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TOXICITY OF DIMETHOATE ON THE SPECIFIC ACTIVITY LEVELS OF DEHYDROGENASE ENZYMES OF FRESH WATER FISH LABEO ROHITA

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ABSTRACT

In this study the fresh water fish Labeo Rohita was exposed to sublethal concentration of Organophosphate compound "Dimethoate" resulted in inhibition of LDH activity in liver, brain and gill tissues. The enzyme NAD specific LDH is associated with cellular metabolic activity and involved in the interconversion of pyruvate to lactate and vice versa under anaerobic condition. The activity of NAD+ ICDH is inhibited due to reduction in oxidative metabolic rate. In TCA cycle the reversible oxidative dehydrogenation of succinic acid into fumaric acid is catalyzed by the enzyme SDH. This is the only reaction in TCA cycle which involves coenzyme flavin (FAD) as an immediate acceptor of electron without participation of NAD (Harper 1985). SDH activity is inhibited by Dimethoate compound. The fish showing a shift towards anaerobic metabolism resulted by the depressed oxidation in mitochondria, the MDH activity is also inhibited in liver, gill and brain tissues after subsequent exposure to Dimethoate. This is the one of the key enzyme of TCA cycle representing the terminal oxidation step the catalyzation of malate to oxaloacetic acid.

I.INTRODUCTION

The widespread use of organophosphorus pesticides in agriculture has increased since the past three decades. These pesticides are entering into the aquatic environment and effects the aquatic animals like fishes. The common property of OP compounds is inhibits the certain enzyme of carbohydrate metabolism in the fish. In the present investigation an attempt has been made to study the impact of technical grade OP compound **Dimethoate** on certain biochemical changes in *LabeoRohita*.

II.MATERIAL AND METHODS

Healthy fresh water edible fish *Labeo Rohita* was selected as the test animal having length 25-35 cm and weighing around 35-50 gms. The fishes were kept in a cement tank for acclimatized with laboratory conditions for 15 days under continuous running tap water and fed with wheat pellets and groundnut cakes. The feeding was stopped one day prior to the starting of experiment. Sublethal concentration of (LC50) Dimethoate is exposed to the fish for 48 hours and tissue samples of liver, brain and gill were quickly isolated and kept in ice-

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jacked petridishes to carry out further experimental work – LDH, SDH, ICDH and MDH were estimated according to Nachlas (et al: 1960) and knornberg and Pricer (1951) respectively. All dehydrogenase enzymes was assayed by the method of Nachlas et al. (1960) with slight modification. The reaction mixture in total volume of 2 ml contained 100 micromoles of phosphate buffer (pH 7.4), 50 micromoles of lithium lactate, 2 micromoles of INT, 0.1 micromoles of NAD and 0.4ml of enzyme source. The reaction mixture was incubated at 29°C for 30 minutes and the reaction was stopped by the addition of 5 ml of glacial acetic acid. The formazan formed was extracted with 5 ml of toluene for overnight at 5°C. The enzyme activity was expressed in micromoles of formazan formed per mg protein per hour.

Table-1: Changes in LDH, SDH, ICDH and MDH activity (micro moles of formazan formed/mg protein/h) in tissues of control and dimethoate exposed fish*Labeo rohita*.

Name of the Enzyme	CONTROL	48 HOURS	72 HOURS
and Tissue			
LDH			
LIVER	2.43 ± 0.04	1.61 ± 0.15	1.42 ± 0.04
		PC = - 33.74	PC = - 41.56
BRAIN	0.69 ± 0.07	0.32 ± 0.01	0.30 ± 0.02
DRAIN		PC= -53.62	PC = -56.52
GILL	0.81 ± 0.02	0.49 ± 0.02	0.45 ± 0.02
		PC = - 39.5	PC = - 44.44
SDH			
LIVER	6.79 ± 0.21	3.12 ± 0.13	2.91 ± 0.12
LIVER		PC = - 54.05	PC = - 57.14
BRAIN	2.62 ± 0.15	1.56 ± 0.21	1.32±0.20
Bidan		PC = -40.46	PC = -49.62
GILL	0.98 ± 0.10	0.57 ±0.08	0.49 ±0.09
		PC = -41.84	PC = -50.00
ICDH			
LIVER	0.39 ± 0.01	0.21 ±0.04	0.19 ±0.03
LIVER		PC = -46.15	PC = -51.28
BRAIN	5.22 ± 0.01	0.11 ± 0.02	0.09 ± 0.009
		PC = -50.23	PC = -59.27
GILL	0.06 ± 0.006	0.039 ± 0.08	0.02 ± 0.008
GILL		PC = -36.06	PC = -67.21
MDH			
LIVER	2.29 ± 0.05	0.98 ±0.01	0.91 ± 0.02

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		PC = -57.20	PC = -60.26
BRAIN	1.12 +±0.08	0.46 ± 0.02	0.41 ± 0.02
		PC = -58.03	PC = -63.39
GILL	0.29 ±0.04	0.09 ± 0.01	0.08 ± 0.01
		PC = -68.96	PC = -72.41

Each Value is mean \pm S.D. of 6 observations. All values are statistically significant from controls at 1% level (p<0.01). PC denotes Percent Change over control.

III.RESULTS AND DISCUSSION

The Table shows the activity of LDH, SDH, ICDH and MDH are presented during 48 hours, 72 hours exposure to Dimethoate, the decrease in the activity of LDH and NAD dependent ICDH was more in the exposed fish at 48 to 72 hours.

The inhibition of TCA cycle enzymes ICDH, SDH, MDH indicates decreased mitochondrial oxidative metabolism due to disfunction in mitochondrial integrity and shift towards anaerobic metabolism. Similar effects of pesticides on dehydrogenase enzymes were reviewed by different research workers. (Murthy *et.al* 1985), (Venkateshwarlu *et.al* 1987), (Shobha Rani *et.al* 1989), (Ganesh *et.al* 1989), (Vatukuru *et. al* 2003), (Tripathi & Singh *et.al* 2004)& (Abdul Naveed *et.al* 2006).

From the above studies, it may be suggested that Dimethoate decreases the oxidative metabolism in the tissues of fish *Labeo Rohita*. The changes in the activity of the carbohydrate metabolism enzymes indicate a decrease in energy metabolism through oxidative pathways and consequently the fish switch over to anaerobiosis as a compensatory adaption to overcome the **Dimethoate** (OP) toxic stress. In conclusion the gluconeogenesis appears to play an important role in *Labeo Rohita* for maintaining the glucose levels during the **Dimethoate** treatment indicating production of glucose from other non-carbohydrates.

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