ENDOSULPHAN-INDUCED CHANGES IN THE CARBOHYDRATE METABOLISM OF A FRESHWATER PRAWN, *MACROBRACHIUM LAMARREI* (H. MILNE EDWARES).

OMKAR, V. B., UPADHYAY and G. S. SHUKLA
Department of Zoology, University of Gorakhpur, Gorakhpur 273001, India.

ENDOSULPHAN is a broad-spectrum, non-systemic organochlorine compound of cyclodiene group, extensively used in place of endrin, to control a variety of pests. It is a mixture of two stereoisomers, α and β, and the latter is highly soluble in soil and other media. The chemical nature and solubility of the metabolite makes endosulphan more potentially toxic to fish and other aquatic organisms[1]. The toxicity of endosulphan to aquatic invertebrates are reported[2-4]. Its effect on oxygen consumption[5], phosphomonoesterases[6], blood glucose[7-8] and other body constituents[9] are also reported. The present authors have evaluated the LC_{50} of endosulphan to the freshwater prawn, *Macrobrachium lamarrei* (H. Milne Edwards)[10]. The alterations in blood glucose and hepatic glycogen values of *M. lamarrei* exposed to endosulphan are reported in this note.

The collection, maintenance, size and weight range of prawns were described earlier[10]. The prawns were exposed to a lethal (96 hr LC_{50}) and two sublethal concentrations (0.4 and 0.8 of 96 hr LC_{50}) of endosulphan for 96 hr and a control was also run side by side. The desired tissues were taken out after every 24 hr to determine blood glucose and hepatic glycogen[11,12]. Each experiment was replicated six times and the data were statistically analysed[13].

Endosulphan induced the hyperglycemia in *M. lamarrei* upto 72 hr of exposure after which hypoglycemic response was recorded. The increase in the blood glucose was significant ($P < 0.05$) in lowest concentration after 24 hr more significant ($P < 0.01$) in highest sublethal concentration after 24 hr and in the lowest concentration after 48 hr and highly significant ($P < 0.001$) in two higher concentrations after 48 hr and in all the three concentrations after 72 hr. The decrease in the blood glucose was significant ($P < 0.05$, $P < 0.01$ and $P < 0.001$) in all the three respective concentrations. The hepatic glycogen was decreased after endosulphan exposure. The decrease in the glycogen was significant ($P < 0.05$) in higher sublethal concentration after 24 hr and in lowest concentration after 48 hr, more significant ($P < 0.01$) in highest concentration after 24 hr, in higher sublethal

Table 1 *Changes in blood glucose and hepatic glycogen of M. lamarrei exposed to endosulphan*

	Concentration of Endosulphan (mg/l)	Exposure time (hr)			
		24	48	72	96
Blood glucose (mg/100 ml blood)	0.000	45.20 ± 2.08[a] (100)[b]	45.34 ± 2.15 (100)	45.42 ± 2.32 (100)	44.90 ± 2.20 (100)
	0.0011	54.69 ± 2.46*[c] (121)	60.76 ± 2.62** (134)	64 95 ± 2.70*** (143)	37.72 ± 2.24* (84)
	0.0022	58.76 ± 2.58* (130)	64.84 ± 2.58*** (143)	68.58 ± 2.82*** (151)	31.88 ± 2.18** (71)
	0.0027	62.38 ± 2.65*** (138)	68.92 ± 2 65*** (152)	72.67 ± 2.86*** (160)	28.74 ± 2.28*** (64)
Hepatic glycogen (mg/g wet tissue)	0.000	7.52 ± 0 65 (100)	7.48 ± 0.66 (100)	7.51 ± 0.59 (100)	7.45 ± 0.61 (100)
	0.0011	6.09 ± 0.62 (81)	5.46 ± 0.54* (73)	4.51 ± 0.48** (60)	3.58 ± 0.38*** (48)
	0.0022	5.41 ± 0.57* (72)	4.64 ± 0.50** (62)	3.45 ± 0.42*** (46)	2.53 ± 0.27*** (34)
	0.0027	4.51 ± 0.48** (60)	3.67 ± 0 43*** (49)	2.63 ± 0.33*** (35)	1.94 ± 0.24*** (26)

[a] Values expressed as mean ± S Em.
[b] Percent change from control (100).
[c] *, **, & *** indicate values significant at $P < 0.05$, $P < 0.01$ and $P < 0.001$ respectively.

concentration after 48 hr and in lowest concentration after 72 hr while in other exposure it was highly significant ($P < 0.001$).

The hyperglycemia followed by a decrease in hepatic glycogen suggests the increased glycogenolysis, possibly by the increased activity of glycogen phosphorylase. It is also possible that endosulphan in some manner stimulates the secretion of sinus gland. However, the hyperglycemia may be a physiological response to meet the critical need of brain for the increased energy in the form of glucose[8]. The increase in blood glucose could possibly furnish the high demand of glucose in brain which would in turn compensate to some extent for any potential decrease in brain glucose. The hypoglycemic response with concomitant decrease in hepatic glycogen might be attributed to the inactivation of the enzymes involved in the carbohydrate metabolism due to endosulphan stress. A similar decrease in hepatic glycogen[14] and increase in blood glucose[7, 8, 14] induced by endosulphan was also reported. Thus, it is probable to conclude that endosulphan stress induces glycogenolysis by increasing the activity of glycogen phosphorylase and the increased synthesis of the sinus gland which in turn causes the hyperglycemia to meet the energy demands due to stress.

11 July 1983, Revised 31 October 1983

1. Gopal, K., Khanna, R. N., Anand, M. and Gupta, G. S. D., *Ind. Health.*, 1982, **20,** 157.
2. Sanders, H. O. and Cope, O. B., *Limnol. Oceanogr.*, 1968, **13,** 112.
3. Sanders, H. O., *U. S. Bur. Sport Fish. Wildl., Tech. Pap.*, 1969, **25,** 1.
4. Gopal, K., Khanna, R. N., Anand, M. and Gupta, G. S. D., *Toxicol. Lett.*, 1981, **7,** 453.
5. Reddy, T. G., and Gomathy, S., *Indian J. Environ. Health.*, 1977, **19,** 360.
6. Shaffi, S. A., *Toxicol. Lett.*, 1980, **6,** 339.
7. Gopal, K., Anand, M., Khanna, R. N. and Misra, D., *J. Adv. Zool.*, 1980, **1,** 68.
8. Misra, D., Khanna, R. N., Anand, M. and Gopal, K., *Chemosphere*, 1980, **9,** 119.
9. Murty, A. S. and Devi, A. P., *Pestic. Biochem. Physiol.*, 1982, **17,** 280.
10. Shukla, G. S. and Omkar, *Crustaceana*, 1982 (In press).
11. Oser, B. L., *Hawks physiological chemistry*, Tata McGraw Hill, New Delhi, 1979, 14th edn., p. 1472.
12. Dubois, M., Giller, K. A., Hamilton, J. K., Rebers, P. A. and Smith, F., *Anal. Chem.*, 1956, **28,** 350.
13. Campbell, R. C., *Statistics for biologists*, Cambridge Univ. Press, London, 1967, p. 242.
14. Singh, N. N. and Srivastava, A. K., *Ecotoxicol. Environ. Safety*, 1981, **5,** 412.

DEVELOPMENTAL PROFILES IN THE ISOZYMES OF α AND β-ESTERASES IN THE EMBRYOGENESIS OF SILKWORM *BOMBYX MORI*.

N. B. KRISHNAMURTHY, S. R. RAMESH and (late) M. R. RAJASEKARASETTY*
Department of Post-Graduate Studies & Research in Zoology University of Mysore, Mysore 570006, India.

"DRAMATIC evidence of changing patterns of gene function is furnished by a number of multibanded systems in which various electrophoretic forms appear and disappear or change in relative concentration as development proceeds"[1]. The use of electrophoretic technique to analyse multibanded systems (*viz* isozymes) during the development has yielded useful information with regard to biochemical variation and nature of gene action in various plants and animals.

Preliminary studies on the ontogenetic variation in acid and alkaline phosphatases have been carried out[2] in two races of silkworm *Bombyx mori*. The present studies were undertaken to analyse ontogenetic differentiation of isozymes of α and β-esterases as a biochemical parameter to understand the genetic programming involved in various stages of development.

The egg stage in silkworm *B. mori* lasts for 168 hr at 27°C with a relative humidity of 70–80%. The eggs of a polyvoltine pure Mysore race of *B. mori* formed the material for the present studies. The eggs were collected on polythene sheets and were maintained at 27 ± 1°C with a humidity range of 70–80%. The eggs (200 mg) homogenized in 0.1 ml of glass distilled water, centrifuged for 5 min at 3000 rpm served as the sample. Fresh samples were prepared for each assay. Electrophoretic assays were made at intervals of 24 hr using polyacrylamide gel as the supporting medium, as described by Rajasekarasetty *et al*[3] with a slight modification where 0.05 ml of the supernatant of fresh homogenate mixed with 0.05 ml of 40% sucrose solution was loaded on each gel and electrophoresed at 150 volts for 3 hr. The tray buffers, staining buffers