INHIBITION AND RECOVERY OF ACETYLCHOLINESTERASE ACTIVITY IN THE NERVOUS TISSUE OF PRAWN, *METAPENAEUS MONOCEROS* (FABRICIUS) EXPOSED TO PHOSPHAMIDON AND METHYLPARATHION

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ABSTRACT

The penaeid prawn, *Metapenaeus monoceros* showed a dose-dependent inhibition of nervous tissue acetylcholinesterase (AChE) during 48 hr exposure to 1.2 and 0.4 ppm of phosphamidon and 0.12 and 0.04 ppm of methylparathion. A progressive recovery of AChE activity from the phosphamidon and methylparathion induced inhibition was observed in this tissue within 7 days after transferring the prawns to toxicant-free water.

INTRODUCTION

BECAUSE of the widespread and indiscriminate use of insecticides in agricultural and public health operations, several non-target species, including some important members of the food chain are adversely affected[1,2]. It is known that most organophosphorous (OP) insecticides are both effective and degradable, presumed to leave no residues[3]. Like other OP insecticides the mode of action of phosphamidon and methylparathion is similar, that is acetylcholinesterase (AChE) inhibition appears to be the primary target. Usually AChE inhibition is regarded as a significant parameter to assess the toxic effects of OP compounds[4]. Our knowledge on the effects of pesticides on marine or brackish water inhabiting invertebrate systems is rather limited. The present paper is on the *in vivo* inhibitory effect of phosphamidon and methylparathion on AChE activity in the nervous tissue of the penaeid prawn, *Metapenaeus monoceros* and its recovery, when transferred to pesticide-free water.

MATERIALS AND METHODS

Penaeid prawns, *M. monoceros* (Fabricius) were collected from the Buckingham Canal, Thummalapenta seacoast, near Kavali. Only intermolt, uninjured prawns of 75 ± 5 mm length and weighing 2.5 ± 0.5 g were selected and acclimatized to laboratory conditions for a week as described earlier[5]. They were fed *ad lib* with oil cake powder. The media having prawns were changed periodically at regular intervals and continuous aeration was provided.

Technical grade phosphamidon (92% w/v) (0, 0-dimethyl-0–1-methyl-2-chloro-2-diethyl-carbomoyl-vinyl phosphate) and methylparathion (80 w/w) (0–0-dimethyl, 0–4 nitrophenyl thiophosphate) were used as test chemicals. A stock solution of 1000 ppm and appropriate working concentrations were prepared[6]. Toxicity studies showed the LC_{50} values to be 1.2 ppm for phosphamidon, 0.12 ppm for methylparathion to the intermolt prawn *M. monoceros* for 48 hr exposure in the static medium[7]. The laboratory acclimatized prawns were exposed to both lethal and sub-lethal concentrations of the two pesticides for 48 hr. During exposure, the control and experimental prawns were not fed and media were not changed. But continuous aeration was provided. After 48 hr exposure in the respective pesticide media the prawns were transferred to pesticide-free media, to study the rate recovery at different time intervals. During reclamation, the pesticide-exposed and control prawns were fed, the media were frequently changed, and continuous aeration maintained. The AChE activity in the nervous tissue was assayed in the control, pesticide-exposed and reclamated prawns according to Metcalf[8] after due standardization[6]. The protein content was then determined[9] and the data subjected to statistical analysis[10].

RESULTS AND DISCUSSION

After due standardization[6], the AChE activity was assayed in the control and experimental prawn nervous tissue. The AChE activity of nervous tissue

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was significantly inhibited in *M. monoceros* following *in vivo* exposure to both phosphamidon and methylparathion at 48 hr under lethal and sub-lethal concentrations (tables 1 and 2). The degree of inhibition is quite considerable and dependent on the concentration of the pesticide. Higher inhibition could be observed at lethal concentrations compared to sub-lethal concentrations. Maximal inhibition was observed in prawns exposed to methylparathion (−63.6%; $P > 0.001$) than to phosphamidon (−53.6%; $P > 0.001$). Most phosphorothionate insecticides are considered latent inhibitors, wherein they are converted to active AChE inhibitors by the microsomal oxidative (desulphurating) systems in the presence of NADH or NADPH[11]. There is a strong evidence to show that methylparathion is metabolically altered to a more active AChE inhibitor by the oxidation of the thiosulphur atom (P = S) to an oxygen atom (P = O). The resulting oxygen analogue (methylparaxon) is several times more potent in inhibiting AChE[12]. The higher rate of inhibition under methylparathion exposure during 48 hr could be due to the active conversion of mehtylparathion to methylparaxon, and the consequent inhibition of AChE is greater when compared with phosphamidon or its converted product. The degree of inhibition is higher at lethal (LC_{50}/48 hr) than sub-lethal (1/3 LC_{50}/48 hr) concentrations. Our results agree with those of Coppage and Matthews[4], who also reported about 72% inhibition of AChE in the ventral nerve-cord of shrimp, *Penaeus monodon* exposed to lethal concentration (LC_{50}/48 hr) of malathion.

Transferring the pesticide-exposed prawns to pesticide-free medium registered a progressive recovery in AChE activity of the nervous tissue. Near control values were obtained on 7 days of reclamation in pesticide-free water of prawns exposed to sub-lethal concentrations of phosphamidon and methylparathion (tables 1 and 2). But when prawns are exposed to lethal concentration, the AChE activity failed to reach control levels indicating inhibitory action of the pesticides to still persist. This trend is greater with methylparathion than with phosphamidon which demonstrates that phosphamidon and methylparathion might either be getting itself retained in the nervous tissue, should have caused some serious damage to the nervous tissue *per se* due to high concentration or that the reclamation period might be short for lethally-exposed prawns. Spontaneous recovery of organophosphate inhibited esterases was reported in vertebrates like fish[13] and invertebrates like housefly, *Musca domestica*[14]. It is interesting to note eel cholinesterase inhibited

Table 1 *Acetylcholinesterase activity in the nervous tissue of prawn M. monoceros exposed to lethal and sub-lethal concentrations of phosphamidon and during reclamation*

Phosphamidon concentration (ppm)	Enzyme activity (μmol of acetylcholine hydrolysed/mg protien/hr)				
	Control	48 hr after exposure	Reclamation period (days)		
			2	4	7
Lethal					
1.2	7.35 ± 0.48	3.41 ± 0.25	4.45 ± 0.32	5.72 ± 0.29	6.93 ± 0.39
	PDC	−53.61	−39.46	−22.18	−5.72
		$P < 0.001$	$P < 0.001$	$P < 0.001$	$P < 0.1$
	PDE		+30.50	+67.74	+103.23
			$P < 0.001$	$P < 0.001$	$P < 0.001$
Sub-lethal					
0.4	7.42 ± 0.51	5.34 ± 0.31	6.03 ± 0.28	6.72 ± 0.29	7.24 ± 0.44
	PDC	−28.03	−18.73	−9.43	−2.43
		$P < 0.001$	$P < 0.001$	$P < 0.01$	NS
	PDE		+12.92	+25.84	+35.58
			$P < 0.001$	$P < 0.001$	$P < 0.001$

Values are mean ± SD of 6 individual observations; PDC = Per cent deviation over control; PDI = Per cent deviation of experimental (48 hr).

Table 2 *Acetylcholinesterase activity in the nervous tissue of prawn M monoceros exposed to lethal and sub-lethal concentrations of methylparathion and during reclamation*

Methylparathion concentration (ppm)	Enzyme activity (μmol of acetylcholine hydrolysed/mg protien/hr)				
	Control	48 hr after exposure	Reclamation period (days)		
			2	4	7
Lethal					
0 12	7.72 ± 0.45	2.81 ± 0 22	4.45 ± 0.38	5 22 ± 0.39	6.60 ± 0 43
	PDC	−63 6	−42.36	−32.38	−14 51
		$P < 0.001$	$P < 0 001$	$P < 0 001$	
		PDE	+58.36	+85.77	+134 88
			$P < 0 001$	$P < 0 001$	$P < 0 001$
Sub-lethal					
0.4	7.54 ± 0.50	4 91 ± 0 29	5 68 ± 0.34	6.05 ± 0.39	6.98 ± 0 45
	PDC	−34.88	−24.67	−19 76	−7 43
		$P < 0 001$	$P < 0.001$	$P < 0.001$	$P < 0.05$
		PDE	+15.68	+23.22	+42 16
			$P < 0.001$	$P < 0 001$	$P < 0 001$

Values are mean ± SD of 6 individual observations; PDC = Per cent deviation over control; PDE = Per cent deviation of experimental (48 hr).

by TEPP showed 45% recovery in 28 days[15]. Coppage *et al*[13] reported almost absolute recovery of brain AChE activity of fish poisoned by malathion in 40 days. Complete (97%) recovery of AChE activity was noticed in *Musca* exposed to malathion[14]. This is attributed to dephosphorylation of the OP compound and resynthesis of the fresh enzyme. A similar situation might also be operating in sub-lethal exposed *M. monoceros* when subjected to reclamation in pesticide-free water. However, lethally-exposed prawns might be requiring still longer period of reclamation in pesticide-free water. In addition, hydrolysis, biodegradation and/or rapid excretion of toxic chemical on transfer of pesticide-exposed prawns to clean water may facilitate quick recovery.

From the present investigation it may be concluded that, interrupted application of pesticides, to fields or waters may be followed as a protection measure to salvage non-target animals from falling a victim to the deleterious effects of pesticides.

ACKNOWLEDGEMENTS

MSR thanks CSIR, New Delhi and KVRR thanks UGC, New Delhi for financial assistance. Thanks are also due to Prof. K. Sasira Basu for encouragement.

6 April 1987

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NEWS

SCIENCE STUDIES: SOME SOCIAL IMPLICATIONS

...What is to be done "if we are going to be counted among those who have sought to alleviate and eradicate the social ills of science and humaity? The most important thing we need to do at the level of perspective and theory is to recognize that the sociological dimension in science studies is not merely a matter of disciplinary politics but part of a Copernican revolution in the social sciences. This revolution, rooted in the works of such social theorists as Karl Marx, Max Weber, Emile Durkheim, and Pyotr Kropotkin, has shifted the individual from the center of the social universe and replaced it with the collectivity. The more profound implications of this shift have yet to make their mark on science studies. As these implications are realized, we will witness the rapid development of still-primitive sociological conceptions of self, mind, and cognition.... Constructivism in science studies has been throttled by scientific orthodoxies. In order to get rid of old baggage, we need to recognize that modern science is an institutionalized cognitive strategy, complete with worldview and a theory of social relations. It developed in, and has changed with, the social changes in modern industrialized society. Let us not mistake the social institution called modern science for the core epistemic strategy it shares with all more or less successful cognitive strategies, the basic rationalities and methods of all human inquiry." [Sal Restivo (Rensselaer Polytechnic Inst., Troy, NY) in *Science, Technology, & Human Values* 12(2): 13–18, Spring 1987. Reproduced with permission from Press Digest, *Current Contents*®, No. 25, June 22, 1987, p. 9. (Published by the Institute for Scientific Information®, Philadelphia, PA, USA.)]

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