

## LETTERS TO THE EDITOR

**EFFECT OF ORGANOPHOSPHORUS PESTICIDE SUMITHION (FENITROTHION) ON ALKALINE PHOSPHATASE ACTIVITY OF FRESHWATER TELEOST, *SAROTHERODON MOSSAMBICUS* (PETERS).**

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THE organophosphorus (OP) pesticide Sumithion produced different physiological changes in the freshwater teleost *S. mossambicus* (Peters) when exposed to a lethal concentration ( $LC_{50}$ -6 mg/l for 48 hr. It was found that exposure to Sumithion depressed tissue respiration<sup>1</sup> and inhibited citric acid cycle enzyme succinic dehydrogenase (SDH) activity<sup>2</sup>. The glycolytic enzyme lactic dehydrogenase<sup>2</sup> (LDH) was increased in Sumithion exposed fish suggesting operation of anaerobic glycolytic pathway. Similarly, changes in blood glucose, muscle, liver glycogen content, and phosphorylase activity<sup>3</sup> were found. The present study reports alkaline phosphatase activity of the fish *S. mossambicus* exposed to lethal ( $LC_{50}$ ) concentration of Sumithion.

Maintenance, size and weight range of fish used in the experiment have been described earlier<sup>2</sup>. The fish were exposed to a concentration sufficient to kill 50% of the test population ( $LC_{50}$ ) within 48 hr of exposure.  $LC_{50}$  value at 48 hr exposure was worked out<sup>4</sup>. The enzyme analysis was carried out in the fish which were found tolerant to  $LC_{50}/48$  hr concentration. Alkaline phosphatase activity was estimated<sup>5</sup> and the inorganic phosphate liberated was determined<sup>6</sup>. Commercial grade Sumithion (fenitiothion 50% w/v was used as the experimental pollutant.

Alkaline phosphatase activity registered a rise in all tissues following exposure to Sumithion. The increase recorded in gill, kidney ( $P < 0.001$ ), liver, muscle, intestine and brain ( $P < 0.05$ ) was statistically significant (table 1). Increase of phosphatase activity in the present study could be attributed to hyperglycemia. Hyperglycemia has been reported to stimulate alkaline phosphatase<sup>7</sup> activity. It was found that exposure of *S. mossambicus* to Sumithion increased blood glucose content, hepatic phosphorylase activity and decreased hepatic glycogen concentration<sup>3</sup>. Since phosphorylation of glucose is an energy requiring process, increase in phosphatase activity which catalyses liberation of inorganic phosphates from phosphate esters like glycerophosphate, phenyl phosphate, etc., is justifiable. Fall in hepatic glycogen concentration and

TABLE I

*Effect of Sumithion on alkaline phosphatase activity in some organs of S. Mossambicus*

Tissue	Control	Sumithion Exposed	% Change
Brain	0.71 ± 0.05	0.78* ± 0.08	+ 10.4
Gill	11.78 ± 1.35	15.43 ± 1.13	+ 31
Muscle	0.76 ± 0.08	0.83* ± 0.05	+ 9.3
Liver	4.63 ± 0.98	5.81* ± 1.26	+ 25.5
Intestine	7.92 ± 1.21	9.34* ± 1.36	+ 18.0
Kidney	23.02 ± 2.36	28.14 ± 1.83	+ 22.3

Activity of the enzyme is expressed as mg of inorganic phosphate formed/g weight of tissue/hr)

Values expressed are mean ± S.D. of 6 individual experiments. Changes after pesticide treatment are statistically Significant \*  $P < 0.05$

increased phosphorylase activity after exposure to Sumithion suggests glycogen as the source of hyperglycemia in the treated fish. Hence it is likely that treatment of Sumithion induced hyperglycemia as a result of glycogenolysis and consequent increase of phosphatase activity to meet energy requirements in order to counter the stress.

11 May 1981

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