HISTOPATHOLOGICAL LESIONS INDUCED BY COPPER SULPHATE IN HEPATOPANCREAS OF MOLLIE-NESIA SP.

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The study of histopathological effects of pollutants on the different organs of fishes is an important basic effort leading to our understanding of true impact of pollutants on the ecosystem because the freshwater fishes show dissimilar pattern of responses when exposed to various metals (Gardner & LaRoche 1973, Hara et al 1976, Mount 1968) Vijayamadhwan and Iwai (1975) reported that the extent of damages vary with body part, nature of metal, medium and duration of tests. In the wake of all these facts it was considered worthwhile to determine the effects of acute and chronic doses of copper sulphate, a common chemical generally used in drinking water and textile industry, on the liver of Mollie-nesia sp.

The living specimens of Mollie-nesia sp. (35 to 45 mm in length) were purchased from an aquarist, Delhi. The fishes were reared for two weeks in glass aquaria for acclimatization. For studying the effects of acute exposure, the fishes survived 24 h test (1000 μg/lit of copper sulphate) were examined; while for chronic toxicity, the fishes were kept 100 μg/lit copper sulphate solution for 25 days. The water was, however, changed every 24 h to maintain the concentration of the metal content during the period of exposure. At the end of relevant experimental period, the surviving fishes from each group were sacrificed and their liver was fixed in Bouin's fluid. The tissues were embedded in paraffin using routine techniques and sections were cut at 6-8 μm and stained with haemotoxyline and eosine.

Histology of liver

Normal: The hepatic parenchyma of Mollie-nesia sp. consists of continuous masses of cells forming cords. Blood sinuses lying frequently between hepatic cords are lined by endothelial cells. They also get covered by connective tissue. A single sinusoid often joins with the other by transverse connection. Polyhydral hepatocytes with a well-defined cell membrane bears a centrally located nucleus with dense cytoplasm. Portal vein gets surrounded by the exocrine pancreas to represent a diffused pancreas. The large pancreatic cells with dark stain are also seen (Plate I, Fig. 1).

Exposed to 1000 (μg/l copper sulphate: The liver of Mollie-nesia sp. when exposed to acute level of copper sulphate shows that the hepatocytes become
The different organs studied in this investigation show dissimilar impact of copper sulphate. LaRoche (1973, p. 40) reported that medium and duration of exposure are considered worthwhile to test copper sulphate, a common contaminant on the liver of fishes.

The fishes (2 cm in length) were exposed for two weeks in glass beakers. After exposure, the fishes were examined, while for some fishes exposed to copper sulphate solution for one week, the concentration at the end of the experiment group were sacrificed and specimens were embedded in paraffin wax, sectioned, stained with haematoxylin and eosin (H & E).

Continuous masses of connective tissue between haptic cords were observed. Polyhydral nuclei are observed in the exocrine pancreas to which dark stain are also observed. Mollienesia sp. when exposed to copper sulphate becomes

**FIG. 1.** (above) Microphotograph of the T.S. of normal liver of Mollienesia sp. x 400

**FIG. 2.** (below) T.S. of liver exposed to acute level of Copper Sulphate

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Microphotograph of T. S. of liver of *Molliesia* sp. exposed to 100 μg/l copper sulphate. x 400

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swollen, due to condensed cytoplasm. The portal vein undergoes partial coagulation of blood. The pancreatic acinar cells lying vicinity of veins become more affected than those of hepatocytes. The cordal arrangement seems to be crumpled due to swollen and stretched hepatocytes. The nuclei of hepatocytes become large (Plate I Fig. 2).

Exposed to 100 μg/l copper sulphate: The vacuolisation of hepatocytes causes serious effects resulting total disruption of hepatic cords. Some of the hepatocytes become completely, while others are partially vacuolated. This leads to forced shifting of nuclei towards periphery and may, therefore, be due to precipitation of cytoplasm. Nuclei get shrunk. The sinusoids also undergo disintegration. The hepatocytes in the vicinity of sinusoids are liable to more toxic hazards than the central ones (Plate II).

Here the hepatic lesions in general are characterised by the destruction of cytoplasmic material of hepatocytes and consequent vacuolisation. A gradual damage is observed in those which are exposed to longer-duration test, showing significant necrosis of hepatocytes and coagulation of blood in sinusoids. Vijayamadhwan and Iwai (1975) suggested that the damages in tests bud of Carassius auratus are mainly due to the permeation of metal. In Mollinesia sp the higher extent of damages in hepatocytes lying in vicinity of sinusoids is probably due to toxic substances present in the incoming blood. Baker (1969) also reported that copper initially induces haenolytic anaemia in pseudopleuronectes americanus and further changes in liver are secondary effects.

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