galls on a leaf varied from 5 to 40 and the young galls were green in colour while the older ones were brown. The mature gall chamber is 4 to 7 mm in diameter, unilocular, enclosing a single nymph inside, unlike the leaf galls of *Garuga pinnata*[3], where more than one nymphal instar occur. Though the galls occur in clusters, the zoocecidia are, however, *unilocular*. The gall cavity is spongy in nature at the nutritive zone of the gall maker and the outer zone is hard.

The size of the gall, which varies from 2 to 12 mm, depends on the developmental stage of the gall maker. When the young and mature galls were cut open, the first and fifth instar nymphs respectively were observed in the gall chambers. The fifth instar nymphs were always surrounded by their white waxy secretions. Exuviae of these nymphs were noticed in fully developed galls indicating their moulting into adults in the nymphal cavity itself. The moulted adults escape from the galls through the apex region on the ventral side of the leaf as in the case of leaf galls on *Ficus glomerata* induced by a *Pauropsylla* sp.[4].

Further studies on the developmental anatomy of this gall is in progress.

Our thanks are due to the Deputy Director, Botanical Survey of India, Coimbatore, for confirming the identity of the host plant. The first author gratefully acknowledges Dr. T. N. Ananthakrishnan, Director, Entomology Research Institute, Loyola College, Madras, for his constant encouragement and guidance.

October 13, 1980.

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TISSUE RESPIRATION IN *SAROTHERODON MOSSAMBICUS* (PETERS) EXPOSED TO SUB-LETHAL CONCENTRATION OF SUMITHION AND SEVIN

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Organophosphate (OP) and Carbamate Insecticides have been reported to reduce whole animal and tissue respiration in Fishes. Exposure of Fish[1] *Lebistes reticulatus* to OP insecticides like malathion, foschlor, dichlorovos inhibited oxygen uptake by 65%, 40% and 30% respectively, while[2] parathion and malathion inhibited *in vitro* O_2 uptake of Blue gill (*Lepomis microchirus*) liver mitochondria in the presence of succinate. The organochlorine and Cyclodiene insecticides namely Chlordane, heptachlor, Kepone, thimet and toxaphene severely inhibited oxygen uptake by Blue gill (*Lepomis microchirus*) liver mitochondria in the presence of succinate[2], but other insecticides like aldrin, dde, dasanit, endrin, lindane, methoxychlor (all organochlorides) parathion, malathion, diazinon (organophosphates), carbofuran and sevin (carbamates) inhibited oxygen uptake in fishes[2]. According to Hiltibran, the inhibition of oxygen uptake was less in the presence of L. Ketoglutarate than in the presence of succinate. Similarly D.D.T. was also found to inhibit oxygen uptake in Blue gill liver mitochondria in the presence of succinate[2]. Earlier experiments with *Tilapia* (Sarotherodon) have revealcd that lethal (Lc 50/48 hours) concentration of sumithion (6 mg/l) and sevin (10 mg/l) reduced 02 uptake of brain, gill, muscle, liver intestine and kidney[3]. The present paper describes tissue respiration of *Sarotherodon mossambicus* (Peters) exposed to sub-lethal concentration of sumithion and sevin.

Maintenance, size and weight range of fish used in the experiments were described earlier. Fish ringer solution with Phosphate buffer at pH 7·5 was used as the suspension medium for the tissues. Oxygen consumption of different tissues was measured in a warburg constant volume respirometer as per procedures given by Umbreit *et al*[5]. Commercial grade sumithion O, O-dimethyl O-(3-methyl-4-nitrophenyl) phosphorothioate from Tata Fison and Co., and sevin (1-naphthyl N-methyl carbamate) EC 50% W.P. from Union Carbide of India were used. The insecticides selected were extensively sprayed by local agricultural workers. Lethal (Lc 50/48 hours) concentration was calculated by Probit method[6] and approximately 1/3 of the Lc 50/48 hours concentration was selected for sub-lethal treatment. It was found that sub-lethal concentration of sumithion and sevin was 2 mg/l and 4 mg/l respectively. Tissue respiration was recorded in fishes exposed to a sub-lethal concentration of sumithion and sevin for 30 days. Similar experiments with normal fish served as controls.

The data on oxygen consumption of brain, gill, muscle, liver, intestine and kidney of normal, 30 day sumithion exposed, 30 day sevin treated fish has been given in Table I. With the exception of gill, metabolic tissues such as brain, liver and muscle exhibited greater reduction of oxygen uptake than osmoregulatory tissues. With sumithion the per cent reduction of O_2 consumption in different tissues was in the following order. Brain > gill > liver > muscle > intestine

TABLE I

Effect of sub-lethal concentration of sumithion and sevin in certain metabolic and osmoregulatory tissues of S. mossambicus

Tissue	Control	30 day sumithion exposed	% change	30 day sevin exposed	% change
1. Brain	638 ± 16·59	431 ± 23·86	−32·44	539 ± 21·13	−15·51
2. Gill	1248 ± 17·51	912 ± 20·16	−27	1115 ± 25·68	−11
3. Muscle	163 ± 10·07	141 ± 9·81	−13·49	148 ± 10·78	− 9·20
4. Liver	549 ± 23·11	415 + 28·10	−24·40	451 + 22·5	−18
5. Intestine	915 ± 21·9	827 + 25·57	−10	841 + 27·14	− 8·01
6. Kidney	1448 + 32·04	1360 ± 38·81	− 6·07	1352 ± 38·23	− 7

Values are expressed as mean ± S.D. of six experiments are significant $P < 0{\cdot}001$ except $P < 0{\cdot}01$.

> kidney. Similarly with sevin, the per cent change from the normal fish was in the following order. Liver > brain > gill > muscle > intestine < kidney. Per cent reduction of O_2 consumption of intestine and kidney did not exhibit any appreciable difference between sumithion and sevin treated fish. The Pesticides *fenitrothion and carbaryl get hydrolysed in water* on standing and yield degradative products like desmethyl sumithion, dimethyl phosphorothioc acid and 1-naphthol respectively. The physiological effects observed in the present study could also be due ot formation of hydrolysed products. The reduced O_2 uptake by tissues observed in the present study indicated decreased oxidation of important substrates such as succinate and pyruvate. The inhibition of Tricarboxylic Acid (TCA) cycle enzyme succinic dehydrogenase (SDH) and triggering of glycolytic enzyme Lactic dehydrogenase (LDH) in sumithion[4] and sevin[7] exposed *S. mossambicus* suggested operation of anaerobic glycolysis in the stressed fish. The decreased O_2 utilisation of gill results 'histotoxic anoxia' in which the gill tissue not only suffers from oxygen 'debt' but also loses effective mechanism for removing CO_2 from the blood. Further it is well known that gill, intestine and kidney are important osmoregulatory tissues in fishes. Inhibition of O_2 uptake in these tissues may lead to disruption of osmoregulation in the fish. The role of Adenosine Triphosphatase (ATPase) activity in maintaining tissue osmolarity has been reported in marine teleosts[8], and inhibition of calcium, magnesium activated ATPase activity[9] in fishes poisoned with parathion and inhibition of high energy phosphate metabolism in rats by carbamate insecticides[10] was found. The decreased respiration of tissues observed in the present study normally leads to decreased oxidation of substrates and as a result energy production will be reduced. Under these conditions ATPase activity will be less and ultimately osmoregulation will be disrupted. *Hence it is likely that presence of even small concen*trations of the insecticides not only disrupts osmoregulatory machinery in the fish but also reduces their survival abilities when exposed for a long period.

December 1, 1980.

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