

Impact of malathion on the biochemical parameters of gobiid fish, *Glossogobius giuris* (Ham)

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Abstract: There is a dearth of information regarding the changes in heart muscle metabolites induced by pesticides. In the present study, the gobiid fish, *Glossogobius giuris*, was exposed to sub lethal concentrations of (0.05, 0.25 and 0.5 ppm) organophosphorus pesticide, malathion for short duration (24 to 96 hr). The cardiac muscles showed maximum depletion of glycogen and cholesterol content during 72 and 96 hr after treatment with 0.5 ppm malathion. Whereas a slight fluctuation of protein and glycogen content was observed in low concentration (0.05 ppm) of malathion. The levels of protein showed a significant decrease at high concentration (0.5 ppm) when treated for longer duration (96 hr). The present study reports metabolic dysfunction in response to malathion toxicity in the fish.

Key words: *Glossogobius giuris*, Malathion, Biochemical components, Heart.

Introduction

Numerous biochemical indices of stress have been proposed to assess the health of non-target organisms exposed to toxic chemicals in aquatic ecosystem (Nimmi, 1990). However, it has been reported that apart from nerve tissue, tissues like blood, liver and gills also contribute information in the detection of toxic symptoms caused by certain groups of pesticides. Christensen and Tucker (1976) reported quantitative changes in carbonic anhydrase activity in channel catfish, *Letaturus punctatus* as a diagnostic sign of pesticide poisoning. Environmental and chemical stress can interfere with physiological and biochemical functions such as growth, development, reproduction and circulatory system in fish. The circulatory system is greatly affected by the water quality and external environmental factors but there is a lack of information about cardiac muscle metabolites of aquatic animals. The present study was designed to understand the impact of sub lethal concentrations (0.05, 0.25 and 0.5 ppm) of malathion on heart muscle glycogen, protein and cholesterol in freshwater gobiid fish, with an exposure period of 24, 48, 72 and 96 hr.

Materials and Methods

The freshwater gobiid fish, (body length 8-10 cms, body wt. 20-25 gm) were collected from local Kelavarpalli dam near Bangalore by using cast and gill nets (mesh: 10 mm) and acclimated lab conditions in glass aquaria (60" x 30" x 20") for 15 days. Fish were fed with standard laboratory diet (Trio) during acclimatization and toxicity test. Commercial grade of malathion [0, 0-dimethyl, S- (1, 2- dicarboethoxy ethyl) phodophorodithioate] was used in the study. Acetone: Ethanol (1:1 v/v) was used as a solvent for the preparation of stock solution, which was further diluted to required concentrations of 0.05, 0.25 and 0.5 ppm in water, was made by adapting the dilution techniques as outlined in APHA (1985).

The acclimatized fish were divided into four experimental groups. The first three groups of fish were placed in freshwater having sub lethal concentrations (0.05, 0.25 and 0.5 ppm, eight fish for each interval in each concentration) of malathion and the fourth group was kept in freshwater with no pesticide and served as control. The acclimated fish were starved for 24 hr prior to their use in the experiment and were not fed during the course of experiments (Dalela *et al.*, 1978). The water was changed after every 24 hr.

Fish from the experimental and control groups were vivisected without anesthesia after an interval of 24, 48, 72 and 96 hr (eight fish in each interval for each concentration). The heart was carefully removed; standard procedures were used for the estimation of glycogen (Hassid and Abraham, 1957), protein (Lowry *et al.*, 1951) and cholesterol (Zlatkis *et al.*, 1953). The results were expressed as mg/g wet weight of the tissue.

Results and Discussion

The changes in glycogen, protein and cholesterol levels in heart muscles of fish after the treatment with malathion are presented in Table 1, 2 and 3 and Fig. 1,2 and 3. While analyzing the changes in the glycogen, protein and cholesterol it became clear that they fluctuated during different intervals of treatment. For instance, after 24 hr of treatment with 0.05 ppm of malathion the muscle glycogen content decreased by 47.93%, whereas higher concentration (0.5 ppm) the quantity of glycogen increased to a maximum. However, after 48 and 72 hr treated with 0.25 ppm malathion, quantity of glycogen increased when compared to control (Fig. 1). A similar change was also observed in the 0.05 and 0.5 ppm malathion exposed fish for 48 hr. However, after 96 hr the quantity of muscle glycogen showed a decreasing trend reaching 20.31% and 21.26% of 0.05 and 0.5ppm exposure respectively (Table 1 and Fig.1.)

The glycogen content was altered in the heart muscles of fish after exposure to 0.5 ppm malathion during 24

Table – 1: Glycogen content (mg/g) of *Glossogobius giuris* exposed to sublethal concentrations of malathion.

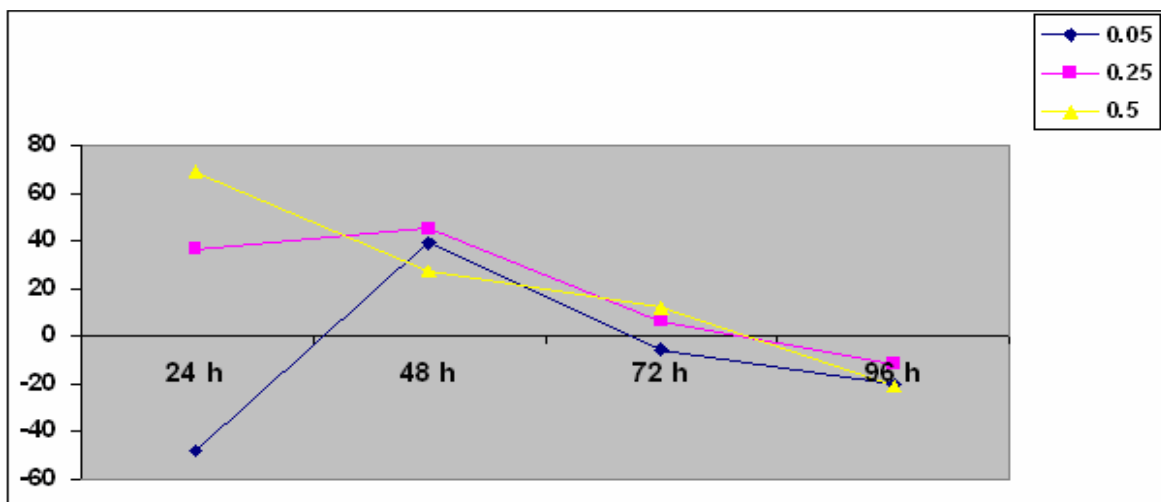
	24hr	48hr	72hr	96hr
Control	2.90±0.72	2.20±0.25	3.30±0.33	3.15±0.47
0.05 ppm	1.51±0.40	3.07±0.30	3.10±0.22	2.51±0.36
0.25 ppm	3.96±0.85	3.20±0.58	3.50±0.45	2.78±0.58
0.5 ppm	4.89±0.71	2.80±0.63	2.90±0.69	2.48±0.34

Table – 2: Protein content (mg/g) of *Glossogobius giuris* exposed to sublethal concentrations of malathion.

	24hr	48hr	72hr	96hr
Control	20.10±0.85	22.00±1.58	19.10±0.13	20.03±2.52
0.05 ppm	14.32±0.98	20.36±0.77	17.62±0.90	14.32±1.48
0.25 ppm	15.46±1.45	24.90±1.23	20.40±1.51	19.71±1.56
0.5 ppm	14.11±1.41	25.76±2.35	20.96±1.40	13.67±1.27

Table – 3: Cholesterol content (mg/g) of *Glossogobius giuris* exposed to sublethal concentrations of malathion.

	24hr	48hr	72hr	96hr
Control	0.75±0.75	0.69±0.14	0.73±0.16	0.81±0.18
0.05 ppm	1.01±0.15	0.97±0.14	0.89±0.20	0.70±0.15
0.25 ppm	0.80±0.22	0.52±0.16	0.70±0.19	0.69±0.19
0.5 ppm	0.60±0.25	0.47±0.08	0.67±0.13	0.63±0.18

**Fig. 1:** Glycogen content (% changes) of *Glossogobius giuris* exposed to sublethal concentrations of malathion.

to 96 hr. As pointed out by Mc Leay and Brown (1979); Veeraiah and Prasad (1998) the decline in muscle glycogen could have resulted because of anaerobic stress. In the present study the depletion of glycogen content in heart might be due to a possible glycogenolysis, resulting in anaerobic glycolysis to cope up with the adverse condition, as reported by Dezwaan and Zandee (1999) and Chaudhari (2000).

The muscle protein showed decreasing trends of 28.75 %, 23.08 % and 29.80% with 0.05, 0.25 and 0.5 ppm of malathion treatment respectively during 24 hr compared to control. After 48 and 72 hr of treatment with 0.25 and 0.5 ppm of malathion the heart muscle protein was slightly elevated

(Fig. 2). However in 96 hr treated fish with 0.05 and 0.5 ppm of malathion, decreasing tendency was seen, reaching a minimum percentage of 29.45 and 32.66 as compared to control (Table 2).

Proteins can be expected to be involved in the compensatory mechanism of stressed organisms (Ramalingam and Ramalingam, 1982). In the present study, when the fish were exposed to malathion (0.5 ppm) for 24 and 96 hr the protein content in the heart were found to have decreased. Krishnamohan *et al.* (1985) and Chandravathy and Reddy (1994) have suggested that decline in the muscle protein content may be due to reduced protein synthesis, increased

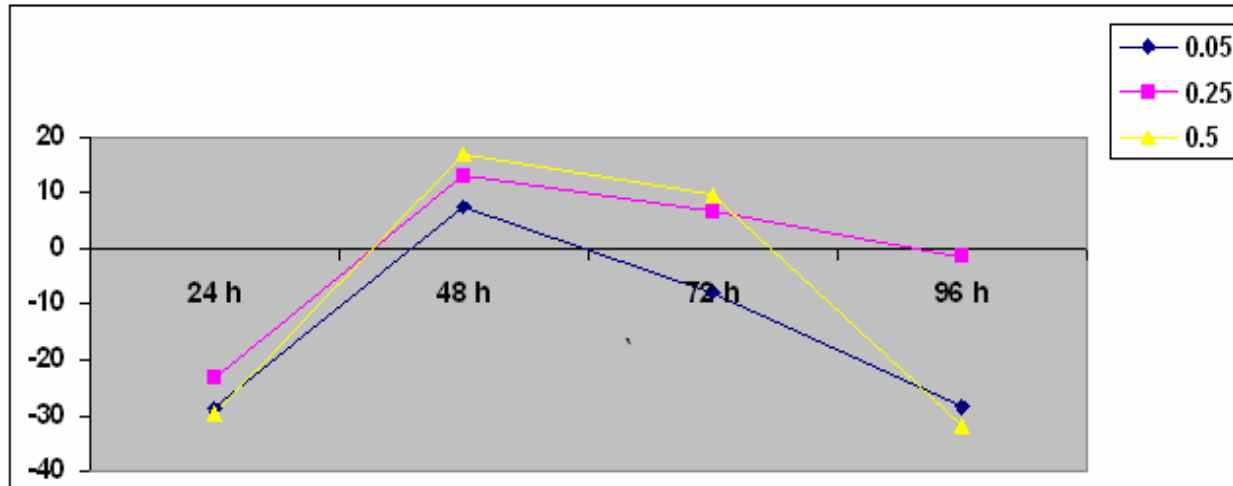


Fig. 2: Protein content (% changes) of *Glossogobius giuris* exposed to sublethal concentrations of malathion.

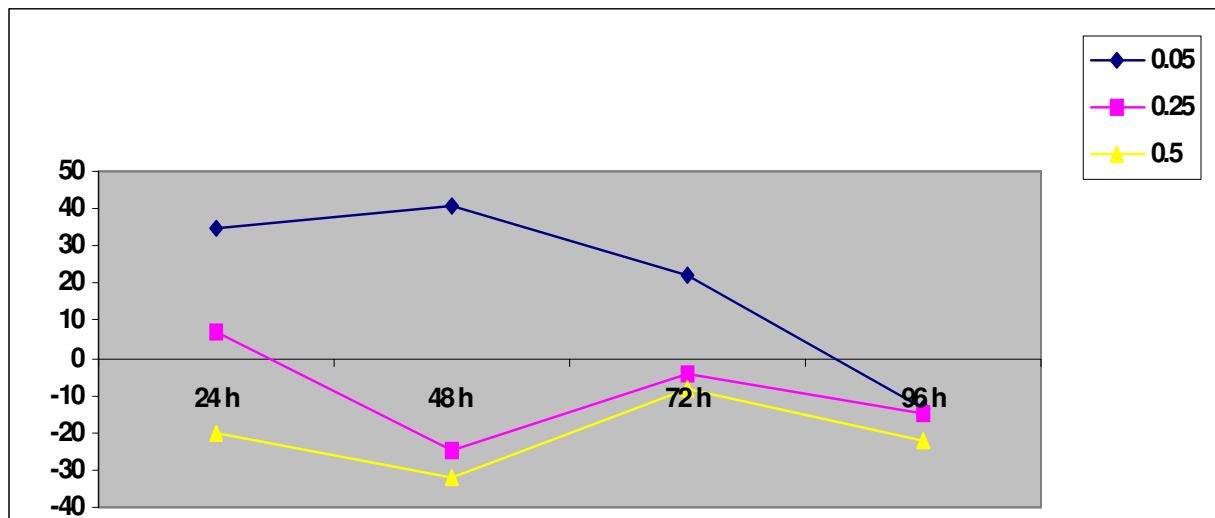


Fig. 3: Cholesterol content (% changes) of *Glossogobius giuris* exposed to sublethal concentrations of malathion.

proteolysis and also due to utilization for metabolic processes under lead toxicity. In the present investigation the protein level gradually decreased in heart of treated fish with 0.5 ppm for 24 and 96 hr. It is presumed that reduction in protein content could be due to its utilization to mitigate the energy demand when the fish are under stress, as reported by Rao *et al.* (1987) and Baskaran *et al.* (1989).

Fig. 3 shows the changing pattern of muscle cholesterol of three concentrations, 0.05 ppm treated fish showed enhancement of muscle cholesterol, which reached a maximum at 24h. However, after 48 hr the quantity increased by a percentage of 40.57 compared to control. A similar fluctuating pattern was observed for 72 hr also (Fig.3). When the fish treated under high concentration of malathion (0.5 ppm) the values of muscle cholesterol started decreasing for 24, 48 and 72 hr. However, by 96 hr it showed a decreasing trend and reaching the minimum percentage of 13.58, 14.81

and 22.22 in 0.05, 0.25 and 0.5 ppm respectively compared to control. This indicates a decreasing trend following malathion treatment. Brycesmith and Waddson (1974) emphasized that reduced ability to metabolise pyruvate would also result in less acetyl co-enzyme 'a', being available from the synthesis of fatty acids and cholesterol than a loss of lipids, is due to inhibited pyruvate metabolism. Fish exposed to sub lethal concentration (0.5 ppm) of malathion for 24 to 96 hr showed reduction in the cholesterol level in the heart (Table 3). Similar result of cholesterol content has been reported in different tissues with pesticide treated fishes (Ghosh and Chatterjee, 1989; Piska *et al.*, 1992).

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