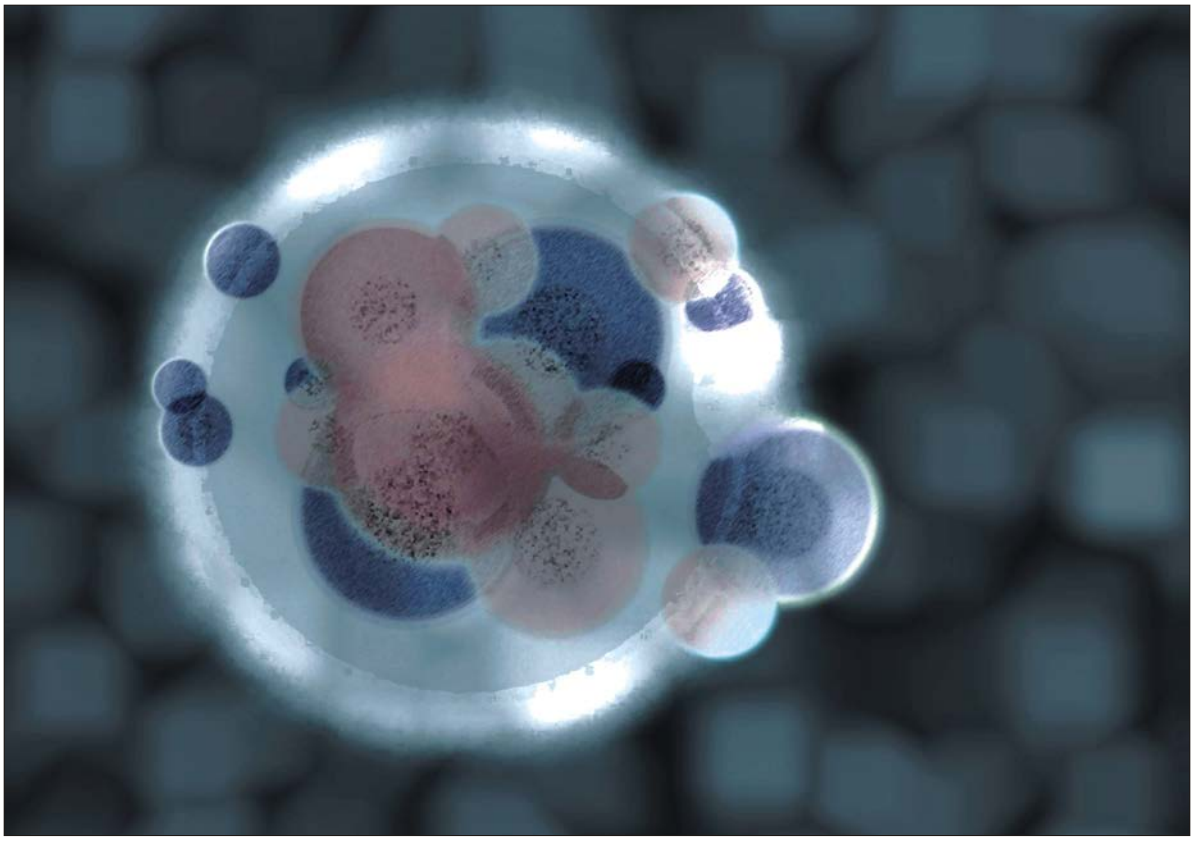

Journal of Biological Sciences

ISSN 1727-3048

science alert

ANSInet
an open access publisher
http://ansinet.com

Journal of Biological Sciences 7 (8): 1473-1477, 2007
ISSN 1727-3048
© 2007 Asian Network for Scientific Information

The Effects of Sub-lethal Doses of Lambda-cyhalothrin on Some Biochemical Characteristics of the African Catfish *Clarias gariepinus*

[1]E. Ogueji Okechukwu and [2]J. Auta
[1]Federal College of Education, Katsina, P.M.B 2041, Katsina State, Nigeria
[2]Department of Biological Science, Ahmadu Bello University, Zaria, Nigeria

Abstract: The impact of long-term exposure to waterborne lambda-cyhalothrin on *Clarias gariepinus* was evaluated through changes of selected biochemical parameters. Fish was exposed to 0.0004, 0.0008 and 0.0016 mg L^{-1} for 8 weeks. The parameters measured were serum glucose, protein, cholesterol, triglyceride, glutamic pyruvic acid transaminase (GPT), Glutamic Oxaloacetic acid Transaminase (GOT) and alkaline phosphatase (ALP).There was significant ($p<0.05$) alterations between the control values and the exposed groups on all parameters except GOT. The alterations in all parameters was significantly ($p<0.05$) dose and time dependent.

Key words: Lambda-cyhalothrin, serum, biochemical parameters, *Clarias gariepinus*, toxicity

INTRODUCTION

Cells contain enzymes that are necessary to their function. When the integrity of a cell is disrupted, enzymes escape into plasma/serum, where their activity can be measured as a useful index of cell integrity (Coppo *et al*., 2002). Modifications in enzyme activity occur by cell death, increase or decrease enzyme production, obstruction of normal excretory route, increased cell membrane permeability, or impair circulation (Kaneko, 1989). Lambda-cyhalothrin (RS)-alpha-cyano-3-phenoxybenzyl 3-(2-chloro3, 3, 3-trifluoropropenyl)-2, 2-dimethyl cyclopropane carboxlate, is a synthetic pyrethroid insecticide and like all pyrethroids, they are neurotoxins (Beat *et al*., 1997). They act on the axons in the peripheral and central nervous systems. They are believed to interfere with sodium channels and the permeability of nerve cells, so affecting the transmission of nerve impulses. Charles and Hance (1968) reported the 96 h LC_{50} for brown trout under laboratory conditions to be from 0.002-0.0028 mg L^{-1}. Bradbury and Coast (1989), also reported that the 96 h LC_{50} value of pyrethroids, determined in laboratory tests, generally lies below 10 μg L^{-1}. Due to the lipophilicity of pyrethroids, they have a high rate of gill absorption, which in turn would contribute in the sensitivity of fish to aqueous pyrethroid exposures. Fish seem to be deficient in the enzyme system that hydrolyses pyrethroid. There metabolism in fish is largely oxidative (Demoute, 1989; Rukiye *et al*., 2003).

Fish have an important role in the food chain; therefore, investigation of the effects of pesticides on fish has a diagnostic significance in evaluation of adverse effect of pesticides to human health (Begun and Vijayaraghavan, 1996). *Clarias gariepinus* is a fresh water fish and an important food supply for humans. The blood of fish is sensitive to pollution induced stress and certain serum chemistry may be used to identify tissue damage (Patil and Kulkarni, 1993). Changes in the biochemical blood profile indicate changes in metabolism and biochemical processes of the organism, resulting from the effect of various pollutants and they make it possible to study the mechanisms of the effect of various pollutants (Luskova *et al*., 2002). Liver is the metabolic centre for detoxification of chemicals. Liver damage was confirmed by changes in the activities of Glutamate-Oxaloacetate Transaminase (GOT) and Glutamate-Pyruvate Transaminase (GPT) activities (Asztalos and Nemesok, 1985). Chronic hepatic disorders and excessive steroids results in increase plasma alkaline phorphatase (ALP) in most animal. During normal bone growth in young animals, a large amount of ALP is found in plasma, also osteopathies result in increase plasma (ALP) (Coppo *et al*., 2002). Increase in blood glucose level is a general response of fish to acute pollutant effects including organophosphates and pyrethroids (Luskova *et al*., 2002). The quantity of protein is dependent on the rate of protein synthesis, or on the rate of its degradation. The quantity of protein may also be affected due to impaired incorporation of amino acids in the poly peptide chains (Singh *et al*., 1996).

The aim of this study was to investigate the serum activities of GOT, GPT, ALP, protein and carbohydrate

Corresponding Author: E. Ogueji Okechukwu, Federal College of Education, Katsina, P.M.B. 2041, Katsina State, Nigeria

metabolisms after exposure of *Clarias gariepinus* juveniles to nominal sub-lethal concentrations of lambda-cyhalothrin (a commonly used insecticide) with a view to access the possible effects of its toxicity.

MATERIALS AND METHODS

Juveniles of *Clarias gariepinus* was purchased from Maigana fish farm in Zaria, Kaduna State Nigeria. The *Clarias* species averaging 14.33±0.50 cm standard length and average body weight of 20.38±1.25 g were used for the study. The fish were conveyed to fisheries laboratory in a portable well-aerated white polythene bag containing water from the Dam. They were held in large water baths (Container) of 160 L capacity at 24.5-25.5°C and acclimatized for two weeks in dechlorinated municipal water. During this period, the fishes were fed with pelleted diet containing 35% crude protein twice per day at 5% body weight. Also, the water in the glass aquaria was changed every alternate day. The fishes were accepted as well as adapted to laboratory conditions when less than 5% death was recorded for the 14 days period and feeding was discontinued 24 h before the start of the experimental run (Reish and Oshida, 1987).

Sub-lethal bioassay: Based on the result of 96 h LC_{50}, which was estimated to be 0.008 mg L^{-1} (Ogueji and Auta, 2006 unpublished) juveniles were exposed to nominal concentrations of Lambda-cyhalothrin for 8 weeks. The concentrations used for chronic study were 0.0004, 0.0008 and 0.0016 mg L^{-1}. Each treatment was in triplicate and there was a control in each case. With the exception of the control tanks, appropriate volumes of the toxicant were added into each tank. The fishes were randomly assigned to give a loading of 10 fish per tank. Fishes were fed to satiation twice daily. The toxicant and test water were renewed at two days intervals to maintain the toxicants strength and the level of dissolved oxygen as well as minimizing the level of ammonia during the experiment. Twelve fishes were sampled at the end of two weeks from concentrations and control and this was repeated at the end of eight weeks.

Biochemical measurements: For biochemical investigations, The caudal peduncle of fish was cut, blood was collected in non-heparinized tubes. The blood was immediately centrifuged at 1500 rpm for 10 min. Serum was then removed and stored at 4°C prior to immediate determination of biochemical parameters, glucose, cholesterol, triglycerides, total protein, Glutamic Pyruvic acid Trasaminase (GPT), glutamic oxaloacetic acid trasaminase (GOT) and alkaline phosphatase (ALP). Blood glucose was estimated using the method of Trinder (1969). Blood cholesterol was measured according to the procedure of Pearson *et al*. (1953). Blood triglyceride was determined using the method of Rice (1970). The method of Lowry *et al*. (1951) was carried out to determine the value of total protein. The activities of blood GPT and GOT were estimated according to the methods of Retiman and Frankel (1957). To determine the activity of blood ALP, Bassey *et al*. (1946) method was used.

Statistical analysis: Graph of probit kill against log concentration was used to determine the 96 h LC_{50}. For the various biochemical parameters, GenStat-Release 4.2 programmes was used to run analysis of variance (ANOVA) and Duncan Multiple Range Test (DMRT) was used to test for differences between different levels of treatment and to separate means respectively, were applicable. Test of significance was at 95% probability.

RESULTS

Analysis of variance (ANOVA) results of sub-lethal exposure to lambda-cyhalothrin indicated significant ($p<0.05$) dose dependent elevations in glucose, triglyceride and GPT levels in the serum (Table 1). On the other hand there was a significant ($p<0.05$) dose dependent inhibition in protein and ALP. No significant difference was recorded for GOT across concentrations. Cholesterol significantly increased ($p<0.05$) in concentrations 0.0004 and 0.0008 mg L^{-1}, but was significantly inhibited in 0.0016 mg L^{-1}. Also GPT activity was significantly ($p<0.05$) elevated in 0.0004 and 0.0008 mg L^{-1} exposed fish (Table 1). The control values of protein and cholesterol were significantly lower than in exposed fish (Table 1). The control values of triglyceride was significantly ($p<0.05$) lower than in 0.0008 and 0.0016 mg L^{-1} exposed fish (Table 1) and that of ALP was significantly lower than in 0.0004 mg L^{-1} but significantly elevated than in 0.0016 mg L^{-1} exposed fish (Table 1). The control values for glucose was significantly lower ($p<0.05$) than in 0.0008 and 0.0016 mg L^{-1}, but significantly elevated than in 0.0004 mg L^{-1} exposed fish.

There were time dependent elevations in the serum values of glucose, protein and ALP (Table 2). On the other hand and within the same time period, there was time dependent significant inhibition in cholesterol, triglyceride, GOT and GPT seral enzymes (Table 2).

DISCUSSION

The significant ($p<0.05$) increase in glucose which was dose and time dependent (Table 1 and 2) may be

Table 1: Means for *C. gariepinus* biochemical parameters after exposure to sub-lethal concentrations of lambda-cyhalothrin

Conc. (mg L^{-1})	Glucose (mg dL^{-1})	Protein (g dL^{-1})	Cholesterol (mg dL^{-1})	Triglyceride (mg dL^{-1})	GOT (IU L^{-1})	GPT (IU L^{-1})	ALP (IU L^{-1})
0.00	54.00^{e}	3.43^{b}	129.00^{d}	142.25^{c}	48.50^{a}	58.75^{a}	17.00^{b}
0.0004	52.75^{c}	3.98^{a}	145.00^{b}	138.25^{d}	47.00^{a}	46.25^{c}	20.25^{a}
0.0008	57.00^{b}	2.95^{c}	161.50^{a}	144.50^{b}	42.50^{b}	51.75^{b}	17.50^{b}
0.0016	60.25^{g}	2.63^{a}	139.50^{c}	150.00^{a}	47.50^{b}	59.25^{a}	15.50^{c}

Means with the same superscript along columns are not significantly different ($p<0.05$)

Table 2: Changes in some biochemical characteristics of *C. gariepinus* due to lambda-cyhalothrin in relation to time

Time (Weeks)	Glucose (mg dL^{-1})	Protein (g dL^{-1})	Cholesterol (mg dL^{-1})	Triglyceride (mg dL^{-1})	GOT (IU L^{-1})	GPT (IU L^{-1})	ALP (IU L^{-1})
2	53.50^{b}	2.49^{b}	185.50^{a}	150.50^{a}	49.00^{a}	58.75^{a}	12.75^{b}
8	58.50^{a}	4.00^{a}	102.00^{b}	137.00^{b}	43.25^{b}	49.25^{b}	22.37^{a}

Means with the same superscript along columns are not significantly different ($p<0.05$)

considered to be manifestation of stress induced by lambda-cyhalothrin. Glucose increase is a general response of fish to acute and sublethal pollutant effects (Verma *et al*., 1983; Ghazaly, 1994; Ceron *et al*., 1997; Luskova *et al*., 2002). Wedemeyer *et al*. (1981) stated that high levels of blood glucose are caused by disorders in carbohydrate metabolism appearing in the condition of physical and chemical stresses. A variety of stressors stimulate the adrenal tissue, resulting in increased level of circulating glucocorticoids (Hontela, *et al*., 1996) and catecholaminies (Nakano and Tomlinson, 1967). Both of these groups of hormones produce hyperglycaemia.

The increase in serum protein was dose and time dependent (Table 1 and 2). Present findings are in agreement with that of other some other workers. For example Oruc and Uner (1999) reported increase in liver protein following exposure to 2, 4-Diamin for 30 days. Salib *et al*. (1984) observed that the protein content in all tissues of malathion exposed *Tilapia mossambica* is slightly higher. They suggested that the fish exposed to pesticides may compensate any possible protein loss by increasing its protein synthesis.

Gill *et al*. (1991) found an increase in liver proteins following Endosulphan intoxication and noted that protein levels in the liver of *Barbus conchonious* could be due to increased protein turnover. They also concluded that compensatory production of enzymes lost as a result of tissue necrosis or to meet increased demand to detoxify the pesticides might have necessitated enhanced synthesis of enzyme proteins (Gill *et al*., 1990). However, the significant decrease in serum protein observed in 0.0008 and 0.0016 mg L^{-1} was in agreement with the work of Ravichandran *et al*. (1994). They reported depletion of protein from 7.9 to 45.0% due to proteolysis after exposing *Oreochromis mossambicus* to sub-lethal concentrations of phenol. The decrease in protein level observed in the two sub-lethal concentrations may be due to their degradation and also their possible utilization for metabolic purpose. Bradbury *et al*. (1987) pointed out that the decreased protein content might also be attributed to the destruction or necrosis of cells and consequent impairment in protein synthesis machinery. The quantity of protein is dependent on the rate of protein synthesis, or on rate of its degradation. The quantity of protein may also be affected due to impaired incorporation of amino acids into polypeptide chains (Ram *et al*., 2003). Cholesterol content in the blood is linked to lipid metabolism and depends on the calorific value of the feed. In this investigation there was a significant ($p<0.05$) serum cholesterol inhibition as time of exposure increased to 8 weeks. The liver is the key organ in the synthesis and excretion of cholesterol, therefore any type of obstruction in the liver either intra or extra hepatic, will cause an increase in total cholesterol levels on the serum. However in chronic conditions such as cirrhosis, that involves considerable destruction of liver cells, the cholesterol levels eventually falls below normal level since decreased synthesis is taking place (Kamath, 1972). There was also a time dependent significant ($p<0.05$) serum triglycende inhibition when compared with control. Khurshid (2003) also reported that total lipid content of young chick embryo exposed to cypermethrin was increased at 200 ppm and decreased at 400 ppm and he concluded that the high dose of 400 ppm of cypermethrin seems to have caused cell death (necrosis or apoptosis or both) in the embryonic tissue. Triglyceride accumulation as observed in fatty liver due effect of toxicants is the result of an imbalance between the rate of synthesis and the rate of release of tricglyceride by the parenchynal cells into the systemic circulation (Gabriel, 1986).

Lombardi (1966) described four general mechanisms that can account for accumulation of triglyceride: The rate of synthesis of hepatic triglyceride is normal, but the liver cell is unable to secrete the triglyceride into the plasma/serum; The secretion is normal, but rate of synthesis is increased; There is both an increase in the rate of synthesis and a block in the secretion of the synthesized triglyceride and The triglyceride synthesis takes place in a compartment of the cell other than the endoplasmic reticulum and thus these pool is not accessible to the normal secretory pathway. It appears that a combination of liver necrosis, affecting the synthesis of triglyceride and blockage of the secretion into the serum was responsible for the inhibition observed

over prolonged exposure periods of fish to lambda-cyhalothrin. There is also time dependent significant ($p<0.05$) serum ALP elevation due lambda-cyhalothrin exposure. Atef (2005) also reported significant elevations of ALP after exposure to cadmium and attributed the increase to liver dysfunction. ALP is made in the liver, membrane-bound close to the biliary canaliculus, secreted into the bile and its increase principally indicates cholestasis. The increase in protein level may also be due to the increase in ALP activity as it plays an important role in protein synthesis (Pilo *et al*., 1972). There was a significant ($p<0.05$) time dependent serum GOT and GPT inhibition and this finding is in agreement with that of some other workers, Oruc and Uner (1999) reported inhibition of seral GPT and GOT enzyme activities following 2 and 30 days of exposure to 2, 4-Diamin to *Cyprinus carpio* and Sadhu *et al*. (1985) reported inhibition of GOT and GPT activities in the serum of *Channa stiatus* following exposure to 0.1 ppm malathion for 10 days. GOT and GPT are important in the diagnosis of heart and liver damage (Dere and Polat, 2001).

The results of this study suggest that sub lethal exposure of *C. garipinus* to lambda-cyhalothrin could lead to alterations in carbohydrate and lipid metabolism and possible organ damage. In the light of the above observations, it is recommended that lambda-cyhalothrin should be used with caution and in a suitable manner, as it could be hazardous to aquatic biota, domestic animals and human beings as well.

REFERENCES

American Public Health Association (APHA), American Water Works Association (AWWA) and Water Environment Federation (WEF), 1992. Standard Methods for the examination of Water and Wastewater, 18th Edn., Washington, DC.

Asztalos, B. and J. Nemcsok, 1985. Effects of pesticides on the LDH activity and isoenzyme pattern of Carp (*Cyprinus carpio*) sera. Compar. Biochem. Physiol., 82: 217-219.

Atef, M.M., 2005. Biochemical effects of short-term cadmium exposure on the fresh water fish. *Oreochromis niloticus*. J. Biol. Sci., 5: 260-265.

Bassey, O.A., O.H. Lowery and M.J. Brock, 1946. A method for the rapid determination of alkaline phosphatase with 5 mm^3 of serum. J. Biol. Chem., 164: 321-329.

Beat, G., W. Andres, S. Gunter and S. Marc., 1997. The pyrethroids permethrin and cyhalothrin are potent inhibitors of the mitochondrial Complex 1. Pharm. Exp. Therapeutic, pp: 281-860.

Begum, G. and S. Vijayaraghavan, 1996. Alterations in protein metabolism of muscle tissue in the fish *Clarias batrachus* (Linn) by commercial grade dimethoate. Bull. Environ. Comtam. Toxicol., 57: 223-228.

Bradbury, S.P., D.M. Symonic, J.R. Coats and G.J. Atchison, 1987. Toxicology of fenvalerete and its constituents isomers to the fathead minnow (*Pimephales promelos*) and blue gill (*Lepomis macrochirus*). Bull. Environ. Cont. Toxicol., 38: 727-735.

Bradbury, S.P. and J.R. Coast, 1989. Toxicokinetics and Toxicodyamics of pyrethroid insecticides in fish. Environ. Toxicol. Chem., 8: 373-380.

Ceron, J.J., E. Sancho, M.D. Ferrando, C. Gutierrez and E. Andreu, 1997. Changes in carbohydrate metabolism in the eel *Anguilla anguilla*, during short term exposure to diazinon. Toxicol. Environ. Chem., 60: 201-210.

Charles, R. and R.J. Hance, 1968. A world compendium crop pesticides constituents technical data. Br. Crop Prot. Council, pp: 203-233.

Coppo, J.A., N.B. Mussart and S.A. Fioranelli, 2002. Physiological variation of enzymatic activities in blood of Bullfrog, *Rana catesbeina* (Shaw, 1802). Rev. Vet., 12/13: 22-27.

Demoute, J.P., 1989. A brief review of the environmental fate and metabolism of pyrethroids. Pest Sci., 27: 375-385.

Dere, E. and F. Polat, 2001. The effect of paraquate on the activity of some enzymes in different tissues of mice (*Mus musculus*). Turk. J. Biol., 25: 323-332.

Gabriel, L.P., 1986. Toxicology of the Liver. In: Casarett and Doulls Toxicology; The Basic Science of Poisons 3rd Edn., Macmillan Publishing Co. New York.

Ghazaly, K.S., 1995. Carbohydrate metabolism in Clarias lazara exposed to three different pesticides. J. Egypt. Ger. Soc. Zool., 16A: 235-251.

Gill, T.S., H. Tewari and J. Pande, 1990. Sublethal effects of an organophosphorus insecticide on certain metabolite levels in a freshwater fish, *Puntius conchonius* Hamilton. Pest. Biochem. Physiol., 36: 290-299

Gill, T.S., H. Tewari and J. Pande, 1991. *In vivo* and *in vitro* effects of cadmium on selected enzymes in different organs of the fish *Barbus conchonius* Ham. (*Rosy barb*). Comp. Biochem. Physiol., 100C: 501-505.

Hontela, A., C. Daniel and A.C. Ricard, 1996. Effect of acute and sublethal exposure to Cadmium on the interregnal and thyroid function in rainbow trout, *Oncorhyncus mykiss*. Aquat. Toxicol., 35: 171-182.

Kamath, 1972. Clinical Biochemistry for Medical Technologists. Churchill/Livingstone, London, pp: 89.

Kaneko, J.J., 1989. Clinical Biochemistry of Domestic Animals, 4th Edn., Academic Press, San Diego, pp: 823.

Khurshid, A., 2003. Cypermethrin, A pyrethroid insecticides induces teratological and biochemical changes in young chick embryos. Pak. J. Biol. Sci., 6: 1698-1705.

Lombardi, B., 1966. Considerations on the pathogenesis of fatty liver. Lab. Invest., 15: 1-20

Lowry, O.H., N.J. Rosebrough, A.L. Farr and R.J. Randall, 1951. Protein measurements with Folin Phenol reagent. J. Biol. Chem., 193: 265-275.

Luskova, V., M. Svoboda and J. Kolarova, 2002. The effects of Diazinon on Blood Plasma Biochemistry in Carp (*Cyprinus carpio* L.) Acta Vet. Brno, 71: 117-123.

Nakano, T. and N. Tomlinson, 1967. Catecholamines and carbohydrate concentration in rainbow trout Salmo gairdneri in physical disturbance. J. Fish. Res. Bd. Can., 24: 1701-1715.

Oruc, E.O. and N. Uner, 1999. Effects of 2,4-Diamin on some parameters of protein and carbohydrate metabolisms in the serum, muscle and liver of *Cyprinus carpio* Environ. Pollu., 105: 267-272.

Patil, M. and R.S. Kulkarni, 1993. Ovarian and hepatic biochemical response to sumaach (acrude form of HCG) in fish, *Notopterus notopterus* Pallas, under pesticide treatment. Geobios, 20: 255-259.

Pearson, S., S. Stern and T.H. Mogavack, 1953. A rapid procedure for the determination of serum cholesterol. J. Clin. Endocrinol., pp: 666.

Pilo, B., M.V. Asnani and R.V. Shah, 1972. Studies on wound healing and repair in pigeon 111. Histochemical studies on acid alkaline phosphatase activity during the process. J. Anim. Physiol., 19: 205-212.

Ram, P.Y., S. Digvijay, S.K. Singh and A. Singh, 2003. Metabolic changes in freshwater fish *Channa punctatus* due to stem-barck extract of *Croton tiglium*. Pak. J. Biol. Sci., 6: 1223-1228.

Ravichandran, S., K. Midhunashanthi and N. Indira, 1994. Impact of phenol on protein metabolism in the freshwater fish *Oreochromis mossambicus*. J. Ecotoxicol. Environ. Monit, 4: 33-37.

Reish, D.L. and P.S. Oshida, 1987. Manual of methods in aquatic environment research Part 10. Short term static bioassay. FAO. Fisheries. Technical Paper 247. FAO Rome, pp: 1-62.

Retiman, S. and F. Frankel, 1957. A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvie acid transaminase. Am. J. Clin. Path., pp: 2856.

Rice, E., 1970. Triglycerides (Natural fats) in serum. Academic Press, New York, Stand. Meth. Clin. Chem. Vol. 6, pp: 215.

Rukiye, V., U.E. Figen, P. Hilal and K. Oner, 2003. Investigation of acute toxicity of deltamethrin on guppies (*Poecilia reticularta*) Ecotoxicol. Environ. Safty, 55: 82-85.

Sadhu, K.A., D.K. Chowdhury and P.K. Mukhopadhyay, 1985. Relationship between serum enzymes, histological features and enzymes in hepatopancreas after sub lethal exposure to malathion and phophamidon in the murrel *Channon striatus* (B.L.). Int. Environ. Studies, 24: 35-41.

Sahib, I.K.A., K.R.S. Sambasiva Rao and K.V. Ramana Rao, 1984. Effect of malathion on protein synthetic potentiality of the tissues of the teleost, *Tilapia mossambica* (Peters), as measured through incorporation of [^{14}C] amino acids. Toxicol. Lett., 20: 63-67

Singh, A., D.K. Singh, T.N. Mishua and R.A. Agarwal, 1996. Mulluscicides of plant origin. Biol. Agric. Hortic., 13: 205-252.

Trinder, P., 1969. Determination of bood glucose using 4-aminophenazone. J. Clin. Pathol,. pp: 22-246.

Verma, S.R., S. Rani, P.I. Tonk and R.C. Dalela, 1983. Pesticide induced dysfunction in carbohydrate metabolism in three freshwater fishes. Environ. Res., 32: 127-133.

Wedemeyer, G., D.J. Mcleay and C.P. Good Year, 1984. Assessing the Tolerance of Fish and Fish Population to Environmental Stress. The Problems and Methods of Monitoring. In: Contaminate Effects on Fisheries on Fisheries. Cairnus, W.V., P.V. Hodson and J.O. Nriagu (Eds.). John Wiley and Son Inc. New York, pp: 164-195.