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LACTIC ACIDOSIS IN DIFFERENT TISSUES OF *SAROTHERODON MOSSAMBICUS* (PETERS) EXPOSED TO SEVIN

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ONSET of oxygen debt[1] resulting in utilization of stored glycogen[2,3] and a shift from aerobic to anaerobic mode of energy release[4,5] have been reported in fishes following exposure to different kinds of pesticides. Animals, which pay off an oxygen debt, tend to accumulate more lactic acid during anaerobiosis[6]. Lactic acid is the end product of anaerobic glycolysis and its accumulation in different tissues above normal levels is indicative of a shift from aerobic to anaerobic metabolic pathway under hypoxic conditions. Changes in lactic acid levels of different tissues following pesticide exposure have not been investigated in detail in fishes except the studies of Shaffi[7] and Verma *et al*[8]. In the present investigation, the patterns of changes in lactic acid levels of different tissues of *Sarotherodon mossambicus* (Peters) following exposure to a carbamate pesticide sevin have been studied.

The carbamate pesticide, sevin (1-Naphthyl N-Methyl carbamate), widely applied in the eradication of cotton pest in Coimbatore District, was used in the present study. Samples of *S. mossambicus* (5–9 g), obtained from local ponds in and around Coimbatore City, were acclimatized to laboratory conditions (29 ± 1°C) in large cement tanks and regularly fed with cooked rice. A stock solution of 1 gram of sevin (available as 10% dust) in 100 ml of distilled water was used to prepare the required concentrations of pesticide-water. The sublethal and lethal concentrations of sevin were assessed by repeated exposure experiments employing the static bioassay method and Probit analysis[9]. Fishes were exposed to 3 ppm (sublethal) of sevin for 48 hr and to 25 ppm (lethal concentration) of sevin for 6 hr in glass jars. A batch of 10 fishes were exposed to each concentration for the prescribed period. Control fishes were also maintained in similar glass jars with pesticide-free tap water.

Lactic acid levels were estimated in blood, liver, muscle and heart of control and pesticide-exposed fishes (after 48 hr of sublethal exposure and after 6 hr of lethal exposure) following the method of Barker and Summerson[10]. Blood sample was collected by cardiac puncture using a pre-chilled hypodermic syringe rinsed with cold heparin. Tissue samples of liver, muscle and heart were dissected by keeping the stunned (by a blow on the head) fish in an iced trough. Lactic acid levels in blood were expressed in mg/100 ml of blood and those of liver, muscle and heart in mg/g of tissue. The lactic acid levels in blood, liver, muscle and heart of control and sevin-exposed *S. mossambicus* are presented in table 1. Data on lactic levels of blood are represented in figure 1 and those of liver, muscle and heart in figure 2.

A perusal of table 1 reveals increased accumulation of lactic acid in the blood of sevin-exposed *S. mossambicus* above normal level resulting in severe 'lactic acidosis'. This probably indicates the operation of anaerobic glycolysis in different tissues and channelling of the final product (lactic acid) into blood. Lactic acidosis in blood appears to be more severe under lethal exposure than sublethal exposure in *S. mossambicus* (table 1). A similar increase in the

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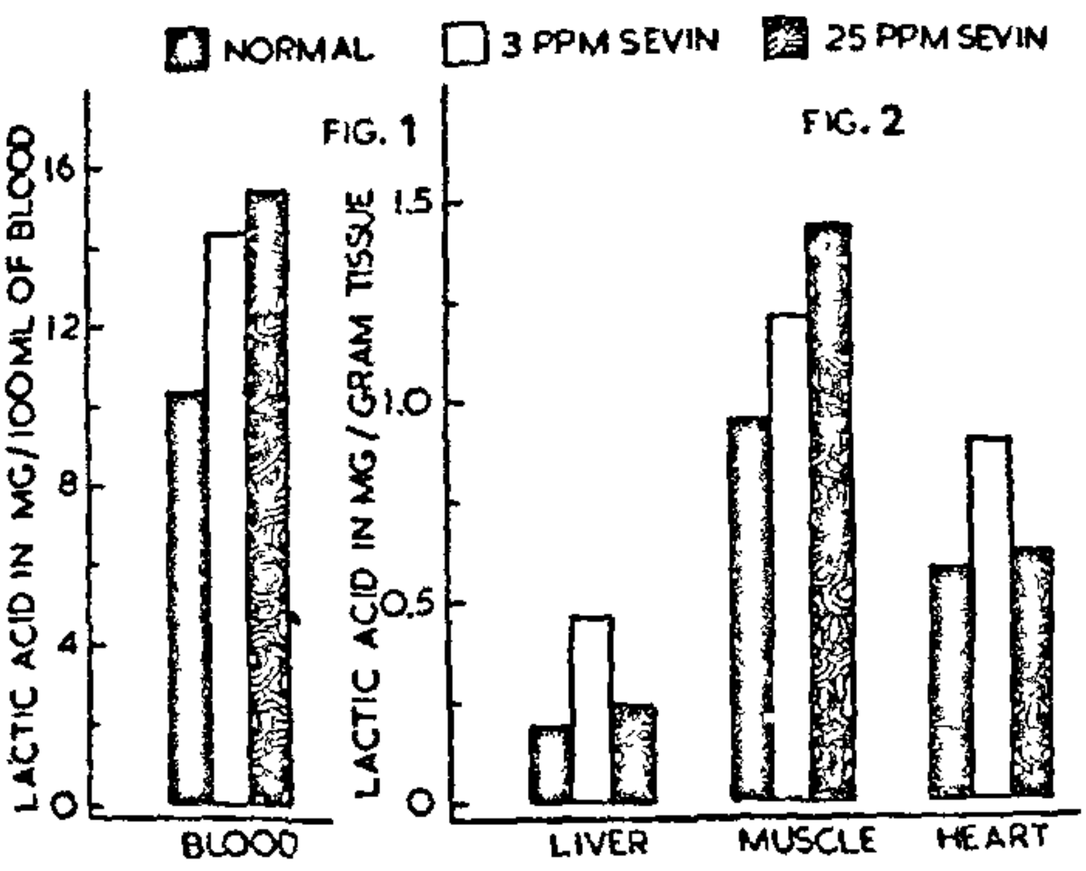
Table 1 Lactic acid levels of blood*, liver†, muscle† and heart† of control and sevin-exposed *S. mossambicus*. Values are means ± S. E. (N)

Tissue	Control	3 ppm sevin-exposed	25 ppm sevin-exposed
Blood	10.27 ± 0.68 (7)	14.24[a] ± 1.21 (7) +39%	15.37[a] ± 0.64 (6) +50%
Liver	0.19 ± 0.01 (7)	0.46[a] ± 0.06 (7) +150%	0.24[b] ± 0.04 (6) +29%
Muscle	0.95 ± 0.07 (7)	1.21[a] ± 0.09 (7) +27%	1.44[a] ± 0.03 (6) +51%
Heart	0.58 ± 0.07 (7)	0.90[a] ± 0.05 (7) +55%	0.62[b] ± 0.10 (6) +7%

*Values expressed as mg lactic acid/100 ml of blood; †Values expressed as mg lactic acid/gm of tissue; [a]Statistically significant at 5% level ($P < 0.05$); [b]Statistically not significant.



Figures 1 and 2. **1.** Lactic acid levels in blood. **2.** Liver, musle and heart of normal and sevin-exposed *S. mossambicus*.

serum lactic acid level has been reported in carp, snake-headed fish and catfish exposed to heptachlor[7] indicating tissue glycogenolysis and in the blood and liver of *Notopterus notopterus*[8] exposed to pulp, paper and mixed pulp-paper wastes.

Comparatively lesser retention of lactic acid in liver and heart tissues of fishes exposed to lethal concentration of sevin indicates that the lactic acid formed in these tissues due to anaerobic glycolysis (utilizing stored glycogen) immediately enters into blood, causing severe lactic acidosis in blood. The onset of severe blood lactic acidosis in *S. mossambicus* (under lethal exposure in the present study) may be one of the causes for the death of the fishes within 24 hr of exposure to lethal concentration of sevin.

The increased retention of lactic acid in liver under sublethal exposure for 48 hr (to about 150%) suggests that under prolonged exposure of the fish to pesticide, lactic acid produced in different tissues is channelled to the metabolically active tissue liver where it could be stored. This stored lactic acid could be used for reconversion into glycogen[8] (glycogenesis) in the liver. Or, the accumulated lactic acid in liver could be reutilized when the fishes return to normoxic conditions, by conversion of the lactic acid into pyruvic acid which will be fed into TCA cycle via acetyl-CoA.

Muscle tissue of *S. mossambicus* appears to be a continuous site of retention of lactic acid (simulating 'diving syndrome' wherein, the onset of blood lactic acidosis is prevented by the sequestering of lactic acid in muscle), showing increased accumulation of muscle lactic acid with increase in concentration of sevin. However, unlike that of 'diving syndrome', sevin-exposure causes severe blood lactic acidosis inspite of heavy retention of lactic acid in the muscle of *S. mossambicus* (table 1).

The very low retention of lactic acid (only 7%) in the heart tissue of *S. mossambicus* exposed to lethal concentration of sevin, inspite of 68% drop in heart glycogen of the same fish under similar exposure[11], could be taken to suggest that the excess lactic acid formed in the heart tissue is flushed off (possibly into the blood), thereby maintaining the heart tissue free from severe lactic acidosis and physiologically fit for increased cardiac function during early periods of pesticide-exposure. Or, the lesser accumulation of lactic acid in the heart tissue of fish under lethal exposure may be due to complete breakdown of glycogen by continuous process of aerobic metabolic pathway, possibly by selective increased oxygen supply to the heart tissue, for increased cardiac

function. This could be of high adaptive value for the fish under pesticide stress.

13 August 1986; Revised 15 October 1986

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EFFECT OF LIV-52 ON BLOOD SUGAR IN BERYLLIUM NITRATE-EXPOSED RATS

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THE toxic effects of beryllium on laboratory animals and humans are well known[1-4]. Aldridge and coworkers[5] studied the mode of its toxic action in rats and rabbits and found that the immediate cause of the death was lowering of blood sugars and liver damage. An Ayurvedic drug, Liv-52 (Himalaya Drug Company, Bombay, India) which is used clinically in various liver disorders[6-9] has also been reported to increase the protective index in beryllium-treated rats[10]. The present investigation, therefore, deals with the effect of oral administration of Liv-52 on the blood sugar and histopathology of liver in beryllium nitrate-exposed rats.

Adult albino rats (150 ± 10 g) of Sprague Dawley strain were selected from the rat colony of the department. All the rats were maintained under uniform husbandry condition of light, temperature and were given pelleted diet (Hindustan Levers, Bombay) and water *ad libitum*. Beryllium nitrate was dissolved at a concentration of 0.316 mg/ml in pyrogen-free distilled water and was injected to the experimental animals intravenously once only at a dose of 0.316 mg/kg body weight (1/10th of LD_{50})[11]. This dose of beryllium nitrate was toxic in pregnant rats[12] and therefore, selected for further studies.

Liv-52 syrup (obtained from Himalaya Drug Company, Bombay) contained the extracts of *Capparis spinosa, Cichorium intybus, Solanum nigrum, Cassia occidentalis, Terminalis arjuna, Achilla millefolium* and *Tamarix gallica.*

The selected animals were divided into four groups of ten each and were treated as follows:
Group 1. Animals were given vehicle only; Group 2. Animals were primed with Liv-52 for 10 days prior to the experiment and thereafter received Liv-

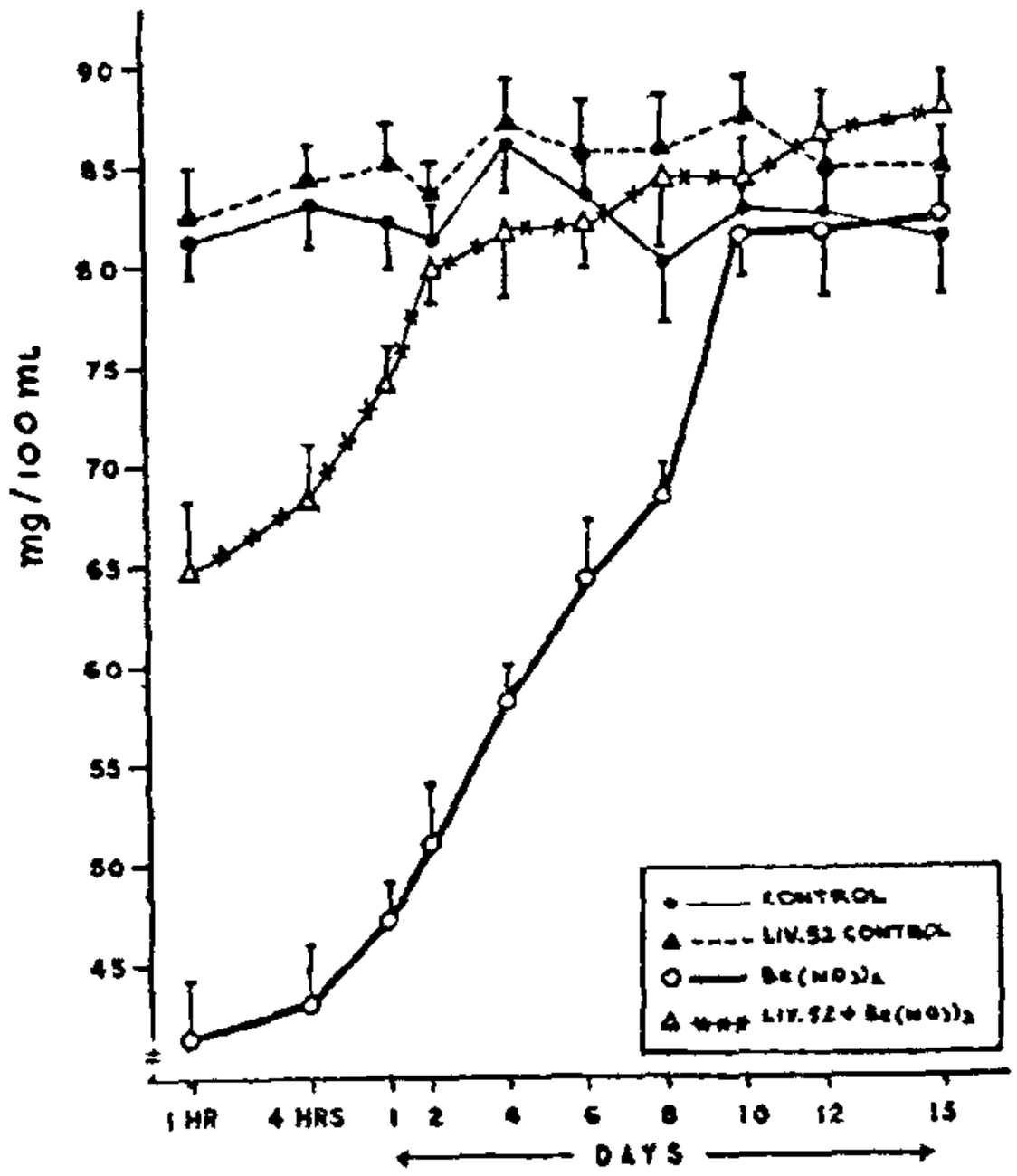

Figure 1. Effect of beryllium nitrate on the blood sugar level in adult rats primed with Liv-52 syrup.