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DIMETHOATE-INDUCED TOXICITY TO A FRESHWATER TELEOST CATLA CATLA (HAM.)

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ABSTRACT- Present communication deals with the effect of dimethoate toxicity on acetylcholine content in the brain of Catla catla (Ham.). A significant decrease in AChE activity has been noticed. Probable reasons for this alteration have been discussed in detail.

Keywords: Teleost, Catla, Dimethoate, Toxicity.

INTRODUCTION

Organophosphates are widely used pesticides. Undoubtedly they have been a boon for agriculture but their extensive use led to ecosystem instability, as they cause toxicity to non-target organisms. (Mullick & Sharma, 2014). These pesticides are highly toxic not only to fish (Chambers and Yarbough, 1974) but also to other organisms which contribute food for the fish (Butler *et al.* 1970). Dimethoate, a widely used organophosphorus insecticide causes severe metabolic disorders in fish. It affects the nervous system by inhibiting acetylcholinesterase (AchE). The enzyme regulates the amount of neurotransmitter acetylcholine (Fukuto, 1971: Hande and Pradhan, 1990: Satyadevan *et al.*1993: Tembhre and Kumar, 1994). In the present study, the toxic effects of dimethoate are examined on the acetylcholinesterase activity and acetylcholine content in the brain of a freshwater major carp. Catla. Catla (Ham.)

MATERIALS AND METHODS

The fish Catla Catla (length 3" to 4" and weight 10 ±2g) was collected from the fish farm. After the treatment of specimens with potassium permanganate solution, the specimens were kept in glass aquaria (capacity 25 liters). The fingerlings were fed regularly with Shalimar fish food. After the acclimatization of the fish to laboratory conditions for about 10 days, they were used as test materials. The water was changed daily with intermittent aeration. The technical grade of dimethoate of 30% purity was used for experimentation.

Dosage mortality studies were conducted at room temperature ranging from 11.7° C to 27.5° C (±2.0° C) in a static water condition as described by Doudoroff *et al*. (1951) for 96 hrs. The pH and hardness of tap water were measured as 7.0 ±0.2: 72 mg/l respectively. 7.0 ±0.5 mg/l dissolved oxygen and 4.1 ±0.2 mg/l carbon dioxide were also observed. As dimethoate is easily soluble in water, the required quantity was added to 10 liters of water in separate glass aquaria having 10 fish each. The LC_{50} value was then calculated according to the method of Duodoroff *et al*. (1951). The LC_{50} value was read at 50% survival. After the determination of the dosage required for LC_{50} for 96 hrs., the fishes were exposed for 96 hrs. at sub-lethal concentrations of dimethoate as the sub-lethal concentration ranges from 1.3 to 2.3 value of the LC_{50} (Konar, 1969). The tissues (brain) from treated and control fishes were isolated and homogenized in 0.25M cold sucrose solution and centrifuged at 1000g for 15 min. The supernatant was used for the assay.

Acetylcholinesterase (AchE) E.C. 3.1.1.7 activity was estimated by the method of Metcalf (1957) and protein content by the method of Lowry *et al*. (1951). The mean values of the normal and dimethoate-treated fish were subjected to statistical analysis (Bailey. 1965).

RESULTS

The dosage response study shows zero mortality (LC_0) at 0.0025 ppm and 100% mortality (LC_{100}) at 0.010 ppm of dimethoate (Table-1). LC_{50} value of dimethoate to Catla Catla for 96 hrs. was determined by interpolation of different concentrations against the percent of mortality of the fish. The LC_{50} value of dimethoate to C. Catla for 96 hrs. was found 0.007 ppm (Fig. A). The brain showed increased inhibition and decreased AchE activity. The phenomenon continued with the increased concentration of toxicants (Table-2). As the concentration of inhibitor increased, accumulation of Ach content was found (Table–3). The activity is expressed in μ Moles of Acetylcholine hydrolyzed/mg protein/h.

Table–1: Toxicity of dimethoate to Catla Catla (Ham.) for 96 hours.

Dose (in ppm)	No. of fish taken	No. of fish died	Percentage Mortality
0.0025	10	-	0.0
0.005	10	2	20
0.009	10	8	80
0.010	10	10	100

Table – 2: Acetylcholinesterase (AchE) activity of the brain of Catla Catla of normal and exposed to different concentrations of dimethoate at 96 hours (Acute exposure).

Control/Concentration of dimethoate	Specific Activity	Relative Percentage	Inhibition Percentage
Control	1.8±0.02	100	-
0.001 ppm	1.7±0.15	92.9	-7.1
0.0025 ppm	1.3±0.10	73.7	-26.3
0.005 ppm	1.1±0.18	61.3	-38.7

Fig. A: LC_{50} of dimethoate to C. catla for 96 hours.

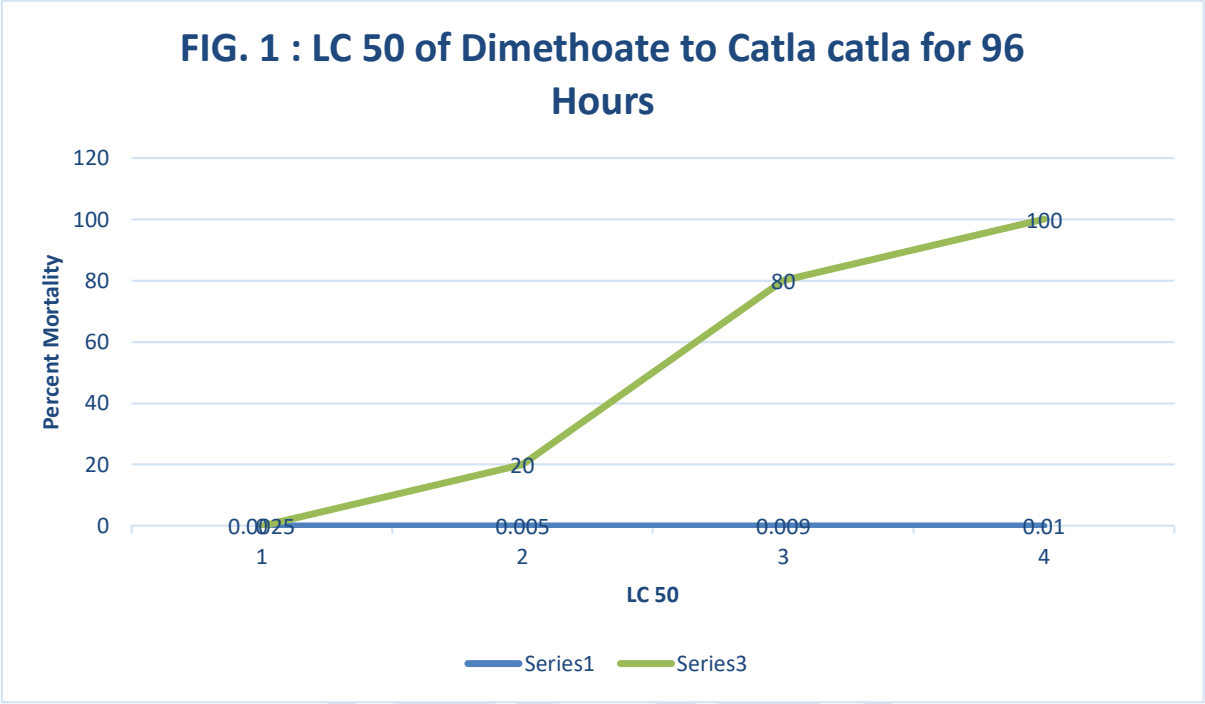

Table – 3: Acetylcholine content (μ moles g wet weight tissue) of the brain of Catla Catla of normal and exposed to different concentrations of dimethoate for 96 hours (Acute exposure).

Control/Concentration of dimethoate	Ach content	Percentage increase
Control	21.15±0.25	-
0.001 ppm	3.70±0.08	12.05
0.0025 ppm	26.28±0.05	24.25
0.0050 ppm	29.48±0.12	39.39

The activity is expressed in μ moles of acetylcholine hydrolyzed mg protein/h.

DISCUSSION

The LC_{50} for acute toxicity of the pesticides depends upon the fish and the duration of exposure. LC_{50} dose of malathion has been found different for different fish for the same duration of 96 hrs. It was 4.0 mg/l for *Clarias batrachus* (Dehadrai. 1990): 15.0 mg/l for *Saccobrachus fossilis* (Verma

et al. 1979): 2.5 mg/l for *Channa Punctatus* (Dubale and Shah. 1975): 0.88 mg/l for *Cirrhinus Mrigala* (Verma *et al*. 1981). Thus it is clear that toxicity of the same chemical differs remarkably in different species (Ruparelia *et al*.1984), Satyadeven *et al*. (1993) reported LC_{50} value of dimethoate to the fish *Cyprinus carpio* as 0.1 ppm for 96 hours of exposure. In the present study, LC_{50} value of the same organophosphate was estimated at 0.007 ppm to Catla Catla for 96 hrs.

Since organophosphorus pesticides are potent inhibitors of acetylcholinesterase enzyme (AchE) and induce neurotoxicity in exposed animals (Briggs and Simons, 1986). These pesticides act by disrupting the passage of impulse across the myoneural junctions by inhibiting the AchE enzyme which modulates the amount of neurotransmitter, acetylcholine (Fest and Schmidt. 1973; Rainsford. 1978). The brain AchE activity in dimethoate-exposed fish has been used in the present investigations as the indicator of OP neurotoxicity.

Rabeni and Stanley (1979) reported that in nature; aerial spraying of acephate, on organophosphate compound has no significant depression of brain AchE activity of Brook Trout and Salmon.

However, Cappage *et al*. (1975) reported that exposure to sub-lethal concentrations of malathion (OP) produced 34% inhibition of brain AchE in *Lagodon rhomboidis*. Sahib et al. (1980) observed the impact of malathion on AchE in *Tilapia mossambica*. They found a maximum of 50.7% inhibition of AchE activity in the brain after 36 hours intervals. Concomitant with decreased AchE activity. The Ach content correspondingly increased after exposure for 36 hours. Shastri and Sharma (1981) studied the effect of sub-lethal concentrations of diazinon enzyme on *Ophiocephalus punctatus*. AchE activity was inhibited from 29 to 42% in the brain of the fishes exposed to diazinon.

Rao and Rao (1982) studied the exposure of fish *Tilapia mossambica* to sub-lethal concentrations of methyl parathion and reported the highest 62% inhibition in AchE activity in brain tissue. They also observed a corresponding increase in Ach content and suggested disruption in nerve impulse conduction. Similar results have been reported by Rao *et al*. (1984) in AchE activity of the liver and brain tissue of malathion, carbaryl, and BHC-exposed *Tilapia mossambica*. They found percent inhibition of AchE activity in brain tissue with malathion, carbaryl, and BHC. 54%, 45%, and 9% respectively. Rao *et al*. (1983) reported- 61.56% inhibition of AchE activity in brain tissue after 48 hours of exposure to methyl parathion in the same fish *T. mossambica*.

Rao and Rao (1989) observed more pronounced inhibitory effects in the brain tissue of *Channa Punctatus* exposed to sub-lethal concentrations of carbaryl and phenthoate and the combined action of C+P on the sensitivity of the AchE system. They reported accumulation of Ach content in tissue could be attributed to the decreased cholinergic transmission as a consequence of exposure to pesticides and their combinations.

Devraj *et al*. (1991) observed maximum 91% inhibition of AchE in brain regions of *Oreochromis mossambicus* exposed to phosalane at the end of 96 hours. Satyadevan *et al*. (1993) reported a maximum of 36.6% inhibition in AchE activity in the brain tissue of *Cyprinus Carpio* exposed to sub-lethal concentrations of dimethoate.

Manju *et al*. (2017) observed maximum 19.2% inhibition of AchE blood serum of *Labeo rohita* exposed to dimethoate at 0.0001 ppm. Venkatewara *et al*. (2021) investigated the toxic effects of malathion on the biochemical parameters of freshwater fish *Labeo rohita*. They observed after 48 hours of exposure the highest % of the decrease in biochemical constituents of all the tissues.

In the present investigation, a significant increase in AchE inhibition (maximum 38.7%) and a concomitant increase in Ach content were noticed. Thus the study supports the findings, several decreases in AchE activity are in agreement with the inhibition of organophosphorus pesticides as they disrupt the nerve impulse transmission of the central and peripheral nervous system by the inhibition of AchE (O. Brein, 1967; Rainford, 1978).

Organophosphorus compounds act by stoichiometrically organophosphorylating the serine at the esteratic site. The linkage formed cannot be split by water. The consequence is an irreversible inhibition of AchE which is rapidly lethal. The cause of accumulation of Ach content is attributed due to not proper hydrolysis of Ach of cholinergic nerves due to inhibition of AchE.

Nachmansohn and Fald (1974) reported the death of an animal occurs when the brain AchE is inhibited by 95%. But in the present study, the maximum inhibition of AchE was found - 38.7% suggestive of the survival of the fish to toxicity at these sub-lethal concentrations of dimethoate. In the normal fish, the brain showed the highest AchE activity indicating its functional significance. Hence the toxic effect of dimethoate is quite obvious by the high inhibition of the AchE enzyme and concomitant increase in ACh content.

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