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Original Research Article DOI: 10.26479/2019.0503.24 TOXICITY OF MONOCROTOPHOS ON THE OXYGEN CONSUMPTION AND GILL HISTOLOGY OF ESTUARINE FISH *MUGIL CEPHALUS*

O. Sathick*, S. Farvin Banu, N. Vasanthi, K. Muthukumaravel

P.G. and Research Department of Zoology, Khadir Mohideen College, Adirampattinam, Tamil Nadu, India.

ABSTRACT: Studies were made to evaluate the effect of sublethal concentration of pesticide, monocrotophos on oxygen consumption and gill histology of *Mugil cephalus*. LC₅₀ values for 96 hours was determined for monocrotophos and was found to be 0.40 ppm. The fishes were exposed to 10% sublethal concentrations of the 96 hours LC₅₀ value for 10, 20, and 30 days along with control. Decrease in O₂ consumption rate was observed in *Mugil cephalus* exposed to 10% sub lethal concentration of pesticide for a period of 10, 20 and 30 days. In this study, the oxygen consumption was gradually decreasing with increasing exposure periods. Maximum decline (-36.94%) over control in the rate of respiration was noticed in 10% sub lethal concentration on 30 days of exposure. Histopathological lesions in the gill, of estuarine fish, *Mugil cephalus* were assessed following 10, 20 and 30 days of exposure to the monocrotophos. Gills of experimental animal were collected, processed and stained with hematoxylin and eosin according to routine histological technique. In the fish exposed to the monocrotophos, the most common lesions were fusion of gill lamellae, detachment of gill epithelium, hyperplasia, and hypertrophy of respiratory epithelium in the gills. The lesions were comparatively most severe in 30 days exposed fish.

KEYWORDS: Mugil cephalus, monocrotophos, oxygen consumption, histology of gill.

Corresponding Author: Dr. O. Sathick* Ph.D.

P.G. and Research Department of Zoology, Khadir Mohideen College, Adirampattinam, Tamil Nadu, India.

1.INTRODUCTION

Pesticides may be categorized as insecticides, herbicides, defolients, fungicides and rodenticides [9]. Pesticides vary in their chemical formulations as well as toxicity, environmental persistence and

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications pathways of action. Among the pesticides, chlorinated hydrocarbons, organophophates and carbamates are commonly used. The presence of pesticides in the aquatic system can obviously lead to multi- fold interaction with other forms of pollution. In India, scores of studies have been undertaken to estimate the acute toxicity level of various pesticides on aquatic fauna [7, 4, 48 & 55]. The rate of oxygen consumption in turn controls the metabolic activities and changes in respiratory rates have been used as the indicator of the stress in pollutant exposed organisms. The oxygen consumption was gradually decreasing with increasing exposure periods as observed by [34 & 13] in Oreochromis mossambicus exposed to sublethal concentrations of Quinolphenols. The rate of oxygen consumption was gradually decreases in all the exposure periods. Toxicants in the environment mainly enter into fish by means of their respiratory system [54]. Changes in oxygen uptake of fishes in response to pesticide exposure are varying in different fishes exposed to a variety of pesticides [25]. The rate of oxygen consumption is found to be increased initially upto 48hours then decreased up to end of the experiment when fish exposed lethal concentration of dimethoate [29]. A review of literature indicates that the effects of pesticides on the proportion of oxygen uptake from water and air by air breathing fishes were studied by only a few workers [5, 36, 18 & 25], therefore, the present work has been undertaken in an air breathing fish, Channa gachua to advance our information in this regard. Fish gills are regarded as a major site of respiration, osmoregulation and excretion and remain in close contact with the external environment and particularly sensitive to changes in the quality of water and considered the primary target of the contaminants. Histologic studies have been considered as the tool for evaluating the toxic effects in target organs of fish in laboratory experiments and in the field experiments [56, 47 & 14]. Gills are the primary site for oxygen uptake in fishes and these delicate organs are in contact with chemical toxins that cause stress exacerbated. Gill lesions as indicators of exposure to toxicants have previously been used in numerous laboratory and field studies around the world [11, 51, 22, 38, 52 & 35]. Gills of Oreochromis niloticus exposed to heavy metals showed mild congestion and edema of the primary lamellae, severe edema, hyperplasia, fusion and focal desquamation of the epithelial lining of the secondary lamellae, epithelial vacuolation of the secondary lamellae, etc. [24]. Hence the present study aimed to investigate the impact of monocrotophos on oxygen consumption and histological structure of gill of Mugil cephalus in order to understand to mode of action, stress response and organ dysfunction.

2. MATERIALS AND METHODS

Mugil cephalus of the weight $(10\pm19g)$ and length $(8\pm0.5 \text{ cm})$ were collected from Agniar estuary, southeast coast of Tamilnadu. The fish were exposed to different concentrations of monocrotophos and mortality was observed for 96 hr. A static renewable bioassay method was adopted for the determination of 96hr median lethal concentration [50]. Probit analysis was followed for the calculations of 96h LC₅₀ [16]. Fish showing an abnormal behavior was removed as soon as possible.

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications In the present study tap water free from chlorine was used which had the following physico – chemical characteristics [3]; temperature 28 ± 0.13 pH 8.4 ± 0.04 , salinity 14.5 ± 0.13 ppt, and D.O.4.6 ±0.2 mg/l. Oxygen consumption was estimated Winkler's method [2]. For histological studies, the gill was removed from the fish and transferred to fresh Bouin's fluid and left in it for 24 hours and processed by adopting the usual micro technique procedure [21].

3. RESULTS AND DISCUSSION

In the present study 96h LC₅₀ value of the monocrotophos during acute exposure to Mugil cephalus was found to be 0.40 ppm. Oxygen consumption of fish, Mugil cephalus exposed to sublethal concentration of monocrotophos. Decrease in O₂ consumption rate was observed in Mugil cephalus exposed to 10% sub lethal concentration of monocrotophos for a period of 10, 20 and 30 days. The rate of oxygen consumption in control Mugil cephalus were 0.579, 0.584 and 0.582 ml/O₂/g/hr at 10,20 and 30 days, respectively. The fish exposed to sub lethal concentrations of monocrotophos shown the oxygen consumption at the rate of 0.518, 0.439 and 0.367ml/O₂/g/hr at 10% sub lethal concentrations of 10, 20 and 30 days respectively (Fig.1). In this study, the oxygen consumption was gradually decreasing with increasing exposure periods. Maximum decline (-36.94%) over control in the rate of respiration was noticed in 10% sublethal concentration on 30 days of exposure (Fig.1). Oxygen consumption of pesticide treated fish showed an initial increase with lower concentration of monocrotophos and decreased with the increasing concentrations. The reduced oxygen consumption could be attributed to gill damage or to hypochronic microcytic anemia as suggested by [40]. Similar decrease in oxygen consumption was observed by [28] in Perca fluviatilis and [45] in Rogor treated Mystus gulio. The decreases in oxygen consumption appear to be a protective measure to ensure that there is a low intake of the toxic substance. Reduced oxygen consumption at higher concentrations of pesticide could also arise as a result of respiratory inhibitory factors that come into play as suggested [41] in Mystus gulio under heavy metal pollutions. Oxygen consumption of the fish was concentration dependent. The initial increase of oxygen uptake is probably to meet energy demand during early periods of exposure, which was followed by gradual decrease in oxygen up taking during the later period of exposure, which may be due to onset of toxicity as suggested by [17]. Oxygen consumption of pesticide treated fish showed decreased with increasing concentrations. The reduced oxygen consumption could be attributed to gill damage or to hypochromic mirocytic anemia as suggested by [8]. Similar decrease in oxygen consumption was observed by [28] in Perca fluviatilis. The decrease in oxygen consumption appears to be a protective measure to ensure that there is a low intake of the toxic substance. Reduced oxygen consumption at higher concentrations of pesticides could also arise as a result of respiratory inhibitory factors that come into play as suggested by [41] in Mystus gulio under heavy metal pollution. The pesticide monocrotophos dissolved in water brings about extensive changes in the physical parameters of water such as salinity, alkalinity and severe depletion in the dissolved oxygen

Sathick et al RJLBPCS 2019www.rjlbpcs.comLife Science Informatics Publicationscontent.So the aquatic systems are drastically altered due to this new stress in the environmentalmedium that brings out such physiological changes and to cope up with the situations.



Figure 1. Changes in the oxygen uptake of *Mugil cephalus* at sublethal concentration of monocrotophos (O₂ml/g/hr)



Plate - 1: Histopathological lesions in the gill of *Mugil cephalus* exposed to 10% sublethal concentration of monocrotophos at different durations

a. Control

SGL - Secondary Gill Lamellae

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b.	10 days treated			
	FSL	-	Fusion of secondary lamellae	
	DE	-	Degeneration of epithelium	
c. 20 days treated				
	FSL	-	Fusion of secondary lamellae	
	DE	-	Degeneration of epithelium	
	Ν	-	Necrosis	
d.	30 days treated			
	FSL	-	Fusion of secondary lamellae	
	ESL	-	Erosion of secondary lamellae	
The structure of gill in Mugil cephalus consist of highly vascular plate like process called primary				

and secondary lamellae. The secondary lamellae of the gill appeared as finger-like structure and are covered with thin layer of epithelial cells. They are very thin, slender and attached on either side of the primary lamellae (Plate. I-Fig.a). In sublethal exposure of monocrotophos, the gill of Mugil *cephalus* showed marked histological changes. Appreciable changes were noticed in the histology of gill after 10 days treatment including fusion of secondary lamellae, degeneration of epithelium and vaculation (Plate. I - Fig.b). The damage was more severe and progressive after 20 days of exposure. The primary and secondary gill lamellae were damaged to a great extent. Hypertrophy, hyperplasia, necrosis and lamellar fusion were observed (Plate. I -Fig.c). However such changes were drastic to the extent that disintegration of lamellar epithelium fusion of secondary lamellae and degenerated secondary lamellae were found in the 30 days treated fish (Plate. I -Fig.d). The histopathological changes observed in the gills of Mugil cephalus in the present study are in good agreement of the reports of [44] and [23]. They observed the bulging of secondary lamellae at the terminal ends, lesions and erosions at the base of lamellae on 12^{th} day of exposure of O. mossambicus to chlorpyrifos. A thick coat of mucus on the gill filaments was persisting on 18th day of exposure. [23], reported that 96-hour fenthion exposure induced gill lesions, including hyperplasia and desquamation of the epithelium and thrombosis in the secondary gill lamellae. Photographs gives histological details of the gills from control fish under high power. The gill consists of a thin epithelium two sets of four holobranchus, forming the sides of pharynx. Each holobranch consist of two hemibranchus and it consists of a row of long thin filaments the primary lamellae and the secondary lamellae. An epithelial tissue covers the gill arch. But at the original of the primary lamellae, the epidermis is much thicker and below this epidermis there is usually an array of lymphoid tissue comprising [53]. A mucoid epidermis covers the primary lamellae. The secondary lamellae consist of an envelope of epithelial cells, supported and separated by pillar cells which are arranged in rows. After exposure to pesticide monocrotophos the secondary lamellar epithelium of the gills was thicker than the control. In the different concentration monocrotophos

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications treated fish the epithelium of the secondary lamellae swelled considerably the lamellae were curved and their blood spaces were constructed the same was observed by [19] and [42]. At the higher concentration the epithelium of the lamellae was swollen and partly detached from the underlying blood space. In some regions the epithelium was partly destroyed. The same results were observed by [49] when the fish rainbow trout treated to pulp and paper mill effluents. After the treatment of 96 hrs pesticide treated fish specific responded were noticed these response include production of copious quantities of mucus on the body surface and especially the gills the results were observed by [12] and further prolonged conditions increased opercular ventilation and coughing rates and gill damage including hyperplasis, necrosis and sloughing of epithelial tissues. [32], analyzed 130 publications in which morphological changes of the respiratory epithelium due to the action of chemicals were registered. Lesions observed less than 10 times were not considered in his statistical analysis, and the wrinkling of the respiratory epithelium is among these lesions. In the present study the pathological effects on gills include the following degenerative changes and these changes that were observed. The pathological changes for pesticide exposure include degeneration of epithelial cells and necrosis in inter lamellar epithelial cells. During prolonged exposure curling of the secondary lamellae, edematous epithelium of secondary lamellae and hypertrophy of both primary and secondary epithelium was observed. [32], reported the most common branchial change that occurred in freshwater fishes rather than in seawater fish. This could be due to the fact that the first ones were hyperosmotic in relation to the environment, facilitating the influx of water through the epithelium lesion, increasing the volume in the oedema, and consequently the detachment. [1], repeated this process as being a decrease of the superficial area of the gills what was necessary to maintain the internal osmotic surrounding regarding the functional loss of the epithelial cells. [37], found that the respiratory epithelium detachment resulted in the increase of the diffusion distance, affecting the gaseous exchanges. This phenomenon has also been described in another type of environmental contamination such as in acid waters [26], heavy metals [39] and salinity [30 & 15]. [31], repeated that these cells stemed from the epithelium of the filament in the interlamellar space and could act as a barrier impeding the diffusion of harmful substances to the blood of the fish. [27], studied the histopathological effects of raw oil on fishes, found that hyperplasia, together with the mucus secretion, protected the gills against future damages caused by intoxicants. This alteration was extremely intense after contamination with heavy metals [39]. The secondary gill lamella is covered by thin sheets of epithelial cells (EC). They are separated by mucous cells (MC) and erythrocytes. They are also separated by a series of peculiar cells called pillar (Pilaster) cells (PC) and supported by a basement membrane. Blood vessels can be seen extended into each secondary gill lamellae. The blood cell has single nucleus, which is flattened in appearance. In the secondary gill lamellae, blood circulates through spaces formed between the individual pillar cells. The epithelium forms a barrier between the fish blood and the surrounding water. The gaseous exchange

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications needed to sustain life takes place through this barrier. The region between two adjacent respiratory lamellae is termed as inter lamellar. [37], did not detect any significant difference in the diameter of the blood spaces surrounded by the pillar cells in fish exposed to the endosulfan organochloride. However, some studies report damages in the pillar cells caused by pollutants [46 & 10]. Such cellular damages are often associated to high doses in which the animals are close to death [32]. [33], reported that in the *M. roosevelti* a different result was obtained after contamination with OP. At the first hour of exposition to the sublethal concentration, the whole structure of the respiratory lamella of M. roosevelti, including the shape of pillar cells, was altered. This collapse of the pillar cells - progressive along the 96 hours of the experiment - was followed by a loss of shape of the erytrocytes, what can indicate osmotic and ionic alterations. The action of methyl parathion causes enzymatic inhibition, blocking the acetylcholinesterasis and other enzymes. It is not the methyl parathion that acts but the methyl paraoxon, which results from enzymatic oxidation mainly in the hepatocytes. The cholinesterase inactivation by methyl paraoxon is taken, chemically, as a phosphorilation, leaving the endogenous acetylcholine free [6]. [10], suggest that the contraction of pillar cells is facilitated by the acetylcholine in the blood. Morphologic alterations of the pillar cells can have several secondary consequences. These cells control the blood pressure of the fish, and changes in the blood pressure and flow can affect the number of irrigated lamellae, the distribution of the blood within the lamellae, the permeability of the branchial epithelium and, as a consequence, the osmorregulatory and gaseous exchange mechanisms [43], causing several physiological disorders. [20], elucidated the effect of organophosphorus pesticide Dimecron on the gill and liver of the fish Etroplus. Dimecron induced branchial congestion in the gill filaments. Edematous fluid lifted the respiratory epithelium in a few secondary lamellae, which were found thickened. The cells between the secondary lamellae were thickened to such an extent that the inter-lamellar spaces occluded, which gave the filament a compact appearance. In the present study the histopathological changes of gills were examined. Gills of the control fish had normal morphological structure. But the gills of fish exposed to 10% sublethal monocrotophous concentration, epithelial hyperplasia and subepithelial edema were found. Similar changes were remained in different exposure time at 10% sublethal concentration of monocrotophos. More pronounced and followed with leukocyte infiltration, slight hypertrophy of chloride cells, as well as lifting and rupture of the respiratory epithelium on some secondary lamellae were observed during prolonged exposure.

4. CONCLUSION

In the present study, it can be stated that monocrotophos exposure during sublethal treatment produces severe toxic effects on the respiratory organ of the estuarine fish *Mugil cephalus*. The finding of the present study indicate that gill structural changes observer serve as "biomarkers" for assessing pesticide toxicity in aquatic environment.

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CONFLICT OF INTEREST

Authors have no conflict of interest.

REFERENCES

- 1. Abel PD and Skid more JF. Toxic effects of an anionie detergent on the gills of rainbow trout.Water Res., 1975; 9: 759-765.
- 2. Annon. Water quality management in aquaculture (CMFRI special publication No.22. CMFRI, Cochin), 1984.
- 3. APHA (American Public Health Association). Standard methods for the Examination of water and wastewater, 20th ed., American Public Health Association, Washington, DC, 1998.
- 4. Arora HC, Shrivastava SK and Seth AK. Bioassay studies of some commercial organic insecticides-Parts I and II; Indian J. Environ. Health. 1971; 13 226-233; 300-306.
- 5. Bakthavathsalam R. Toxicity and Physiological Impact of Three Selected Pesticides in an Air Breathing Fish, Anabas testudineus. Ph.D. Thesis, Annamalai University, Tamilnadu, India, 1980.
- 6. Barberá C. Agricultural pesticides. Omega editions, 1976.
- 7. Basak PK and Konar SK. Estimation of safe concentration of insecticides, a new method tested on DDT and BHC. J. Inland Fish. Soc. India, 1977; 9, 19-29.
- 8. Baskaran P and Palanichamy S. Impact of agricultural (ammonium chloride) fertilizer on physiology and biochemistry of fresh water teleost fish, Oreochromis mossambicus, J.Ecobio., 1990; 2:97-106.
- 9. Benson WR. The chemistry of pesticides. Annals of the New York Academy of Sciences, 1969; 160: 7-29.
- 10. Bettex-Galland M and Hughes GM. Contractile filamentous material in the pillar cells of fish gills. J. Cell. Sci., 1973; 13, 359-370.
- 11. Dalzell DJB and Macfarlane NAA. The toxicity of iron to brown trout and effects on the gills: A comparison of two grades of iron sulphate. J. Fish. Biol. 1999; 55:301-15.
- 12. Dandy JWT. Activity response to chlorine in the brook trout, Salvelinus fontinulis (Mitchell). Can. J. Zool. 1972; 50, 405 - 410.
- 13. Dharmalata and Namitha Joshi. Toxicity and Respiratory responses of Heteropneustes fossilis exposed to zinc chloride and fly ash leachate. Him. J Environ. Zool. 2002; 16(1):87-90.

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications
14. Dutta HM. A composite approach for evaluation of the effects of pesticides on fish. In: Munshi JSD, Dutta HM (eds), Fish Morphology, 1996; pp.334. New York, Science Publishers Inc.

- 15. Fanta E, Luvizotto MF and Meyer AP. Gill structure of the Antartic fishes Notothenia (Gobionotothen) gibberifrons and Trematomus newnesi (Nototheniidae) stressed by salinity changes and some behavioural consequences. Antartic Record (Nankyoku Shiryô), 1995; 39: (1), 25-39.
- 16. Finney DJ. Probit Analysis. Cambridge Univ. Press, London, 1971.
- 17. Fry FJ. Fish and Fisheries of India, Hindustan Publishing Corporation, New Delhi, 1971.
- Ganapathyraman S. Impact of Organophorus Pesticide, Monocrotophos on Dual Mode Respiration and Protein Metabolism in the Air Breathing Fish, *Channa punctatus* (Bloch.).M. Phil. Dissertation, Annamalai University, Tamilnadu, 1987.
- 19. Girija M. Effect of heptachlor and dichlorovas on structure and function of gill tissue of a fresh water teleost *Tilapia mossambica* (Peters) Ph.D thesis S.V. University, Tirupathi, India, 1987.
- Gopalakrishnan KS. Studies on the effects of some pesticides on the fish *Etroplus maculates* (Bloch), Ph.D Thesis, Cochin University of Science and Technology, Kerala, India, 1990; 124-125.
- Gurr E. Methods of analytical histology and Histochemistry. Leonard Hill Ltd., London, 1959;
 45-49.
- 22. Heath AC. Water pollution and fish physiology, 2nd Edition. Lewis, Boca Raton, FL, 1995.
- 23. Jauch D. Gill lesions in Cichlid fishes after intoxication with the insecticides. Fenthion. Experimentia. 1979; 35, 371-372.
- Kaoud HA, Zaki MM, Dahshan AR, Saeid S and Zorba HY. Amelioration the toxic effects of cadmium exposure in Nile Tilapia (*Oreochromis niloticus*) by using Lemnagibba L. Life Science J. 2011; 8(1): 185–95.
- 25. Karuppiah D. Toxicity and Effects of a Carbamate Pesticide Sevin on Some Physiological Aspects of a Freshwater, Edible Air Breathing Fish, *Channa striatus* (Bloch). Ph. D. Thesis, Bharathiar University, Coimbatore, Tamil Nadu, 1996.
- Kawall HG. Effects of acidic waters on Gymnocorymbus ternetzy (Boulenger, 1895) (Pisces: Characidae). MSc Thesis, Federal University of Paraná, Curitiba, Brasil, 1993.
- 27. Khan RA and Kiceniuk J. Histopathological effects of crude oil on Atlantic cod following chronic exposure. Can. J. Zool., 1984; 62, 2038-2043.
- 28. Lars Collivin. The effect of copper on maximum respiration rate and growth Rate of perch, *Perca fluviatilis* L.water Res. 1984; 18, 139-144.
- 29. Lokhande MV. Oxygen consumption and behaviour surveillance in the freshwater fish *Rasbora daniconius* exposed to dimethoate. International Journal of Fisheries and Aquatic Studies 2017; 5(2): 712-716.

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications
30. Luvizotto MF. Effect of different salinities on chloride cells and secretory cells of the gill epithelium of the Antarctic fish *Nototheniops nudifons (Lonnberg, 1905)*. MSc Thesis, Federal University of Paraná, Curitiba, Brasil, 1994.

- Magor BG. Gill histopathology of juvenile Oncorhynchus kisutch exposed to suspended wood debris. Can. J. Zool., 1988; 66, 2164-2169.
- Mallat J. Fish gill structural changes induced by toxicants and other irritants; A statistical review.
 Can. J. Fish. Aquat. Sci. 1985; 42: 630-648.
- 33. Marcelo Rubens Machado and Edith Fanta. Effects of the organophosphorus methyl parathion on the branchial epithelium of a freshwater fish Metynnis roosevelt. Braz. arch. biol. technol. 2003; 46 (3) pp.361-372.
- 34. Mathivanan R. Effect of sublethal concentration of quionolphenols on selected respiratory and biochemical parameters in the fresh water fish, *Oreochromis mossambicus*. J Ecotoxicol. Environ. Monit. 2004; 14(1):57-64.
- 35. Maurya PK and Malik DS. Bioaccumulation of heavy metals in tissues of selected fish species from Ganga river, India, and risk assessment for human health. Hum Ecol Risk Assess Int J. 2018; 1-19.
- 36. Natarajan GM. Changes in the dual mode gas exchange and some blood parameters in the air breahting fish, *Channa striatus* (Bleeker) following lethal (LC₅₀/48hrs) exposure to metasystok (Dimeton). Curr. Sci., 1981; 50(1): 40-41.
- 37. Nowak B. histological changes in gills induced by residues of endosulfan. Aquat Toxicoll., 1992;23: 63-84.
- Oliveira CA, Belger L, Pelletier E and Rouleau C. Histopathological evidence of inorganic mercury and methyl mercury toxicity in the Arctic charr (*Salvelinus alpinus*). Environ Res. 2002; 90:217–225.
- 39. Oliveira Ribeiro CA, Turcati NM, Carvalho CS, Cardoso R and Fanta E. Toxic effect of HgCl2 on the structure of the gill arches of Trichomycterus brasiliensis (Pisces, Siluroidei). Ann. II Symposium on Environment, Univ. Salgado de Oliveira, Niterói, Brasil, 1994.
- 40. Palanichamy S and Baskaran P. Selected Biochemical and physiological Responses of the fish *Channa striatus* as Biomonitor to assess Heavy metal pollution in fresh water environment J. Exotoxical Environ. Monit. 1995; 5 (2) 131-138.
- 41. Rafia Sultana and Uma Devi. Oxygen consumption in a cat fish *Mystus gulio* (Ham) exposed to heavy metals. J.Environ, Biol. 1995; 16 (3), 207 –210.
- 42. Ramamurthy K. Impact of heptachlor on haematological, histological and selected biochemical parameters on fresh water edible fish *Channa punctatus* (Bloch) Ph.D thesis S.V. University, Tirupathi, India, 1988.
- 43. Randall D. Blood flow through gills. In-Gills. Cambridge University Press. 1982; pp.173-192.

Sathick et al RJLBPCS 2019 www.rjlbpcs.com Life Science Informatics Publications
44. Rao JV, Rani CHS, Kavitha P, Rao RN and Madhavendra SS. Toxicity of chlorpyrifos to the fish *Oreochromis mossambicus*, Bull. Environ. Contam. Toxicol. 2003; 70, 985-992.

- 45. Sathick O, Raveendran S and Muthukumaravel K. Effect of pesticide rogor on oxygen consumption and gill histology of the estuarine fish *Mystus gulio*, J. Ecotoxicol. Environ. Monit. 2011; 21(5). 409-414.
- 46. Schimid OJ and Mann H. Action of a detergent (dodecylbenzenesulphonate) on the gills of the trout. Nature, 1961; 192, 675.
- 47. Schwaiger J, Bucher F, Ferling H, Kalbfus W and Negele R. A prolonged toxicity study on the effect of sublethal concentrations of bis(tri-n-butylin)oxide (TBTO): Histopathological and histochemical findings in rainbow trout (*Onchorhynchus mykiss*). Aquat. Toxicol. 1992; 23:31–48.
- 48. Sharma HC, Chauhan HS and Ram S. Probability analysis of rainfall for crop planning. Journal of Agricultural Engineering, 1979; 16(3): 87-92.
- 49. Soivio A, Nikunen E and Tuurala H. Acute response to sodium hypochlorite in rainbow trout acclimatized to pulp and paper mill effluents. Aquatic Toxicology. 1988; 13. 77-87.
- 50. Sprague JB. The RBC's of pollutant bio-assay using fish. Biochemical method for assessment of water quality. Amer soc. Test. Mat. 1973; 528 630.
- 51. Teh SJ, Adams SM and Hinton DE. Histopatologic biomarkers in feral freshwater fish populations exposed to types of contaminant stress.Aquat.Toxicol. 1997; 37, 51 70.
- 52. Thophon S, Kruatrache M, Upatham ES, Pokethitiyook P Sahaphong S and Jaritkhuan S. Histopathological alterations of white *Seabass Latescalcarifer*, in acute and subchronic cadmium exposure. Environ. Pollut. 2003; 121:307–320.
- 53. Tilak KS, Veerahah K and Milton orema Raju J. Toxicity and effects of Ammonia, Nitrite, Nitrate and Histopathological changes in the gill of freshwater fish *Cyprinus carpio J*. Ecotoxicol, Environ. Monit. 2006; 16 (6) 527 – 532.
- 54. Tovell PWA, Howes D and Newsome CS. Absorption, metabolism and excretion by gold fish of the anionic detergent, Sodium lauryl sulphate. Toxicol. 1975; 6:17-29.
- 55. Verma SR, Tonk IP and Dalela RC. In vivo enzymatic dysfunction induced by some aquatic pollutants in a fish, *Sarcobranchus fossilis*. J. Environ. Biol., 1980; 1: 1.
- Wester PW and Canton JH. The usefulness of histopathology in aquatic toxicity studies. Comp Biochem. Physiol. C. Comp. Pharmacol. Toxicol. 1991; 100:115–7.