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EFFECT OF DIMETHOATE ON THE LEVEL OF GLYCOGEN IN TISSUES OF FRESHWATER FISH *PUNTIUS TICTO* (HAM)

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ABSTRACT

Freshwater fish *Puntius ticto,* exposed to lethal and two sublethal concentrations (5.012ppm; 2.506 ppm and 1.253ppm) of dimethoate for 96hr and 60 days respectively. Glycogen content was analyzed from different tissues after exposure period. The significant decrease in the glycogen content was observed in gills, kidney and brain; moderate decline in gonads, muscles and slight changes takes place in liver and intestine.

INTRODUCTION

Indiscriminate use of pesticides in agriculture to control the crop pests has indirectly created the problem of pollution in aquatic ecosystem. These pesticides are also injurious to non-target organisms like fish. Among pesticides the organophosphorous pesticides are most preferred to eradicate pests and insects due to their low resistance in the environment, this resulted in contamination of freshwater bodies. Various fish species have been studied in context of shown uptake and accumulation of many contaminants or toxicants such as pesticides (Herger *et al.*,1995), which may causes physiological and biochemical changes in the freshwater fauna by influencing the activities of enzymes and metabolites (Koundinya,1978). The biochemical changes in different organs/tissues of fish due to toxicity stress of heavy metals and pesticides have been reported by number of workers Khan *et al.*,1992; Balint *et al.*,1995; Das *et al.*,1999; Rao and Ramaneshwari, 2000; Khare and Singh, 2002). A very little work has been done on the toxic effects of pesticides on biochemical contents in the tissues of *Puntius ticto*. Therefore the present work is carried out to study the effect of dimethoate pesticide on biochemical contents during acute and chronic exposure.

MATERIALS AND METHODS

Puntius ticto, a freshwater fish were collected from the fresh water sources around Aurangabad city (M.S., India). They were acclimatized to the laboratory conditions, for a period a period of two weeks. During acclimatization they were fed on alternate days with pieces of live earthworms. The LC_{50} values are determined by following the guidelines given by Annon (1975) and Finney's Probit Analysis Method (1971). The acclimated fishes were exposed to lethal concentration (5.012ppm) for 96hr and two sublethal concentrations (2.506ppm and 1.253ppm) for 60 days. Simultaneously a control group of healthy fishes were maintained under identical conditions. The fishes were sacrificed immediately at the end of exposure period and different tissues were processed for the biochemical estimations. Glycogen content was estimated by using Anthrone Reagent Method (De zwann and Zandee,1972).

RESULTS AND DISCUSSION

The glycogen content was analyzed from the tissues of experimental and control fish. The result shows that significant decrease of glycogen content in the gills, kidney and brain. Moderate decline was observed in gonads, muscles whereas slight change found in liver and intestine.

The chronic exposure results when compared, showed that there is decrease in the amount of glycogen in all tissues at 2.506ppm exposure; whereas increased glycogen content was observed at 1.253ppm exposure (Table 1). The glycogen decreased progressively throughout the exposure period (Table 1) in all tissues.During stress an organism needs sufficient energy which is supplied from reserve material *i.e.* glycogen. Significant decrease of glycogen was observed in

Table 1: Fluctuations in glycogen content in *Puntius ticto* to dimethoate toxicity exposure

Sr. No.	Tissues	Control	Letal	% change	Sub-lethal (5.012 pm)	% change	Sub-lethal (2.506 ppm)	% change (1.253 ppm)
	ovary	0.396 ± 0.0089	0.1032 ± 0.0364	-73.94	0.1389*** ± 0.0297	-64.92	0.1687*** ± 0.0298	-57.4
2	Testis	0.4982 ± 0.0059	0.1985*** ± 0.0297	-60.16	0.1687*** ± 0.0298	-66.14	0.2289*** ± 0.0298	-54.17
3	Intestine	0.8217 ± 0.0150	0.3731*** ± 0.0238	-54.59	0.4565*** ± 0.0595	-44.44	0.6351** ± 0.0595	-22.71
4	Muscles	0.6252 ± 0.0112	0.2580*** ± 0.0298	-58.73	0.1389*** ± 0.0298	-77.78	0.2481*** ± 0.0149	-60.32
5	Gills	0.5379 ± 0.0124	0.2084*** ± ± 0.0172	-61.26	0.0992*** ± 0.0595	-81.56	0.1270*** ± 0.0119	-76.39
6	Kidney	0.9448 ± 0.0259	0.4565*** ± 0.0595	-51.68	0.1489*** ± 0.1002	-84.24	0.2878*** ± 0.0298	-69.54
7	Brain	0.2243 ± 0.0215	0.1171* ± 0.0595	-47.79	0.0397*** ± 0.0059	-82.3	0.0635*** ± 0.0059	-71.69
8	Liver	0.722 ± 0.0155	0.3374*** ± 0.0595	-53.27	0.3771*** ± 0.0227	-47.77	0.5756*** ± 0.0149	-20.28

The values are expressed in mg/100 mg dry weight (mean S.D.); * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$.

gills, kidney and brain because these organs are more active and they require large amount of energy. This energy demand is solved by utilizing reserve food material in the form of glycogen. It also appears that vigorous struggling may enhance muscle activity which may probably contribute to glycogen breakdown *i.e.* glycogenolysis. Kabeer (1979) suggested that the decrease in glycogen content in Malathion treated freshwater fish *Tilapia mossambica* may be due to decrease in glycogen synthesis. Grant and Schoetteger (1972) reported organochlorine contaminants blocked the glycogenolysis in fish. Rao and Rao (1979) also reported decrease in glycogen content of liver after methyl parathion treatment in *Tilapia mossambica*. Mane *et al.,* (1986) studied fenthion induced biochemical changes in lamellibranch mollusk *Indonaia caerules* .They reported constant decrease in glycogen and lipid in certain tissues and stated it may due to their utilization for energy. Studies indicating such depletion in fish models (Mishra and Srivastava, 1984) during organophosphorous toxicity often excellent support to the decreasing levels of glycogen in the present study. Nasreen *et al.,* (1994) also observed marked depletion in the glycogen content in all the tissues to phenol exposure in *Channa punctatus*. Similar results were observed by Shakoori *et al.,* (1996);Das *et al.,* (1999);Shobha *et al.,* (2007) reported decrease in glycogen in muscle, gills, liver heart and kidney of *Catla catla,* when exposed to cadmium chloride and stated that glycogen reserves are being used to meet the stress through glycolysis or hexose monophosphate pathway. It is assumed that decrease in glycogen content may be due to inhibition of hormones which contribute to glycogen synthesis. Jha and Pandey (1989) stated that depletion in glycogen might be due to rapid glycogenolysis and inhibition of glycogenesis through activation of glycogen phosphorylase and depression of transferase. According to Chezhian *et al.,* (2010) decreased level of glycogen may be due to the induced activation of adrenal pituitary glucocorticoid hormones which stimulate the hepatic glucose production thereby elevating blood glucose level to meet the critical need of energy under effluent stress. During present study glycogen level in all tissues decreased continuously when the concentration of dimethoate and period of exposure increases. Similar results were observed by Singh and Bhati (1994); Jones and Kumar (1996);Rawat *et al.,* (2002). The fish showed stress condition during the exposure period as fast swimming, fast opercular movements, dashing with the walls of aquarium, reduced feeding etc. So during such type of stress conditions the glycogen reserves are decreased to meet energy demand by the process of glycogenolysis. The decrease in the glycogen content in tissues of *P. ticto* can be due to its enhanced utilization as an immediate source to meet energy demands under pesticide stress. It could also be due to the prevalence of hypoxic or anoxic condition. Under hypoxic conditions the animal derives its energy from anaerobic breakdown of glucose which is available to the cells by the increased glycogenolysis (Chandravathy and Reddy, 1995). Similar results also found by Valarmathi and Azariah (2002); Vutukuru (2005); Venkatramana *et al.,* (2006); Muley *et al.,* (2007).

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