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Effect on Neurotransmitter Substances Exposed to Synthetic Pyrethroid (Lambda-Cyhalothrin) in Freshwater Catfish

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ABSTRACT

Numerous pollutants found in the environment are known to have neurotoxic effects. In fish and animals, these neurotoxins have been shown to alter neurotransmitter levels and cause brain dysfunction. Lambda-cyhalothrin is a widely used agricultural pyrethroid that is extremely hazardous to fish and aquatic invertebrates due to its strong adsorption to soil sediments. As a result, it is shown to be persistent in the soil environment. Catfish, which are widely cultivated in rice-paddy slurries, give farmers an extra revenue stream. However, both commercially raised fish and fish raised in paddy fields run the risk of being directly exposed to toxicants used for field pest control. The fishes exhibited notable inhibition of the AChE enzyme after being exposed to a sub-lethal quantity of the pyrethroids for an extended period of time. Inhibition of AChE activity was also observed in the brain, liver, muscle and plasma of fingerling channel catfish when exposed to synthetic pyrethroids.

INTRODUCTION

Pesticides have positive effects on the economy by increasing food and fiber output and reducing vector-borne diseases, but they can have negative effects on human and environmental health. There is now ample proof that many agricultural chemicals do have the potential to harm people and other living things, as well as to have unintended negative environmental impacts (Igbedioh, 1991; Forget, 1993; Jeyaratnam, 1985). Due to extensive research on dosage response relationships that have produced safe and cost-effective pesticide application levels, the impacts of pesticides on aquatic ecosystems are comparatively well-known (Muirhead-Thomson, 1971). Because they are a great source of protein for human diets, fish play a significant role in aquaculture. The impact of pesticides on growth, survival and fertility of fishes is an important concern today since pesticides hamper the normal physiological processes of the organisms.

A pesticide should ideally kill the intended pests but not humans or other non-target species. Pesticides have a tendency to reduce soil fertility, and in recent years, their concentration in food items has increased significantly (Tsumuki et al., 1970). Additionally, pesticides can have either an agonistic or antagonistic effect, changing the expression of certain genes (Kavlock et al., 1996). The indirect or secondary impacts of pesticides are a further topic of concern in addition to these direct consequences. The ecological consequences of hazardous chemicals that arise from a decrease in the activity and/or density of species susceptible to the toxic material are known as indirect impacts.

Synthetic pyrethroids have a high affinity to bind to the soil sediments. Lambda-cyhalothin, a commonly used agricultural pyrethroid, has strong adsorption to soil sediments; thus found to be persistent in the soil environment. The offsite movement of these compounds into surface waters is of major concern today (Walters et al., 2002; Weston et al., 2004; Bacey et al., 2005).

Even at sub-lethal level, the synthetic pyrethroid Lambda-cyhalothrin demonstrated a notable suppression of the malate dehydrogenase activity of the different Clarias batrachus tissues (Suman Gulati et al., 2025). Catfish, which are widely cultivated in rice-paddy slurries, give farmers an extra revenue stream (Teugels, 1984). Saravanan et al. (2009) investigated changes caused by lambda-cyhalothrin in freshwater fish. However, fish raised in rice fields run the risk of being directly exposed to the toxicants used to control pests in the crops. Fish kept in commercial ponds, however, may also be at risk of being exposed to other pesticides from the surrounding farms, which frequently contaminate the pond (Nettleton et al., 1990).

Studies in biochemistry, physiology, and pharmacology have shown that biogenic amines play a role in controlling a number of central nervous system functions, including blood pressure regulation, motor coordination, and body temperature. Any deviation from the amines' usual concentration causes a

disruption in the central nervous system's functions, which in turn causes convulsive seizures (Chase and Murphy, 1973).

Acetylcholine esterase (AChE), an enzyme found in synaptic areas, catalyzes the conversion of acetylcholine into choline and acetic acid, which mediates impulse transmission. Thus, when the enzyme acetyicholine esterase is blocked, acetylcholine is bound to accumulate at neuro-motor junctions, which ultimately results in hyper excitability (Kabeer Ahammad and Rao, 1980). A long term exposure to a sub-lethal concentration of a combination of two pyrethroids on the fishes showed significant inhibition of the AChE enzyme. Inhibition of AChE activity was also observed in the brain, liver, muscle and plasma of fingerling channel catfish when exposed to synthetic pyrethroids (Straus and Chambers, 1995).

Monoamine oxidase (MAO) is an important catabolic enzyme that plays important role in the degradation of biogenic amines into their corresponding aldehydes and acids. Monoamine oxidase is a flavoprotein. Increase in the concentration of biogenic amines produces a stimulation of the cerebral activity while a decrease produces a depressant effect. Thus to maintain a stable balance in the concentration of these biogenic amines, normal functioning of the enzymes such as monoamine oxidase is required. Various studies have revealed an alteration in the biogenic amine concentration due to the influence of various organophosphorous and organochloride pesticides.

Thus, this study evaluates the toxicity effect of synthetic pyrethroid lambda-cyhalothrin on neurotransmitter substance of the freshwater catfish *Clarias batrachus*.

MATERIALS AND METHODS:

Assay of Specific Activity of Neurotransmitter Substances:

Specific activity of Acetylcholine esterase (AChE):

Acetylcholine esterase activity was assayed in the tissues and serum following the method of Ellman *et al*. (1961). The specific activity of AChE was determined as nanomoles of substrate hydrolysed/minute/mg protein. The activity of serum AChE was expressed as IU/L

Specific activity of Monoamine Oxidase (MAO):

Activity of monoamine oxidase was determined by following the method of Srivastava *et al*. (1979). The specific enzyme activity was expressed as µmoles of benzaldehyde liberated/min/mg protein.

RESULTS:

The changes in the levels of the enzyme acetyl-choline esterase (AChE) in the ovary, muscle, liver, heart and brain tissues of the pyrethroid-exposed fishes are tabulated (Table 1a & 1b).

The ovarian and muscle tissue of the fishes exposed to the higher sub-lethal concentration of the pyrethroid showed a significant decline ($P<0.05$) in the AChE content from the 15th day of exposure in comparison to the AChE content of the control group of fishes. However in fishes exposed to the lower sub-lethal concentration of the pyrethroid, a significant decline ($P<0.05$) was witnessed in the AChE content on the 30th and 45th day of exposure, while the 15th day did not show any significant change in the AChE content of these tissues.

In the brain tissue, a significant decline ($P<0.05$) was witnessed in the AChE content on the 15th day of exposure in fishes of both experimental groups. This was followed by an elevation in the AChE content on the 30th day. However, the 45th day of exposure to the pyrethroid showed a significant decline ($P<0.05$) in the AChE content when compared to the control group of fishes. A decline of 60.23% and 53.18% was witnessed in the brain AChE content of the fishes exposed to the higher and lower sub-lethal concentration of the pyrethroid respectively after 45 days of exposure.

In the hepatic tissue of fishes of both experimental groups, the 15th, 30th and 45th day of exposure showed significant decline ($P<0.05$) in the AChE content when compared to the control group of fishes.

In the cardiac tissue of fishes exposed to the higher sub-lethal concentration of the pyrethroid, a significant decline ($P<0.05$) was witnessed in the AChE content only on the 45th day of exposure, while no significant change was recorded on the 15th and 30th day of exposure to the pyrethroid. On the other hand, in comparison to the control group of fishes, the fishes exposed to the lower sub-lethal concentration of the pyrethroid showed no significant variation in the AChE content of the cardiac tissue throughout the exposure period.

Alterations in the levels of the enzyme monoamine oxidase in the ovary, muscle, liver, heart and brain tissues of the experimental animals is tabulated (Table 2a & 2b).

The brain tissue of fishes of both experimental groups showed a significant ($P<0.05$) and gradual increase in the monoamine oxidase levels on the 15th, 30th and 45th day of exposure to the pyrethroid. An elevation of 202.22% and 137.88% was witnessed in the monoamine oxidase levels of the brain tissue of fishes exposed to the higher and lower sub-lethal concentration of the pyrethroid respectively.

In the hepatic tissue, a significant elevation ($P<0.05$) was witnessed in the monoamine oxidase levels on the 30th and 45th day of exposure, while the 15th day did not show any significant change in the enzyme levels in fishes of both experimental groups in comparison to the control group of fishes. A similar trend was also witnessed in the monoamine oxidase levels of the cardiac tissue in experimental fishes of both groups. However, both the tissues showed an overall significant ($P<0.01$) change in the enzyme levels in fishes of both experimental groups.

A different trend was witnessed in the muscle tissue. An initial significant decline ($P<0.05$) was witnessed in the monoamine oxidase levels on the 15th and 30th day of exposure to the two different sub-lethal concentrations of the pyrethroid. However, on the 45th day, a significant elevation ($P<0.05$) was witnessed in the enzyme level of the muscle tissue in fishes of both experimental groups in comparison to the control group of fishes.

DISCUSSION

In a study similar to this one, Reddy and Philip (1994) found a significant decrease in AChE activity in a variety of Cyprinus carpio tissues exposed to the toxic effects of cypermethin. Under malathion toxicity, Labeo rohita and Tilapia mossambica brain tissue likewise showed a considerable decrease in AChE activity (Mathivanan and Bhaskaran, 2002). The brain tissue of Clarias batrachus treated to endosulfan and cypermethin also demonstrated a notable inhibition of acetylcholine esterase, according to Anand Kumar and Reddy (1997).

The present study findings are in line with the earlier findings of Mathivanan and Bhaskaran (2002) who reported a significant decline in the Ache activity of brain tissues of Labeo rohita and Tilapia mossambica.

Channa punctatus, when exposed to organophosphorous pesticide showed injury in the ventricular myocardium and inhibition of AChE (Gaur and Kumar, 1993). Biochemical studies have suggested that insecticides inhibit AChE because they mimic the substrate acetylecholine, bind to the specific receptors and react with the esterase enzyme in the same way as the normal substrate and thus inhibit its further activity with normal substrate molecules interfering with normal nervous functioning (Rathore and Singh, 2000).

The current study also discovered that the different tissues of fish exposed to lambda-cyhalothrin had significantly higher levels of monoamine oxidase (MAO), an enzyme that breaks down monoamines in the biogenic amine pathway. Under phosalone activity, Devaraj et al. (1991) similarly reported elevated MAO activity in the Oreochromis mossambicus brain. Additionally, Shaffi (1995) documented elevated MAO levels in several brain tissue regions of Labeo rohita, Clarias batrachus, and Channa punctatus that were exposed to sub-lethal doses of lead and mercury.

Increased levels of monoamine oxidase in the brain tissue could be one of the major reasons for the damage witnessed in the tissues of animals subjected to sub-lethal concentrations of lambda-cyhalothrin in the present study.

REFERENCES

- Anand Kumar V, Reddy SLN. Acetylcholinesterase inhibition in freshwater fish, *Clarias batrachus* (Linn), as a diagnosis of environmental pollution by endosulfan and cypermethrin. *J Aquat Biol*. 1997;12(2):42–6.
- Bacey J, Spurlock F, Starner K, Feng H, Hsu J, White J. Residues and toxicity of esfenvalerate and permethrin in water and sediment in tributaries of the Sacramento and San Joaquin Rivers, California, USA. Bull Environ Contam Toxicol. 2005;74(5):864-71. doi:10.1007/s00128-005-0661-8.
- Chase TN, Murphy DL. Serotonin and central nervous system function. Annu Rev Pharmacol. 1973;13:181-97. doi:10.1146/annurev.pa.13.040173.001145. PMID:4200208.
- Devaraj P, Selvarajan VR, Durairaj S. Relationship between acetylcholinesterase and monoamine oxidase in brain regions of *Oreochromis mossambicus* exposed to phosalone. *Indian J Exp Biol*. 1991 Aug;29(8):790–2. PMID: 1769726.
- Ellman GL, Courtney KD, Andres V Jr, Feather-Stone RM. A new and rapid colorimetric determination of acetylcholinesterase activity. Biochem Pharmacol. 1961 Jul;7:88-95. doi: 10.1016/0006-2952(61)90145-9. PMID: 13726518.
- Forget G. Balancing the need for pesticides with the risk to human health. In: Forget G, Goodman T, de Villiers A, editors. *Impact of pesticide use on health in developing countries*. Ottawa: IDRC; 1993.
- Gaur M, Kumar S. Effect of an organophosphorous pesticide on AchE enzyme kinetics in normal and injured myocardium of the heart of *Channa punctatus*. *Biomed Res*. 1993;4(2):171–9.
- Igbedioh SO. Effects of agricultural pesticides on humans, animals, and higher plants in developing countries. Arch Environ Health. 1991 Jul-Aug;46(4):218-24. doi:10.1080/00039896.1991.9937452. PMID: 2069430.
- Jeyaratnam J. Health problems of pesticide usage in the Third World. Br J Ind Med. 1985 Aug;42(8):505-6. doi:10.1136/oem.42.8.505. PMID:4016001; PMCID:PMC1007519.
- Kavlock RJ, Daston GP, DeRosa C, Fenner-Crisp P, Gray LE, Kaattari S, et al. Research needs for the risk assessment of health and environmental effects of endocrine disruptors: a report of the U.S. EPA-sponsored workshop. Environ Health Perspect. 1996 Aug;104 Suppl 4:715-40. doi:10.1289/ehp.96104s4715. PMID:8880000; PMCID:PMC1469675.
- Mathivanan R, Bhaskaran R. AchE activity in brain tissue of *Labeo rohita* (Hamilton) and *Tilapia mossambica* (Peters) in malathion toxicity: in vivo and in vitro studies. *Indian J Environ Toxicol*. 2002;12(2):70–2.
- Muirhead-Thomson RC. *Pesticides and Freshwater Fauna*. London and New York: Academic Press; 1971. 248 p. Review by Caspers H. *Int Rev Hydrobiol*. 1972;57(4):635–6. doi:10.1002/iroh.19720570427.
- Nettleton JA, Allen WH, Klatt LV, Ratnayake WMN, Ackman RG. Nutrients and chemical residues in one- to two-pound Mississippi farm-raised channel catfish (*Ictalurus punctatus*). J Food Sci. 1990;55(4):954-8. doi:10.1111/j.1365-2621.1990.tb01573.x.
- Rathore V, Singh S. Evaluation of serum protein due to toxicity of kinadon in the fish *Channa punctatus* (Bloch). *Bionotes*. 1999;1(2):41.
- Reddy PM, Philip GH. In vivo inhibition of AChE and ATPase activities in the tissues of freshwater fish, *Cyprinus carpio* exposed to technical grade cypermethrin. *Bull Environ Contam Toxicol*. 1994 Apr;52(4):619–26. doi:10.1007/BF00194152.
- Sahib KA, Rao KV. Toxicity of malathion to the freshwater fish *Tilapia mossambica*. Bull Environ Contam Toxicol. 1980;24:870-4.
- Saravanan R, Revathi K, Murthy PB. Lambda-cyhalothrin induced alterations in *Clarias batrachus*. *J Environ Biol*. 2009;30(2):265–70.
- Shaffi S. Sublethal effect of mercury and lead on monoamine oxidase in different regions of the brain in three freshwater teleosts. *Rev Esp Fisiol*. 1995;51(3):125–8. Available from: https://revistas.unav.edu/index.php/ref/article/view/46499
- Srivastava VK, Pandey R, Gupta RC, Barthwal JP, Kishore K. MAO inhibitory and anticonvulsant activity of some new 1-(thiazol-2-yl)-2-methyl-4-(substituted benzylidene)-5-imidazolones. *J Indian Chem Soc*. 1979;56:1024–6.
- Straus DL, Chambers JE. Inhibition of acetylcholinesterase and aliesterases of fingerling channel catfish by chlorpyrifos, parathion, and S,S,S-tributyl phosphorotrithioate (DEF). Aquat Toxicol. 1995;33(3-4):311-24. doi:10.1016/0166-445X(95)00024-X.
- Suman Gulati, Priyadarssini M, Logeswari E, Revathi K. Effect of Lambda-cyhalothrin on the activity of glycolytic pathway and mitochondrial oxidative enzymes. *The Bioscan*. 2025;20(Suppl 2):873-9. doi:10.63001/tbs.2025.v20.i02.S2.pp873-879
- Teugels GG. The nomenclature of African *Clarias* species used in aquaculture. Aquaculture. 1984;38(4):373-4. doi:10.1016/0044-8486(84)90342-9.
- Tsumuki H, Saito T, Miyata T, Iyatomi K. Acute and subacute toxicity of organophosphorus insecticides to mammals. In: O'Brien RD, Yamamoto I, editors. *Biochemical toxicology of insecticides*. New York, London: Academic Press; 1970. p. 65-73.
- Walters J, Kim D, Goh KS. Preliminary results of pesticide analysis of monthly surface water monitoring for the red imported fire ant project in Orange County, March 1999 through August 2002. Sacramento, CA: California Department of Pesticide Regulation; 2002.
- Weston DP, You J, Lydy MJ. Distribution and toxicity of sediment-associated pesticides in agriculture-dominated water bodies of California's Central Valley. Environ Sci Technol. 2004 May 1;38(10):2752-9. doi:10.1021/es0352193.

TABLES:

Table 1a: Effect of Lambda-cyhalothrin at higher sub-lethal concentration (5.768 ppm) on AChE content in various tissues of *Clarias batrachus*

Tissue	F Value	P value	Control	15 Experimental Days	30 Experimental Days	45 Experimental Days	Recovery
Ovary	6.281	0.001**	0.09^{b} ± 0.02	0.07^{ab} ± 0.02 (-22.22)	0.06^{a} ± 0.02 (-33.33)	0.04^{a} ± 0.02 (-55.55)	0.06^{ab} ± 0.01
Muscle	48.48	0.000**	0.25^{c} ± 0.02	0.21^{b} ± 0.02 (-16.00)	0.14^{a} ± 0.02 (-44.00)	0.15^{a} ± 0.01 (-40.00)	0.23^{bc}± 0.01
Liver	34.32	0.000**	0.22^{d} ± 0.02	0.16^{c} ± 0.02 (-27.27)	0.13^{ab} ± 0.02 (-40.90)	0.11^{a} ± 0.02 (-50.00)	0.15^{bc} ± 0.01
Heart	11.34	0.000***	0.24^{b} ± 0.02	0.23^{b} ± 0.02 (-4.16)	0.23^{b} ± 0.02 (-4.16)	0.18^{a} ± 0.02 (-25.00)	0.23^{b}± 0.02
Brain	71.936	0.000**	1.71^{d} ± 0.31	0.93^{b} ± 0.07 (-45.61)	1.21^{c} ± 0.19 (-29.24)	0.68^{a} ± 0.03 (-60.23)	1.44^{c} ± 0.21

Table 1b: Effect of Lambda-cyhalothrin at lower sub-lethal concentration (2.884 ppm) on AChE content in various tissues of *Clarias batrachus*

Tissue	F Value	P value	Control	15 Experimental Days	30 Experimental Days	45 Experimental Days	Recovery
Ovary	5.5411	0.002**	0.095^{b} ± 0.02	0.05^{a} ± 0.02 (-44.44)	0.05^{a} ± 0.02 (-44.44)	0.05^{a} ± 0.02 (-44.44)	0.06^{a} ± 0.01
Muscle	35.57	0.000**	0.25^{b} ± 0.02	0.24^{b} ± 0.02 (-4.00)	0.17^{a} ± 0.02 (-32.00)	0.15^{a} ± 0.02 (-40.00)	0.23^{b} ± 0.01
Liver	24.52	0.000**	0.22^{c} ± 0.03	0.16^{b} ± 0.01 (-27.27)	0.15^{ab} ± 0.01 (-31.81)	0.12^{a} ± 0.02 (-45.45)	0.16^{b} ± 0.01
Heart	2.62	0.058^{NS}	0.24^{a} ± 0.02	0.24^{a} ± 0.02 (0.00)	0.24^{a} ± 0.02 (0.00)	0.21^{a} ± 0.02 (-12.50)	0.23^{a} ± 0.02
Brain	144.66	0.000**	1.735^{b} ± 0.27	0.99^{c} ± 0.08 (-42.77)	1.33^{a} ± 0.16 (-23.12)	0.81^{e} ± 0.09 (-53.18)	1.57^{d} ± 0.23

Table 2a: Effect of lambda cyhalothrin at higher sub-lethal concentration (5.768 ppm)

Tissue	F Value	P value	Control	Experimental Days (15)	Experimental Days (30)	Experimental Days (45)	Recovery
Muscle	50.92	0.000**	2.522^{c} ± 0.229	1.477^{a}± 0.270 (-42.62)	2.022^{b} ± 0.157 (-19.82)	3.025^{d} ± 0.212 (+19.94)	2.560^{c} ± 0.105
Liver	179.56	0.000**	8.435^{a} ± 0.249	8.503^{a} ± 0.235 (+0.84)	9.872^{c} ± 0.188 (+17.03)	11.393^{d} ± 0.243 (+83.48)	9.413^{b} ± 0.182
Heart	35.87	0.000**	2.488^{a}± 0.236	2.517^{a} ± 0.212 (+1.165)	3.125^{b} ± 0.248 (+25.60)	3.883^{c} ± 0.307 (+56.06)	3.073^{b} ± 0.121

Brain	31.40	0.000**	0.540^{a} ± 0.171	0.957^{b} ± 0.189 (+77.22)	1.432^{c} ± 0.248 (+165.18)	1.632^{c} ± 0.174 (+202.22)	0.925^{b} ± 0.156

Table 2b: Effect of lambda cyhalothrin at lower sub-lethal concentration (2.884 ppm)

Tissue	**F Value**	**P value**	**Control**	**Experimental Days (15)**	**Experimental Days (30)**	**Experimental Days (45)**	**Recovery**
Muscle	17.20	0.000**	2.448^{b} ± 0.175	1.912^{a}± 0.380 (-21.89)	2.305^{b} ± 0.205 (-5.84)	2.903^{c} ± 0.152 (+18.58)	2.670^{bc} ±0.077
Liver	53.56	0.000**	8.497^{a} ± 0.239	8.612^{a} ± 0.241 (+1.35)	9.263^{b} ± 0.227 (+9.01)	10.083^{c} ± 0.176 (+18.86)	9.052^{b} ± 0.161
Heart	25.39	0.000**	2.480^{a} ± 0.192	2.605^{ab} ± 0.172 (+5.04)	2.923^{b} ± 0.168 (+17.86)	3.483^{c} ± 0.281 (+40.44)	2.758^{ab} ± 0.082
Brain	17.01	0.000**	0.557^{a} ± 0.178	0.662^{a} ± 0.192 (+18.85)	0.995^{b} ± 0.153 (+78.63)	1.325^{c} ± 0.246 (+137.88)	0.793^{ab} ± 0.103