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1. Huxley, J. S., *Nature (London)*, 1924, **114,** 856.
2. Lowry, O. H., Rosenbrough, N. J., Farr, A. L. and Randall, R. J., *J. Biol. Chem.*, 1951, **193,** 265.
3. Dubois, M., Gilles, K. A., Hamilton, J. K., Rebers, P. A. and Smith, F., *Anal. Chem.*, 1956, **28**, 351.
4. Bray, H. C. and Thrope, W. V., *Meth. Biochem. Analysis*, 1954, **1,** 27.
5. Humphries, E. C., *Mineral compounds and ash analysis; in modern methods of plant analysis*, (eds) K. Peach and M. V. Tracey; Springer-Verlag, Berlin, 1956, p. 542.
6. McNeill, S. and Southwood, T. R. E., *Symposium of the phyto chemical society*, 1978, Reading 1977.
7. Beck, S. D., *Biol. Bull.*, 1956, **10,** 219.
8. Hartfield, L. D., Frazier, J. L. and Ferreira, J., *Physiol. Entomol.*, 1982, **7,** 15.
9. Van Emden, H. F., *Aphid host plant relationships; Some recent studies in prespectives in aphid biology*, (ed.). A. D. Lowe; *Ent. Soc. New Zealand. Bull.*, No. 2. 1973, p. 54.

SIGNIFICANCE OF AMINOTRANSFERASE ACTIVITY OF THE FRESHWATER TELEOST, *OREOCHROMIS MOSSAMBICUS* (TREWAVAS) UNDER LINDANE TOXICITY

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THE indiscriminate and excess use of organochlorine insecticides are known to cause serious effects on non-target animals[1-3]. Lindane is an organochlorine insecticide and is expected to show similar behaviour. But its effect on metabolic and physiological alterations particularly on fish enzyme systems needs elucidation. The aspartate (AAT) and alanine (AlAT) aminotransferases are known to play a strategic role in mobilising L-amino acids for gluconeogenesis and also function as links between carbohydrate and protein metabolisms under altered physiological, pathological and induced environmental stress conditions[4-6].

Because of this unique property, AAT and AlAT enzyme systems were determined in metabolically and functionally active tissues of lindane exposed fish, *Oreochromis mossambicus*, as a function of time.

Healthy, living specimens of *O. mossambicus* were collected from local freshwater tanks. Before experimentation, the fish were allowed to acclimate to laboratory conditions for a week. The test water characteristics were, temperature, $20 \pm 3°C$; pH, 7.2; hardness, 160 ppm (as $CaCO_3$); alkalinity, 87 ppm (as $CaCO_3$) and dissolved oxygen, 7.5 ppm. Preliminary toxicity tests[7] showed that LC 50 of lindane to fish was 0.15 ppm[8].

Two hundred fish measuring 20 ± 3 cm in length and 8 ± 2 g in weight were selected, divided into six equal groups and exposed to lethal (LC 50 0.15 ppm) and sublethal (.0.05 ppm) concentrations of lindane for 12, 24 and 48 hr. After each exposure the fish were stunned by a blow on the head and the tissues like, brain, liver, muscle and gill were isolated and homogenized in 0.25 M sucrose solution. The homogenates were centrifuged at 1000 g for 20 min and the clear supernatants were used as the source of enzymes. The enzyme activity levels in the tissues were estimated by the method of Reitman and Frankel[9] after due standardization and the protein content determined by Lowry's method[10].

The tissue specific AlAT activity recorded maximum elevation at 48 hr of exposure in both concentrations of lindane and the trend is liver, brain, muscle, gill (table 1), while the AAT activity, though elevated at 48 hr exposure, show some variability at 12 and 24 hr of exposure (table 1). The lyotropic series of tissue specific AAT activity levels of fish exposed to both concentrations of lindane is as follows:

On lethal exposure: muscle > liver > gill > brain
On sublethal exposure: muscle > liver > brain > gill

AAT and AlAT enzymes increase in the four tissues at different levels in both concentrations of lindane indicating that the fish is under toxic stress and energy crisis caused by lindane thus promoting the utilization of amino acids for energy synthesis[4,11]. This suggests that the tissue glycogen might be insufficient to meet the lindane toxic stress and hence the operation of gluconeogenesis to mitigate the lindane toxic stress. Irrespective of the concentration, lindane can affect the tissues almost equally at 48 hr of exposure period.

The present results indicate that under lindane exposure, in both concentrations and at different time

Table 1 *Aspartate (AAT) and Alanine (AlAT) Aminotransferase activity levels in the tissues of O. mossambicus, control (C) and exposed to lethal (L) and sublethal (SL) concentrations of lindane as a function of time.*

		Experimental					
		Lethal Exposure			Sublethal Exposure		
Tissue	C	12 hr	24 hr	48 hr	12 hr	24 hr	48 hr
AAT activity							
Brain	1.88 ± 0.13	0.70[b] ± 0.08 (−34)	1.92[b] ± 0.05 (+16)	2.42[a] ± 0.04 (+34)	0.55[b] ± 0.03 (−46)	1.61[b] ± 0.04 (−5)	1.98[b] ± 0.13 (+15)
Liver	1.54 ± 0.10	1.50[b] ± 0.11 (−3)	1.69[b] ± 0.13 (+9)	2.54[a] ± 0.12 (+64)	1.34[b] ± 0.09 (−13)	1.58[b] ± 0.12 (+4)	1.93[a] ± 0.15 (+25)
Muscle	1.67 ± 0.22	1.20[a] ± 0.07 (−40)	1.96[b] ± 0.10 (+5)	2.92[a] ± 0.13 (+75)	1.13[a] ± 0.05 (−42)	1.70[b] ± 0.07 (+3)	2.24[a] ± 0.15 (+34)
Gill	1.49 ± 0.26	1.56[b] ± 0.14 (+3)	1.62[b] ± 0.13 (+7)	1.93[a] ± 0.18 (+29)	1.50[b] ± 0.08 (+1)	1.59[b] ± 0.07 (+5)	1.82[b] ± 0.20 (+22)
AlAT activity							
Brain	4.26 + 0.36	5.98[b] ± 0.18 (+40)	6.05[a] ± 0.22 (+61.7)	9.69[a] ± 0.62 (+126)	5.90[b] ± 0.23 (+38)	6.63[a] ± 0.22 (+55)	8.51[a] ± 0.74 (+98)
Liver	4.64 ± 0.78	6.13[b] ± 0.44 (+27)	8.69[b] ± 0.49 (+80)	10.37[a] ± 0.66 (+145)	5.89[b] ± 0.19 (+22)	7.28[b] ± 0.22 (+51)	9.93[a] ± 0.19 (+106)
Muscle	1.94 ± 0.14	2.14[b] ± 0.13 (+41)	3.47[a] ± 0.11 (+82)	4.63[a] ± 0.38 (+113)	2.69[b] ± 0.13 (+48)	3.29[b] ± 0.14 (+73)	3.72[a] ± 0.70 (+91)
Gill	1.34 ± 0.09	1.58[b] ± 0.12 (+17)	1.66[b] ± 0.08 (+23)	1.93[a] ± 0.10 (+43)	1.46[b] + 0.08 (+8)	1.51[b] ± 0.11 (+12)	1.63[b] ± 0.07 (+21)

Values are significant at 5% level. [a]$P < 0.001$ [b]Not Significant
The values are expressed as μmol of pyruvate formed/mg protein/hr. Each value is mean ± S.D of six individual observations.
The values in the parantheses are percent change over control.

Table 2 *AAT/AlAT ratios in the selected tissues of fish, Oreochromis mossambicus exposed to lethal and sublethal concentrations of lindane as a function of time.*

		Experimental					
		Lethal Exposure			Sublethal Exposure		
Tissue	Control	12 hr	24 hr	48 hr	12 hr	24 hr	48 hr
Brain	0.441	0.117	0.317	0.249	0.093	0.243	0.233
Liver	0.332	0.247	0.195	0.245	0.228	0.217	0.194
Muscle	0.861	0.561	0.565	0.631	0.420	0.517	0.602
Gill	1.112	0.987	0.975	1.000	1.027	1.053	1.017

intervals, the activity profiles of AAT and AlAT followed the same trend as that of the control tissues, but with a difference in the activities (table 1). The AAT/AlAT ratio values (table 2) clearly demonstrate that lindane at 2 concentrations imposes a stress condition. The high levels of AlAT and AAT, more so of AlAT, under lindane exposure suggest that the fish shows adaptability to lindane toxicity.

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1. Gupta, R. C. and Gupta, P. K., *Prog. Ecol.*, 1977, **5/7**, 173.
2. Attri, B. S., *Indian J. Environ. Prot.*, 1981, **1**, 86.
3. Barbehenn, K. Yle, R. and Reichel, W. C., *J. Toxicol. Environ. Health.*, 1981, **8**, 325.
4. Nichol, C. A. and Rosen, F., In: *Advances in enzyme regulation*, Vol. I (ed.) B. Weber, Pergamon Press, New York, 1963, 341.
5. Knox, W. E. and Greengard, O., In: *Advances in enzyme regulation*, Vol. III (ed.) B. Weber, Pergamon Press, New York, 1965, 247.
6. Harper, H. A., Rodwell, V. M. and Mayes, P. A., In: *Review of physiological chemistry* (Lange Medical

Publications, Maruzan, Asia (PVL) Ltd. 1979, 282.
7. Finney, D. J., In: *Probit analysis*, 2nd edition (Cambridge University Press, London) 1964, p. 20.
8. Narasimha Murthy, B., Ph.D., thesis, S.V. University, Tirupati, India, 1983.
9. Reitman, S. and Frankel, A., *Am. J. Clin. Pathol.*, 1957, **28**, 138.
10. Lowry, O. H., Rosebrough, N. J., Farr, A. L. and Randall, R. J., *J. Biol. Chem.*, 1951, **193**, 265.
11. Kacew, S., Sinsal, R. L., Hrdina, P. D. and Ling, G. M., *J. Pharmac. Exp. Ther.*, 1972, **181**, 234.

A CASE OF CHROMOSOME POLYMORPHISM IN *RATTUS RATTUS*

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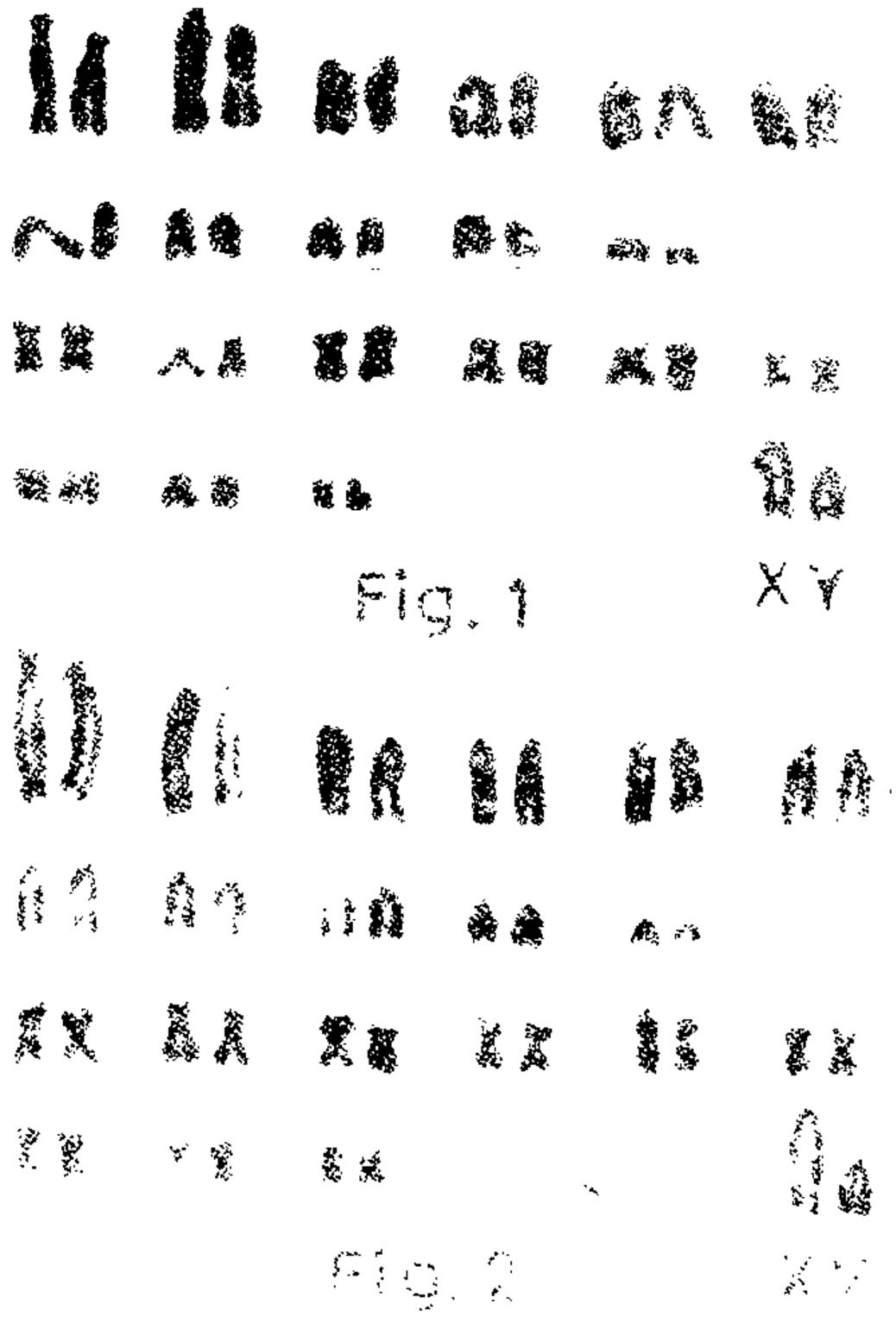
THE common house rat *Rattus rattus* is unique for its chromosomal polymorphism which may be incidental to the geographic distribution of the species. Yosida *et al*[1,2] who described three geographic variants,the Asian ($2n = 42$), the Ceylonese ($2n = 40$) and the Oceanian ($2n = 38$) reported that the Asian type is the ancestral form from which the Ceylonese and the Oceanian types have evolved by way of chromosome fusion. The *Rattus* polymorphism was perhaps contributed by the supernumeraries[3–7].

Ten male and nine female rats were collected from the Gopalpur area. The bone marrow preparations were made following Colchicine-Giemsa-air-drying technique. The morphology of the chromosomes was determined following the procedure of Levan *et al*[8] on 10 well-spread plates from each individual.

The karyotypes of nine male and nine female individuals consisted of 42 chromosomes with 11 pairs of uniarmed (inclusive of X and Y) and 9 pairs of biarmed elements. The first pair of chromosomes (figure 1) is a submetacentric one with a small short arm and a relative length of 9.4%. The uniarmed chromosomes have a relative length range of 6.5% and 2.3% while the biarmed chromosomes ranged between 4.8% and 3.4%. In one of the male individuals, however, an extra uniarmed element was observed in its karyotype whereby the $2n = 43$ (figure 2). Interestingly enough the extra element resembled the smallest pair of acrocentric chromosomes in its size.



Figures 1, 2. 1. Karyotype of normal *R. rattus* male with $2n = 42$, **2.** Karyotype of male with $2n = 43$.

It has been shown that chromosome no. 1 of this species of rat is polymorphic with regard to the centromeric position[3,9]. It is believed that the karyotype where the first pair of chromosome is acrocentric is primitive and the karyotype where the first pair is submetacentric is a derived one consequent upon chromosomal reorganisation.

Most authors have shown supernumeraries in the black rats to be small metacentrics. However, we have found an extra acrocentric element which can compare with the last pair of acrocentrics of the normal karyotypes. A single case of subtelocentric supernumerary has so far been reported from Hokkaido (Japan), and its origin[7], it is claimed, is rather recent and independent of the metacentries.

The effect of extra elements, while being morphologically negative, can yet have visible influence on the physiology or behaviour of the host. White[10], however, felt that a single supernumerary confers an adaptive advantage on the individual while a higher