Histopathological changes in liver of *Heteropneustes fossilis* exposed to cypermethrin

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Abstract: Cypermethrin was administered to *Heteropneustes fossilis* in chronic concentration to determine lesion of liver as indicators of tissue damage. The cypermethrin dose used was ¼ of 96 hr LC₅₀. Histopathological changes in liver ranged from vacuolization, necrosis, fibrosis of perivascular region and disposition of yellow brown grains at different time of exposure viz; 20°, 30°, 40° and 60° days

Key words: *Heteropneustes fossilis*, Liver, Cypermethrin, Histopathology

Introduction

The pyrethroid pesticide cypermethrin is used extensively to control pests. Bradbury and Coats (1989) observed that pyrethroid insecticides are extremely toxic to fish during aqueous exposure. Pyrethroids are readily metabolized and have relatively short half life. Liver is an established organ in fishes (Braunbeck and Volke, 1993) and plays an important roles in uptake, accumulation biotransformation and excretion of xenobiotics (Couch, 1975; Gluth et al., 1985; Pentreath, 1976; Kohler, 1990). Functional changes are known to be reflected in structural changes of the organ. While most of studies are restricted to the mortality and growth rate of fishes, only a few studies have focused on their effect on different organ system. However, histopathological derangements in fish on account of cypermethrin poisoning are still lacking. The present paper reports histopathological changes in the liver as a result of exposing *Heteropneustes fossilis* to sublethal concentration of cypermethrin.

Materials and Methods

The liver histopathology of cypermethrin (commercial grade 25% EC) was studied by static bioassay using tap water as dilution medium, having pH 7.20 and hardness of 200ppm as CaCO₃. The hardness was measured according to APHA (1995). Prior to conducting the bioassay for histopathology, a toxicity bioassay was run in the same water to estimate the 96hr LC₅₀ value of cypermethrin for *H. fossilis* and the same was found to be 0.046 ppm. The ¼ of LC₅₀, i.e., 0.012ppm was used in the sublethal bioassays, the sublethal concentration was, however, changed every third day. In all, 60 healthy fish, 6.00±0.5cm in length were acclimatized to laboratory conditions for 7 days, before their use in bioassays for liver pathology. They were then divided into 2 groups of 30 fish each constituting control and sublethal bioassay sets. The liver pathology was examined after 20°, 30°, 40° and 60° days in 5 treated specimens each time. An equal number of control fish were examined at similar intervals for histological comparison. The liver was removed and fixed in Bouin’s solution and then processed for microtome sectioning at 8µm and stained with hematoxylin and eosin and silver impregnation and mounted in DPX.

Results and Discussion

The histopathological changes were more evident in specimens exposed to cypermethrin and were not observed in the control fish. The liver cells in *H. fossilis* are polygonal containing spherical central nucleus (Fig.1). After 20 days of exposure the hepatocytes became irregular and lose their polygonal shape. Some cells exhibited cloudy swelling, their contour becoming indistinguishable. There were many regions in the liver where cells were highly vacuolated. Many cells had exhibited pycnosis (Fig. 2 and 3). At the end of 30 days treatment, pronounced structural changes in hepatocytes such as focal necrosis, pycnosis and darkly stained specks of necrotic nuclei were observed (Fig.4). After 40 days of exposure, intensive vacuolation in cytoplasm and pynotic nuclei were also observed (Fig.5). After completion of 60 days treatment, extensive vacuolated pycnosis and necrosis were highly evident at some points and likely initiation of fibrosis was also observed. The silver impregnation of liver cells, indicate perivascular fibrosis (Fig. 6 and 7).

The majority of insecticides are biotransformed in metabolites by liver through various enzyme systems and as a consequence of this process, liver undergoes different levels of damages. The cypermethrin induced hepatocyte pathologies are same with those reported by earlier workers under influence of different pesticides (Amminikutty and Rege, 1977; Mandal and Kushershtha, 1980; Kumar and Pant, 1981).

Shakoori et al. (1988) observed hypertrophy of hepatocytes in rodents after administration of cypermethrin in diet over a period of 6 months. Ahmed et al. (1989) reported extensive degenerative changes such as cloudy swelling, fatty...
Fig. 1: Showing liver of control fish; hepatocytes (single head arrow), epithelial cell (double head arrow) H and E, 1600x

Fig. 2: After 20 days of exposure, liver shows cloudy swelling (single head arrow), focal necrosis (black arrow) and hypertrophy of hepatocytes. H and E, 400x

Fig. 3: Shows vacuolated hepatocytes with pyknotic nuclei (single head arrow) H and E, 600x

Fig. 4: After 30 days, liver shows granulated cytoplasm (head of arrow), normal hepatocytes (single head arrow) and pyknotic nuclei 600x

Fig. 5: After 40 days of exposure, liver shows the pyknotic nuclei (single head arrow), vacuolation of cytoplasm (double head arrow), RBC (block arrow). H and E.600x

Fig. 6: After 60 days of exposure, liver exhibits extensive vacuolation of hepatocytes (single head arrow), pyknotic nuclei (double head arrow) and necrosis. 600x
Histopathological changes in liver of fish exposed to cypermethrin

Fig. 7: Showing fibrosis in portal areas. Silver impregnation 400x degeneration and necrosis of hepatocytes in rats exposed to sub acute dose of cypermethrin. Similar observations were also made by El-Toukhy and Girgis (1993) and Luty et al. (2000) after administration of larvin and cypermethrin to mice. Shrunken and pyknotic nuclei indicated that cells became hypo functional and at the end, necrosis was extensive. The stagnation of bile inside the hepatocytes signifies affected metabolism (Fanta et al., 2003). Couch (1975) reported perivascular lesions in liver of fishes exposed to organic contaminants and pesticides. According to Gingerich (1982) the vacuolization of hepatocytes might indicate an imbalance between rate of synthesis and rate of release of substance in hepatocytes. In this study, all effects that were observed in the liver reduce the general state of health of H. fossilis at sublethal concentration. It may therefore, be said that a sublethal concentration may be safe however, it can not be used indiscriminately.

References
Amminikutty, C.K and M.S. Rege: Effects of acute and chronic exposure to pesticides, thiodan 35 EC and agalol 3 on the liver of widow tetra.