



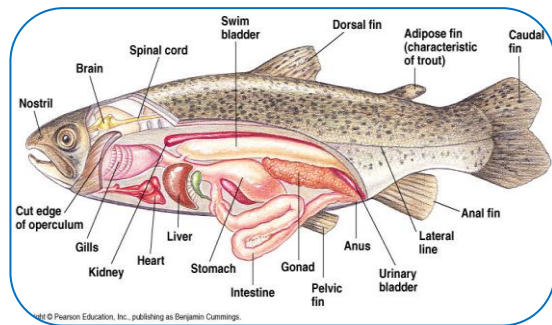
## EFFECTS OF CHEMICAL ON GILL, LIVER AND KIDNEY OF FRESH WATER FISH CIRRHINUS MRIGALA (HAMILTON)

Shazia Rahman

Research Scholar , L.N.M.U, Darbhanga.

### ABSTRACT :

*Cirrhinus Mrigala (Hamilton), the Indian fresh water big carp fish, has been exposed to 96 h optimistic concentrations of the Sublethal(Static, 96 h LC50). The static values of LC50 for 24, 48 and 96h were 14,260 mg/L, 13,710 mg/L and 11,900 mg/L. Vital tissues such as gill, liver and kidney have been isolated and examined for their histological changes. Damages of the fish that were exposed to sub-lethal confident concentration were found in primary and secondary lamellae. Determined damage was more in relation to sublethal exposure in gills in the fish exposed to higher concentration. The fusion of the neighbouring secondary gill lamellae and the necrosis of the main gill lamellae was distinctly shortened and clubbed. There have been several important degenerative changes to *Cirrhinus Mrigala* fish liver exposed to the sublethal confidential concentration. Changes in the shape and size of hepatocytes, breakdown and degeneration of hepatic cells, development of more vacuoles and so on entail pathological changes in the liver. Vacuolization, degeneration of cell membrane, damage to haemopoietic tissue and renal tubes and nuclei hypertrophy were changes in the kidney of fish *Cirrhinus Mrigala* which were exposed to sublethal confident concentration, etc. The adjustments that occurred were addressed with the available literature in depth in the photographs.*



**KEYWORDS :** *Cirrhinus Mrigala, confidor, gill, Liver, Kidney, haemopoietic tissue and renal tubules.*

### INTRODUCTION :

The population density, industrialization and farming practises have risen over the last few decades, resulting in more and more waste entering fresh waters. In recent decades, pollution of fresh water with various contaminants has become a problem. The natural aquatic water supply may be polluted extensively by heavy metals from the home, industry or other industrialised activities (Velez, 1998). The ecologically sound equilibrium and the diversity of marine ecosystems are impaired by heavy metals (Farombi et al., 2007). Strong metals and chemicals are harmful to animals and cause many death and/or sublethal disease in both invertebrate and vertebrate aquatic animals (Wilbur, 1969) in heart, kidneys, reproductive system, respiratory system or nerve system. Heavy metals can accumulate in the fish tissue and cause morphological alterations (Monteiro et al., 2005). A global trend is being followed to supplement physical and chemical parameters with biomarkers in aquatic pollution monitoring to determine the harmful effects of contaminants on aquatic species (Abdel et al., 2012). In the diagnosis, aetiology and disease prevention, histopathology is of primary importance. There is still no data on tropical fish and effects on various fish tissues (Mela et al. 2007).

Poultry is susceptible and their tissue is vulnerable to pathological effects at various levels of pesticides (Murthy 1986). The metabolic and physiologic processes of the species are affected by toxicants, but such experiments on their own do not satisfy a full understanding of toxic stress pathological conditions of tissues. Therefore an insight into histological research is useful. The significance of studying the histopathological changes in different fish organs caused by different pesticides has been well reported. (Eller, 1971; Smith et al., 1972; Bansal, 1979; Mallatt, 1985; Roy et al., 1986; Richmonds and Dutta, 1989; Roy and Munshi, 1987–1991; Tilak et al., 2001 and Veeraiah, 2001).

Historopathological changes of the Cirrhinus Myigala confidant concentration (96h, LC50), which have been recorded in gill, liver and kidney tissues over 96 hour, were therefore attempted.

A relatively recent systemic chloride, nicotinyl insecticide, is Confidor (Imidaclopride). The tobacco toxin 'nicotine' is chemically related. The nervous system functions like nicotine (Caroline Cox, 2001). The chemistry acts by preventing the stimuli transmitted through the test organism's nervous system.

## MATERIALS AND METHODS

Freshwater fish Cirrhinus Mrigala (Hamilton) of size ranging from 5 to 6±2Cm were acclimatized in laboratory conditions for one week. Static system for exposure of 24, 48, and 96 hours was used for experiments with confidant commercial grade toxicity. The 24, 48 and 96 hours of LC50 values included 14.260 mg/l, 13.710 mg/i respectively and 11.900 mg/l. During 96 hours, fish were exposed to the concentration of confidant sub-lethal (96 h, LC50). One day before the experiment, the feeding of fish was halted. Fish were randomly selected for histopathological analysis at the end of each exposure cycle.

The essential tissues such as gill, liver and kidney were monitored and fish exposed. In order to rinse and clean the tissues, the saline physiological solution (0.75% NaCl) was used. They have been set in the fluid of the watery boe. They went through various alcohol sequences. They were cleared in paraffin wax in xylene. Parts of Ehrlich hematoxylin / eosin were cut to 6 µl of the thickness, dissolved in 70 percent alcohol and balsamed (Humason, 1972). Sections were mounted in Canada. The diapositives were viewed by microscope. Possible changes in tissues of confidentially handled fish were detected and the Olympus Microscope was used as photomographs.

## RESULTS AND DISCUSSION

### Pathology of gill

For any pollutant, Gills are the main pathway of entry. The triggers have shown major degenerative changes in the exposed fish's gills. In primary and secondary gill lamellae, damage of gill Cirrhinus Mrigala fish that was exposed to sublethal confidential concentration was observed. Secondary Imellae have been reduced and fused, vascular degeneration, primary gill tip bumps, Primary lamellae necrotics, hyperplasia, and hypertrophy were observed. (Fig. 1A and B).

(1971) Munshi and Singh, Mallatt (1985); Roy et al. (1986) Richmonds et Dutta (1989), Powell and al. (1992), Dutta et al. (1993) and Tilak and Veeraiah (2001) documented historological changes to gills of fish from pesticides and chemicals. (2001). The epithelial layer of the secondary lamellae of the gill of fish is a barrier between the fish blood and surrounding water. This barrier is used to improve life-supporting exchange and to thicken the respiratory function of this organ by the use of physical, chemical or biological substances (Eller 1971). Due to damage from the secondary lamellae, the propagation potential will be limited, and fish subject to certain forms of hypoxia will therefore be presented (Hughes et al., 1976). Jyotsnaetal has reported similar results in fish exposed to chlorine and malathion. (1984).(2008). Similar changes were also observed in Vijayalakshmi, Tilak (1996), Tilak and Veeraiah (2001) in the gills of the Cirrhinus Mrigala fish exposed to chloropyrifos.

For increased focus and prolonged visibility, the club and spatial forming of the adjoining two gilletts and the epithelic layer of secondary lamb occurred. This raising and swelling epithelium can be used to protect the internal structure from contaminated water (Dutta, 1995).

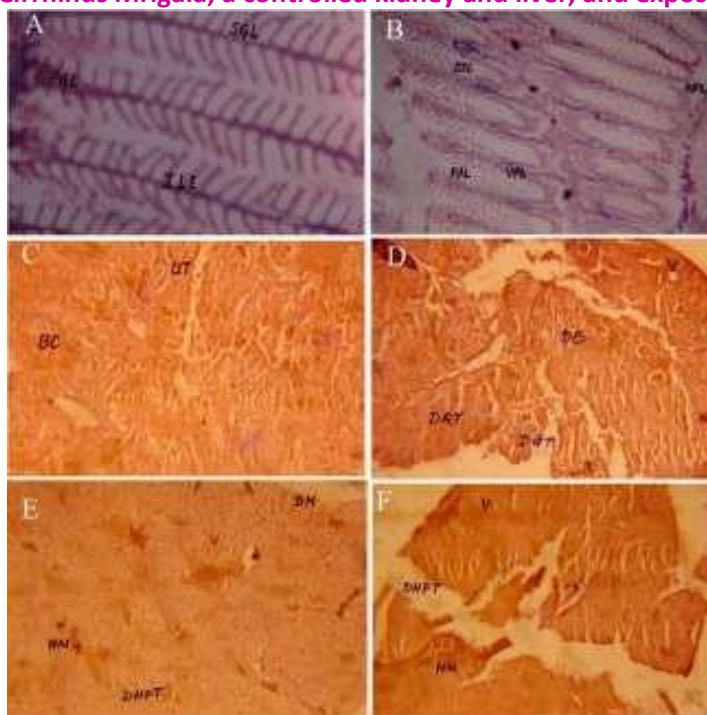
In certain cases, hyperplasia can be modified to shield underlying tissues from irritating by any

organism (Meissner and Diamandospoules, 1977). However, increased epithelial layer thickness, which involve hyperplasia of mucous cells and melting of neighbouring secondary lamellae, will not only reduce the surface available for oxygen extraction, but also increase the gap between water and blood diffusion from oxygen (Skidmore & Tovell, 1972). Thus, while the protective role of hyperplasia may in fact exist, it also may inhibit the respiratory, secretory and excreta functions (Eller, 1975). Change in gills observed by this study can lead to the death of fish as a result of serious physiological problems (Richmonds and Dutta, 1989; Dutta et al., 1993).

**Liver:** Life changes of the *Cirrhinus Mrigala* fish were characterized by vacuolization of the cytoplasm and hepatocyte degeneration on the periphery and changes in the form of the hepatocytes. The reduction in the hepatic cell diameter was due to cell shrinking. The nuclei became pyknotic and eccentric (Fig. 1C and D).

In detoxification, the liver plays a significant function. For the bulk of toxicants, this is the site of biotransformation. The liver of *Channa punctatus* treated with the same endrin has been found by Sastry and Sharma (1978). Changes in fish liver caused by the pesticide can be considered as a contamination stress index for the identification of fish (Cough, 1975). The histopathological changes resulting from malathion exposure can affect the functionality of the liver, which can contribute to the dysfunction of many fish organ systems. The death of fish will lead to changes in species (Dutta et coll., 1993b). This can lead to a change in population structure.

**Figure 1: T.S. *Cirrhinus Mrigala*, a controlled kidney and liver, and exposed fish.**



**Figure 1: A. Control Gill, B. Sub-lethal exposure Gill, C. Control Kidney, D. Sub-lethal exposure of Kidney E. Control liver and F. Sub-lethal exposure of liver.**

**Kidney:** Microscopy research revealed cell and renal tubular necrosis, cloudy renal tubular swellings, cytoplasm degeneration within pyknotic nuclei and disorganization of the connective tissues (Hamopoetic tissue), in which the kidney of fish *Cirrhinus Mrigala* was treated with the sub-lethal concentration. The cell membrane was also disintegrated, nuclei hypertrophy, vacuoles and glomerulin swelling (Fig.1).

Similar findings were observed in gold fish at chronic exposure to DDT until some point (Rudd and

Linelly, 1956). Khillare and Wagh (1987), which illustrated the long-term toxicity of freshwater fish stigma Barbus' kidney, also revealed the same findings. Earlier in the *Ophiocephalus punctatus* treated with endrin (Matheur 1969) degeneration of epithelial cells and loss of parenchymatic cells of renal tubules was observed. The abnormality of the renal tubes in *Cirrhinus Mrigala* was also produced by heptachlor (Konar, 1970). In hemopoietic tissue that involves extreme necrosis, cloudy swelling in renal tubules, cell hypertrophy and granular cytoplasm, *Cirrhinus Mrigala* was highly degenerative in the cypermethrine exposed (Veeraiah 2001).

## CONCLUSIONS

The widely grown fish *Cirrhinus Mrigala* in this area is greatly affected by pesticide contamination from nearby fields of agriculture and irrigation channels. The compounds build up in the adipose tissue and are then absorbed in the body by various tissues. This leads to improvements in anatomy or physiology. In certain cases, therefore, fish eventually die. Consumers may have a health issue with the fish infected by the pesticide. The quantity of pesticide must therefore be controlled in aquatic medium in order to avoid fish death.

## REFERENCES:

1. Anthony Reddy P, Veeraiah K, Tata Rao S and Ch Vivek; THE EFFECT OF CONFIDOR ON HISTOLOGY OF THE GILL,LIVER AND KIDNEY OF FISH LABEO ROHITA(HAMILTON); INTERNATIONAL JOURNAL OF BIOASSAYS ISSN:2278-778X CODEN:IJBNHY
2. Dutta HM, CR Richmonds and T Zeno. Effects of diazinon on the gills of bluegill sunfish *Lepomis macrochirus*. J. Environ. Toxic. Oncol, 1993a, 12(4): 219 – 227.
3. Dutta HM, S Adhikar, NK Singh, PK Raj and JSD Munshi. Histopathological changes induced by malathion in the liver of fresh of Water Catfish, *Heteropneustes fossilis* (Bloch). Bull. Environ. Contam. Toxicol, 1993b, 51: 895 – 900.
4. Eller LL. Gill lesions in freshwater teleosts. In: Riubelin W.E ogalo, G. (ed). The pathology of fishes. Univer. Wis-press., 1975, 305-330.
5. Khillare VK and Wagh SB. Effects of the long term of toxicity on kidney of the freshwater fish *Barbus stigma*. 1987, Abs. Proc. 8th, A.E.B. Ses and Symp Jammu P: 3.
6. Burrow RE. Effects of accumulated excretory product on hatchery reared Salmonids. U.S. Fish wildlife Serv. Res. Rep. 1949, 66: 1- 12.
7. Richmonds C and H M Dutta. Histopathological changes induced by Malathion in the gills of bluegill *Leopomis macrochirus*. Bull. Envrion. Contam. Toxicol, 1989, 43: 123 – 130.