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Methylparathion Induced Alterations in the Tissue Carbohydrate Catabolism of Marine Prawn, *Metapenaeus monoceros*

M. Srinivasulu Reddy and K. V. Ramana Rao

Division of Toxicology, Department of Marine Zoology, Sri Venkateswara University Post-Graduate Centre, Kavali 524 202, India

The organophosphorous insecticides have superseded the organochlorines owing to their rapid biodegradability and shorter persistence in the environment. But as a consequence of their indiscriminate and widespread use in agriculture and public health, these insecticides ultimately reach the aquatic environment and affect the life therein. The organophosphorous insecticides are believed to be nerve poisons and block synaptic transmission in the cholinergic part of the nervous system (O'Brien 1967; Metcalf 1971; Reddy & Rao 1988a), which lead to the excessive accumulation of acetylcholine at nerve cell junctions. But studies on the lethality of these chemicals did not find a relationship between acetylcholinesterase inhibition and death of the organism (Schoor & Brausch 1980). Thus it is believed that some other systems are also involved in the toxicity of pesticides. Thus, keeping in mind the toxicity of methylparathion to Metapenaeus monoceros and various reports on other pesticide - induced alterations in the carbohydrate catabolism, it was thought desirable to investigate the possible effect of methylparathion on the carbohydrate catabolism of M. monoceros.

MATERIALS AND METHODS

M. monoceros were collected from the Buckingham Canal near Kavali seacoast, Andhra Pradesh, India. Only intermolt prawns (75 ± 5 mm in length and 2.5 ± 0.5 g weight) were selected and acclimatized to laboratory conditions for a week at constant salinity of 15 ± 1 ppt, pH 7.1 ± 0.2 and temperature of 23 ± 2°C. They were fed ad libitum diet of oil cake powder, which was stopped before 24 hr of experiments. The media

Send reprint requests to Dr.M.S.Reddy at the above address.

in which prawns were placed was changed periodically at regular intervals and continuous aeration was provided.

Technical grade methylparathion (80% w/v; 0-0-dimethyl, 0-4 nitrophenyl thiophosphate) was used as test chemical. 100 specimens of three batches were exposed to 10, 20 and 30 µg/L of methylparathion in plastic containers of fifteen liters capacity. Suitable controls were maintained without pesticide. The whole set was aerated continuously. The treatments were continued upto 5 days and the desired tissues were taken out from control and experimental animals after 2, 3, 4 and 5 days of exposure, and were used for biochemical analysis. The glycogen content was estimated by the method of Carrol et al (1956) and the blood glucose level was determined by the method of Nelson-Somogyi as given in Oser (1979). Each experiment was replicated six times and the data were subjected to statistical analysis as per Bailey (1965).

RESULTS AND DISCUSSION

Prawns are active animals and are very sensitive to any change in their environment. During exposure to different concentrations of methylparathion, the prawns became restless and showed fast erratic swimming (hyperexcitability) and which was followed by tremor in the appendages, restriction of appendage movement, loss of coordination and subsequently equilibrium, violent action of chelate legs (pressing one another through their chelate legs) and ultimately death.

The glycogen content in the hepatopancreas and muscle of M. monoceros depleted significantly after exposure to different concentrations of methylparathion. Methylparathion exposure of M. monoceros induced hyperglycemia at times upto 4days of exposure at all the concentrations, after which hypoglycemia was recorded (Tables 1 & 2).

Methylparathion is principally an anticholinesterase. These studies show that methylparathion specifically decreased the glycogen reserves of the hepatopancreas and the muscle tissue of M. monoceros and induced hyperglycemia during exposures upto 4 days. After 5 days of continuous exposure, hypoglycemia was observed. It is obvious from the results obtained in the present investigation that the decrease in glycogen reserves of the hepatopancreas was more pronounced than that of the muscle, which clearly indicates that the hepatic glycogen was more rapidly utilized than that of

Table 1. Changes in the glycogen content of hepatopancreas and muscle of control and methylparathion exposed Metapenaeus monoceros (Each value is mean ± SD of 6 individual observations)

Methyl parathion concentration (µg/L)	Exposure period (in days)			
	2	3	4	5
	Hepatic glycogen (mg/g wet wt of tissue)			
00	11.24 ±1.25	11.49 ±1.32	11.45 ±1.34	11.51 ±1.15
10	7.08 ±0.89 (-37)	6.82 ±0.83 (-41)	6.54 ±0.78 (-43)	6.02 ±0.59 (-48)
20	6.81 ±0.84 (-39)	6.01 ±0.73 (-48)	5.24 ±0.68 (-54)	4.73 ±0.62 (-59)
30	6.04 ±0.72 (-46)	5.74 ±0.62 (-50)	4.89 ±0.51 (-57)	4.42 ±0.43 (-62)
	Muscle glycogen (mg/g wet wt of tissue)			
00	2.51 ±0.24	2.64 ±0.20	2.49 ±0.18	2.58 ±0.22
10	1.84 ±0.18 (-27)	1.73 ±0.17 (-35)	1.58 ±0.13 (-37)	1.40 ±0.13 (-46)
20	1.62 ±0.15 (-35)	1.53 ±0.15 (-42)	1.39 ±0.12 (-44)	1.27 ±0.12 (-50)
30	1.52 ±0.12 (-39)	1.38 ±0.11 (-48)	1.26 ±0.10 (-49)	1.12 ±0.09 (-57)

All values are statistically significant at $P < 0.001$.

Table 2. Changes in hemolymph glucose of control and methylparathion exposed Metapenaeus monoceros (Each value is mean ± SD of 6 observations)

Methyl-parathion concentration (µg/L)	Exposure period (in days) 2	3	4	5
00	50.44 ±3.37	52.43 ±2.82	51.18 ±2.88	48.94 ±2.58
10	71.82 ±3.84 (+42)	79.48 ±4.04 (+52)	85.33 ±4.12 (+67)	22.89 ±0.28 (-53)
20	84.18 ±3.93 (+67)	96.74 ±4.12 (+85)	99.38 ±4.19 (+94)	20.18 ±0.26 (-59)
30	95.43 ±4.08 (+89)	107.83 ±5.14 (+106)	113.36 ±5.29 (+121)	17.36 ±0.21 (-65)

Values are expressed in mgs of glucose/100 ml of hemolymph. All values are statistically significant at $P < 0.001$.

muscle glycogen. The hepatopancreas of crustaceans is analogous to the liver of vertebrates and is involved in the synthesis and degradation of several molecules involved in metabolism (Chang & O'Connor 1983). So, methylparathion first initiates the process of glycogenolysis at the hepatopancreas and later at the muscle tissue. Similar depletions of the glycogen reserves of hepatopancreas and muscle tissues were found in prawns, Penaeus indicus (Reddy & Rao 1988b), Metapenaeus monoceros (Reddy 1986; Reddy & Rao 1988c), Macrobrachium lamerrei (Omkar & Shukla 1984) and crab, Oziotelphusa senex senex (Reddy et al. 1982; 1983) after exposure to different organophosphorous insecticides. The decrease in the glycogen content observed in the present study might be due to the prevalence of hypoxic or anoxic conditions, which normally increase carbohydrate and glycogen utilization (Dezwaan & Zande 1972). A decrease in whole animal respiration, tissue respiratory potentials

and a condition similar to hypoxia was already noticed in M. monoceros and P. indicus after exposure to phosphamidon (Reddy 1986; Reddy et al. 1986). The decrease in the glycogen reserves with an increase in hemolymph glucose level may be ascribed to the decreased glycogen synthesis resulting from the decreased activity levels of glycogen synthetase or the increased glycogenolysis perhaps resulting from the enhanced activity of glycogen phosphorylase, due to methylparathion induced stress condition. The increased glycogen phosphorylase activity levels in the tissues of M. monoceros during pesticide exposure reveals that the glycogen utilization is very high in the glycolytic pathway than in the control tissues (Reddy 1986). Reddy and Rao (1988b;c) observed a decline in the glycogen content in the midgut gland, muscle and gill of prawns, M. monoceros and P. indicus exposed to phosphamidon and suggested that it may be due to either a reduction in glycogenesis or increased glycogen utilization through the glycolytic pathway. This has been further confirmed by an increase in the aldolase activity levels and subsequently tissue glycolytic potentials (Reddy & Rao 1988c). Nagabhushanam and Kulkarni (1981) observed an inverse relation between midgut gland glycogen and hemolymph glucose level of a freshwater prawn, Macrobrachium kistnensis after exposure to heavy metal pollutants such as copper sulphate and zinc sulphate and suggested their interconversion according to energy requirements. The decrease in glycogen synthesis as a consequence of the decreased activity of glycogen synthetase may be justified on the basis of the fact that glycogen synthetase is a serine containing enzyme and methylparathion is one of the organophosphorous insecticides, which is reported to be a potent inhibitor of serine containing enzymes (Bell et al. 1970). In crustacean tissues, glycogen phosphorylase is responsible for the degradation of glycogen and also shown to alter the carbohydrate levels of hepatopancreas and muscle and hemolymph of crabs (Ramamurthi & Venkataramanaiah 1982) and lobster (Cowgill 1956). The activity of glycogen phosphorylase is regulated by the neuroendocrine principle of the eyestalk (sinus gland complex) in the crustaceans (Ramamurthi et al. 1968). Hence it is possible that methylparathion exposure of M. monoceros initiates the synthesis of glycogen phosphorylase at the tissues which induces glycogenolysis. Methylparathion exposure causes accumulation of acetylcholine at the synaptic junctions by inhibiting AChE activity in M. monoceros, which in turn stimulates the secretion of the sinus gland of the eyestalk and

thus activates phosphorylase activity. Likewise, Brzezinski and Ludwicki (1973) reported that organophosphorous insecticides causes accumulation of acetylcholine at synaptic junctions with a simultaneous increase in the secretion of catecholamines in the mammals. The hyperglycemia after methylparathion exposure may be attributed to the mobilization of hepatic and muscle tissue glycogen reserves. The hyperglycemia may be a physiological response to meet the critical need of the brain tissue for increased energy in the form of glucose during methylparathion exposure. Hyperglycemic condition may be generally due to nonspecific response to pesticide induced stress (Selbergeld 1974). Hypoglycemic condition after 5th day of methylparathion exposure, with a concomitant decrease in tissue glycogen reserves may be ascribed to the rapid utilization of glycogen and its allied carbohydrates for energy requirement of M. monoceros during methylparathion augmented stress condition.

Methylparathion exposure of M. monoceros causes widespread physiological disturbances like inhibition of AChE (Reddy & Rao 1988a) and decreases the total protein content and increase in tissue free amino acid pool (Reddy & Rao 1986), which ultimately lead derangement in tissue metabolic pathways. The physiological, ecological and biochemical significance of methylparathion induced alterations in carbohydrate catabolic profiles of marine prawn, may be taken as indices for determining the environmental pollution by insecticides. Such major alterations, including hormonal imbalance by affecting the secretion of the sinus gland X-organ complex, should have serious consequences for the general body metabolic profiles and energy conservation of the prawn, M. monoceros.

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