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Changes in Acetylcholine and associated Acetylcholinesterase activity induced by some organophosphates in a freshwater fish Catla catla (Hamilton).

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Abstract

Inhibition of Acetylcholinesterase activity in freshwater fish Catla catla is demonstrated in the present study using AchE as substrate. The fish on acute exposure to 1/5, 1/3, and 2/3 concentrations of LC_{50} value of dimethoate and malathion showed an increased percentage of inhibition in acetylcholinesterase activity with the increase in dose. This suggests that organophosphates induce a decrease in cholinergic transmission and consequent accumulation of acetylcholine in the tissues, namely, the brain, gill, and liver. Thus, disrupting normal physiology, and behaviour and ultimately may cause the death of the fish. The highest inhibition in AchE activity was observed in the brain followed by the gill and liver. It has been revealed that dimethoate caused a high percentage of inhibition in enzyme activity as compared to malathion in all experimental tissues.

Keywords: Dimethoate, Malathion, Catla catla, Acetylcholine, Acetylcholinesterase.

Introduction

Intensive use of pesticides in agriculture has witnessed a marked increase in soil and water pollution. (Mullick *et al*. 2014). Pesticides have been a boon to agricultural productivity, yet they also cause toxicity to non-target organisms, (Shoaib *et al*. 2016).

Organochlorines are replaced by organophosphates as they are less persistent. Even very small amounts of these toxic chemicals change the quality of water and badly affect fish and other aquatic organisms. (Dey & Saha, 2014), (Dhasarathan *et al*. 2000). They alter the biochemical and physiological metabolic processes of the non-target organisms. Organophosphates impair the nervous transmission of the organisms as they tend to inhibit Acetylcholinesterase activity irreversibly. The enzyme involved in terminating the action of the neurotransmitter

Acetylcholine (Ach) is most often studied. (Singh & Kumar, 2000), (Mushigeri and David, 2005).

Acetylcholine and nor-adrenaline are the two main transmitter substances in the vertebrate nervous system. Acetylcholinesterase enzyme is situated on the postsynaptic membrane which hydrolyses the acetylcholine to Choline and Acetic Acid. The choline is reabsorbed into a synaptic knob to be recycled into acetylcholine by synthetic pathways in the vesicles. (Taylor *et al*. 1998).

Organophosphates are potent neurotoxic agents which inhibit acetylcholinesterase activity, causing accumulation of acetylcholine (Ach) at nerve synapsis that leads to subsequent disruption of neural transmission. Several workers (Aldrige, W. N. 1971), (Coppage *et al*.1975), (Sahib and Ramana Rao, 1980), (Rath and Mishra, 1981), Reddy *et al*. (1992), Korami *et al*. (2000) Hence, Acetylcholinesterase acts as sensitive biomarker for the presence of organophosphate pollutants, (Pazhanisamy, K and N. Indra, 2007). Therefore, AchE can be a good diagnostic tool in biomonitoring programme.

Freshwater fish Catla catla fast-growing food fish is subjected to investigation. dimethoate and malathion are among the widely used pesticides in India. Hence, the present study was carried out to evaluate the comparative toxic effects of dimethoate & malathion in the brain, gill, and liver tissue of fish under acute exposure to pesticides.

MATERIAL AND METHODS

The fish Catla catla (length 3” to 4” and weight 10 ±2 g) was collected from the fish form. Treatment of potassium permanganate solution was given to specimens before they were kept in glass aquaria (capacity 25 lits) for acclimatization. Specimens acclimatized for 10 days were used as test materials. The fingerlings were fed regularly with Shalimar fish food. Dimethoate (30% purity) and malathion (EC50%) were used for the present study.

Dosage mortality studies were conducted at room temperature ranging from 13.7° C to 27.5° C (±2° 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 at 7.0 ± 0.2; 72 mg/l respectively. As dimethoate and malathion both were easily soluble in water, the required quantity was added to 10 liters of water in separate glass aquaria having 10 fish each. Then LC_{50} value for 96 hrs was calculated by the method of Doudoroff *et al*. (1951)

To study the acute toxicity of dimethoate and malathion on fish three sub-lethal concentrations (2/3, 1/3, 1/5) of LC_{50} value were taken. (Konar, 1969).

Estimation of Acetylcholine (Ach)

For Ach estimation, the colorimetric method of Metcalf (1951) was used. The incubation medium was prepared in 5 sets of test tubes. Each set contains 4 test tubes. Tissues after weighing & teasing were kept in boiling water both to inactivate the enzyme and to release bound Ach. Then tissues after cooling homogenized in 2 ml of distilled water and 2 ml of alkaline hydroxylamine hydrochloride and 1 ml of diluted HCL added to this. After centrifugation of this mixture, the supernatant was collected. 1 ml Ferric-chloride was added to the supernatant. The optical density was measured at 540 nm in a spectrophotometer against a blank. Estimated values of Ach content were expressed as µ moles/g wet-weight tissue.

Estimation of AchE activity

Acetylcholinesterase activity (AchE) E.C. 3.1.1.7 was estimated by the method of Metcalf (1951) & protein content by the method of Lowery *et. al*. (1951).

Tissues from control and treated fishes were homogenized in 0.25 M cold sucrose solution and centrifuged at 1000 g for 15 minutes. The supernatant was used for enzyme assay. Acetylcholine iodide was used as substrate. 5 sets of test tubes were prepared. 1 ml of homogenate was taken in the test tube and then 12 µ m of AChI, and 100 µ m of sodium phosphate buffer (pH 7.4) were added to the reaction mixture. After incubation for 30 minutes at 37° C, the reaction was stopped by adding 2 ml of alkaline hydroxylamine hydrochloride solution. 1 ml of HCL (1:1HCL : H_2O) added to this solution. The reaction mixture was then filtered. To develop colour 1 ml of 0.37 M Ferric Chloride solution was added to the clear filtrate and colour was reed at 540 nm in a spectrophotometer. (Systronic, 169 model) using blank (homogenate). The experimental, except distilled water substitutes such as µ M of Acetylcholine, hydrolyzed/mg protein/hour.

The mean values of control and treated fishes were subjected to statistical analysis (Bailey, 1965).

Results

The LC_{50} values of dimethoate and malathion for 96 hrs were found as 0.007 ppm and 0.047 ppm respectively for Catla catla. The upper and lower confidence limits for both LC_{50} values were calculated to be 5.045 and - 0.396 for dimethoate and 4.095 and - 0.296 for malathion. To study acute toxicity 3 sub-lethal concentrations were taken for dimethoate as 0.001 ppm (1/5), 0.0025 ppm (1/3), 0.0050 ppm (2/3); and for malathion as 0.009 ppm (1/5), 0.015 ppm (1/3) 0.031 ppm (2/3).

Accumulation of Ach in the tissues brain, gill, and liver on 96 hrs. exposure of both pesticides for different sub-lethal concentrations are shown in Table 1, 2, 3 Ach content in control fish

was observed maximum in the brain, it was slightly decreased in Gill while it was observed approximately 50% decreased in the liver as compared to brain.

On acute exposure to different concentrations of pesticides, an increase in Ach content was observed with the increase in the concentration of dose. Brain tissue exposed to the highest sub-lethal dose of dimethoate showed the highest increase in ACh content (39.39%), while in the case of malathion, brain tissue showed the highest increase in ACh content (31.18%) on exposure to the highest sub-lethal dose. (Table – 1; Fig. 1)

Gill tissue of fishes exposed to the highest sub-lethal dose of dimethoate and malathion showed maximum accumulation of Ach content of 34.49% and 27.18% respectively. (Table – 2; Fig. 2)

For the same exposure of sub-lethal dose (2/3 of LC_{50}) to dimethoate and malathion liver tissue showed a maximum increase in Ach content of 12.75% and 11.45% respectively. (Table – 3; Fig. 3)

On average after acute exposure (96 hrs.) for both pesticides brain tissue showed the highest increase in Ach accumulation followed by gill and liver.

AChE activity

Acetylcholinesterase activity in the brain, gill, and liver tissue of fish exposed to dimethoate and malathion is shown in Tables 4, 5, and 6. Dose-dependent significant inhibition in AchE activity observed in all three tissues % inhibition increases as the dose increases.

In brain tissue, maximum 38.7% and 31.8% inhibition in AchE activity were observed in fish exposed to the highest sub-lethal dose of Dimethoate and Malathion respectively. (Table – 4; Fig. 4)

For gill tissue maximum inhibition of AchE activity for the same dose & same exposure time was observed at 33.6% and 29.9% for dimethoate and malathion respectively. (Table – 5; Fig. 5)

The liver showed a maximum increase in inhibition of AchE activity for the same exposure of dimethoate and malathion, 14.9% and 13.8% respectively. (Table – 6; Fig. 6)

Results show that percent inhibition in AchE activity in brain, gill, and liver tissue exposed to dimethoate is comparatively high than for malathion, when exposure time, dose & tissues taken for observation were the same. It may be inferred that dimethoate is more toxic than Malathion to fresh-water fish Catla catla.

Discussion

In the present study a significant increase in Ach content was observed in different tissues of fish Catla catla in the order brain > gill > liver exposed to 96 hours. Increased Ach was found during exposure to both pesticides dimethoate and malathion. Results showed a corresponding increase in inhibition of Acetylcholinesterase activity with the increased value of Ach. As the organophosphates are potent inhibitors of the enzyme AchE, they bind the active site and prevent the breakdown of Ach (Koelle, 1963; Aldrige, 1971) resulting blocking of synaptic transmission in the cholinergic nerves. The inhibitory effect of pesticides on AchE activity was not supported by Rabeni & Stanley, (1979) as they reported that in nature, aerial spraying of acephate, an organophosphate compound has no significant depression of brain AchE activity of book trout and Salmon. However, Akter *et. al*. (2020) observed AchE activity in the brain of Heteropneutes fossils and showed significant ($P < 0.05$) inhibition compared with the control group.

However, Sancho. Ferrando and Andreu (1998) reported a 57% decline in AcHE activity in European eel (Anguilla Anguilla) exposed to 0.04 ppm fenitrothion (an organophosphate) while a 51% reduction in AchE activity was reported for 0.02 ppm. Chuiko (2000) carried out a comparative study on 11 freshwater teleost species. He observed in vitro inhibition of brain and serum AchE by DDVP (as an organophosphate pesticide). Similar in vitro inhibition of AchE activity by organophosphates was also reported by (Valbonesi, Brunelli, Mattioli, Rossi & Fabbri, 2011; Rodrigues *et al*. 2011; Colovic, Krstic, Uscumlie & Vasic, 2013). Reza *et al*. (2017) also showed significant inhibition of AchE activity in *Labeo rohita* at 216.7 ± 11.0, 207.3 ± 5.0, and 146.7 ± 5.5 nmol/min/mg proteins after exposure to Envoy 50 SC, Samcup 20 EC and Dursban 20 EC, respectively. *Labeo rohita* showed higher enzymatic inhibition (51.49%) in comparison to B. gonionotus (19.60%).

Findings of the present study also interpret a significant increase in inhibition of AchE activity, Highest inhibition of AchE in brain tissue exposed to both organophosphate pesticides and the consequent highest accumulation of Ach content may be due to the decreased ionic composition in the tissues of Catla catla.

The inhibitory activity of AchE in Catla catla under organophosphates stress offers support to the present observation. Reddy *et. al*. (1992) was of the view that a high level of inhibition of AchE activity with a concomitant increase in Ach content in the tissues is due to inhibition in the integral activity of the central nervous system and Ach accumulated in the brain and other tissues. Damage in the central nervous system may result in uncontrolled hormonal release and consequently degeneration of many biochemical and physiological functions (Corbett, 1974). As gill tissue showed also a high level of reduction in AchE activity and consequent increase in Ach content. This may be explained as gills are involved in the osmoregulation process in the

fish and greater uptake of pesticides may be related to this function, (Mushigiri & David, 2005). As they are in direct contact with the ambient medium and when they are exposed to pesticides, gills rapidly accumulate the pesticide, (Lock *et. al*. 1981). The liver is the primary organ for detoxification (Hutter *et. al*. 1969), therefore, it is expected that pesticides would reach there. In the present study, the liver should maximum increase in inhibition of AchE activity at 14.9% and 13.8% for dimethoate and malathion respectively, and a corresponding increase in Ach content. The study supports the findings of Manju *et. al*. (2017), who observed maximum 19.2% inhibition of AchE in the blood serum of *Labeo rohita* exposed to dimethoate at 0.0001 ppm. Venkatewara *et. al*. (2021) observed 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.

AchE activity is an important biomarker for organophosphates and carbamates pesticides than other contaminants. These pesticides bind the active site and prevent the breakdown of Ach (Long 1963 & Aldrige, 1971), causing the blocking of synaptic transmission in cholinergic nerves. AchE hydrolysis acetylcholine, a neurotransmitter liberated at synapses. The passage of nerve impulses from one nerve cell to another across the synaptic gap is facilitated by neurotransmitters. If AchE is inhibited, Ach accumulates and nerve impulses cannot be stopped, causing prolonged muscle contraction, as a consequence paralysis or death may occur.

Conclusion

Thus, exposure to dimethoate and malathion causes inhibition of AchE activity and accumulation of Ach at synaptic junctions in Catla catla. This may create widespread disturbance in the normal physiology of the organism.

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Tables and Figures

Table: 1 Acetylcholine Content of the Brain of Catla catla on Normal and Exposed to different concentrations of Dimethoate and Malathion at 96 hours

Control/ concentration of Dimethoate	Biochemical parameters of Dimethoate		Control/ concentration of Malathion	Biochemical parameters of Malathion	
	Ach content (µM/g wet weight tissue	Percentage increase		Ach content (µM/g wet weight tissue	Percentage increase
Control	21.15±0.25	-	Control	20.20±0.16	-
0.001 ppm	23.70±0.08	12.05	0.009 ppm	22.10±0.25	9.40
0.0025 ppm	26.28±0.05	24.25	0.015 ppm	24.20±0.08	19.80

0.0050 ppm	29.48±0.12	39.39	0.031 ppm	26.50±0.10	31.18

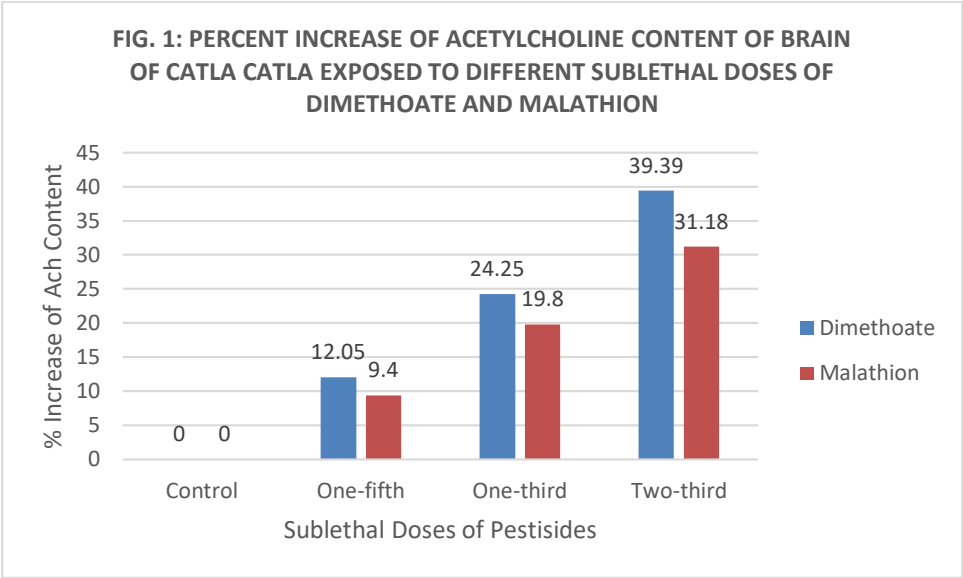

Table: 2 Acetylcholine Content of Gill of Catla catla on Normal and Exposed to different concentrations of Dimethoate and Malathion at 96 hours					
Control/ concentration of Dimethoate	Biochemical parameters of Dimethoate		Control/ concentration of Malathion	Biochemical parameters of Malathion	
	Ach content (µM/g wet weight tissue	Percentage increase		Ach content (µM/g wet weight tissue	Percentage increase
Control	20.15±0.05	-	Control	20.05±0.20	-
0.001 ppm	22.50±0.10	11.66	0.009 ppm	21.48±0.08	7.13
0.0025 ppm	24.70±0.12	22.58	0.015 ppm	23.28±0.15	16.10
0.0050 ppm	27.10±0.25	34.49	0.031 ppm	25.50±0.25	27.18

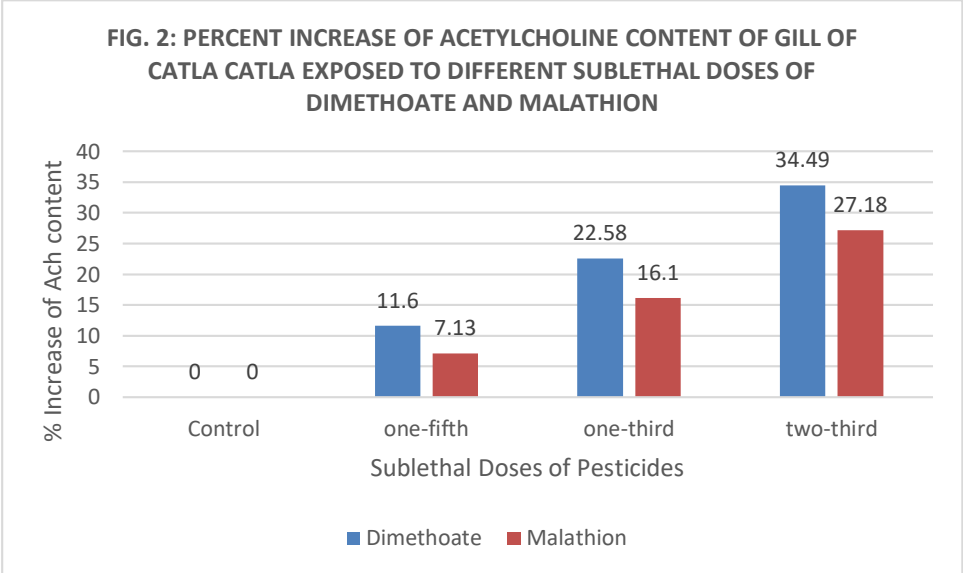

Table: 3 Acetylcholine Content of the Liver of Catla catla of Normal and Exposed to different concentrations of Dimethoate and Malathion at 96 hours.					
Control/ concentration of Dimethoate	Biochemical parameters of Dimethoate		Control/concentration of Malathion	Biochemical parameters of Malathion	
	Ach content (µM/g wet weight tissue	Percentage increase		Ach content (µM/g wet weight tissue	Percentage increase
Control	10.20±0.05	-	Control	9.6±0.06	-
0.001 ppm	10.60±0.09	3.92	0.009 ppm	9.8±0.04	2.08
0.0025 ppm	10.76±0.16	5.49	0.015 ppm	10.03±0.12	4.48
0.0050 ppm	11.50±0.22	12.75	0.031 ppm	10.70±0.04	11.45

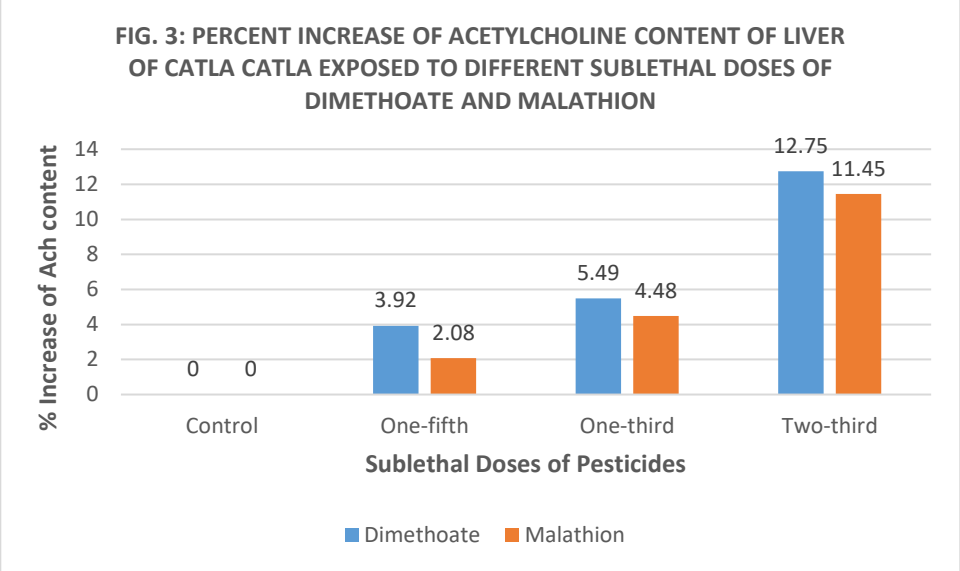

Table: 4 Acetylcholinesterase (AchE) Activity of the Brain of Catla catla on Normal and Exposed to different concentrations of Dimethoate and Malathion at 96 hours.							
Control/concentration of Dimethoate	Biochemical parameters of Dimethoate			Control/concentration of Malathion	Biochemical parameters Malathion		
	Specific activity	Relative %	inhibition %		Specific activity	Relative %	inhibition %
Control	1.8± 0.02	100.0	-	Control	1.9± 0.02	100.0	-
0.001 ppm	1.7± 0.15	92.9	-7.1	0.009 ppm	1.8± 0.10	94.3	-5.7
0.0025 ppm	1.3± 0.10	73.7	-26.3	0.015 ppm	1.5± 0.05	76.9	-23.1
0.0050 ppm	1.1± 0.18	61.3	-38.7	0.031 ppm	1.3± 0.04	68.2	-31.8

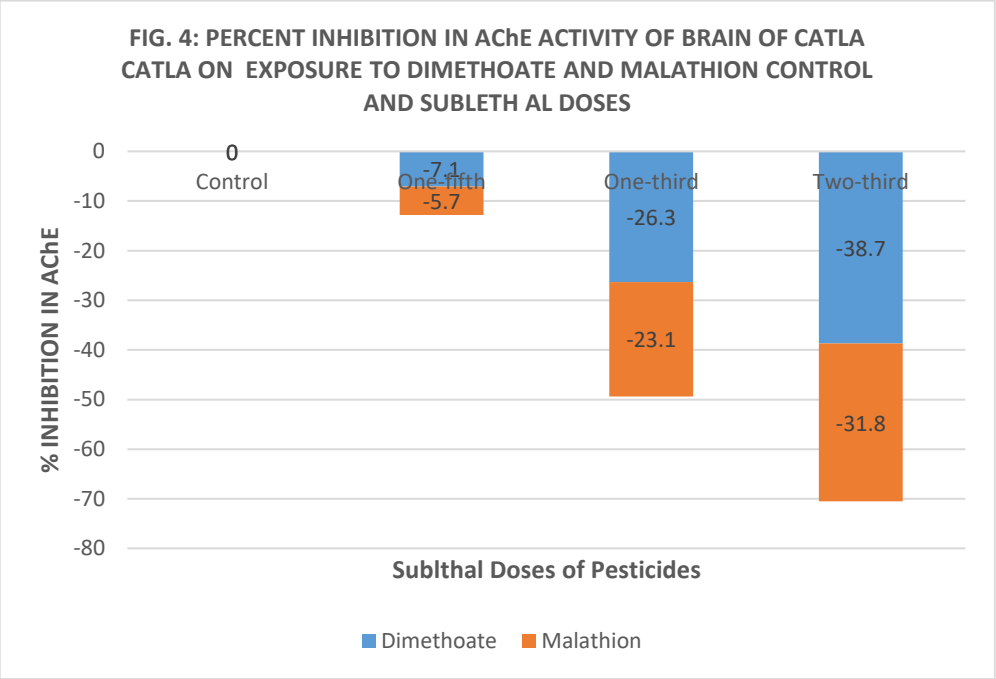

Table: 5 Acetylcholinesterase (AChE) Activity of Gill of Catla catla on Normal and Exposed to different concentrations of Dimethoate and Malathion at 96 hours

Control/concentration of Dimethoate	Biochemical parameters of Dimethoate			Control/concentration of Malathion	Biochemical parameters Malathion		
	Specific activity	Relative %	inhibition %		Specific activity	Relative %	inhibition %
Control	1.7± 0.10	100.0	-	Control	1.8± 0.03	100.0	-
0.001 ppm	1.5± 0.15	93.2	-6.8	0.009 ppm	1.7± 0.05	94.8	-5.2
0.0025 ppm	1.3± 0.20	77.1	-22.9	0.015 ppm	1.4± 0.15	77.9	-22.1
0.0050 ppm	1.1± 0.18	66.4	-33.6	0.031 ppm	1.3± 0.18	70.1	-29.9

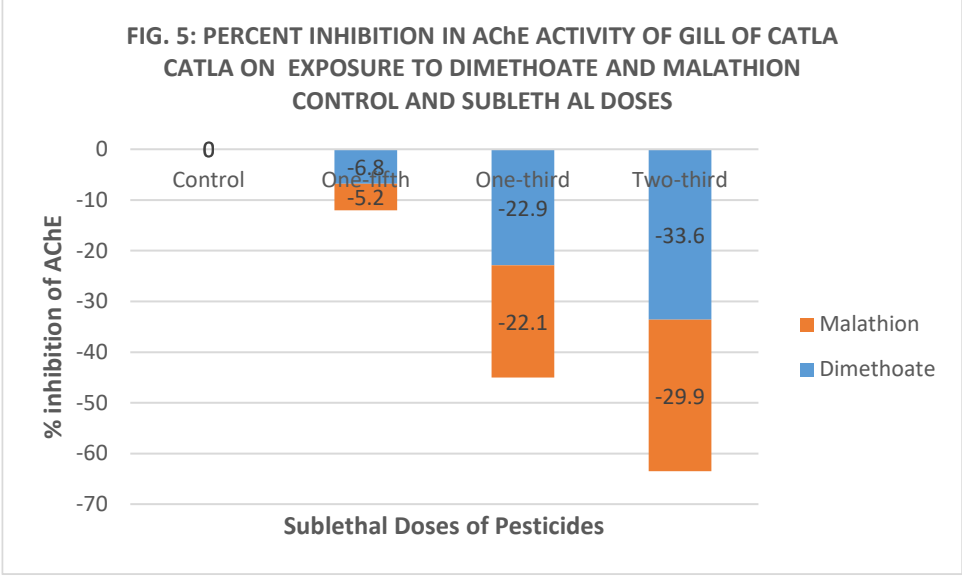

Table: 6 Acetylcholinesterase (AchE) Activity of the Liver of Catla catla on Normal and Exposed to different concentrations of Dimethoate and Malathion at 96 hours.							
Control/concentration of Dimethoate	Biochemical parameters of Dimethoate			Control/concentration of Malathion	Biochemical parameters Malathion		
	Specific activity	Relative %	inhibition %		Specific activity	Relative %	inhibition %
Control	0.47± 0.10	100.0	-	Control	0.48± 0.07	100.0	-
0.001 ppm	0.45± 0.20	96.4	-3.6	0.009 ppm	0.47± 0.02	97.1	-2.9
0.0025 ppm	0.43± 0.05	90.8	-9.2	0.015 ppm	0.44± 0.06	91.4	-8.6
0.0050 ppm	0.40± 0.15	85.1	-14.9	0.031 ppm	0.41± 0.18	86.2	-13.8