports that chlordecone induced oxidative stress on gill as well as in liver by significant ($p < 0.05$) reduction in the activities of antioxidant enzymes with concomitant increase in the levels of hydrogen peroxide and lipid peroxidation. The present study also showed a significant reduction in the marker enzyme, alkaline phosphatase in gill and liver and it could be due to decreased state of inter and intracellular membrane transport and possibly this could be also due to the toxicity of chlordecone. Histopathological observations reveal chlordecone-treated gill with destruction of primary and secondary lamellae, upliftment of gill epithelium and reduction in the number of chloride cells. Similarly exposure to chlordecone critically affected the architecture of hepatocytes with enucleated cells, cytoplasmic vacuolization and hepatic necrosis. Thus toxicity of chlordecone by reducing the activities of antioxidant enzymes resulted in oxidative imbalance in the vital organs as gill and liver of the fish, *Etroplus maculatus*.

KEYWORDS: *Etroplus*, Gill; Liver, ROS, Antioxidant Enzymes, Lipid Peroxidation, Alkaline Phosphatase