CHAPTER VI
Modern agricultural practices have reached a stage where a halt in the use of these pesticides would mean to reduce the agricultural production. The organo chlorine insecticides which are ubiquitous in nature have become an integral part in the tissues of animals. Recognition of the incorporation of the parent compound or their metabolites in lower organisms in the tissues of
fishes, birds, wild animals and humans was recorded to cause serious morphological alterations in vital tissues of the organisms even at very low levels (Amminikutty and Rege, 1977; Dikshith, 1978; Jayantha Rao, 1982; Madhu, 1983; Girija moses and Jayantha Rao, 1985; Radhaiah et al., 1986; Nadamuni Chetty et al., 1988; Vijay Joseph and Jayantha Rao, 1990).

In general, any chemical substance can cause damage/injury to an animal, if taken beyond the safe level. The liver is a main organ for pesticide accumulation to a greater extent (Edwards, 1973). Johanson (1973) and Verma et al. (1974) pointed out that a prolonged period of exposure to chemical compounds with very low concentration results in the accumulation of more pesticide in the organs.

Compounds that enter the body via the intestinal lymphatic system after oral feeding, by pass the liver, accordingly they are not subjected initially either to the detoxifying reactions of the liver or to excrete via the biliary system. In effect, compounds transported by oral feeding can be distributed to all parts of the body in their unmetabolised form (Turner and Shanks, 1980).

Histological alterations in the tissue of BHC poisoned rats have been studied to some extent (Barros and Saliba,
1978; Dikshit et al., 1978; Shivanandappa et al., 1981).

(The investigation of histopathological effects have not been pursued with the same vigour compared to biochemical aspects) A large number of reports in the toxicity of organo chlorine insecticides to various animal species have been published (Pawar and Katdare, 1984; Zaidu et al., 1985; Anand et al., 1986; Fox et al., 1986). Histopathological changes in different organs of rat, fish and mouse were reported after acute and chronic sublethal exposure to endosulfan) (Satyaprasad, 1983; Kulshrestha, 1984). Some more workers reported on organo chlorine insecticides and pointed out architectural damage to the gill, kidney, liver and intestine of various animals (Madhu, 1983; Philip, 1984; Radhaiah, 1985; Girija Moses, 1987). Somasundaram et al. (1978) pointed out that dieldrin caused histopathological changes in the skin and increased liver protein. Reports on tumorogenic potential of endosulfan in mouse are un equivocal (Ionnes et al., 1969; Thorpe and Walker, 1973). Many scientists reported on histopathological studies under insecticidal and herbicidal treatment in different animals (Kim et al., 1985; Ehan et al, 1986; Korolev et al., 1986; Anthony et al., 1987; Ozata and Atalay, 1987; Ramalingam, 1988; Sonnenschein et al., 1989; Varshneya et al., 1989).
The histopathological studies would help in assessing the extent of pollution in the ecosystem or animal by pesticides and offer an exceptional opportunity to detect the effect of pollutant in various organs and organ systems of an organism. To have a clear understanding, as to how these chemicals cause injury to the tissue, it is essential to have an insight into the histopathological analysis of the tissues. Thus these histopathological studies have a way for understanding the pathological condition of the animal (Jayantha Rao, 1984). In view of this an attempt was made to study the toxic effects of chlordane under sublethal treatment in albino rat.

OBSERVATIONS:

The liver of rat comprises a continuous mass of hepatic cells (hepatic parenchyma) arranged in cords. There is no clear division of the hepatic cells into lobules as in the case of other mammals. The hepatocytes are large in size and possessing centrally placed nuclei. The pancreatic tissue is distinct with a well organized cellularity and a large number of blood sinusoids in the hepatic mass (Figure - 16).
HISTOPATHOLOGICAL LESIONS IN LIVER UNDER CHLORDANE INTOXICATION:

The microscopic observation of the liver sections of the experimental animals showed hypertrophy of hepatocytes (Figure - 17) and moderate degenerative changes in liver cells of single dose administered rats.

In the case of double dose chlordane administered rats clear cloudy swelling (Figure - 19), necrosis and pycnotic nuclei in hepatocytes (Figure - 20) and moderate nuclear degeneration (Figure - 21) were evidently seen in the liver.

Most of the rats administered with multiple dose of chlordane exhibited rupture in the blood vessel (Figure-22), edematous condition and nuclear degeneration (Figure - 23) and binucleated nature of hepatocytes and pushing of nuclei were commonly observed. However, the severity in the damage of the liver was more in multiple dose administered rats rather than single and double dose.

DISCUSSION:

It is clearly indicated that the chlordane induced pronounced pathological changes in liver of rat exposed to single, double and multiple doses (Figures 17 to 24).
Severe architectural lesions were observed in multiple dose administered rats. Since the liver is the major metabolic center to detoxify the pesticide, it was adversely affected with hypertrophy of hepatocytes, degenerative changes, cloudy swelling, necrosis and pycnotic nuclei, pushing of nuclei to the periphery of hepatocytes, emptied hepatocytes and damage in the blood vessels of liver (Figures 17 to 24).

A few reports pertaining to pathogenesis in rats, mice and other non-target organisms with different pesticides were also documented earlier. Phillip (1984) reported congestion of portal vessels and central vein, swollen periportal hepatic cells showing parenchymatous degeneration, severe fatty change in periportal cells in field mice treated with B.H.C. Conspicuous changes in the cytoplasm such as increased peripheral granulation, plasma inclusions, cytoplasmic hyalinisation and cytoplasmic vacuolation have been observed in the liver of rats treated with B.H.C. and lindane (Muralidhara, 1981). Exposure of male rats to parathion (2.6 mg/kg), lindane (17.6 mg/kg) and their combination through oral incubation daily for a period of 90 days induced swelling, focal necrosis and degenerating nuclei in hepatocytes besides congestion of blood vessels of portal triads and mild proliferation of fibroblasts around
Repeated administration of ethyl parathion (50%) and methyl parathion plus DDT (30%) over a period of 90 days produced pathological lesions in the testis and liver of albino rats (Datta and Dikshith, 1973). Degenerative changes of seminiferous tubules showed oedema, presence of multinucleated giant cells and enlarged interstitium. Hepatic damage included as sinusoidal congestion, cellular vacuolation and foamy appearance. Inflammation of bile duct epithelium and irregular necrosis occurred. Baker et al. (1972) reported proliferation of smooth endoplasmic reticulum as well as liver cell enlargement and bile stasis with mirex administration.

Intraperitoneal administration of diazinon (21.6 mg/kg) showed feathery degeneration in liver and some of the hepatocytes were shown to possess pycnotic nuclei in rats (Dikshith et al., 1975). Anthony et al. (1987) reported vacuolation and lipid accumulation in rats administered with diazinon. Mean lethal doses of methyl parathion also induced severe histopathological changes in rat liver (Sonnenschein et al., 1989). A single sublethal dose administration of vacor, a rodenticide caused congestion, dilatation of sinusoids and necrosis (Usha devi and

the bile ducts (Dikshith et al., 1978):
Similar changes have also been reported following zinc phosphide poisoning on rats (Johnson and Elbert voss, 1952) and subchronic administration of dieldrin on rabbits (Hurkat, 1978).

Ramalingam et al. (1988) reported severe histopathological changes on administration of DDT in chickens. These lesions include degenerative changes and vacuolation in liver, disruption of absorptive layer of intestine and severe damage in renal tubules of kidney.

Sublethal concentration of aldrin induced vacuolation, disintegration of cytoplasm, necrosis in the liver of frog, Rana hexadactyla (Vijay Joseph, 1989).

Various histopathological changes were reported in different fishes exposed to various pesticides (Bhattacharya et al., 1975; Jayantha Rao, 1982; Madhu, 1983; Prasada Rao, 1987; Radhaiah, 1988; Kulashrestha and Jauhar, 1984). These changes include cloudy swelling, peripancreatic edema, necrosis, pushing of nuclei to the periphery of hepatocytes, pycnotic nuclei binucleated condition and degeneration of cytoplasm in hepatocytes.

However, the increase in the liver fresh weight (data not included) in the present investigation, hyper trophy of
hepatocytes, increase in the glutamine-S-transferase (see Chapter IV) of the liver could possibly be due to a compensatory adaptive mechanism.

The results suggest that the chlordane totally incurs a deficit in liver function and causes necrosis of the organ. It is interesting to note that sublethal chlordane toxicity can cause a serious dysfunction of liver. On extrapolation to field trials, these effects if occurred in wild animals including human beings would often evoke a serious concern among conservationists. Therefore great care should be taken while repeated spraying of such chemicals in fields.
FIGURE - 16

Microphotograph of control rat liver (H & E) x 50 showing hexagonal hepatocytes (H) with centrally placed nucleus (N).

FIGURE - 17

Microphotograph of single dose chlordane administered rat liver (H & E) x 50 showing hyper trophied hepatocytes (HH).
FIGURE - 18

Microphotograph of double dose chlordane administered rat liver (H & E) x 50 - showing cloudy swelling (CS) of hepatocytes and moderate cellular degeneration (CDG) in hepatocytes.
FIGURE - 19

Microphotograph of double dose administered rat liver (H & E) x 100 showing cloudy swelling (CS) and cellular degenerative changes (CDG); Ruptured hepatocytes (RH) and onset of cytoplasmic degeneration (CYD) at higher magnification.

FIGURE - 20

Microphotograph of rat liver under double dose chlordane administration (H & E) x 100 - showing necrosis in hepatocytes (NH); pycnotic nuclei (PN).
FIGURE - 21

Microphotograph of rat liver under double dose chlordane administration (H & E) x 100 - showing nuclear degeneration (NDG) in hepatocytes; pycnotic nuclei (PN).
Microphotograph of multiple chlordane dose administered rat liver (H & E) x 200 - showing ruptured blood vessel (RBV), cellular disorganization (CDO).

Microphotograph showing multiple chlordane administered rat liver (H & E) x 200 - showing nuclear degeneration (NDG), Binucleated condition (BNC) and edematons condition (EC) in hepatocytes.
FIGURE - 24

Microphotograph of multiple chlordane administered rat liver (H & E) x 200 - showing pushing of nuclei to periphery (PNP) of hepatocytes, Nuclear degeneration (NDG); Nuclear fragmentation (NF) and binucleated condition (BNC).
Fig. 24