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Research Article

EFFECT OF DELTAMETHRIN IN THE INDIAN MAJOR CARP CIPRINUS CARPIO WITH SPECIAL REFERENCE TO CHOLINERGIC ACTIVITIES

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ABSTRACT

Effect of deltamethrin in the Indian major carp *Ciprinus carpio* with special reference to cholinergic activity was studied. The fishes were randomly divided into 3 groups having 6 in each group: (1) Control (2) Deltamethrin-induced toxic group (5ppm) (3) Deltamethrin-induced toxic group (10 ppm). Deltamethrin-induced toxic groups increased the Acetylcholine (ACh) and decreased the levels Acetyl cholinesterase (AChE) in Brain of fish. Exposure of fishes to 5ppm and 10ppm sub lethal concentrations of deltamethrin caused rigorous changes in the acetyl cholinesterase activity. Utmost activity was observed in the fish exposed to 10ppm sub lethal concentration.

INTRODUCTION

Acetylcholine is an organic chemical that functions in the brain and body of many types of animals, including fish, as a neurotransmitter-a chemical released by nerve cells to send signals to other cells. Its name is derived from its chemical structure: it is an ester of acetic acid and choline. Parts in the body that use or are affected by acetylcholine are referred to as cholinergic. Substances that interfere with acetylcholine activity are called anticholinergics. Acetylcholine is synthesized in certain neurons by the enzyme choline acetyltransferase from the compounds choline and acetyl-CoA. Cholinergic neurons are capable of producing ACh. An example of a central cholinergic area is the nucleus basalis of Meynert in the basal forebrain (Siegal, A. and Sapru, H.N. 2006)

The enzyme acetylcholinesterase converts acetylcholine into the inactive metabolites choline and acetate. This enzyme is abundant in the synaptic cleft, and its role in rapidly clearing free acetylcholine from the synapse is essential for proper muscle function. Certain neurotoxins work by inhibiting acetylcholinesterase, thus leading to excess acetylcholine at the neuromuscular junction, causing paralysis of the muscles needed for breathing and stopping the beating of the heart.

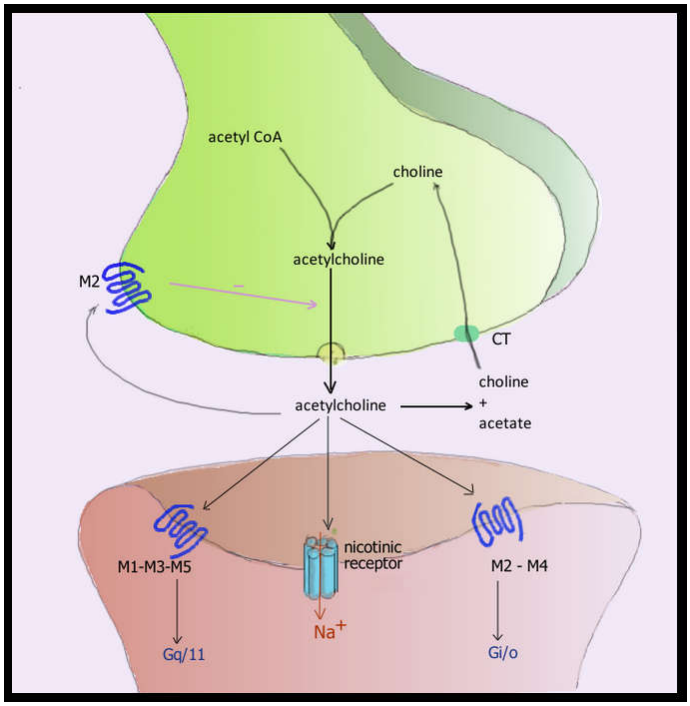

Fig 1 Acetylcholine processing in a synapse. After release acetylcholine is broken down by the enzyme

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Acetylcholine is the neurotransmitter used at the neuromuscular junction-in other words, it is the chemical that motor neurons of the nervous system release in order to activate muscles. This property means that drugs that affect cholinergic systems can have very dangerous effects ranging from paralysis to convulsions. Acetylcholine is also used as a neurotransmitter in the autonomic nervous system, both as an internal transmitter for the sympathetic nervous system and as the final product released by the parasympathetic nervous system. The main function of AChE is the rapid hydrolysis of the neurotransmitter, whereas BChE has no known specific natural substrate, although it is able to hydrolyse acetylcholine. Acetylcholinesterase hydrolyses the neurotransmitter, acetylcholine, thereby ending transmission of nerve impulses at the synapses of cholinergic neurons in the central and peripheral nervous system. Organophosphorus compounds are widely used for agriculture and domestic purpose for controlling insect pests (Videira *et al.,* 2001). Due to their rapid breakdown in water and their low environmental persistence, organophosphorus pesticides have largely replaced the use of organochlorines in recent years (Li and Zhang, 2001). Organophosphorus insecticides are known to inhibit acetylcholinesterase, which plays an important role in neurotransmission at cholinergic synapses by rapid hydrolysis of neurotransmitter acetylcholine to choline and acetate (Soreq and Zakut, 1993). These inhibit the action of the cholinesterase by phosphorylating or carbamylating the active centre of the enzymes. Cholinesterases (ChEs) are a ubiquitous class of serine hydrolases which physiologically remove acetylcholine from the synaptic cleft. ChEs are widely distributed among vertebrate and invertebrate animals (Bocquene et al., 1997).

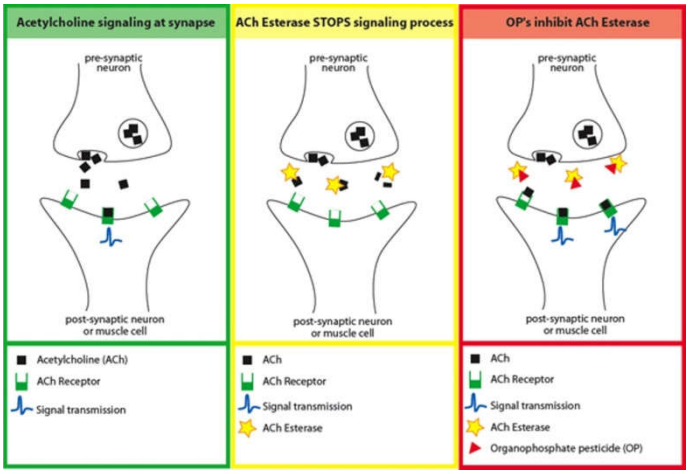


Fig 2 Acetylcholine signaling process from pre-synaptic neuron to post-synaptic neuron

Acetylcholinesterase hydrolyses the neurotransmitter, acetylcholine thereby ending transmission of nerve impulses at the synapses of cholinergic neurons in the central and peripheral nervous system. It has been suggested that BChE acts as a scavenging enzyme in the detoxification of natural compounds (Massoulie *et al*., 1993). Choline, on the other hand, cannot be synthesized by the nervous tissue, but it has to be derived from the diet and delivered to the nerve cell via the blood stream (Schwartz, 1991).

Hence, the present study is undertaken to examine the toxic effect of different concentrations of deltamethrin on selected biochemical parameters in functionally different types of fish organs with particular reference to cholinergic activity.

MATERIALS AND METHODS

Experimental animals

The tests were performed in a concrete holding tanks, glass aquaria, constant supply of water and good lighting system. The indoor tanks were filled with tap water and aerated for 3 days to help reduce the chlorine content. About 300 active test specimens ranging between 5 and 10 cm standard length were transported to the laboratory from a farm. The specimens were acclimatized to laboratory conditions for 7 days in the indoor holding tanks. The pH, dissolved oxygen concentration and temperature of water in the tanks were monitored.

Preliminary tests were conducted to provide guidance on range of concentration of pesticide to use in the bioassay. A stock solution of 25 mg/l was prepared from the original product concentration of 12.5 g/l. From the stock solution, the test solutions were prepared using distilled water. The specimens were not fed a day prior to and during toxicity tests to reduce faecal and excess food contaminating the test solution. The nominal test concentrations were 5ppm&10ppm with six replicates each. The results from the toxicity tests were analyzed, using a World Health Organisation (WHO). The concentrations used were converted by the programme to log dose and the number of dead fishes to mortality Probit values. A plot of these two parameters was made from which the LC_{50} was estimated. The fishes were maintained according to the ethical guidelines.

Selection of Pesticide

Deltamethrin, a synthetic pyrethroid pesticide, was selected for the present study. It was obtained as commercial grade chemical from Sigma chemicals, USA.

Experimental design

The fishes were divided into 3 groups, each consisted of 6 and used for studying the effects of different concentrations of deltamethrin.

Group 1 - Control
Group 2 - 5ppm concentration
Group 3 - 10ppm concentration

Isolation of Tissues

The animals were sacrificed. Brain was separated and frozen in liquid nitrogen (-180^{0}C) and stored at -4^{0}C until further use. At the time of analyses the tissues were thawed and selected parameters were estimated by employing standard methods.

Procurement of Chemicals

All chemicals used in the present study were Analar grade (AR) and obtained from the following scientific companies: Sigma (St. Louis, MO, USA), Fisher (Pittsburg, PA, USA), Merck (Mumbai, India), Ranbaxy (New Delhi, India), Qualigens (Mumbai, India).

Toxicity in aquatic fish for LC_{50}

Fishes were reared and acclimatized to the laboratory conditions in the glass aquaria containing aerated tap water avoiding overcrowding. The water was renewed every day and

the fishes were fed with poultry feed on alternate day. During the experimentation fishes were not fed. The physico-chemical characteristics of the test water such as temperature (22±2^{0}C), water hardness (420 mg/lit), pH (7±0.2) and Dissolved oxygen (4.27 ml/lit) were analyzed during the experiments according to standard methods suggested by APHA, (1989). The stock solution of pesticide, endosulfan (35%) was prepared by dissolving a known concentration of this pesticide into the distilled water and required concentrations were made from the stock solution. The acute toxicity as widely accepted for the period of 24, 48, 72 and 96 hrs have been carried out in the present investigation and the observations were made on the percentage mortality of the fish. The LC_{50} values were calculated by using regression equation method of probit analysis (Hubert and Schoch, 1984).

Bioche mical Analyses

Cholinergic Metabolism

Acetyl cholinesterase (E.C. 3.1.1.7) activity

Acetyl cholinesterase activity was assayed by the method of Ellman *et al*., (1961).

Acetylcholine (ACh) content

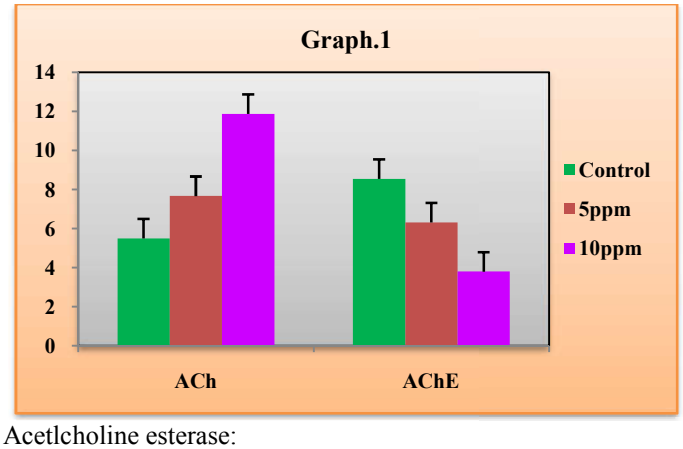
Acetylcholine content was estimated by the method of Hestrin (1949) as given by Augustinson (1957).

Statistical treatment of data

All assays were carried out with six separate replicates from each group. The mean, standard error (SE) and Analysis of Variance (ANOVA) were done using SPSS statistical software for different parameters. Difference between control and experimental assays was considered as significant at $\rho<0.05$

RESULTS

An increase in the content of Acetylcholine is observed throughout dealing with different concentrations of deltamethrin (Tables 1). Acetylcholine esterase levels were decreased in the brain of fish when compared control with experimental groups (Tables 2) and Graph 1.



Acetlcholine esterase:
Control fish brain > 5 ppm deltamethrin > 10 ppm deltamethrin
Acetyl choline:
Control fish brain < 5 ppm deltamethrin < 10 ppm deltamethrin

DISCUSSION

Considerable information is available on the distribution and neurophysiological role of ACh and AChE in many regions of the CNS (Feldberg, 1945; Feldberg and Vogt, 1948; Burgen and Chipman, 1951; Hebb, 1955; Hebb and Silver, 1956; Crossland, 1960; Hebb and Krnjevic, 1962; Whittaker, 1963; Ryall *et al*., 1964; Kasa *et al*., 1970; Pepeu, 1972). The active substance was shown to be acetylcholine (ACh) (Loewi and Navratil, 1926; Dale, 1938; Crossland, 1960; Eccles, 1964; Ryall and De Groat, 1972), which was already known to have powerful biological action (Dale, 1938). ACh, the primary transmitter of the cholinergic system is one of the low molecular weight transmitter substances that are not an amino acid or derivative of aminoacid (Sami Ikonen, 2001). ACh acts as the transmitter at the junctions between motor nerves and skeletal muscle (Brown *et al*., 1936). It was shown that certain spinal cells presented into motor axons are strongly excited by ACh and the synaptic transmission of these cells is enhanced by anticholinesterases and blocked by ACh antagonists (Eccles *et al*., 1954, 1956). In the synaptic terminals, ACh that is stored in the synaptic vesicles is released into the synaptic cleft as a result of an action potential (Caulfield, 1993). ACh diffuses to the pre- and post synaptic membranes where it binds to the receptors. The cholinergic receptors in the mammalian central nervous system can be divided into two groups: muscarinic and nicotinic receptors, based on the ability of two natural alkaloids, muscarine and nicotine, which mimic the effects of ACh (Dale, 1938; Arneric *et al*., 1995). Nicotinic receptors are directly gated ion channels that are usually considered as mediators of acetylcholine in autonomic nervous system ganglia. Muscarinic receptors are the main type of cholinergic receptors in the central nervous system (Caulfield, 1993). Nicotinic actions are typically quick in onset and short-lasting, and are blocked by an excess of nicotine or by curare and curare-like agents. By contrast, muscarinic actions tend to be slow in onset and are prolonged and blocked by atropine and related drug compounds. In general, the parasympathetic system acts on its effector organs by muscarinic transmission. Whereas nicotinic actions are seen characteristically at skeletal neuromuscular junction and in autonomic ganglia, but ACh may act in both ways on the same cell, as in sympathetic ganglia; and the released ACh is normally taken up by the nerve endings. It is hydrolyzed by cholinesterases, and about half of the choline thus formed is immediately absorbed into the nerve endings by an active process that is blocked by hemicholinium-3. This choline is then re synthesized into ACh. In the presence of anticholinesterase agents accumulated exogenous ACh is probably not taken up by the nerve endings since it cannot be released by preganglionic stimulation (Katz *et al*., 1973). In several areas of the nervous system the actions of ACh can be reproduced with nicotine and blocked by dihydro-β-erythroidine, for example in medulla (Salmoiraghi and Steiner, 1963), lateral geniculate (Curtis and Davis, 1963; Phillis *et al*., 1968), cerebellum (McCance and Phillis, 1968). There is a good correlation between ACh content, choline acetyltransferase and AChE activity in all areas of brain but it is found to be very poor in the dorsal root fibers of cerebellum (Silver, 1967). In the cholinergic system, the inactivation of ACh is performed mainly by a specialized enzyme, acetylcholineesterase (AChE). This enzyme is located in the post-synaptic membrane of the synapse, and catalyses the

conversion of ACh molecule and water into acetic acid and choline, which can be returned back to the presynaptic terminal by reuptake process (Taylor, 1991). AChE is found in synapses of central and peripheral nervous systems, as well as neuromuscular junction which play a major role in normal neuronal and neuromuscular transmission (Brimijoin, 1986; Abiola *et al*., 1988). The composition and removal of AChE from the synapse by collagenase, demonstrate that the enzyme is located in the outer basal lamina (Schumacher *et al*., 1986). The active site of AChE contains anionic site and esteratic site. The anionic site is composed of glutamic acid residue which interacts with the nitrogen and choline moiety of acetylcholine, whereas esteratic site contains serine residue which is acetylated by acetylcholine during enzymatic hydrolysis (Derache, 1977). Inhibition of AChE that is responsible for the degradation of acetylcholine will result in excessive stimulation of cholinergic nerves. This will result in tremors, convulsion and finally the death of the aquatic organism (Baxter *et al*.,1997). Several factors seem to be involved in affecting the AChE activity caused by OPs such as length to time and exposure concentrations (Uncer *et al*., 2006).

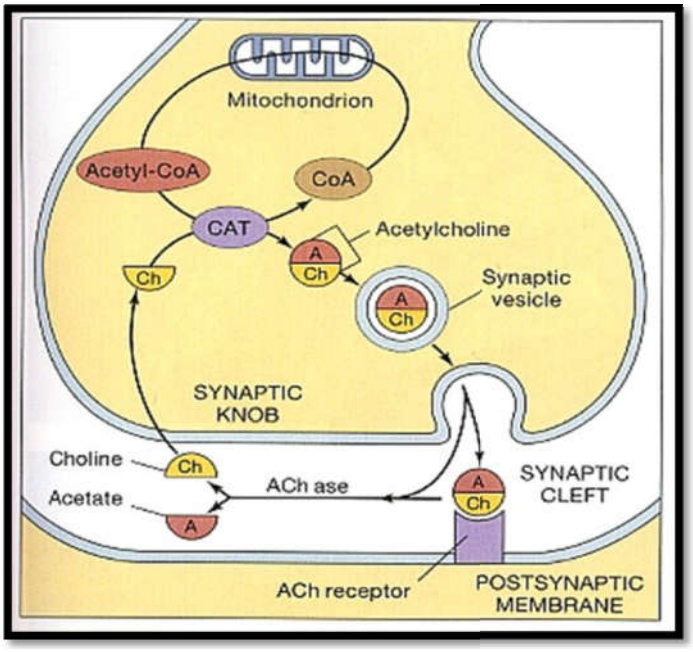


Fig 3 Degradation of acetylcholine in synaptic cleft

Inhibition of AChE, impairs cholinergic nerve impulses and may result in death of organisms (Salles *et al*., 2006). As OPs exert their action by inhibiting AChE activity, measurement of AChE activity in the brain of fish has been described as a method for diagnosing anti cholinesterase pesticides in aquatic solutions (Zinkl *et al*., 1987; Sams *et al*., 2000). Inhibition of AChE was accompanied by an increase in acetylcholine levels (Brzezinski and Ludwicki, 1973). This condition can lead to increase of catecholamines which can affect the activity of enzymes involved in glycogenolysis and glycogen synthesis. Continuous stress may affect the synthesis site of AChE or decrease the levels of excess AChE. Mortality of fish may be due to inhibition of other enzymes, especially those taking part in carbohydrate and protein metabolisms. The inhibitory effect on AChE activity indicates that insecticides might interfere in vital processes like energy metabolism of nerve cells (Ansari *et al*., 1987). Consequently, inhibition of AChE leads to paralysis and death. AChE inhibition in brain, was observed earlier, when fish was exposed to other organophosphorous insecticides like chlorpyrifos and profenfos (Kumar and Chapman, 2001). Quite interestingly, the changes in ACh content and AChE activity occurred in fish brain. This indicates that the deltamethrin effect proceeds through generalized changes in brain of fish in addition to the specific impact on the target areas. Taking into consideration of neuronal damage as reported earlier, it is clear that Acetylcholine is accumulated due to AChE inhibition, which subsequently causes specific neurobehavioral symptoms.

Taking into consideration the role of cholinergic system as a segment of the multiple transmitter systems of the brain, an effort has been made in the present study to inspect the changes in the levels of acetylcholinesterase activity and acetylcholine content in the fish brain on exposure of different concentrations of deltamethrin.

Table 1 Alterations in the Acetylcholine (ACh) content in Brain of *Ciprinus carpio* after insecticide deltamethrin exposure.

Brain	Control	5ppm	10ppm
Acetylcholine (ACh)	5.495 ±0.348	7.668* ±0.452 (39.54)	11.874* ±0.775 (54.85)

(Values are expressed in µ moles of Acetylcholine/g wet wt of tissue)
All the values are mean, ±SE of six individual observations.
Values in '( )'parentheses are % change over control.
*Values are significant at P < 0.05 in Scheffe test.

Table 2 Changes in the Acetylcholinesterase (AChE) activity in Brain of *Ciprinus carpio* after insecticide deltamethrin exposure.

Brain	Control	5ppm	10ppm
Acetylcholinesterase (AChE)	8.546 ±0.250	6.317* ±0.356 (-26.08)	3.796* ±0.128 (-39.90)

(Values are expressed in µ moles of Acetylthiocholine iodide hydrolyzed/mg protein/hr)
All the values are mean, ±SE of six individual observations.
Values in '( )'parentheses are % change over control.
*Values are significant at P < 0.05 in Scheffe test.

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