Histopathology
The science that deals with the study of tissues or microscopic anatomy is called histology. Histology, the microanatomy of specific tissues, has been successfully employed as a diagnostic tool in medical and veterinary science since the first cellular investigations were carried out in the mid nineteenth century (Virchow, 1858).

Histopathology is mainly directed to study the effect of chemicals, pesticides and other pathogens on the structural components of the living system and the ways in which cells and tissues respond to injury. A chemical or a derivative acting directly on the cell most frequently causes chemical cytotoxicity by altering its environment. The cells in turn respond histopathologically by degeneration, proliferation, inflammation and repair. The chemical affects the cell by altering the external environment, oxygen and nutrient transport system or the endocrine and immune system.

Water pollution induces histopathological changes in fish. As an indicator of exposure to pollutants, histology represents a useful tool to assess the degree of pollution, particularly for lethal and chronic effects. The structural changes of the organ at microscopic, cellular and organelle level lead to alterations in functional systems (Jagadeesan and Mativanan, 1999). The severity of histological damages in any particular aquatic organism is directly proportional to the concentration of a pollutant in the medium. Moreover, the histopathological picture of the organs can corroborate with the biochemical changes accounting for the functional disruptions in the activity of the organs due to cellular damage. Vijayamadhavan
and Iwai, (1975) have reported that the extent of damage varies with organs, nature of pollutant, medium and duration of tests. Fishes are particularly sensitive to different pesticide concentration and their tissues are more prone to pathological effects (Murty, 1986). Harjit and Toor, (1997) stated that even low concentrations of some pesticides have harmful effect on the tissues of fishes. Seifter, (2001) have reported that mortality of fishes occurs due to the pathological lesions caused by pesticides.

Susceptibility to chemical injury varies greatly in the tissues and cells of the same animal. The extent of severity of tissue damage of a particular compound depends on the toxic potentiality of the organisms (Tilak et al., 2001). It is even greater in different animal groups. However, the location of the major damage may be determined by the mode of action of the chemical. The mode of action of each poison and the pattern of tissue vulnerability has been well defined and the toxic level of each agent at which a fairly standard distinctive pattern of tissue damage has been studied.

Degeneration is the most common symptom seen within the cell or population of cells. This may be produced by reduced blood and nutrient supply or endocrine deficiency. Intracellular accumulation like water, fat and proteins is seen very often, ultimately killing the cells, which are dead and still form a part of the living body. This leads to necrosis. Histologically necrosis is characterized by a sequence of morphological changes, which takes several hours to develop after actual death of cells. Gross necrotic changes are of three types.
1. Liguetactine necrosis: Resulting from rapid enzymatic digestion of cells.

2. Coagulative necrosis: The result of ischemia or loss of blood supply to an area.

3. Fat necrosis: The area has soapy fat consistency. In section, the stages of necrosis are best assessed in terms of the nucleus. They are,

I Pyknosis: Nucleus is shrunken and very dark. Rupture of the nuclear membrane and fragmentation of nuclear chromatin

II Karyolysis: characterized by fading and dissolving of nucleus leaving a ghost out line.

A few reports are available on the damage caused to different internal organs of an organism exposed to pesticides and heavy metals (Anitha Kumari and Ramkumar, 1997; Kapila and Ragothaman, 1999; Tilak et al., 2001; Santhakumar et al., 2001 and Vardhani Gowri, 2002). Gupta and Singh (1982) studied the impact of BHC on gills, liver and intestine of Trichogaster fasciatus and have shown that gills exhibited greater damage compared to liver and intestine.

Histopathological studies were carried in different fishes under the toxicant as observed in different fishes like, rainbow trout Oncorhynchus mykiss (Walsh and Ribelin, 1975); snake head, Channa punctats (Dubale and Shah, 1979); Walking cat fish, Clarias batrachus (Mandal and Kulshrestha, 1980); Saratherodon mossambicus, (Shukla et al., 1984)., Puntius sorana, (Amita and
Roshan, 1989); Cirrhinus mrigala, (Roy and Munshi, 1991); Glossagobius giuris, (Venkataramana et al., 2001) and Channa marulius, (Bijay, 2002); Channel catfish, Ictalurus punctatus, (Areechon and Plumb, 1990). Radhaiah et al., (1986) reported that, malathion induce necrosis, hyperplasia and oedema of gills and with vacuolation in liver of fish Tilapia mossambica, exposed to heptachlor. Similar changes have been reported in kidney of fish, Labeo rohita on exposure to heptachlor (Konar, 1970).

Jayantha Rao et al., (1984, 1985) reported that phosphomidon was found to be hepatotoxic and induced hepatopathy in freshwater fish, Tilapia mossambica. Eller, (1971), reported histopathological changes in gill, liver, pancreas of trout, exposed to aldrin. Under chronic conditions, gill and liver showed extensive damage and also significant damage in gastro intestinal tract of fish Channa gachua observed under acute toxicity of aldrin (Qureshi and Alvi, 1986). Tilak et al., (2001) observed marked histopathological changes in the tissues of fish, Labeo rohita. The gill showed hydropsy, lamellar clubbing and sever necrotic lamellar degeneration in the secondary gill lamellae on exposure to chlorpyrifos. Histopathological changes like increased shrinkage of striated muscle, number of vacuoles, and disposition of nucleus were observed by Venkataramana et al., (2001) in the fish, Glossagobius giuris exposed to malathion.

Dhanapakiam and Juliet Premlatha, (1994) observed hypertrophy of renal coils and degeneration of renal components in freshwater fish, Cyprinus carpio
exposed to malathion. Khan et al., (1994), reported that the winter flounder, *Pleuronectes americanaus* living adjacent to pulp and paper mill exhibited hyperplasia of gills, vacuolation in the liver, multifocal hemosiderosis in the spleen and liver. Several authors reported that, untreated effluent discharged from pulp and paper mills into receiving waters is known to be toxic to some aquatic organisms. Manifestation of toxicity in fish include fin necrosis, increase of parasites, change in physiology, kidney tumors, neoplastic lesions in the liver and skin tumors (Khan et al., 1992; Lindejoo and Thulin, 1990; Muckittrick et al., 1991; Bucher et al., 1992; Lindstrom and Oikari, 1990; Myers et al., 1987; Hawkins et al., 1990; Moore, 1991; Muralidharan et al., 2000 and Vardhani and Gowri, 2002).

Mandal and Kulshrestha, (1983) and Vinod Ghanathay, (1989) studied histopathological changes in *Clarias batrachus* and *Channa punctatus* exposed to sumithion and BHC respectively. Similar histopathological studies were carried out by Sowbhagya, (1991) and Vijayander Reddy, (1993) in fishes exposed to paper mill effluents and chromium respectively. Thorat, (2001) reported histopathological changes in the intestine of the fish, *Catla catla* exposed to endosulfan. Anitha and Ramkumar, (1997 a & b) revealed degenerative changes in the serosa, mucosa and submucosa layers, focal necrosis, proliferation and desquamation of the superficial part of villi in the fishes *Channa punctatus* and *Heteropneustes fossilis* collected from polluted Hussainsagar Lake. Effect of many dyes was studied by Saraswathi and Padmavathi, (1994), and reported extensive damage of the intestinal tissue in fish, *Cyprinus carpio*. 
Subhadra Banerjee and Bhattacharya (1994), reported the drastic histopathological changes i.e., degeneration and dispersion of chromoffin tissue, kidney lesions, karyolysis, dilation and shrinkage of Bowman’s capsule and glomerulus in *Channa punctatus* on exposure to mercury and ammonia. Cadmium effect on the histology of kidney and gill was studied by Ooi and Law, (1989) and Kapila and Ragothaman, (1999) respectively reported necrosis, damage of the renal tissue, disturbance in basement membrane, degeneration of gill lamella, cyst formation, swelling of base and increased interlamellar space in gill. Dubale and Shah, (1979) observed precipitation of both the cytoplasmic and nuclear material, vacuolation of hepatocytes and reported that the damage increased with the concentration and time of exposure to malathion. Anees, (1980) studied hepatic pathology of *Channa punctatus* exposed to sub lethal level of diazinon, methyl parathion, which resulted vacuolation of tissue.

Toxicants impair the metabolic and physiological activities of the organisms but such studies alone do not satisfy the complete understanding of pathological condition of tissues under toxic stress. Hence, it is useful to have an insight into histological analysis regarding the extent damage of the tissues like gill, liver, intestine and kidney when dimethoate enters the body *Cyprinus carpio*. The foregoing literature clearly documents that the pesticides cause structural changes in different organs of freshwater animals. The histological studies of the organs of freshwater fishes subjected to dimethoate were observed in the present study.
RESULTS

Histology of Gill (Control)

Gills are the vital organs for respiration in fish which establish a direct contact with the medium through which a pollutant largely enters into the body, (Bijay, 2002; Throat, 2001; Santakumar et al., 2001 and Vijayalakshmi and Tilak, 1996). The teleosts have four pairs of gill arches. The normal structure of the gill lamellae are flat leaf like structure laterally compressed and situated alternately on either side of the inter brachial septum. The primary gill lamellae consist of centrally placed rod like supporting axis with a row of secondary gill lamellae present on each side of it. The secondary gill lamellae also turned as respiratory lamellae are highly vascularized and covered with a thin layer of simple squamous epithelial cells separated by mucus cells. Blood vessels can be seen extended into each secondary gill lamellae. The blood cell has single nucleus, which is flat in appearance. The region between two adjacent respiratory lamellae is turned as inter lamellar region (Plate 1 Fig. 1 & 2).

Histological changes in the Gill of exposed fish

Fish on exposure to the lethal concentration of dimethoate, on day 1, the enlargement of the base of primary gill lamellae was observed (Plate, 1 and Fig. 3 & 4). On day second lamellar oedema and secondary gill lamellae clubbing at the distal end was seen leading towards telangiectatic secondary lamellae (Plate, 1 and Fig. 5 & 6). On day 3\textsuperscript{rd} lamellar telangiectasis was observed, which continued up to day 4 (Plate, 1 and Fig. 5 & 6). Lamellar hypertrophy and lamellar
LEGEND FOR FIGURES

Plate : 1 :

Fig. 1 : Section of gill of control fish, *Cyprinus carpio* showing normal gill filament

PGL = Primary gill lamellae,
SGL = Secondary gill lamellae and
ILS = Inter lamellar space.
GR = Gill rackers
BSG = Basal Gill Filament
H and E. × 100.

Fig. 2 : Section of gill of control fish, *Cyprinus carpio* showing a single gill filament. H and E. × 400.

Fig: 3 and 4 : Section of gill of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 24 hr showing early Lamellar clubbing (LC), slightly Telangiectatic (T) and Lamellar oedema (LE).

H and E. × 100 and × 200.

Fig. 5 and 6 : Section of gill of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 48 hr showing Telangiectatic secondary lamellae (TSL). H and E. × 400.

(H and E = Hematoxylin and Eosin)
**LEGEND FOR FIGURES**

Plate: 2

**Fig. 1 and 2:** Section of gill of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 μl/l) for 72 hr showing more Telangiectatic secondary lamellae (TSL), Hyperplasia (HP) and Lamellar oedema (LE).
H and E. × 200 and × 400.

**Fig. 3 and 4:** Section of gill of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 μl/l) for 96 hr showing sever Telangiectatic secondary lamellae (TSL) and Lamellar fusion (LF).
H and E. × 200.

**Fig. 5 and 6:** Section of gill of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 μl/l) for 96 hr showing single filament with desquamation of secondary lamellae (DSL), epithelial capillary separation (ECS)
H and E. × 200.

*(H and E = Hematoxylin and Eosin)*
**LEGEND FOR FIGURES**

Plate : 3 :

**Fig. 1:** Section of gill of control fish, *Cyprinus carpio* showing normal structure

- PGL = Primary gill lamellae,
- SGL = Secondary gill lamellae and
- ILS = Inter lamellar space. H and E. X 200.

**Fig. 2:** Section of gill of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 μl/l) for day 1 showing loss of secondary gill lamellae and necrosis (N) leading to hyperplasia (HP).

H and E. × 200.

**Fig. 3 and 4:** Section of gill of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 μl/l) for 5th day showing Telangiectatic secondary lamellae (TSL), lamellar oedema (LE), Hyperplasia (HP)

H and E. × 400.

**Fig. 5 and 6:** Section of gill of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 μl/l) for 5th day showing Hyperplasia (HP), and damage of basal gill filament (BGF).

H and E. × 200 and × 400.
LEGEND FOR FIGURES

Plate : 4 :

**Fig. 1 and 2** : Section of gill of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 µl/l) for 10\(^{th}\) day showing recovery in structure of gill filament with mild lamellar fusion (LF) of secondary lamellae. H and E. × 200.

**Fig. 3 and 4** : Section of gill of fish, showing recovery in structure of gill filament for 15\(^{th}\) day showing recovery in structure of gill filament. H and E. × 200 and ×100.

**Fig. 5 and 6** : Section of gill of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing sever Telangiectatic secondary lamellae (TSL) and Lamellar fusion (LF).
H and E. × 400 and × 100.

\(\text{H and E = Hematoxylin and Eosin}\)
hyperplasia was observed. Fusion of these lamellae was noticed all along their length on day 4. The cells seemed to have undergone a clear increased hyperplasia further desquamation of the hyperplasic epithelium and capillaries with loss of the lamellar structure of the gill were seen all along the gill filaments (Plate, 2 and Fig. 1, 2, 3, 4, 5, & 6).

In the sublethal concentration of dimethoate a mild degree of degenerative changes and sign of shrinkage in the primary gill lamellae was observed at day 1 exposure. The slight damage to the base of secondary lamellae and inter lamellar tissue was observed (Plate, 3 and Fig. 2 & 3). On day 5 the changes were more marked when compared to day 1 (Plate, 3 and Fig. 4). The degeneration of epithelial cells encapsulating primary and secondary gill lamellae with necrosis. Fusion of secondary lamellar, bubbling of primary gill lamellae, atrophy is also observed. The excess secretion of mucus and necrosis of basal filament was observed. But on further exposures, for 10 and 15 days the gill structure was just similar to that of control fish except mild degree of precipitation of mucus over the gill lamellae (Plate, 4 and Fig. 2, 4 & 6).

**Histology of Intestine (Control)**

Intestine consists of four layers viz., serosa, muscularis externa submucosa and mucosa. The serosa is made up of loose connective tissue. Next to serosa is muscularis externa. It is distinguished into outer longitudinally arranged muscle fibers, whereas the inner layer is composed of circular muscle fibers. Submucosa is made up of loose connective tissue, blood vessels and capillaries. The
**LEGEND FOR FIGURES**

Plate : 5

**Fig. 1:** Section of intestine of control fish, *Cyprinus carpio* showing a full view of intestinal layers i.e.,

\[
\begin{align*}
S & = \text{Serosa}, & V & = \text{Villi} \\
\text{LMF} & = \text{Longitudinal muscle fibers}, & L & = \text{Lumen} \\
\text{CMF} & = \text{Circular muscle fibers and} \\
\text{H and E. } & \times 100.
\end{align*}
\]

**Fig. 2 and 3:** Section of intestine of control fish, *Cyprinus carpio* showing enlarged intestinal layers

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\begin{align*}
S & = \text{Serosa}, & V & = \text{Villi} \\
\text{LMF} & = \text{Longitudinal muscle fibers}, & \text{SM} & = \text{Mucus cell} \\
\text{CMF} & = \text{Circular muscle fibers}, & \text{LP} & = \text{Lamina propria} \\
\text{H and E. } & \times 400.
\end{align*}
\]

**Fig. 4:** Section of intestine of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 24 hr showing full view of damaged intestinal layers.

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\text{H and E. } \times 100.
\]

**Fig. 5 and 6:** Section of intestine of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 48 hr showing damage of Serosa (S), mucous cell become enlarged and filled with secondary material (SM).

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\text{H and E. } \times 400. \\
\text{(H and E = Hematoxylin and Eosin)}
\]
LEGEND FOR FIGURES

Plate : 6 :
Fig. 1: Section of intestine of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 72 hr showing more damage of intestinal layers. Bulging and rupture of the tips of prominent villi (V) and sub mucus region (SIM) along with degeneration of epithelial cells (DEC). H and E. × 400.

Fig. 2 and 3: Section of intestine of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing goblet cells (GC), Vacuolization (V) and necrosis (N). H and E. × 400.

Fig. 4: Section of intestine of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing maximum damage of intestinal layers and villi H and E × 400.

Fig. 5 and 6: Section of intestine of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing Vacuolization and necrosis of epithelial cell (NEC), Necrosis (N) and degeneration of villi. H and E. × 400.

(H and E = Hematoxylin and Eosin)
LEGEND FOR FIGURES

Plate : 7 :

**Fig. 1**: Section of intestine of control fish, *Cyprinus carpio* showing a entire view of intestinal layers i.e.,
CMF = Circular muscle fibers,
LP = Lamina propria,
V = Villi and
L = Lumen

H and E. × 200.

**Fig. 2 and 3**: Section of intestine of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 µl/l) for 1st and 5th day showing damage of intestinal layers. H and E. × 100 and × 400.

**Fig. 4**: Section of intestine of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 µl/l) for 10th day showing less damage of intestinal layers. H and E. × 400 and × 200.

**Fig. 5**: Section of intestine of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate (EC) (7 µl/l) for 15th day showing entire view of recovery of intestinal layers. H and E. × 200.

(H and E = Hematoxylin and Eosin)
submucosa is followed by innermost mucosa, which is divisible into lamina propria and epithelial layer. The lamina propria is vascular and is made up of areolar connective tissue. The epithelial layer lining the lumen of the gut is made up of columnar epithelium and is thrown into deep mucosal folds. Mucosa comprises of various glands. The submucosa is reduced having bundles of longitudinal muscle fibers. In intestine the mucosal folds are produced into prominent slender folds called villi, which have intestinal glands. The submucosa extends into villi forming lamina propria. The villi are covered with epithelial lining consisting of prismatic cells with basal nuclei containing goblet cells (Plate, 5 and Fig. 1, 2 & 3).

**Histological changes in the Intestine of exposed fish**

The intestine of fish, *Cyprinus carpio* exposed to lethal concentration dimethoate shows the degeneration of cytoplasm of columnar epithelial cells. The goblet cells degenerated indicating non-lubricant surface, creating fringed or serrated margin. The basal nuclei of columnar and lymphatic cells appear to be highly active amounting to carcinogenic condition of villi. The lumen is reduced owing to the shrinkage of intestinal structural composition. The compactness with increased hyperplasia caused the shortening of villi and lodge shaped narrowness of lumen. Lymphatic cells are found to be hyperactive. Lamina propria is considerably diminished. The lumen of intestine is further reduced. The goblet cells showed vacuolization and degradation followed by their appearance with highly swollen nuclei. The epithelial columnar cells of villi were distinct in the
control fish. In the exposed fish up to 96 h homogenous mass of necrosed cells were seen. The flaked shaped goblet cells, found in the control fish, were increased and almost rounded due to loss of neck on account of necrosis of epithelial cells. At focal areas, the perforations of villi and their rupture were more on 3rd and 4th day of exposure of dimethoate (Plate, 5 & 6 Fig. 5, 6 & 1, 2, 3, 4, 5).

In sublethal concentrations of dimethoate the intestine exhibited, hypertrophy and necrosis of epithelial cells in the lumen of the intestine on 1st day. On 5th day cellular exudates in the lumen of intestine (Plate, 7 and Fig. 1, 2 &3 ). The circular and longitudinal muscles were desquamated. After 10th days recovery tendency was seen. On 15th day the intestine showed maximum recovery in structure (Plate, 7 and Fig. 4 & 5).

**Histology of Liver (Control)**

The liver of fish comprises a continuous mass of large hexagonal hepatic cells (Hepatic parenchyma). Hepatic cells are of polygonal shape containing clear spherical nucleus. They are located among sinusoids forming cord like structures known as hepatic cell cords. In fish these structures are generally obscure. Bile canaliculus is centrally located in each cord. There is no clear division of hepatic cells into lobules. These cells contained granular cytoplasm and with distinct nuclei either exocentric or slightly centrally placed. Hepatic cells have many vital functions other than the secretion of bile. They play an important role in protein, lipid and carbohydrate metabolism. They serve as storage site for some nutrients. Detoxification is another important function. A large number of blood sinusoids and lipid glycogen granules are found in the hepatic mass (Plate, 8 & Fig.1, 2 & 3).
**LEGEND FOR FIGURES**

Plate: 8:

**Fig. 1 and 2**: Section of liver of control fish, *Cyprinus carpio* showing normal structure

- HP = Hepatocytes,
- N = Nucleus and

**Fig. 3 and 4**: Section of gill of control fish, *Cyprinus carpio* a part of liver enlarged showing polygonal hepatocytes (HP) and nucleus (N) and Blood Vessels (BV). H and E. × 400 and × 100.

**Fig. 5 and 6**: Section of liver of fish, *Cyprinus carpio* exposed to lethal concentrations of dimethoate (EC) (36.22 μl/l) for 24 hr showing cytoplasmic degeneration, shape of hepatocytes was changed (HP) and damage of blood vessel (BV). H and E. × 100.

*(H and E = Hematoxylin and Eosin)*
LEGEND FOR FIGURES

Plate: 9

Fig. 1 and 2: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 48 hr showing thicken of blood vessels, and hepatocytes atrophy (HA).
H and E. × 100 and × 200.

Fig. 3 and 4: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 72 hr showing, cytoplasmic degeneration, and swollen hepatocytes.
H and E. × 400.

Fig. 5 and 6: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing sever necrosis, cytoplasmic degeneration, vacuolization and granulisation of interlobular duct with nuclear hypertrophy.
H and E. × 400.

(H and E = Hematoxylin and Eosin)
**LEGEND FOR FIGURES**

**Plate : 10**

**Fig. 1 and 2**: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate EC (7 μl/l) for 1\(^{st}\) day showing slight necrosis (NC), damage of blood vessel (BV).
H and E. × 200

**Fig. 3**: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate EC (7 μl/l) for 5\(^{th}\) day showing diffused necrosis (NC), cytoplasmic degeneration, severe damage of blood vessel (BV) and vacuolization of hepatic cells (VZ).
H and E. × 400.

**Fig. 4 and 5**: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate EC (7 μl/l) for 10\(^{th}\) and 15\(^{th}\) day showing recovery structure.
H and E. × 100

**Fig. 6**: Section of liver of fish, *Cyprinus carpio* exposed to dimethoate EC (7 μl/l) for 15\(^{th}\) day showing recovery structure.
H and E. × 1000

*(H and E = Hematoxylin and Eosin)*
Histological changes in the Liver of exposed fish

On day 1 of exposure to the lethal concentration of dimethoate, the liver of fish exhibited enlarged nuclei and vacuolization in hepatic cells. Liver cords were seen disarrayed (Plate, 8 and Fig. 5 & 6). On day 2 of exposure, the parenchymatous nature of the liver was greatly disrupted with congested blood vessels. The hepatocyte cell membranes were ruptured and granular degeneration was evident in most of the hepatocytes. Nuclei became slightly hypertrophic (Plate, 9 and Fig. 1 & 2). Further on day 3 severe degrees of atrophic changes were noticed in the liver cords. Hemorrhagic condition was prominent with heavy vacuolization in the liver tissue. At some regions exfoliation and congregation of hepatocytic nuclei and focal necrosis were seen (Plate, 9 and Fig. 3 & 4). This was followed by the severe degree of vacuolization, shrinkage of hepatocytes, atrophy, cytoplasmic degeneration, rupture of blood vessels, diffused necrosis, dissolution of laminar structure and cytoplasmic disintegration in hepatocytes on day 4 of exposure (Plate, 9 and Fig. 5 & 6).

Compared to the structure of the liver of control fish, exposed to sublethal concentration of dimethoate initially exhibited few changes like slight disarray of liver lobes, mild degree of degeneration of cytoplasm, occasional blood clots and congregation of nuclei at day 1 (Plate, 10 and Fig. 1 & 2) and cloudy swelling of hepatocytes, granulization of cytoplasm, hypertrophic and pyknotic nuclei on day 5 (Plate, 10 and Fig. 3 & 4). However, on further exposure to day 10 certain degree of reorganization in the structure of liver cords was observed. The nuclei
appeared normal, with a very little degree of cytoplasmic vacuolization (Plate, 10, & Fig. 5). At 15 days of exposure, no significant changes were seen different from controls, except a slight degree of hyperchromatic condition of the nuclei (Plate, 10 and Fig. 4, 5 & 6).

**Histology of Kidney (Control)**

The basic unit of kidney in fish consists of a renal corpuscle, Bowman’s capsule and glomerulus and various segment of the renal tubules, namely proximal tubule, intermediate segment, distal tubule and collecting duct. Proximal tubules have prominent brush borders bathed in the vascular bed in the interstitial tissues. Distal tubules and collecting ducts, both devoid of brush borders, and are sparsely distributed. The intermediate segments between proximal and distal tubules are rarely seen. The renal corpuscles are located in close vicinity of renal tubules and blood vessels in the interstitial tissue. Pigments and leucocytes are very common in the interstitial tissue (Plate, 11 and Fig. 1, 2 & 3).

**Histological changes in the Kidney of exposed fish**

In lethal concentration of dimethoate the kidney showed reduction in renal cell number in the proximal and distal collecting tubules, which have resulted in narrowness of lumen. The tubular cells have undergone hypertrophy and some of the renal tubules have lost their normal shape. Vacuolization due to degeneration of cytoplasm is quite obvious. The nuclei of epithelial cells have become quite dominant and are found infiltrating into the surrounding tissue. The perforation of
LEGEND FOR FIGURES

Plate : 11

Fig. 1 and 2 : Section of kidney of control fish, *Cyprinus carpio* showing normal structure
P = Proximal tubule G = Glomerulus
BV = Blood vessels DT = Distal tubule
UT = Uriniferous tubule
H and E. × 40 and × 400.

Fig. 3 : Section of kidney of control fish, *Cyprinus carpio* showing a enlarged Proximal tubules (P) with Blood vessels (BV) and Uriniferous tubule (UT)
H and E. × 1000.

Fig. 4 Section of kidney of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 μl/l) for 24 hr showing Vacuolization (VZ) and damage of distal tubule (DT). H and E × 400.

Fig. 5 and 6: Section of kidney of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 μl/l) for 48 hr showing Glomerulus shrinkage (G), Vacuolization (VZ), tubular degeneration and intra lumen vacuolization (ILV).
H and E. × 400.

(H and E = Hematoxylin and Eosin)
LEGEND FOR FIGURES

Plate: 12

**Fig. 1 and 2:** Section of kidney of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 72 hr showing maximum damages like Necrosis of distal tubule (DT), Glomerulus shrinkage (G), Vacuolization (VZ) and tubular degeneration. H and E. × 400.

**Fig. 3 and 4:** Section of kidney of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing degeneration of Interstitial tissue (IT), tubular degeneration, Necrosis of proximal tubule (N) and Vacuolization (VZ). H and E. × 400.

**Fig. 5 and 6:** Section of kidney of fish, *Cyprinus carpio* exposed to dimethoate (EC) (36.22 µl/l) for 96 hr showing degeneration of Interstitial tissue (IT), tubular and Bowmen’s capsule degeneration, Necrosis and enlargement of distal tubule (DT) and Vacuolization (VZ). H and E. × 400.

(H and E = Hematoxylin and Eosin)
LEGEND FOR FIGURES

Plate : 13

Fig. 1 and 2: Section of kidney of fish, *Cyprinus carpio* exposed to sublethal concentrations of dimethoate EC (7 μl/l) for 1st and 5th day showing desquamation and degeneration of tubules, necrosis (N) and Glomerulus shrinkage (G).

H and E. × 400 and × 200.

Fig. 3 and 4: Section of kidney of fish, *Cyprinus carpio* exposed to sub lethal concentration of dimethoate EC (7 μl/l) for 10th and 15th day showing recovery structure, cellular damages were reduced.

H and E. × 200.

*(H and E = Hematoxylin and Eosin)*
Plate-13

Image 1: BC, G, UT

Image 2: UT, G

Image 3: UT, G, DT

Image 4: UT, DT
kidney tubules is commonly observed. The kidney demonstrated hyperplasia, 
vacuolization, degeneration and necrosis leading to the complete necrosis. 
Cubiodal epithelial cells lining the tubules showed complete vacuolization with 
degenerating cytoplasm and more nuclear division and their disorderly scattering 
nature. The hemopoietic tissue was fully studded with lymphatic cells at the 
highest rate of nuclear division. The lumen of the tubules was found to be dilated. 
Kidney tubules were also found to be perforated (Plate, 12 and Fig. 1, 2, 5 & 6).

In sublethal concentration of dimethoate the kidney of the fish exhibited a 
mild degree of changes. 1\textsuperscript{st}, 5\textsuperscript{th} and 10\textsuperscript{th} day of shows more changes i.e., epithelial 
cells of the tubules which showed desquamation, irregular orientation of the 
nuclei in the cells, lumen of the tubules became wider as a result of flattening of 
epithelial cells. Ruptures of the tubules were quite prominent, cell fragments 
could be seen inside the lumen of some tubules. Vacuolar degeneration was seen 
in the few tubules. Hemopoeitic tissue was degenerated. But on 15\textsuperscript{th} day kidney 
showed recovery tendency (Plate, 13 and Fig. 4 & 5). Glomerular cells attained 
normalcy in structure. Cytoplasm appeared clear and vacuolization and karyolysis 
of cell was completely reduced. Necrotic changes in uriniferous tubules were 
reduced. Clumping of damaged blood cells was seen.

DISCUSSION

Most of the pollutants have telling effects on the tissues in aquatic 
organism. The most common route of entry of the water soluble toxicants in fishes 
is the gills (Holden, 1973). In the present study compared to the controls, the
progressive degenerative changes in the gills of fish exposed to the lethal concentration of dimethoate. The changes include swellings of the base of the secondary gill lamellae, lamellar oedema, fusion of primary and secondary gill lamellae, erosion of superficial cells, hypertrophy and hyperplasia, nuclear pyknosis and the ultimate disintegration of secondary gill lamellae were observed. Thus the structural changes in the gill filaments particularly the secondary gill lamellae, offer a favorable material for the studies on the effect of toxic substances because they have a key position in the body of the fish due to their role in the transport of oxygen.

Gills perform numerous functions, which include respiration, osmoregulation, excretion of nitrogenous waste products, and acid-base balance (Heath, 1987). Previous histopathological studies of fish exposed to pollutants have shown that fish gills are primary markers for aquatic pollution. Therefore, functional impairment of gills caused by pollutants can significantly damage the health of fish. For this reason, fish gills are considered to be the most appropriate indicators of water pollution levels (Alazemi, et al., 1996).

The progressive degenerative changes in the gills of fish following exposure to the lethal concentration of dimethoate suggest that the major route of entry of pesticide is the respiratory system (Gill et al., 1988). The changes in the secondary gill lamellae indicate that the death of fish exposed to the lethal concentration might have occurred due to the failure of gaseous exchange across the respiratory epithelium. This is evident from the present study that there was
significant decrease in the oxygen consumption of fish. Areechon and Plumb (1990) reported that high concentration of malathion resulted in separation of the epithelial layer covering the secondary lamellae from the pillar cell system. This has increased the gap between water and blood and finally the fish died of tissue hypoxia. Similar histopathological changes have been reported in the gills of *Colisa fasciatus* exposed to lindane (Verma *et al.*, 1975); *Channa punctatus* to methoxy ethyl mercuric chloride (Sastry and Rao, 1983); *Rasbora daniconius* to mercury, (Ashok and Vinod 1995) and *Labeo rohita* to chloropyrifos, (Tilak *et al.*, 2001). However, the damage in the pillar cells was well marked at higher concentrations, which was also reported in, *Anabas testudineus* exposed to monocrotophos. (Santhakumar *et al.*, 2001); rainbow trout exposed to zinc sulfate, (Skidmore and Tovell, 1972); *Labeo rohita*, exposed to mercury (Jagadeesan, 1999) and *Cirrhinus mrigala* exposed to malathion (Roy and Datta, 1991).

Prashanth, (2002) reported lamellar oedema, which is most frequent following exposure to cypermethrin. Complete oedematous separation of the respiratory epithelium of primary and secondary epithelium lamellae with necrosis of lamellar epithelial cells may cause the respiratory and osmoregulation distress. Supporting to the present investigation Vinod Ghanathay, (1989) reported enlargement of secondary gill lamellae and inflammation of interlamellar epithelium in *Channa punctatus* on exposure to BHC. He also observed increased numbers of mucous cells in the secondary lamellae of the experimental fish as compared to control fish, indicating the defensive response of the fish to the
toxicant. The pillar cell system also appeared to be collapsed and pilaster columns were seen curled. Pools of congested blood were also seen within the subepithelial space. Collapse of the pillar cells system is believed to occur when a fall in the hydrostatic pressure causes this system to fail as a vascular endoskeleton (Santhakumar et al., 2001 and Bijya, 2002). Due to the formation of subepithelial space, the blood supply between the pilaster cells and epithelial lining as well as water balance is affected (Vijayalakshmi and Tilak, 1996). Degeneration of epithelial cells indicates the damage in the gill lamellae which reduces the activity of gill. This consequence is also likely to limit the respiratory capacity of the gills.

The fusion of secondary lamellar in gill may take place as an ultimate result of massive lamellar hyperplasia, which results in a solid fusion of many or all of the lamellar capillaries within a mass of hyperplasic epithelium. Lamellar hyperplasia is more long term response of the malphigian cells, often to lower levels of irritation (Ronald J Roberts, 1989). Cells are principally derived from the primary lamellae. They migrate distally, often in the early stages, resulting in an accumulation of cells at the leading edge of the secondary lamella, known colloquially as clubbing of the lamella. Seifter, (2001) have shown that secondary lamellar swelling occurs, associated to a degree with hypertrophy of individual epithelial cells and alteration underlying pillar cell architecture. Thickening of lamellae due to inflammation of epithelial cells results in the lifting and dissociation of epithelium. This reduces the availability of water space and constricts the blood capillaries (Kapila and Ragothaman, 1999).
A characteristic pathological change observed in the gill, associated with physical trauma, is the condition known as lamellar telangiectasis. Telangiectatic secondary lamellae may occur in association with metabolic waste or chemical pollution. It is recognised grossly by the presence of small red spots on the secondary lamellae, results dilation of lamellar capillary and pooling of the blood. If there are many telangiectatic lamellae, respiratory function may be impaired (Seifter, 2001). The dominance of telangiectatic tissue is observed at 48 to 96 hrs of exposure. Histologically it is obvious that the lesion has its genesis in the rupture of the retaining pillar, or pilaster cells, which normally join the dorsal surface of secondary lamellae to the ventral. The result is dilation of the lamellar capillary and pooling of the blood, which fuses with adjacent lamellae as reported by Ronald J Roberts, (1989). Bulging of secondary lamellae were observed in may species, like Gambusia affinis, (Veena Sakthivel and Gaikwad, 2002); Channa marulius (Bijay, 2002); Anabas testudineus, (Santhakumar et al., 2001); Labeo rohita, (Tilak, et al., 2001); Boleophthalmus dussumeri, (Kapila and Ragothaman, 1999); Labeo rohita, (Jagadeesan 1999) and Punitus stigma, (Khillare and Davane, 1998).

Hyperplasia and fusion of gill filaments due to separation of epithelium reduces the surface area available for gaseous and other exchanges (Skidmore and Tovel, 1972). In fish, the respiratory epithelium is the barrier between the blood and the surrounding water through which respiratory exchanges take place (Narain et al., 1990). Hyperplasia of lamellar epithelium is generally due to an
increase in numbers and migration of the malphigian cells of the primary lamella. Hyperplasia is a long term response of the malphigion cells, often to lower levels of irritations. Cells are principally derived from the primary lamellae. They migrate distally, often in the early stages resulting in an accumulation of cells at the leading edge of the secondary lamella, known colloquially as ‘clubbing’ of the lamellae. There may be an increase in numbers of mucous cells at the base of the lamellae. Eventually the intercellular space may be filled with new cells and the respiratory area greatly reduced (Ronald J Roberts, 1989). Any damage to this epithelium affects not only ventilatory process but also other vital process like ion exchange, secretary and excretory function of the gills (Narain et al., 1990; Bijay, 2002 and Sarita and Sudha, 2002).

Toxic stress induced by the malathion in the gill epithelium leads to events like increased influx of hydrogen ions which reduces the pH of the blood and thus decreases the oxygen carrying capacity of the hemoglobin (Santhakumar et al., 2001). On the other hand the ion regulatory and excretory functions of the gills were hampered. Epithelial damage disturbs the exchange of ammonium and bicarbonate ions of the blood with sodium and chloride ions of the medium, which normally occurs across the gill epithelium of fish (Love, 1980).

Gill in the fish is perhaps the most sensitive organ for pollutants in the water. Since gills are not only the respiratory but also the osmoregulatory organs of the fish. The cellular damage induced by dimethoate might also impair the oxygen consumption and osmoregulatory function of fish as evidenced from the
decreased oxidative metabolism uptake of vital ions and the associated ATPase activities, which also could be one of the possible reasons for the death of the fish. Similar reason also suggested by Areechon and Plumb, (1990) for the death of the *Ictalurus punctatus* on exposure to malathion. Similar observations made in *Rainbow trout*, (Walsh and Ribelin, 1975), snake head *Channa punctatus*, (Dubale and Shah, 1979), walking cat fish *Clarious batrachus* (Mandal and Kulshrestha, 1983), *Sarotherodon mossambicus* (Shukla *et al.*, 1984). The progressive dissolution of gill structure in the fish exposed to lethal concentration provides a good support for the progressive decrease in the soluble and structural protein levels in this organ.

In the sublethal treatment of dimethoate the changes in the gills was totally different. On 1\textsuperscript{st} and 5\textsuperscript{th} day hyperplasia, fusion of gill epithelium due to separation of epithelium, necrosis of gill epithelium, degeneration of pilaster cells and telangiectatic in secondary lamellae was observed (Plate 3, Fig. 3 & 4). Slow recovery on day 10 of exposure was observed (Plate 4, Fig. 2 & 3) attributing to longer days of exposure which clearly indicate that the fish is adapting to the surrounding toxicant media. On 15\textsuperscript{th} day of exposure gill structure showed maximum recovery (Plate 4, and Fig. 4). Fukuda, (1983) and Goldes *et al.*, (1988) have both shown complete recovery from severe reactive hyperplasia in less than a month when exposed to lower concentration of toxicants. The progressive recovery in gill of *Labeo rohita* exposed to mercury has also been noticed (Jagadeesan and Mathivanan, 1999).
The histopathology of the intestine is influenced to a considerable extent by the presence of pesticide within lumen. The intestine of the fish exposed to lethal and sublethal concentration of dimethoate showed increase in the mucosal cell activity, degenerative changes in its structure, hypertrophy of epithelial cells, swelling of lamina propria, fusion of villi due to excessive hypertrophy and oedemec lamina propria ultimately leading to rapture of villi at their tips. The damage was greater towards the sides of villi. The damage was greater at high dose and with the time of exposure. The lamina propria separated from the basement membrane inhibiting blood supply to epithelial layers.

A very specific lesion is associated with infectious haematopoietic necrosis where there is acute necrosis of eosinophil granule cells, which forms a discrete and distinctive submucosal layer in the gut. Intestinal layers such as submucosa, muscular layer and serosa on day 1 of exposure showed oedema and which was increased with time of exposure. Thus intestinal wall was found to be sensitive to dimethoate. Increase in goblet cells indicates that the toxicants trigger mucous cell activity. Focal necrosis, proliferation of villi, desquamation of the superficial parts of villi and necrosis of the tips of villi have been observed. The increase in the blood vessels of the intestine might not be due to only allergic responses but also to anemia triggered by dimethoate. At some places the epithelial lining of the villi was ruptured and the disruption of the intestinal brush border may result in impaired intestinal absorption. The similar observations were reported in other animals (Anith Kumari and Ramkumar, 1997a & b; Saraswathi and Padmavati, 1994).
The eosinophil granular cells (EGC) often form a very conspicuous layer from which they move into villi or even into the submucosa in certain conditions. The degeneration of epithelial cells and rupture of villi in the intestine of *Heteropneustes fossilis* due to phospamidon was reported by Konar, (1979). Similar findings was reported by Anita Kumari and Ramkumar, (1997a) in *Channa Striatus* and *Heteropneustes fossilis* exposed to polluted water of Hussainsagar lake. Histopathological effects of insecticides on intestine of fish have been studied by several authors (Thorat, 2001, Saraswathi and Padmavathi, 1994, Amita and Roshanlal, 1989 and Bakthavathsalam et al., 1984).

Gupta and Singh† (1982) observed the degeneration of the epithelium, shrinkage and atrophy in the lamina propria and vacuolation in different layers of stomach and intestine in the fish, *Trichogaster fasciatus* when exposed to 1.5 ppm sublethal and 3.6 ppm lethal concentrations of BHC for 24 and 96 hrs. In *Clarias batrachus* exposed to sublethal concentration of dichlorvos found marked dilation of blood vessels of submucosa and degeneration of intestinal folds after 10 days of exposure (Banerjee, 1982). These changes continued further when exposed to 20 and 30 days. Lakota et al., (1983) exposed the fry of *Cyprinus carpio* to 1-10 ppm to lindane, DDT and toxaphene and reported the formation of histological lesions in the intestine.

During sublethal exposure of dimethoate on 1 and 5th day, the intestine showed damages in the mucosa, submucosa, serosa, villi cells and also blood vessels (Plate, 7 and Fig 2 &3). But on day 10, intestine (Plate, 7 and Fig. 5)
showed some recovery tendency. The damages of epithelial layers, villi and blood vessels are reduced comparatively on day 1 and 5, it was also continued till days 15 of exposure. On 15th day, a full recovery of intestine was seen. This could be due to long term exposure of dimethoate during which recovery was noticed in the tissues. Thus dimethoate has traversed through the delicate mucous membrane resulting into sloughing of the epithelium or excess exudation of mucus from the goblet cells. In lower concentration of dimethoate skin first reacted by exuding enough mucous and thus the metabolic rate decreased resulting in less entry of toxicant into intestine (Amita & Roshan, 1989).

A wide range of causes can damage the liver because of its multiple metabolic functions; such damaged can have serious effects on the metabolism of the entire animal. The liver of the fish does not show the diversity of pathology seen in higher animals probably as a result of the lack of kupffer cells in the liver sinusoids. However, it is susceptible to a number of toxic and metabolic differences. Acute and extensive necrosis of liver cells may occur in toxic condition (Ronald, 1978).

Liver is involved in the metabolism of most toxicants, which can usually be detoxified, but many of them can be bioactivities and in turn becomes more toxic. The toxicology of liver is complicated by the variety of liver injuries caused. The liver has a high concentration of xenobiotic metabolizing enzymes, some of which activate the toxicants to induce lesions locally (Sastry and Rao, 1983). Toxicants induced changes in the liver of fishes can be regarded as an
In the present investigation the appearance of degenerative changes in the liver of fish exposed to the lethal concentration of dimethoate support the metabolic disorders observed in it. The disarrayed liver cords, vacuolation of cytoplasm, swollen hepatic cells, hepatic cells necrosis, dilated sinusoids, coagulation of blood cells, hepatic granuloma, cirrhosis followed by the shrinkage of hepatocytes and dissociation of laminar structure serves as degree of nuclear atrophy were observed, which suggests that the depletion in its glycogen reserves. Acute pathological changes in the liver due to insecticides have been reported by a number of workers (Ammenikutty and Rege, 1978; Anees, 1980 and Vardhani and Gowri, 2002). These pathological changes may be associated with the accumulation of the pesticide (Moriarty, 1975). Mandal and Kulshrestha (1980) reported histopathology of liver exposed to malathion which suggests the changes in the liver characterized by necrosis, loss of shape of original hepatic cells, rupture and disintegration of cell boundaries which leads to the formation of multinucleated giant cells. Similar responses were also observed in the fish subjected to malathion treatment (Areechon and Plumb, 1990; Shukla et al., 1984). The necrosis of hepatocytes vacuolization and swelling of liver cords were noticed by some workers in different fishes treated with various toxicants. The liver of blue gills treated with methoxychlor showed cell vacuolization and swelling of liver cords. Other investigators in different fishes treated with various toxicants also noticed these changes. The liver of blue gills treated with methoxychlor showed cell vacuolization (Kennedy et al., 1970).
Cirrhosis is a diffuse increase in the fibrous tissue of the liver, usually associated with chronic damage and destruction of hepatocytes. The damage can result from a wide range of stimuli, from longstanding biliary obstruction, heavy metal or pesticide poisoning to chronic parasitism. The mercuric chloride treated *Channa punctatus* showed vacuolization of hepatocytes, necrosis, and rupture of cell membrane (Sastry and Rao, 1983). Vinod Ghanathay (1989) reported vacuolation of connective tissue and grouping of hepatocytes culminating in focal necrosis, etc in *Channa punctatus* on exposure to BHC. Abhagupta and Singh (1982) observed dilation of sinusoids, deformation of hepatic cells and necrosis in *Tricogaster fasciatus* on exposure to BHC. Bhattacharya *et al.*, (1975) reported swollen liver cells with irregular surface in *Clarius batrachus* exposed to various concentration of aldrin. The cells were either binucleated or the nucleus was enlarged. Degenerative changes were shown by rupture and vacuolation of hepatic cells, some times with the appearance of inter cellular spaces indicating a sever necrotic condition. The damage caused to liver was more in the fishes at lethal concentrations of dimethoate but the damage at sublethal concentration was not significant.

It is not generally possible to differentiate liver necrosis on a zonal basis in relation to its lobular disposition. Mandal and Kulshrestha, (1980) reported changes like necrosis and binucleated hepatocytes in the liver of fish, *Clarius batrachus* exposed to 1 ppm of malathion between 45 and 90 days of exposure. Eller, (1971) observed enlarged nuclei and in others acidophilic pigmentation with
eccentric nuclei, in the trout exposed to aldrine. Several reports on pesticide
toxicity reveal such changes in the liver of fish as noticed by Vinod Ghanathay,
1989; Sastry and Malik, 1979).

Jayantha Rao et al., (1985) stated that the concentration of pesticide is
more important in bringing the histological changes in the liver of fish; hence
these changes could be used as a tool for assessing the toxic effects of the
pesticides in aquatic environment. The differences in the degree of liver damages
noticed in the concentrations of the pesticide in the present study may be due to its
mode of action, accumulation, persistence and concentration.

Dimethoate exposure induced marked abnormality in the kidney initiated
with disruption of tubular organization. Thereafter degeneration of tubular
epithelial cells and lymphocytic infiltration was evident. Most of these
pathological changes persisted with vacuolation, clotting of blood in some
sinusoids and glomerular degeneration.

Pesticide accumulates preferentially in the kidney when the body burden of
pesticide increases and new proteins such as metallothionein are synthesized in
the liver and kidney to over come the stress induced by pesticide as reported by
Ooi and Law, (1989). The membranous organelles, such as mitochondria,
endoplasmic reticulum and nuclear envelope, are most easily affected by
dimethoate in which disorganization, rearrangement and malfunction may occur.
Thus, the proximal tubules which posses numerous mitochondria rather than the
distal tubules are easily damaged by dimethoate. The collecting ducts are usually
more resistant to dimethoate exposure. The injuries to collecting ducts are only obvious in the fish exposed to lethal concentration of dimethoate.

Histological changes in the glomerulus are principally proliferative, i.e. an increase in the number of cells of the glomerulus or membranes, where there is a change in the appearance of the capillary wall. The appearance of atrophic or pyknotic nuclei in fish kidney increases with the increase of time course. The phenomenon of nuclear changes in fish is probably similar to that found in other animals (Copius-Peereboom and Copius-Peereboom-Stegeman, 1981). It has been suggested that nuclear changes are induced preceding atrophy and necrosis of cells in other animals. At the beginning, the change may probably from part of a defuse mechanism, leading to the synthesis of metallothionein. However, during prolonged treatment, further accumulation of dimethoate causes a condensation of nuclear material to form direly stained pyknotic nuclei.

The dilation of the lumen of the kidney tubules, degeneration in the hemopoietic tissue rupture in the collecting tubules and necrosis as observed in the present investigation. Such symptoms induced by pesticide in the kidney was observed by many workers in the freshwater fish (Kumar and Pant, 1984; Srivastava and Srivastava 1981; Casillas et al., 1983; Sukumar and Karpagaganapathy, 1986; Gill et al., 1988 and Vardhani and Gowri, 2002). Gupta and Singh (1982) reported degeneration and dissolution of epithelial cells of renal tubules and hypertrophy and necrosis of renal cells of the kidney of *Trichogastre fasciates* exposed to sublethal concentrations of BHC compound. Similar
observations were made by Csepai (1978) in *Cyprinus carpio* exposed to Anthio 40 EC, satox and basudin. The deformation of renal tubules was observed in *Anabas testudineus* chronically exposed to furadon (Bakthavathsalam *et al.*, 1984).

Glomerulonephritis is observed frequently, especially in lethal dose with histopathological changes including thickening of bowman’s capsule, diffuse thickening of glomerular basement membrane and thickening and fibrous of the glomerular tuft as observed by Prashanth, (2002). According to Dubale and Shah (1981) the renal tubules of kidney are the first to be affected by pesticide stress. Rashatwar and Ilyas (1984) reported the histolopathological changes in kidney leads to cloudy swelling of renal tubules in *Nemachellus denisoni* exposed to phosphamidon.

The tubules of the kidney occasionally show pathological features, which are of significant. But much more frequently, tubular necrosis of fibrous occurs as a result of a degenerative process taking place in the haemopoitic tissues. In the present study also the swelling of renal tubules in acute exposure was evident. Changes like vacuolation of epithelial cells of renal tubules and pronounced enlargement of the tubules were observed at sublethal concentration and prolonged exposure to dimethoate.

Necrosis and vacuolation were observed by Dhanapakiam and Premalatha, (1994) in *Cyprinus carpio* exposed to malathion. Sastry and Sharma, (1979) observed a number of striking changes in the histological structure of the kidney of *Channa punctatus* exposed to sub lethal concentration of 0.01 ppm of aldrin for
a span of 30 days and found that the shrinkage of glomerulus was the visible sign of intoxication. Konar, (1979) observed shrinkage and degeneration of glomerulus and vacuolation of tubules in carp chronically treated with hepatochlor. Vinod Ghanathay, (1989) studied histopathological changes in the kidney of *Channa punctatus*, exposed to BHC. He observed that in kidney tissues, after 5 days of exposure to BHC, the glomeruli were shrunken, but some of them were slightly vacuolated and on the 10th day there was a cloudy swelling and hydropic degeneration of interstitial tissues. On the 15th day, they reported that most of the glomeruli were shown complete necrosis and the tubular epithelium was fibrosed.

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 this process the pesticide are excreted through kidney and appears to cause considerable damage.

In view of the literature cited above, it is apparent that in the present investigation, dimethoate at lethal and in sublethal concentration caused considerable histological damages to the organs studied and extend support to the earlier mentioned alterations in oxygen consumption, hematological aspects, ions and protein metabolism.