

Impact of Methyl Parathion on the Tissue NH_3 -Changes in the Fish *Tilapia mossambica* (Peters)

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The level of glutamate dehydrogenase declined with a concomitant decrease in the levels of free ammonia and urea while the glutamine content increased in muscle, gill and liver tissues of methyl parathion exposed fishes. These biochemical changes at the tissue level suggest the possibility of ammonia recycling to counteract the methyl parathion toxic stress.

Key Words: Methyl parathion, Glutamate dehydrogenase, *Tilapia mossambica*, Glutamine

Introduction

High production and use of different pesticide formulations (MacDonald & Deichmann 1970), are a source of environmental contamination (Legator et al. 1969, Mark 1969). There is an increasing concern about their toxic hazards due to their indiscriminate use in the environment (Matsumara et al. 1972). Many attempts were made to evaluate these hazards on biota (Palmer et al. 1972) as is evident from the reports on fish kills due to the extensive use of organophosphorus pesticides (Holden 1974). Though there is some literature on the toxic effects of pesticides on the physiological and metabolic changes in fishes, yet no attempt was made to study the nitrogen excretion which is very important for the survival of the fish. The

present paper deals with the sublethal effect of methyl parathion, on the selected tissues in relation to the fate of ammonia and its derivatives. The fish, *Tilapia mossambica* was chosen as an experimental animal because of its availability, edibility and quick adaptability to laboratory conditions.

Material and Methods

The details of maintenance and acclimation of *T. mossambica* are the same as described earlier (Siva Prasad & Ramana Rao 1979). The technical grade methyl parathion of 80% purity obtained from Bharat Pulverising Mills Pvt. Ltd., Bombay was used for the study. The standard was prepared by taking 1 mg per 1 ml as equivalent to 1000 ppm.

LC values were determined by probit analysis (Finney et al. 1965). LC₅₀ was found to be 0.3 ppm for 48 hr. Hence, 0.1 ppm concentration of methyl parathion was selected, since it is sublethal concentration, and the fishes were exposed for 48 hr. The troughs containing methyl parathion-exposed fishes (MPE) were aerated frequently to prevent hypoxic condition of the medium. Controls received similar treatment except the addition of MP.

After exposure, three tissues, viz., muscle (Red), gill and liver were isolated and kept in cold. For Glutamate dehydrogenase (GDH) (EC 1.4.1.3), the tissues were homogenised in 0.25M cold sucrose solution using Yorco tissue homogeniser (Yorco Scientific Industries,

Delhi) and centrifuged at 600 g for 15 min. The supernatant was used for the enzyme assay.

The GDH was estimated by the method of Lee and Lardy (1965) after due standardisation. The free ammonia, urea and glutamine contents were estimated by the methods described by Bergmeyer (1965), Natelson (1971) and Colowick and Kaplan (1967) respectively. The protein content in the tissues was estimated by using Folin-phenol reagent (Lowry et al. 1951). The data was subjected to statistical analysis (Bailey 1965).

Results and Discussion

The data in table 1 show a decrease in the ammonia and urea contents in the three tissues of MPE fishes. Curiously,

Table 1 Changes in Ammonia, Urea, Glutamine contents and Glutamate dehydrogenase activity in the selected tissues of controls and methyl parathion-exposed (MPE) fish *Tilapia mossambica* (Peters)

Nitrogenous product/enzyme	Muscle		Gill		Liver	
	Control	MPE	Control	MPE	Control	MPE
Ammonia (μ moles of ammonia/g wet weight)	17.242 \pm 4.761	10.125 \pm 1.964 -41% $P < 0.05$	2.424 \pm 0.161	1.059 \pm 0.424 -56% $P < 0.005$	4.471 \pm 0.651	2.675 \pm 0.907 -40% $P < 0.05$
Urea (μ moles of urea/g wet weight)	5.518 \pm 0.806	3.977 \pm 0.229 -28% $P < 0.025$	10.288 \pm 0.85	9.258 \pm 0.44 -10% N.S.	17.871 \pm 1.008	15.779 \pm 0.964 -12% N.S.
Glutamine (μ moles of glutamine/g wet weight)	103.082 \pm 3.082	184.132 \pm 4.967 + 79% $P < 0.001$	33.219 \pm 1.712	48.973 \pm 2.054 + 47% $P < 0.001$	147.854 \pm 14.187	162.1 \pm 5.141 + 10% N.S.
Glutamate dehydrogenase (μ moles of formazan formed/mg protein/hr)	0.0257 \pm 0.0013	0.0134 \pm 0.0007 -48% $P < 0.001$	0.0046 \pm 0.0007	0.0035 \pm 0.0004 -24% $P < 0.05$	0.0531 \pm 0.0029	0.0468 \pm 0.0021 -12% $P < 0.05$

Each value is a mean of six individual observations

+ or - indicates per cent increase or decrease over control

\pm indicates standard deviation

P = 't' test; N.S. = indicates not significant

the glutamine content showed a remarkable elevation in the muscle and gill tissues of MPE fishes. The GDH activity decreased in the three tissues, most conspicuously in muscle and gill tissues.

Since GDH is known to perform oxidative deamination (Harper 1977), the decrease in the level of GDH activity corroborates with the decrease in the ammonia content in the tissues of MPE fishes suggestive of less availability of ammonia. As GDH is a regulatory enzyme (Cohn & Stumpf 1978) and since ammonia content decreased in MPE fishes, it is possible that deamination process is brought to the minimum thereby suggesting that the liberated ammonia might be utilised in a different way. The decrease in urea level was found to be not significant. However, the presence of urea is notable, since teleosts are known to synthesise urea (Wakell et al. 1973). Earlier studies on malathion toxicity in the same species showed an increase in the rate of protein synthesis (Kabeer 1979). Hence, the significant increase in glutamine content observed in the present study suggests that the

ammonia formed during toxic stress might be converted to glutamine and stored in the tissue, to be utilised for amino acid and protein synthesis under impending circumstances. The inter conversion of glutamine to glutamate is well documented (Harper 1977).

Thus, it can be reasonably suggested that methyl parathion may interact with the biochemical sequences of nitrogen metabolism. However, the possible occurrence of ammonia recycling and increase in amino acid and protein synthesis suggests that the animal is able to adapt to the toxic stress of the sublethal concentration of methyl parathion by enhancing the synthetic potential.

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