Chapter-VI

HISTOPATHOLOGY
HISTOPATHOLOGICAL CHANGES IN THE GILL, LIVER, AND KIDNEY OF THE FISH LABEO ROHITA (HAMILTON) EXPOSED TO CYHALOTHRIN

The toxicity of any pollutant is either acute or chronic. The chronic studies include both histochemistry and pathology. Although, toxicant impairs the metabolic and physiological activities of the organisms, physiological studies alone don’t satisfy the complete understanding unless pathological conditions are studied in the tissues under toxic stress. Hence, it is useful to have an insight into the histological analysis. The extent of severity of tissue damage is a consequence of the concentration of the toxicant and is time dependent. Also, the severity of damage depends on the toxic potentiality of a particular compound or pesticide accumulated in the tissue (Jayantha Rao, 1984; Karuppasamy, 2000; Tilak et al., 2005).

The susceptibility of animal tissue to different chemical agents may vary from animal to animal and also within the same animal among the different tissues of the individual. Incorporation of the parent and/or their metabolites in lower organisms in the tissues of fishes, birds and mammals have been recorded to cause serious morphological alterations in vital tissues even at the very low concentrations (Chakraburthy and Konar, 1974; Mathur et al., 1981; Tilak et al., 2001).

Aquatic vertebrates are susceptible to non-target effects, because of their relatively restricted mobility, due to reduced pesticide dispersion leading to lengthy periods of exposure (Smith and Straltar, 1986). Fishes are particularly sensitive to a wide variety of pesticide chemicals, and toxic concentrations may raise not only from spillage of agricultural practices if their use is excessive but also from several other sources. Apart from causing death either directly or due to starvation by destruction of food organisms, many pesticides have been shown to effect growth rate, reproduction and behaviour with the evidence of tissue
damage. Fenvalerate induced histopathological and histochemical changes in the liver of the cat fish *Clarias gariepinus* (Sastry *et al.*, 1979; Sakar *et al.*, 2005).

Modes of action of different chemicals varies leading to varied effects on various body tissues. Some toxins exert their effect locally at the portal of entry, resulting in damage to external surface of the body. Some toxins, when ingested, effect the different regions of gastro intestinal tract. There exists a different group of toxins that do not cause deleterious effects of the portal or entry but they chemical with their varied mode of action affect different tissues thereby bringing about certain architectural changes ultimately culminating in either death of the organism or making the organism less labile for its survival.

Changes similar to pesticide-induced histopathological effects were in the liver of Blue gills starved for 24 h. Similarly, while there were distinct pathological changes in the liver of fish exposed to 0.1 mg pcp/L for 24 h, those that were exposed for 96 h had normal liver (Owen and Rosso, 1981; Venkatesam and Subramaian, 2007). Hence, it is difficult to appreciate the real significance of pesticide induced changes. (Sprague, 1971)underlined that lack of basic information on fish histology makes the interpretation of any observed change difficult.

Eller (1971) described endrin-induced histopathological changes in cutthroat trout and reviewed the gill lesions in fresh water teleosts. Couch (1975) reviewed the histopathological effects of pesticides and related chemicals on the livers of fish and controlled that many of them were non-specific. No characteristic trends of pathological changes for any class of pesticides were seen. On the other hand, examining the spots that were exposed to toxanphene, the control and treated fish may be identified on the basis of gill changes alone (Lowe., 1964). The lamellae were slender and delicate in the controls and clubbed and distally thickened in the exposed fish the changes observed were sever necrosis cloudy swelling and granular vacuoles (Sakar, 2001; Tilak *et al.*, 2007).
Although major advances have been made in recent years in science, the histology and histopathology of fish and other aquatic invertebrates are still to be studied when with mammals (Rand and Petrocelli, 1984; Sakar et al., 2001; Sankar et al., 2005).

A number of pathological changes have been reported in fishes exposed to different organochlorine and organophosphate pesticides (Lowe, 1966; Konar, 1970; Natarajan, 1981; Jayantha Rao, 1982; Jayantha Rao et al., 1985 Girija, 1987; Rama Murthy, 1988; Anita Susan, 1994; Vijaya Lakshmi, 1996; Yacobu, 1999; Ramana Kumari, 1999; Karuppasamy, 2000; Veeraiah, 2001; Tilak et al., 2001a; Tilak et al., 2001b; Tilak et al., 2002; Tilak et al., 2005; Tilak et al., 2007; Venkatesam and Subramaian, 2007).

Sudha Sing and Asja Mehrotra (1999) observed severe damage in the outermost layer serosa and muscle layers, necrosis in intestinal villi and increase in the number and size of mucus cells, under sub-lethal exposure of carbaryl for a period of one month. Ramachandra Mohan (2000) observed significant reduction in the ovarian weight and diameter of developing oocytes and also degeneration of growing oocytes and resorption and yolkly oocytes exhibited atresia in Glassogobius giuris under sub-lethal exposure of malathion.

In the present study, an attempt has been made to observe possible histopathological changes in certain vital tissues like gill, liver, and kidney of the fish Labeo rohita (Hamilton) exposed to sub-lethal and lethal concentrations of cyhalothrin for 8 days.
MATERIAL AND METHODS FOR HISTOPATHOLOGY

Freshwater fish *Labo rohita* was acclimatized to laboratory conditions for 10 days. They were exposed to sub-lethal and lethal concentrations of cyhalothrin for 4 days. At the end of the exposure period, fish were randomly selected for histopathological examination.

Gill, liver, and kidney tissues were isolated from normal (not exposed to the toxicant) and experimental fish. Physiological saline solution (0.75% NaCl) was used to rinse and clean the tissue. They were fixed in aqueous Bouins solution for 48 h, processed through graded series of alcohols cleared in xylene and embedded in paraffin wax. Gills alone were processed by double embedding technique. Sections were cut of 6 μ (microns) thickness; stained with Ehrlich hamatoxillin and Eosin (dissolved in 70% alcohol) (Humason, 1972) and were mounted in *Canada balsam*. Photo monographs were taken with the help of Intel play microscope attached to computer and analyzed.

OBSERVATION AND DISCUSSION

General histology of fish gill:

Teleosts have five pairs of gill arches. In the front four pairs, the slender gill filaments form two lines facing towards the back and these two lines are joined to each other at the base by a gill septum. The last pair of gill arches generally transforms into the pharyngeal bone and does not play a role in respiration.

Numerous semicircular secondary gill lamellae are lined up along both sides of the gill filament. The surface of the gill lamellae is covered with simple squamous epithelial cells and many capillaries separated by pillar cells run parallel along the surface. Numerous semicircular secondary gill lamellae are lined up along both sides of the primary gill lamellae (Plate VI.1&VI.2 Fig 1). The primary gill lamellae consists of centrally placed rod like supporting axis
(SA) with blood vessels on either side. The secondary lamellae, also termed as respiratory lamellae (RL), are highly vascularised and covered with a thin layer of epithelial cells (EC). Blood vessels (BV) are extended into each of the secondary gill filaments. The blood cells of the secondary gill lamellae have a single nucleus which are flattened in appearance. The region between the two adjacent secondary gill lamellae is known as inter lamellar region.

Pathology of Gill tissue under Cyhalothrin toxicity:

Cyhalothrin has induced marked pathological changes in fish gills. The changes include the bulging of tips of primary gill filaments. The secondary gill filaments lost their original shape and curling of secondary gill filaments was also observed. The pillar cell nucleus showed necrosis and development of vacuoles in the secondary gill epithelium. There is a tendency of fusion of disorganised secondary gill filaments (Plate VI.1&VI.2 Fig 2, 3.).

The damage of gills of fish exposed to the higher concentrations (lethal doses) were severe. Shortened and clubbing of ends of the secondary gill lamellae, fusion of adjacent secondary gill lamellae and necrosis in the primary lamellae were well marked. Hyperplasia and hypertrophy of nuclei were also seen. Besides these changes pyknotic nuclei, vacuolization and degeneration of epithelial cells and pillar cells and lifting of the epithelial layer from the secondary lamellae were also significant.

The epithelial layer of secondary lamellae of gill of fish forms a barrier between the fish blood and surrounding water. Gaseous exchange needed to sustain life takes place through this barrier and any thickening induced by physical, chemical or biological agents hinders the respiratory function of this organ (Eller, 1971).

In fish, gill is the first organ to which the pollutant comes into contact. Hence, it is more vulnerable to damage than any other tissue. The proliferative gill lesions are often observed after exposure of fish to water soluble toxicants.
LEGEND FOR FIGURES

Plate VI.1

Fig.1. Control: Normal Gill lamella of *Labeo rohita* after 24 hrs

- **PGL**: Primary gill lamella
- **SGL**: Secondary gill lamella
- **PC**: Pillar cell
- **CA**: Central axis

Fig.2. Sublethal: Normal Gill lamella of *Labeo rohita* exposed for 24 hrs to sublethal concentration of Cyhalothrin.

- **PGL**: Primary gill lamella
- **HGL**: Hyperplasia in gill lamella
- **EC**: Erythrocyte
- **DGSL**: Degenerated Secondary lamella

Fig.3. Lethal: Normal Gill lamella of *Labeo rohita* exposed for 24 hrs to lethal concentration of Cyhalothrin.

- **DGSL**: Degenerated Secondary lamella
- **HGL**: Hyperplasia in gill lamella
- **MC**: Mucus cell
- **ASL**: Atrophy of Secondary lamella
LEGEND FOR FIGURES

Plate VI.2

Fig.1. Control: Gill lamella of *Labeo rohita* after 96 hrs.

- PGL : Primary gill lamella
- SGL : Secondary gill lamella
- PC  : Pillar cell
- CA  : Central axis

Fig.2. Sublethal: Gill lamella of *Labeo rohita* exposed for 96 hrs to sublethal concentration of Cyhalothrin.

- PGL : Primary gill lamella
- HGL : Hyperplasia in gill lamella
- EC  : Erythrocyte
- DGSL : Degenerated Secondary lamella

Fig.3. Lethal: Gill lamella of *Labeo rohita* exposed for 96 hrs to lethal concentration of Cyhalothrin.

- DGSL : Degenerated Secondary lamella
- HGL : Hyperplasia in gill lamella
- MC  : Mucus cella
- ASL : Atrophy of Secondary lamella
The nutritional gill disease consists of lamellar epithelial hyperplasia with eventual fusion of secondary lamellae near the tips of gill filaments (Cowey and Roberts, 1978). The biological function of the inflammatory response is to destroy “WALL OFF” irritating substances so that damaged tissue may be healed.

A number of pathological changes have been reported in fish exposed to different organochlorine and organophosphorous and synthetic pyrethroid compounds. Exposure of ‘Sockeye’ salmon fry to the butoxy ethyl ester of 2, 4-D for 96 hrs (1 mg/l) resulted in hypertrophy and hyperplasia of the epithelial cells of the gills (McBride, 1981). Eller (1971) described endrin induced histopathological changes in cut throat trout and reviewed the gill lesions in freshwater teleosts.

Baticodos et al., (1991) reported slight hyperplasia of gill epithelium in pinaeus monodon exposed to gusathon, a commonly used organo phosphate. Inflammatory alterations of lamellar epithelium and hyperplasia were reported in the gills of freshwater major carp Cirrhinus mrigala (Hamilton) during 48 h exposure to sub-lethal dose of malathion (Roy and Datta, 1991). Edema with lifting of lamellar epithelium and hyperplasia of lamellar epithelium were observed in the gills of all cat fish containing residues of endosulfan (Nowak and Barbara, 1992). Similar findings were noted in the gills of rainbow trout on exposure to zinc sulphate by Skidmore and Tovell (1972).,Karuppasamy.(2000). Sakar.et al.,(2005).,Tilak.et al.,(2005). Sunitha and Sahai (1983) reported swellings of inflammation in almost all the respiratory lamellae of gills of Rasbora daniconius on exposure to HCH.

Anitha Kumari and Shree Ram Kumar (1997) observed decreased carbohydrate activity in the secondary lamellae and also in the respiratory epithelium of the fresh water teleost Channa punctatus under exposure to the polluted water of Hussain Sagar and states that the degeneration of respiratory epithelium and damages of gill tissue causes a decrease in energy metabolism.
Hyperplasia of gill filaments, fusion of gill filaments due to separation of epithelium, necrosis of gill epithelium, degeneration of pillar cells, development of vacuoles in the epithelium are the pathological changes observed in cyhalothrin (Dicofol 18.5 % E.C) exposed fish. Similar changes were observed in rainbow trout exposed to sub-lethal concentrations of monocrotophos (Vijayalakshmi, 1994) to fenvalerate in *Labeo rohita* by Tilak *et al.*, (2001a, 2001b) to cypermethrin in *Labeo rohita* (Veeraiah, 2001), Copper Sulphate in *Oreochromis mossambicus* Vekatesam and Subramaian, (2007), Butachlor and Machete in *Channa punctatus* (Tilak.,et.,al.,2007).

**General histology of Liver:**

The surface of liver is covered with serous membrane and some connective tissue extends inward into parenchyma. It is composed of parenchymal cells (hepatic cells) and lattice fibres, which support the former. Hepatic cells are roundish polygonal, 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. Fairly large quantities of lipid glycogen granules are also observed in the cytoplasm of fish hepatic cells (Plate VI.3 & VI.4 Fig.1.). 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 sites for some nutrients and detoxification is an another function attributed to them.

**Pathology of Liver tissue under Cyhalothrin toxicity**

Cyhalothrin has induced discrete pathological changes in the liver tissue of the fish *Labeo rohita* These changes include degeneration of cytoplasm in hepatocytes, atrophy, formation of vacuoles, rupture in blood vessels, necrosis and disappearance of hepatocytic cell wall and disposition of hepatic cords (Plate VI.3 & VI.4 Fig. 2, 3).
Liver, the first organ to face any foreign molecule through portal circulation is subjected to more damage (Jayantha Rao, 1982). Liver is an important organ of detoxification which breaks down toxic substances and metabolites of administered substances. This breakdown is carried out by endoplasmic reticulum of hepatocytes. Due to these reasons the hepatic cells are damaged severely.
LEGEND FOR FIGURES

Plate VI.3

Fig.1. Control: Normal structure of Liver in *Labeo rohita* after 24 hrs.

- **HC**: Hepatic Cell
- **N**: Nucleus
- **BC**: Bile canaliculus
- **LGG**: Lipid and glycogen granules

Fig.2. Sublethal: Normal structure of Liver in *Labeo rohita*, exposed for 24 hrs to sublethal concentration of Cyhalothrin.

- **FV**: Formation of vacuoles
- **ABS**: Appearance of Blood streaks among hepatocytes
- **BC**: Bile canaliculus
- **DGHP**: Degenerated hepato pancreatic tissue

Fig.3. Lethal: Normal Structural of Liver in *Labeo rohita*, exposed for 24 hrs to lethal concentration of Cyhalothrin.

- **FV**: Formation of vacuoles
- **ABS**: Appearance of Blood streaks among hepatocytes
- **BC**: Bile canaliculus
- **DGHP**: Degenerated hepato pancreatic tissue
LEGEND FOR FIGURES

Plate VI.4

Fig.1. **Control:** Normal structure of Liver in *Labeo rohita* after 96 hrs.

- **HC** : Hepatic Cell
- **N** : Nucleus
- **BC** : Bile canaliculus
- **LGG** : Lipid and glycogen granules

Fig.2. **Sublethal:** Normal structure of Liver in *Labeo rohita*, exposed for 96 hrs to sublethal concentration of Cyhalothrin

- **FV** : Formation of vacuoles
- **ABS** : Appearance of Blood streaks among hepatocytes
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Fig.3. **Lethal:** Normal Structural of Liver in *Labeo rohita* exposed for 96 hrs to lethal concentration of Cyhalothrin.

- **FV** : Formation of vacuoles
- **ABS** : Appearance of Blood streaks among hepatocytes
- **BC** : Bile canaliculus
- **DGHP** : Degenerated hepato pancreatic tissue
Mathur (1965) reported that in fish *Ophiocephalus punctatus* exposed to the toxicant resulted in vacuolation and necrosis in liver. Dubale and Shah (1979) reported that *Channa punctatus* under malathion toxicity showed the degenerative changes in liver. Rashatwar and Ilyas (1984) reported that in teleost fish *Nemachelius denesoni* (Day) exposure to phosphamidon caused highly vacuolated and cloudy swelling and even the connective tissue was damaged in liver. Ansari *et al.*, (1987) reported significant alterations in the hepatic cell count and the nucleocyton plasmic index in the liver of zebra fish *Brachydanio rerio* (cyprinid) exposed to 0.9 mg/l concentration of malathion.

A few other reports are available which deal with the other than organo phosphate pesticides’ effect on histology. Cruz (1989) reported that formalene treatment caused cloudy swelling, haemorrhage, deposition of pigments and necrosis in liver of milk fish Chanoes fingerlings. Radhaiah and Jayantha Rao (1992) reported moderate cytoplasmic degeneration in hepatocytes, formation of vacuoles, rupture in blood vessels and appearance of blood cells among hepatocytes, formation of vacuoles, picnotic nuclei in the liver of *T. mossambica* exposed to fenvalerate. Similar changes were observed in three Indian major carps *Catla catla, Labeo rohita* and *Cirrhinus mrigala* exposed to fenvalerate by Anita Susan (1994) and Vijayalakshmi (1994) observed the same changes in *Labeo rohita* under fenvalerate and monocrotophos synergistic exposure. Yacobu (1999) reported the same observations in *Ctenopharyngodon idellus* exposed to fenvalerate. Anitha Kumari and Shree Ram Kumar (1997) observed an uneven distribution of carbohydrate content and a drastical decrease in the hepatic cells of the freshwater teleost upon exposure to polluted waters of Hussain Sagar lake. Tilak *et al.*, 2001a, 2001b reported the same degenerative changes in *Labeo rohita* and *Ctenopharyngodon idellus* under chlorpyrifos and fenvalerate toxicities. Tilak.,et.,al.,(2005) *Catla catla* under Chlorpyrifos toxicities.. Tilak.,et.,al.,(2007) *Channa punctatus* exposed to sub-lethal concentration of Butachlor.
Cyhalothrin has induced discrete pathological changes in the liver tissues of all the fish *Labeo rohita*. The pathological changes noticed in the liver might effect the physiological activity of the fish such as reduction in enzyme synthesis (Sastry and Sharma, 1979; Rashatwar and Ilyas, 1984). This reduces the functional ability of liver which indirectly effects all metabolic activities of the organism.

The degenerative changes are intensified in lethal exposures. They include degeneration of cytoplasm in hepatocytes, atrophy, formation of vacuoles, rupture in blood vessels, necrosis and disappearance of hepatocyte wall disposition of hepatic cords decrease in size of nucleus pyknotic and vacuolar degeneration within the nucleus was evident (Plate VI.3 & VI.4 Fig. 2, 3).

**General Histology of fish Kidney**

Teleostean kidney consists of head and body kidneys. Head kidney is the anterior portion of the kidney and consists of lymphoid tissue. Body kidney is composed of many nephrons and interstitial lymphoid tissue. The interstitial tissue is the major haematopoitic tissue in the body. Each nephron consists of two parts, the glomerulus and the urinary tubule. The glomerulus capsule consists of an inner and outer layer of single flattened epithelia. Renal tubule consists of single layer of epithelial cells. Mesangium fills the space between the loops of glomerular capillaries.

Renal tubules are thin and short in the neck segment. The proximal convoluted segment is divided into two parts i.e. segment I and segment II. The renal tubules are composed of cuboidal epithelial cells with densely arranged microvilli in the tubular lumen. In segment II, renal tubules are composed of cuboidal epithelial cells. Cilia and microvilli are found in the tubular lumen. In the distal convoluted segment, epithelial cells have no microvilli. The cells of this segment are stained with eosin more faintly than those of proximal convoluted
segment (Plate VI.5 & VI.6, Fig.1). Thus, it is easy to distinguish between proximal and distal convoluted segments under light microscopy (Oguri, 1982).

**Pathology of Kidney tissue under Cyhalothrin toxicity**

Renal tissues of the fish *Labeo rohita* under cyhalothrin toxicity evidenced marked pathological changes. Highly degenerative changes were observed in haemopoitic tissue which include severe necrosis, cloudy swelling in renal tubules, cellular hypertrophy and granular cytoplasm. The epithelial cells of the distal convoluted tubule decreased in size. The interstitial renal tissue was less effected. Renal interstitial tissue showed formation of vacuoles and cellular contours were not clearly distinguished (Plate VI.5 & VI.6, Fig. 2, 3).

From the body of fish, the waste products are eliminated through kidney. The non-detoxified pesticide molecules must be eliminated through the kidney of fish and hence, it is susceptible to chemical compounds when exposed to lethal or sublethal dose of cyhalothrin while it was eliminated through kidney might have caused degenerative changes in renal tubule and glomeruli, i.e. necrosis in the proximal tubules with development of vacuoles (Plate VI.5 & VI.6, Fig. 2, 3).

Srivatsava and Srivatsava (1982) reported the bursting of glomeruli and tubules and degeneration of cellular boundaries and clumping of glomeruli at some places in the kidney of *Cirrhinus mrigala* exposed to urea. Cloudy swelling of renal tubule, marked loss of haemopoitic tissue, shrinkage of glomeruli were reported in *Nemachilus denisoni* (Day) exposed to phosphamidon (Rashatwar and Ilyas, 1984). In monocrotrophos treated mice thickening of glomerular basement membrane, tubular degeneration and compensatory dilation and the fecal collection of chronic inflammatory cells in the interstitial tissue were reported by Malaya Gupta et al., (1988).
LEGEND FOR FIGURES

Plate VI.5

Fig.1. Control: Normal structure of Kidney in *Labeo rohita* after 24 hrs.

PCS : Proximal Convolut ed Segment
DCS : Distal Convolut ed tubule

Fig.2. Sublethal: Normal structure of Kidney in *Labeo rohita*, exposed for 24 hrs to sublethal concentration of Cyhalothrin.

DART : Degeneration and atrophy in renal tubules
DGHT E : Degenerating haemopoietic tissue with erythrocytes
FV RIT : Formation of Vacuoles in the renal interstitial tissue

Fig.3. Lethal: Normal Structural of Kidney in *Labeo rohita* exposed for 24 hrs to lethal concentration of Cyhalothrin.

DL : Decreased Lumen in tubules
DGHT E : Degenerating haemopoietic tissue with erythrocytes
FV RIT : Formation of Vacuoles in the renal interstitial tissue
LEGEND FOR FIGURES

Plate VI.6

**Fig.1. Control:** Normal structure of Kidney in *Labeo rohita* after 96 h.

- PCS : Proximal Convoluted Segment
- DCS : Distal Convoluted tubule

**Fig.2. Sublethal:** Normal structure of Kidney in *Labeo rohita*, exposed for 96 h to sublethal concentration of Cyhalothrin.

- DART : Degeneration and atrophy in renal tubules
- DGHTE : Degenerating haemopoietic tissue with erythrocytes
- FVIRIT : Formation of Vacuoles in the renal interstitial tissue

**Fig.3. Lethal:** Normal Structural of Kidney in *Labeo rohita* exposed for 96 h to lethal concentration of Cyhalothrin.

- DL : Decreased Lumen in tubules
- DGHTE : Degenerating haemopoietic tissue with erythrocytes
- FVIRIT : Formation of Vacuoles in the renal interstitial tissue
Degenerative changes in epithelial cells of proximal tubules and haemopoitic tissues, severe necrosis in the proximal tubules leading to the formation of vacuoles, degenerative changes in epithelial cells of collecting tubules of *Tilapia mossambica* exposed to fenvalerate has been reported by Radhaiah (1988). Cytological break down of glomeruli was reported in kidneys of the stickle back *Gasterosteus aculeatus* exposed to dissolved cadmium (Oronsaye, 1989). Anitha Kumari and Shriram Kumar (1997) observed mild activity of carbohydrates in the cytoplasm, nuclei and the luminar border of the proximal and distal tubules in the kidney of the fresh water teleost *Channa punctatus* under exposure to the polluted water of the Hussain Sagar Lake.

The present observations are in agreement with the reports of Goel and Veenagarg, 1980; Mandal and Kulshreshtha, 1980; Dubale and Awasthi, 1982; Malaya Guptha et al., 1988; Ramana Kumari, 1999; Yacobu, 1998; Tilak et al., 2001a, Tilak et al., 2001b and Tilak et al., 2002) who observed renal damage, rupture in the glomeruli and reduced renal tubules and its lumen in *Channa punctatus* exposed to Dabb. El-Zalabani and Soliman (1981) and Feng et al., (1982) also reported necrosis in renal epithelium, swelling of mitochondria in the renal tubules in animals administered with methothrin and pyrethrin respectively. Such sort of pathological conditions causing dis-function of kidney tissue have been reported under pesticide toxicity by Radhaiah (1985and1988); Ramamurthy.(1988); Karuppasamy.(2002), Tilak.,et al.,(2005.,2007), Venkatesam. (2007).

Thus, when fish is exposed to pesticide, they suffer irreparable architectural changes in various vital organs making the fish less fit for better survival. These histopathological changes can alter various physiological activities of the fish.