CHAPTER-7. HISTOPATHOLOGICAL ABNORMALITIES OF SOME TISSUES OF *A. testudineus* ON EXPOSURE TO OPME

7.1 INTRODUCTION:

Adverse effect of toxicants leading to histopathological changes on the tissues for longer period of time serves to identify the cause & possibly the causative agent of death. Industrial effluents, pesticides and heavy metals bring about a number of histopathological abnormalities in different tissues of fishes. Several workers (Benoit et al., 1976; Hawkins et al., 1980; Rhodes et al., 1985; Dutta & Sinha, 1988) have reported histopathological disorders in important tissues of fishes, such as liver, kidney, gill, testis, ovary and intestine on exposure to cadmium. Since gills of fish are exposed to the aquatic environment, they are extremely susceptible to the aquatic pollutant & any adverse effect is easily reflected in the structure of gill. Das & Kaviraj (1994) observed complete necrosis and sloughing of secondary lamellar epithelium of the gill of common carp treated with cadmium. Pandey & Saxena (1992) reported histopathological changes in kidney & intestine of *Labeo rohita* fingerlings due to copper intoxication. Several workers have reported morphological changes in the liver, kidney & testis due to various pesticide intoxication (Sarin & Saxena, 1978; Dikshith et al., 1980). In
Plate 4 — Photomicrograph showing gill lamellae of *A. testudineus* after 90 days' exposure to 5% OPME x 100

- b. Disintegrated gill lamellae.
- c. Clumping of epithelial cells.
Plate 5 – Photomicrograph of T.S. of intestine of *A.testudineus* after 90 days exposure to 5% OPME x 100

a. Serosa.

b. Villi
Plate 6 – Photomicrograph of Section of Liver of *A. testudineus* after 90 days exposure to 5% OPME x 100

a. Space.
b. Hypertrophy
mucous secreting cells. There was breakage of mucosal epithelial cells of villi in the intestine of *A. testudineus* exposed to OPME (5%) for 90 days (Plate- 5)

After 90 days exposure to 5% OPME the hepatic cells of a *A. testudineus* were found to be ruptured & vacuolated. Necrosis of hepatic cells & hypertrophied intralobular vein was observed (Plate -6)

### 7.4 DISCUSSION:

Heavy metals like cadmium and mercury has been found to induce hyperplasia of the secondary lamellae epithelium of gills (Kaviraj & Das, 1995; Paulose 1989). Das & Kaviraj (1994) noted complete necrosis & sloughing of secondary lamellar epithelium of the common carp treated with cadmium. Saxena & Pandey (1993) observed histopathological changes in the gill of copper sulphate treated fingerlings of *Labeo rohita*. Mathiessen and Brafield (1973) noted histopathological changes in the gill of *Gasterosteus aculeatus* treated with Zinc. Temmink et al., (1983) have reported hyperplasia in the gills of Rainbow trout, *Salmo gardeneri* under the stress of chloromate. Turala & Scivio (1982) noted structural changes in the secondary lamellae of the gill of *Salmo gairdeneri* exposed to dehydrobietic acid Zinc. All these observations corroborate the present finding that toxicant damage the gills of fishes. Damaged gills lead to respiratory impairment and death in many species of fishes.
Hypertrophy of mucosal cells with increased amount of mucin granules and ruptured goblet cells at the tip of villi were observed in the intestine of *C. punctatus* exposed to sublethal mercury chloride (Sastri & Gupta, 1978). Pande & Saxena (1992) have found increase in this size of mucous secreting goblet cells of *Labeo rohita* on exposure to copper. Pandey et al., (1994) noted copious secretion of mucous from the goblet cell, edematous separation of the mucous from submucosa and necrotic changes in the intestinal mucosa of *Liza parsia* under the stress of mercury. Lata & Srivastava (1983) have reported changes in the intestine of *Puntius sophore* (Hamilton) under the stress of three carpet dying chemicals. Srivastava & Tripathy (1981) reported histopathological changes in *Esomis dendricus* (Hamilton) under sodium & Ammonium Sulphate stress. Toxicity to cadmium & Copper to a fresh water fish has been reported by Shivraj and Patil (1988). Zinc and Copper sulphate salts also damage the intestine of fishes (wong, 1977).

The intestine is an organ of digestion & absorption of food materials. Any change in the histopathological structure of intestine adversely effect digestion & assimilation of food leading to death.

Jabde (1993) found liver necrosis in teleost fish *Noemachelous aureus* on exposure to lethal and sublethal concentration of cypermethrin. Jain & Mishra (1994) observed hypertrophy & vacuolar degenerations of hepatocytes of *Puntius ticto* due to exposure to atrazine. Pandey et al., (1994) noted dilation of bile canaliculi, copious secretion of
bile and increased granulation in hepatocytes in the liver of *Liza parsia* under the stress of mercury. The liver of *Etroplus maculatus* was effected by DDT (Padgaonkar and Pranab, 1993). Baronia and Sahani (1984) and Baronia et al., (1991) have studied the histopathological changes in the liver of albino rat under the stress of DDT and Carbaryl respectively.

Thus it can be concluded that toxicants including OPME causes necrosis of the hepatic cells in animals including fishes. This leads to impaired functioning of the liver, leading to various type of biochemical & Physiological disorders.