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HISTOPATHOLOGICAL CHANGES IN THE GILL OF THE FISH *CYPRINUS CARPIO* EXPOSED TO PERMETHRIN, A SYNTHETIC PYRETHROID OF CLASS I TYPE

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ABSTRACT: The gill of the fish is the primary target organ culminating the toxic affect ultimately leading to death of the fish. The histopathological changes in the gill of the fish Cyprinus carpio after exposure to Permethrin a synthetic Pyrethroid of class I type and the result showed that vascular degeneration cloudy swelling, necrosis and other pathological changes.

Key words: Cyprinus carpio, Permethrin, synthetic Pyrethroid class I type, Necrosis.

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INTRODUCTION

'Water water every where, nor even a drop to drink' may be changed as water water every where nor even a drop of pure water to drink. Due to rapid advanced modern culture, money to earn in quick time has resultant damage to mothers earth, certainly not a proper 'placental' connection at the present juncture. Mans activities make the environment none other than contamination, rendering unfit, of the natural resources of which water is the major component.

One of the activities of humans, recognised as anthropological activities viz., agriculture and aquaculture by profession to earn the currency, use of 'cides' to combat damage by the pests have to be reduced at a minimal level is none other than the usage of chemicals to increase the produce.

The resultant of such is a change in the ambient water quality, resulting contamination, rendering unfit for its usage.

One of the important 'pesticides' are none other than synthetic Pyrethroid a new class of organic compounds, sharing the qualities of both organochlorines and organophosphorous, a modified forms of natural pyrethrins which are of different types targeted on plant enemies which maintain an imbalance of production and damage, Permethrin is one of the kind of the synthetic Pyrethroid the present work is aimed to study its effect on the entry of the toxicant in the nektonic fish, a water dweller and conduit of energy in the ecosystem. While entering, the contaminant as a toxicant also changed the architectural contour of the gills, the modified pharyngeal pouches and is the route of entry of dissolved oxygen through them may be impaired and in fact impaired that the present study resulted, to the extent of severity of tissue damage due to toxic potentiality, and the damage which are directly proportional.

MATERIALS AND METHODS

Fresh water fish, the common carp *Cyprinus carpio*, (size 3-5 cm; length 3-6 g in weight) acclimatized in the laboratory conditions for one week. They were exposed to sub lethal and lethal concentrations of Permethrin technical grade (0.24 μ g/L and 2.4 μ g/L) for 8 days in the laboratory. At the end of the exposure period, the gills of the fish were randomly selected for histopathological examination.

Gill tissue was isolated from normal (not exposed to toxicant) and experimental fish. Physiological saline solution (0.75% NaCl) was used to rinse and clean it. They were fixed in aqueous Bouins solution for 48 h, processed through graded series of alcohols cleared in xylene and double embedded technique in paraffin wax. Section were cut 6 μ (microns) thickness; stained with Ehrlich haematoxillin and Eosin (dissolved in 70% alcohol) Humason [15] and were mounted in Canada balsam. The photographs at 200x magnification were taken with computer aided microscope (Intel play Qx3, Intel Corporation, made in China).

RESULTS 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 the 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 semi circular secondary gill lamellae are lined up along both sides of the primary gill lamellae (Fig.1).

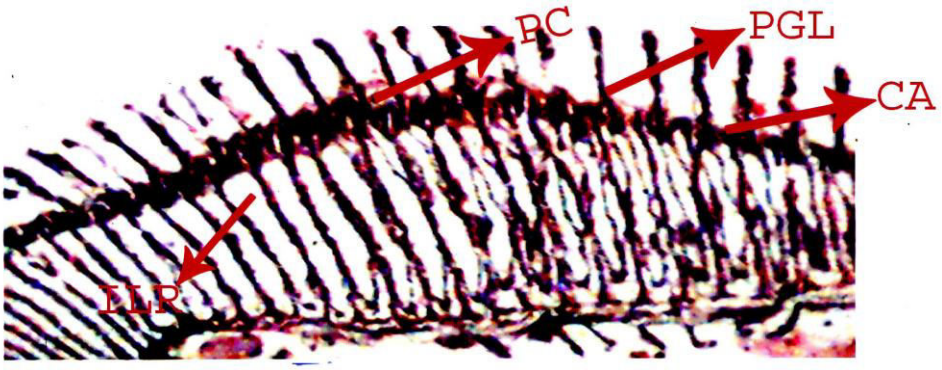

Fig.1 Control Gill of *Cyprinus carpio*

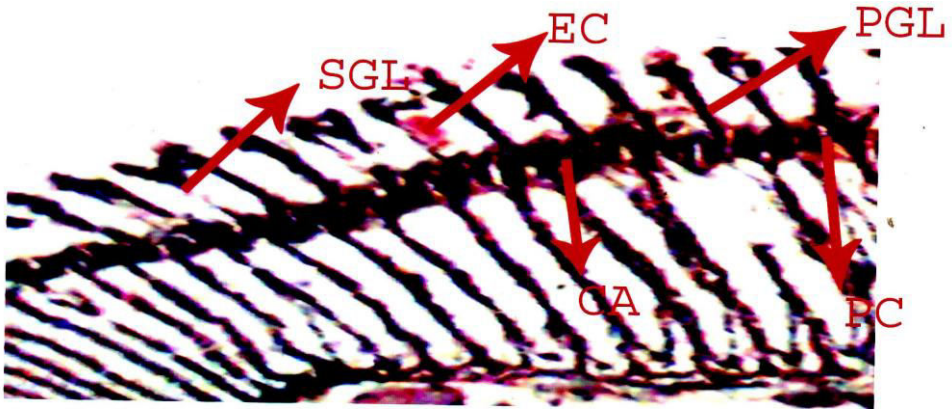

Fig.2 Sublethal exposed Gill of *Cyprinus carpio*

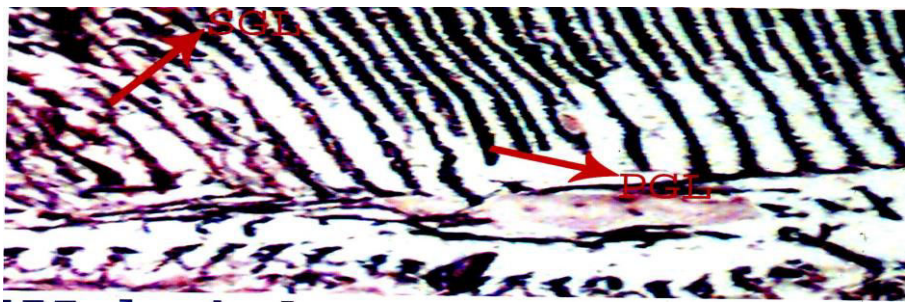

Fig.3 Lethal exposed Gill of *Cyprinus carpio*

The photographs at 200x magnification were taken with computer aided microscope (Intel play Qx3, Intel Corporation, made in China).

(PGL) Primary gill lamella
(SGL) Secondary gill lamella
(PC) Pillar cell
(ILR) Inter lamellar region
(CA) Central axis
(EC) Erythrocyte

The primary gill lamellae consist 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 is flattened in appearance. The region between the two adjacent secondary gill lamellae is known as inter lamellar region.

Pathology of Gill Tissue under Permethrin toxicity

Permethrin has induced marked pathological changes in the gills (Fig 2). 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.

The damage of gills of fish exposed to the higher concentrations (lethal doses) were severe (Fig.3). 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 [11].

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. The nutritional gill disease consists of lamellar epithelial hyperplasia with eventual fusion of secondary lamellae near the tips of gill filaments Cowey & Roberts [9]. 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, organophosphorus and synthetic pyrethroid compounds. Eller [11] described endrin induced histopathological changes in cut throut trout and reviewed the gill lesions in fresh water teleosts. Others have reported on these lines to different pesticides, as reviewed by Ullah and Jallil [27] and Sankar Murthy [22].

Baticodos et al [6] reported slight hyperplasia of gill epithelium in *Penaeus monodon* exposed to gusathion, commonly used organophosphate. Inflammatory alterations of lamellar epithelium and hyperplasia were reported in the gills of fresh water major carp *Cirrhinus mrigala* (Hamilton) during 48 hours exposure to sublethal dose of malathion, Roy & Datta [19]. Oedema with lifting of lamellar epithelium and hyperplasia of lamellar epithelium were observed in the gills of all cat fish containing residues of endosulfan Nowak – Barbara [18]. Similar findings were noted in the gills of rainbow trout on exposure to zinc sulphate by Skidmore and Tovell [23]. Sunitha and Sahai [24] reported swellings of inflammation in almost all the respiratory lamellae of gills of *Rasbora daniconius* on exposure.

Anitha and Kumar [3] observed decreased carbohydrate activity in the secondary lamellae and also in the respiratory epithelium of the freshwater 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 exposed to sublethal concentrations of different chemicals reported by Vijaya Lakshmi and Tilak [29]; Tilak et al [25, 26] and Veeraiah [28].

Evans [12] reported the actual mechanism how O_2 is impaired in the gill, which clearly explains the way of lacking proper neuronal control and consumption is monitored, by the enzyme carbonic anhydrase. Chloride cells are involved for the exchange of HCO_3^- ions which in turn take O_2 from the aqueous medium. The study emphasized when the fish is exposed to the toxicant continuously, gill epithelium is damaged there by intake is reduced (Fig.4, 5 & 6).

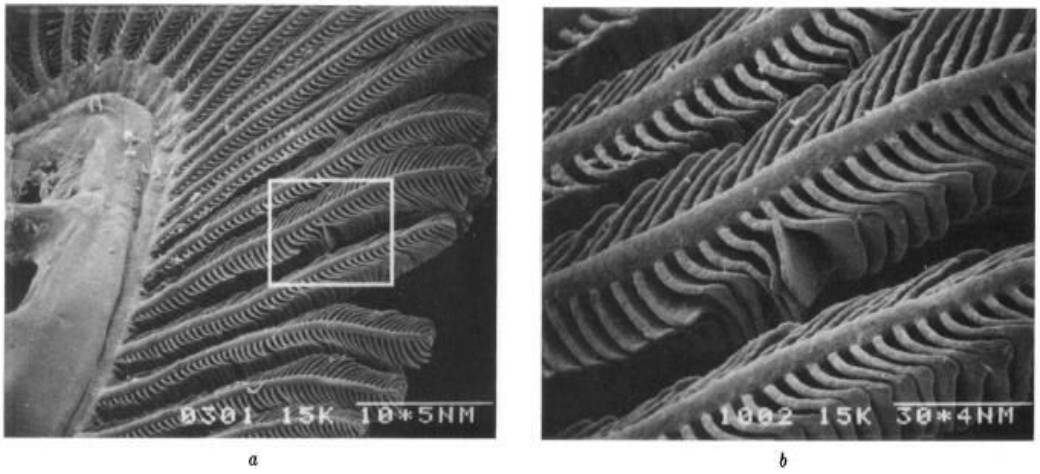

Figure 4: Scanning electron micrographs of (a) branchial arch and filaments of gill from the teleost, Opsanus beta; calibration line is 1 mm (10 x 105 nm); (b) filament and secondary lamellae of gill from the teleost, Opsanus beta; calibration line is 300 ,um (30 x 104 nm).

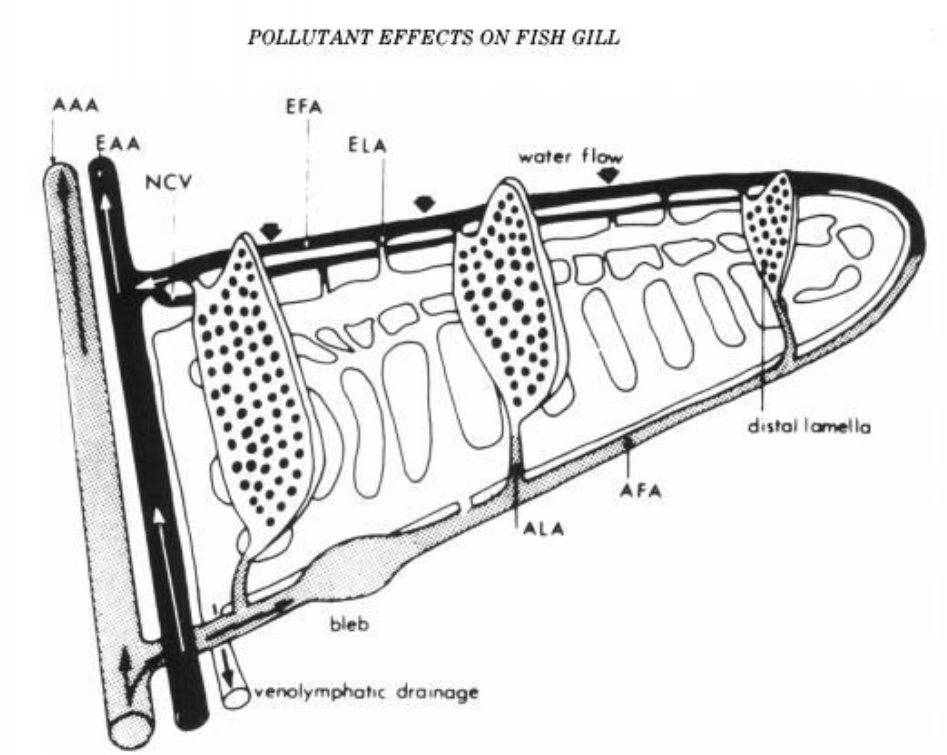

Figure 5: Diagram of vascularization of gill arch, filament, and lamellae of a typical teleost fish. (AAA) afferent arch artery; (AFA) afferent filament artery; (ALA) afferent lamellar artery; (EAA) efferent arch artery; (EFA) efferent filament artery; (ELA) efferent lamellar artery; (NCV) nutritive companion vessel; (CVS) central venolymphatic sinus.

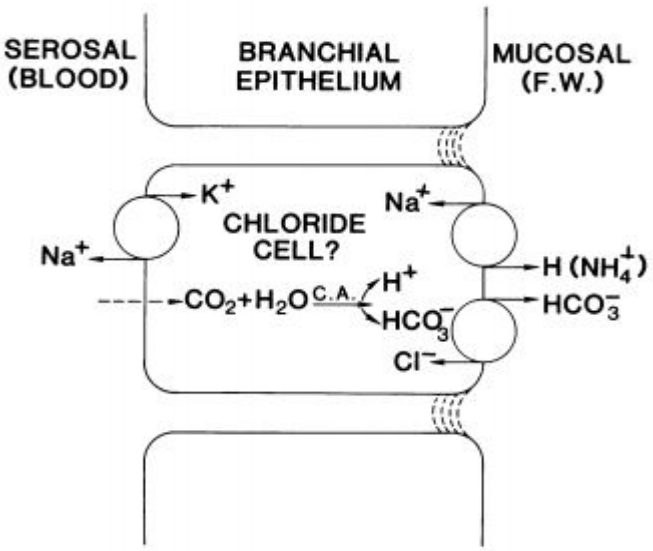

Figure 6. Current model of pathways for Na+ and Cl- transport by the freshwater teleost branchial epithelium. Four dotted lines at the apical junction between adjacent cells denote "tight junctions" characteristic of freshwater gills. C.A. indicates intracellular carbonic anhydrase.

Histopathological damage to gill surfaces in the present study may be attributed to high accumulation of pesticides in the gill, isolation due to elevated mucus secretion increase of ventilation volume and decreased gill oxygen efficiency as reported by Bradbury et al [4] and Bradbury and Coats [5].

Changes in the gill architecture under Permethrin would alter the diffusing capacity of the gill with consequent hypoxic and anoxic conditions then respiration may become problematic task for the fish Hassan et al [14] opined that the epithelial lifting and hyperplasia lowered the rate of gaseous exchange due to its natural mechanism to increase the pollutant blood defence distance along with necrosis and excessive mucus secretion, oxygen deficiency occurred. Neelima et al [17], reposted the pathology of gill tissue exposed to cypermethrin in the fish *Cyprinus carpio,* due to toxicant exposure the changes include epithelial lifting bulging of tips of primary gill filaments, degenerated secondary lamellae curling of secondary gill filaments atrophy, secondary lamella and fusion of secondary gill filaments.

Cengiz [8] reported histopathological effects of deltamethrin in the gill of the common carp after acute exposure at a concentration of 0.029 and 0.041 mg/L and the changes observed are desquamation necrosis aneurysm in secondary lamellae, lifting of the lamellar epithelium, oedema epithelial hyperplasia and fusion of the secondary lamellae.

Rao et al [20] reported degenerate changes in mosquito fish, *Gambusia affinis* in relation to sublethal exposure of chloropyriphos an organophosphate.

Similar such reports of other non-synthetic Pyrethroids are by Cengiz [8]; De Silva and Samayawardhenar [10]; Rodruiquez et al [21], Camargo and Martinez [7] and Evans et al [13].

The work of the synthetic Pyrethroids, Cypermethrin of the fish and resulted pathological changes of the gill are reported by Neelima et al [17], are similar such type, but both of the toxicants in the present study and the report belong to different class of synthetic Pyrethroids, may be damage not dependent on type of chemical class.

Histopathological changes of the common carp *Cyprinus carprio* exposed to paraquat and observed changes like hyperplasia oedema pyknosis and necrosis in gills Hassson et al [14].

Andem et al [1] reported histopathological changes of changes of synthetic pyrethroid cypermethrin exposed to African cat fish *Clarias geriepinus* finger lings and primary lamellae and secondary lamellae are not damaged in the control where as in the exposed fish gills showed a significant damage finally leading to haemorrage.

Anita Susan et al [2] reported histopathological changes in the three Indian major carps exposed to fenvelarate and reported significant changes in gills such as hyperplasia, desquamation and necrosis etc.

Nazia Khatun et al [16] reported for 30 days exposure of sublethal concentration of Chlorophyrifos in the cat fish *Heteropneustes fossilis,* resulting in some degenerative changes of gills.

It is the type of the toxicant, not the matter much but the entry point of the water to the fish is damaged severally, rendering the fish unsuitable to live in such stressful condition and is different for each different chemical.

The present study will be an indices for the other commonly cultured fishes, the Indian major carps where necessary precautions have to be taken to avoid Contamination.

CONCLUSION

It is concluded that when fishes are exposed to pesticides they suffer severe architectural changes in gill which after various physiological activities of the fish succumb to death. The fish are responding directly to the toxicant as much as the secondary effects caused by stress in order to meet the demand of the oxygen for energy synthesis.

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