LETTER TO THE EDITOR

EFFECTS OF TECHNICAL AND COMMERCIAL GRADE MALATHION ON NITROGEN METABOLISM OF THE TELEOST, *TILAPIA MOSSAMBICA* (PETERS)

Sir,

(Received on January 29, 1983)

The biological activity of the organophosphorus insecticides is attributed to the inhibition of the cholinesterases (9). Besides inhibiting the target enzyme, the organophosphorus esters can also influence other physiological events, thus resulting in various hazards to the biota like causing a new disease "Handigodu Syndrome" (10) and affecting growth and reproduction in fish (5). Literature pertaining to the comparative effects of technical and commercial grade malathion on nitrogen metabolism in the fish is not readily available. Hence this report intends to give such a comparative evaluatoin.

Fish (*T. mossambica*) weighing 8 ± 2 g were collected from the local reservoirs and acclimatized to laboratory conditions for a week. They were fed daily with groundnut cake and frog leg muscle twice a week. The technical grade (tgM) and commercial grade (cgM) malathion solutions obtained from Cyanamid India Ltd., Bombay, were prepared as 1000 *ppm* stock solutions in acetone. Required dilutions were made with tap water. Acetone in the quantity used was not toxic to fish. The commercial grade malathion contains only 50% of the active ingredient and 50% of unknown oils and solvents as against technical grade malathion which contains 95% active ingredient and only 5% of unknown oils and solvents. LC_{50} values for *T. mossambica* were determined by probit analysis (4) and were found to be 5.59 *mg/L* for tgM and 0.887 *mg/L* for cgM respectively in 48 hr exposure study. 1/3 of the LC_{50} values were taken for estimation of biochemical parameters like glutamate dehydrogenase (GDH) (7), free ammonia (1), urea (8) and glutamine (2) levels.

Findings are summarized in Table I. There is a general decrease in the level of GDH and in the amounts of ammonia and urea of the three tissues in cgM and tgM exposed fish. However, the quantity of glutamine elevated in cgM and tgM incomparison with normal level. Except changes in urea of muscle and gill all changes between normal and cgM and tgM exposed fish were statistically significant. The decrease in GDH activity and in ammonia and urea levels indicate the diversion of nitrogen metabo-

lism towards decreased ammonia production which in turn leads to higher glutamine level. This could be used for various synthetic processes under stressful toxicity condition.

The presence of higher urea level in the hepatic tissue in comparison with other tissues ($P<0.001$), confirms the operation of ornithine cycle in the liver tissue (6). The occurence of urea in muscle and gill might be due to its mobilization from liver, since the presence of urea cycle enzymes in these tissues has not been demonstrated.

In the present study, the potency of toxic stress induced by cgM is found to be more than that of tgM (Table I), suggesting greater effectiveness of cgM over tgM with respect to certain parameters. The observed greater potency of cgM is most

TABLE I : Changes in the levels of glutamate dehydrogenase (GDH), glutamine, ammonia and urea in the fish, *T. mossambica* induced by exposure to malathion preparations for 48 hrs.

The values were expressed in μ*moles* of formazan/*mg* protein/hr for GDH, and as $\mu M/g$ wet wt. tissue for glutamine, ammonia are urea.

Each value is the mean ± S.D. of 6 individual observations. tgM = Malathion technical grade; cgM = Malathion commercial grade; Percent increase or decrease is given in the paranthesis.

Estimations	*Treatment*	*Tissue*		
		Muscle	*Gill*	*Liver*
Glutamate dehydrogenase	Nil (control)	0.116±0.003	0.052±0.005	0.368±0.008
	tgM	0.101±0.003[a] (—13)	0.041±0.002[a] (—21)	0.296±0.072[a] (—19)
	cgM	0.101±0.002[a] (—13)	0.038±0.005[a] (—26)	0.290±0.012[a] (—21)
Glutamine	Nil (control)	97.95±2.26	32.56±2.54	128.08±5.30
	tgM	132.96±8.05[a] (+36)	41.25±4.74[a] (+27)	182.12±11.05[a] (+42)
	cgM	152.06±2.72[a•] (+55)	43.26±3.78[a] (+34)	200.21±5.58[a•] (+56)
Ammonia	Nil (control)	17.27±0.03	15.94±0.31	18.72±0.13
	tgM	16.94±0.10[c] (+2)	14.23±0.04[a] (—11)	15.76±0.06[a] (—16)
	cgM	16.62±0.19[a•] (—4)	13.93±0.06[a•] (—12)	15.12±0.06[a•] (—19)
Urea	Nil (control)	5.01±1.68	5.71±1.64	12.52±1.87
	tgM	4.56±0.13[d] (—9)	5.24±0.10[d] (—8)	9.89±0.19[b] (—21)
	cgM	4.27±1.46[d] (—14)	5.06±0.20[d••] (—11)	9.73±0.66[b] (—22)

Probability of difference from control (t-test) :
(a = $P<0.001$; b = $P<0.005$; c = $P<0.025$; d = NS)
and of difference from tgM group ($^{\bullet}P<0.001$; $^{\bullet\bullet}P<0.05$).

probably due to presence of emulsifier system in cgM, since the emulsifier may lead to a better penetration of malathion. Whether with tgM or cgM, changes in the GDH, ammonia or urea seem to be limited in magnitude. However, there is a sharp increase in the glutamine content of all tissues, particularly in liver which suggests that ammonia is being salvaged and recycled towards glutamine formation to meet the energy crisis under the pesticide intoxication.

ACKNOWLEDGEMENTS

The authors thank CSIR and ICMR, New Delhi for financial assistance, Cyanamid India Ltd., Bombay for providing commercial and technical grade malathion samples and to Prof. K. Sasira Babu for his encouragement.

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