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Histopathological Changes in Liver and Kidney of *Mus booduga* Following Oral Benzenehexachloride (BHC) Feeding

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The organochlorine (OC) insecticides have been of interest for years because of their insecticidal properties and their potential toxicological activities in a wide variety of organisms including man. These insecticides which are ubiquitous in nature have become an integral part of the tissues of animals (Gupta et al., 1982). Prolonged exposure to chemical compounds with very low concentrations result in the accumulation of more pesticide in the organs (Dikshith et al., 1973). Toxicity tests that include histopathological evaluation were reported for OC insecticides (Barros and Saliba, 1978; Dikshith et al., 1978; Shivanandappa and krishnakumari, 1981).

Compounds transported by oral feeding can be distributed to all parts of the body in their unmetabolised form (Turner and Shanks, 1980). Susceptibility to chemical injury varies greatly in the tissues of the same animal. For this reason two different tissues namely, liver and kidney were studied. In view of the value of histopathology in toxicity tests, the present study is carried out in the mice, Mus booduga.

MATERIALS AND METHODS

Adult mice, Mus booduga were acclimatised to laboratory conditions for 10 days before pesticide treatment. Mice in the weight range of 10-12 g were selected for the experiment. Technical grade Benzenehexachloride (BHC: 1,2,3,4,5,6-Hexachlorocyclohexane) obtained from Mico Farm Chemicals, Madras, India was dissolved in a minimum volume of corn oil and was administrated orally at a dose of 50mg/kg b.w. daily for 1, 5 and 15 days. The controls were given isovolumetric amount (0.02 ml)

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of corn iol.

After the stipulated period, liver and kidney tissues from experimental and control animals were isolated and rinsed in physiological saline to remove debris and fixed in 10% formalin for 24h. subsquently the tissues were washed in running tap water for 12h, dehydrated in alcohol, cleared in methyl benzoate and embedded in paraffin wax (paraffin embedding method). Sections of 5-6 u were cut in rotary microtome and stained by Hematoxylin and Eosin (Harris, 1900) in order to observe histological changes.

RESULTS

Lesions observed in liver:

1 Day : Congestion of portal vessels and central vein was observed. The hepatic cells surrounding the portal areas were swollen and revealed parenchymatous degeneration (Fig. 1) and in some vacuolar degeneration was observed.

5 Days: Severe congestion of central vein and portal vessels were noticed. Severe fatty changes were conspicuous in periportal cells (Fig. 2). The centrolobular cells revealed mild fatty changes.

15 Days: Congestion of central vein and portal vessels were noticed. The periportal hepatocytes and the centrolobular cells revealed only granular degeneration.

Lesions observed in kidney:

1 Day: Congestion of blood vessels and glomerular tufts (Fig. 3) were seen

5 Days: The tubules were swollen and revealed fatty changes. In a few cases interstitial haemorrhages in the medulla were noticed. Cystic dilation of the tubules were evident. Hyaline casts were seen in the tubles (Fig. 4).

15 Days: Congestion of blood vessels and glomeruli were noticed. The epithelial cells in the glomeruli revealed vacuolation. The tubules still showed fatty changes. Cystic dilation of the tubules and interstitial haemorrhages were still conspicuous.

DISCUSSION

From the above results it is evident that BHC induced marked histological changes in both the tissues namely

liver and kidney at all periods of pesticide treatment. The extent of liver damage observed in the present investigation indicates that chronic exposure always causes impairment to the architecture of the tissue. since liver is involved in detoxification of pesticides (Hurtterer et al., 1969) it is susceptible to a greater degree of disruption in its structural organisation due to toxic stress. Necrotic changes around porta hepatis characterizing a perilobular necrosis in animals poisoned with 900 ppm dietary BHC were reported (Barros and Saliba, 1978). Apart from this Dikshith et al., (1978) observed swelling of hepatocytes, congestion of blood vessels of the protal triad and mild proliferation of fibroblasts around the bile ducts. Hepatocyte hepertropy and hyperplasia, balloning cells (couchman bodies) and vacuolization in periportal and centrolobular areas were also reported in rats treated with 750 and 1500 ppm BHC (shivanandappa and Krishnakumari, 1981). The various changes noticed in the present study might be attributed to the action of BHC on liver cell membranes, microsomes and mitochondria (Shmanandappa and Krishnakumari, 1981; Yunzhen and Yixiang, 1984).

Interesting alterations were also noticed in the kidney of BHC fed mice. As kidney forms the main organ of excretion the changes noticed are quite prominent. Earlier King (1962) reported congestion of the kindney tubules and necrosis of tubular epithelium following DDT treatment. Hyaline degeneration of the nephrocytes resulting in the structural destruction of the convoluted tubules were also reported (Barros and Saliba, 1978). Marked degenerative changes in the renal tubules were observed in animals treated with DDT (Buhler et al., 1969).

The proximal tubule in mammals and fishes is involved in reabsorption and lysosomal degradation of macromolecules (Hickman and Trump, 1969). After reabsorption, more macromolecules may form intracellular droplets or dense bodies in higher vertebrates (Rollason and Brewer, 1984). During excretion the pesticides passed through kidney and appears to have caused considerable damage to this tissue. The lesions noticed in the present study indicate that BHC may be mildly nephrotoxic.

The changes observed in the present study are due to chronic exposure of mice to low concentration of BHC. the histological studies clearly demonstrate that the tissue structural integrity is disrupted to a large extent indicating that BHC even in sublethal concentration can cause deleterious effects.

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