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STUDIES ON BLOOD BIOCHEMICAL COMPOSITION OF POLLUTANT EXPOSED CRAB, *PARATELPHUSA JACQUEMONTII* (RATHBUN)

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As few pesticides like organophosphates are considered to be inhibitors of AchE, it could be concluded that the reduction in carbohydrate metabolism in treated crabs is due to general lowering of the metabolic activities There was increased protein breakdown to yield more energy to face the toxic impact. This was further corroborated through the increased levels of free amino acids. These free amino acids might be fed into TcA cycle as keto acids by way of transamination since transaminases are known to elevate during pesticide intoxication. The increased levels of amino acids might also be due to increased synthetic potentiality.

INTRODUCTION

Environmental pollutants bring about damage to different organs or disturb the physiological and biochemical processes with in the organism following the exposure to them. Sheshadri & Khanna (1982) have reported changes in proteins and glutathione concentration due to lead toxicity in rats.

O'Braines (1967) has accounted in detail the mode of action and metabolism of carbamate pesticides. Bhagyalakshmi (1961) has reported the drastic effects of sumithion on metabolism of freshwater crab, *Oziotelphusa senex senex*. Rajender (1982) has observed the effect of monocrotophos, sumicidin, benidiocrab and quinalphes on normal metabolism of the nervous tissues and haemolymph of the cockroach *P. americana*. Thus it seems that much work has been done in fishes and other invertebrates, but no information is available on the toxic effects of the locally used pesticides and fertilizers on the freshwater crab *P. jacquemontii*.

In present investigation attempts have been made to study changes in some important biochemical components of freshwater crab, *P. jacquemontii* exposed to sublethal concentrations of different toxicants, as HCH, Thimet, Ammonium sulphate and Mercuric chloride for 24, 48, 96, 168, 360 and 720 hrs time exposures.

MATERIAL AND METHODS

The crabs were collected and maintained in laboratory. Intermoult crabs of same size and weight were divided into different groups each having equal number of males and females. The crabs were acclimated for about a week, were fed with dried prawns alternate day. The water of container was replaced every day.

The crabs were divided into following groups :

Group I : Initial control (IC) sacrificed at the start of the experiment.

Group II : Simultaneous control (SC, exposed to only solvent sacrificed at the end of the experiment.

Group III : Crabs exposed to four pollutants (HCH, Thimet, Ammonium sulphate and Mercuric chloride) lethal concentrations, Lc_{50}/96 hr at 24 hr (HCH 54.11; Thimet 7 5, Ammonium sulphate 2080, Mercuric chloride 0.67 mg/lit).

Group IV V, VI & VII : Crabs exposed for 48, 96, 168, 360 and 720 hrs respectively.

All groups had 50 crabs each, of which 25 were males and 25 females.

Crab were exposed to 96 hrs Lc_{50} concentration and were sacrificed at 24, 48, 96, 168, 360 and 720 hrs. Haemolymph was drawn from the coxa of the walking leg.

Glucose was estimated by using the method of Semogyi as given Hawk *et al.* (1954). Free amino acids were estimated by Danielson (1958) method.

RESULTS AND DISCUSSION

It was very clear from Table I that there was decrease in the blood glucose level after 48 hrs. The per cent decrease in Mercuric chloride was faster

Table I. Changes in blood of *P. jacquemontii* exposed to four pollutants (Mean ± SD)

Time interval (hours)	Control	HCH	Thimet	Ammonium sulphate	Mercuric chloride
24	15 26	58.76	50 20	54 69	62 38
	± 2.16	± 2.08	± 2.58	± 2.46	± 2 65
		(+ 29 82)	(+10 91)	(+20 83)	(37 82)
48	45 38	64 84	55.34	60.76	68.92
	± 2.60	± 2 58	± 2.15	± 2.62	± 2 65
		(+41.60)	(+24.15)	(+33.89)	(+53.04)
96	48.16	65.50	56.42	59.95	69.67
	± 2.18	± 2.18	± 2.32	± 2.70	± 2.90
		(+36.18)	(+17.15)	(+24.48)	(+29.38)
168	45.82	56.78	49.72	51.11	59.28
	± 2 19	± 2.76	± 2.14	± 2.67	± 2.90
		(+23.91)	(+ 8.51)	(+11.55)	(+29.30)
360	45.28	41.44	42.88	42.02	35.55
	± 2 01	± 2.88	± 2 00	± 2.08	± 2.44
		(— 8 48)	(— 5.30)	(— 7.20)	(—21.46)
720	45.26	31.88	40.90	39.72	28.74
	± 2.48	± 2.18	± 2.20	± 2 44	± 2.28
		(—29.56)	(— 9 63)	(—12.24)	(—36.50)

Paranthesis figures are percent change

than HCH, Ammonium sulphate and Thimet.

The free amino acid (FAA) level in haemolymph increased after HCH.

Table II. Changes in blood amino acid of *P. jacquemontii* exposed to four pollutant (Mean ± SD).

Time interval (hours)	Control	HCH	Thimet	Ammonium sulphate	Mercuric chloride
24	4 88 ± 0.24	5.21 ± 0.27 (+ 7.76)	5.01 ± 0 27 (+ 7.76)	5.15 ± 0.51 (+ 5 32)	5.43 ± 5.43 (+ 7 17)
48	4.45 ± 0.15	5.32 ± 0.42 (+19.32)	5.07 ± 0.25 (+13.48)	5.21 ± 0.28 (+17.07)	5.52 ± 0 41 (+23 59)
96	4.45 ± 0.15	5.45 ± 0.50 (+22.47)	5.33 ± 0.42 (+19.77)	5.29 ± 0.32 (+18.87)	5 65 ± 0.[illegible]2 (+26.96)
168	4.41 ± 0.30	5.64 ± 0.32 (+27.89)	5.43 ± 0.33 (+23.12)	5.44 ± 0.40 (+2[illegible].58)	5.72 ± 0.25 (+29 70)
360	4.34 ± 0.10	5.64 ± 0.42 (+29.95)	5.63 ± 0.22 (+29.72)	5.55 ± 0 50 (+27 88)	5.74 ± 0.25 (+31 79)
720	4 08 ± 0.26	5.72 ± 0.25 (+40.19)	5.65 ± 0.44 (+[illegible]8.48)	5 68 ± 0 43 (+39.21)	6.11 ± 0 17 (+49 75)

Paranthesis figure is percent change

Thimet, Ammonium sulphate and Mercuric chloride exposure period (Tabie II) The increase was directly proportional to the exposure period. The increase was higher in Mercuric chloride and HCH than Ammonium sulphate and Thimet.

The hyperglycemia followed by a decrease in hepatic glycogen suggests the increased glycogenolysis possibly by the increased activity of glycogen phosphorylase. It is also possible that pollutants in some manner stimulate the secretion of sinus gland. However, the hyperglycemia may be a physiological response to meet the critical need of brain for the increase energy in the form of glucose (Shukla & Upadhayaya, 1983). The increase in blood glucose could furnish the high demand of glucose in the brain which could in turn compensate of some extent for any potential decrease in brain glucose.

A similar decrease in hepatic glycogen (Koundinya & Ramamurthi, 1979; Singh & Srivastava, 1981) and increase in blood glucose (Weise *et al*., 1964; Shaffi, 1980; Singh & Srivastava, 1981) induced by endosulphan and for other presticides HCH (Bhagyalakshmi *et al*., 1982; Reddy *et al*, 1983).

The increase in total free amino acid content may suggest the existence of heavy drain on the metabolites during exposure The toxic impact caused by pollutants may be the reason for elevation of free amino acid content. Such observations were reported by Kabeer *et al*. (1978) in case of a pelecypod *Lamellidens marginalis* exposed to sublethal Malathion concentrations Protein content decreased in hepatopancreas, muscle and gill of fresh water crab *P. jacquemontii* reflects enhanced proteolytic activity and broken down the tissue proteins to justify increased energy demands as reflected in increased level of total free amino acids, which may be fed into TcA cycle as keto acids for energy production (Kabeer *et ai*., 1978). This possibility is further investigated by Schafer (1967). Shakoori *et al*. (1976), Dikshith *et al*. (1978), Bhagyalakshmi (1981), have also reported decreased in protein and increase in total free amino acids after napthalene exposure of oyster, *Ostred edulis*. In addition, studies of McKim *et al* (19'0). Lane & Scura (1970), Sakaguchi & Hamaguchi (1975) have also revealed marked variations in the activity of enzymes involved in transamination in fishes, at similar situations.

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