5. Kumar, V. S. D., Personal Communication, 1985.
6. Veerraju, P., Babu, M. S., Jaikishan, G., Naidu, J. M. and Blake, N. M., *Hum. Hered.*, 1982, **32**, 240.
7. Reddy, A. P., Mukherjee, B. N. and Basu, A., *J. Indian Anthrop. Soc.*, 1983, **18**, 195.
8. Naidu, J. M., Mohrenweiser, H. W. and Neel, J. V., *Hum. Hered.*, 1985, **35**, 148.
9. Ghosh, A. K., *Indian J. Phys. Anthrop. Hum. Genet.*, 1977, **3**, 73.
10. Rao, P. J. M., Ph.D. thesis, Osmania University, Hyderabad, 1987.
11. Muralidhar, B., Ramesh, A. and Goud, J. D., *Gene Geol.*, 1988, (in press).
12. Goud, J. D. and Rao, P. R., *Anthrop. Anz.*, 1979, **37**, 1.
13. Pingle, U., Mukherjee, B. N. and Das, S. K., *Z. Morphol. Anthropol.*, 1981, **72**, 339.
14. Parr, C. W., Bagster, I. A. and Welch, S. G., *Biochem. Genet.*, 1977, **15**, 109.

INFLUENCE OF SEX HORMONES ON SERUM TRANSAMINASES DURING EXPERIMENTAL LIVER INJURY IN RATS

SUDHIR KUMAR and S. V. S. RANA
Department of Zoology, Institute of Advanced Studies, Meerut University, Meerut 250 004, India.

SEVERAL deviations from the normal metabolic function of the liver occur in a wide array of toxic abuses. They result in marked swelling as well as eventual rupture of subcellular organelles. These events lead to activation, inactivation or leakage of enzymes contained within the organelles. Therefore, efflux of enzymes is a sensitive index of hepatotoxicity. Glutamic pyruvic transaminase (GPT, EC 2.6.1.2) and glutamic oxaloacetic transaminase (GOT, EC 2.6.1.1) are of significant diagnostic value. They have been extensively studied during acute or chronic intoxication of the liver[1-4]. Similarly, they may serve as reliable markers of liver function improved by liver protecting agents. A simultaneous study of histological lesions almost confirms the therapeutic value of the drug/agent. In a recent study, sex hormones have been found to exert a protective influence on histological lesions induced by halogenalkanes[5]. The present study was undertaken envisaging a similar influence on liver function also. This report describes the effects of testosterone and progesterone on serum transaminases (GOT, GPT) in rats treated with the hepatotoxins carbon tetrachloride (CCl_4), hexachlorobenzene (HCB) and trichloroethylene (TCE).

Ninety-day-old 45 male and 45 female Wistar rats (*Rattus rattus* albino) weighing 100 ± 10 g were selected from the laboratory stock. They were kept on a 12 h light/dark cycle and fed on laboratory chow obtained from M/s Lipton India Pvt. Ltd., Bangalore, and tap water *ad libitum*. Each rat was housed separately in woven wire cages under standard laboratory conditions (room temp. = 25 ± 3°C, humidity $60 \pm 10\%$) and administered the respective halogenalkane/sex hormone, as in table 1.

After the scheduled treatments, the rats were anaesthetized with diethyl ether and blood was aspirated from the caudal vein. Transminases were estimated in the serum by the method of Reitman and Frankel[6]. Student's *t* test was applied for statistical inferences[7]. Data were further analysed for intergroup comparisons by analysis of variance.

Release of GOT into the serum was much higher in CCl_4-treated rats than in control rats. Hexachlorobenzene (HCB) and TCE treatments also resulted in increased serum GOT. Although testosterone inhibited enzyme efflux in CCl_4- and HCB-treated rats, the hormone did not significantly inhibit enzyme release caused by TCE. Progesterone also failed to inhibit enzyme efflux in TCE- and HCB-treated rats.

Results for GPT show that hormone failed to inhibit enzyme efflux in almost all the treatments. However, testosterone was much more effective than progesterone in all the treatments. Non-significant results were, however, obtained against HCB (table 2).

Sex-related differences in the metabolism of xenobiotics are widely known now. Moreover, it has been reported that liver of males are more sensitive to carbon tetrachloride[8] whereas trichloroethylene induces greater fatty infiltration in liver of females[9]. Quantitative profiles established in the liver of male and female rats have also shown changes in the activities of enzymes[10]. CCl_4 and TCE both are known to induce serum transaminases[11,12]. These observations seem to point to the differential action of sex hormones. The present data for GOT show that the hormones may not interfere at all with the toxic effects of trichloroethylene, whereas they are effective against carbon tetrachloride and hexachlorobenzene. The data for GPT again cast doubt on their efficacy against hexachlorobenzene, parti-

Table 1 *Experimental design*

Group No	Treatment	No and sex of rats employed	Dose administered/ kg body wt	Vehicle/route	Duration/ schedule
1	Saline	5(M)+5(F)	0 25 ml	Intraperitoneal	Each alternate day for 30 days
2	CCl_4	5(M)+5(F)	0.25 ml	Olive oil/ intraperitoneal	Each alternate day for 30 days
3	CCl_4 + testosterone propionate	10(M)	0 25 ml + 0.25 mg	Olive oil/ intraperitoneal	Alternate to each other regularly for 30 days.
4.	CCl_4 + progesterone	10(F)	0 25 ml + 0.25 mg	Olive oil/ intraperitoneal	Alternate to each other regularly for 30 days
5.	Trichloroethylene	5(M)+5(F)	0.25 ml	Olive oil/ intraperitoneal	Each alternate day for 30 days
6.	Trichloroethylene + testosterone propionate	10(M)	0.25 ml + 0.25 mg	Olive oil/ intraperitoneal	Alternate to each other regularly for 30 days.
7.	Trichloroethylene + progesterone	10(F)	0.25 ml + 0 25 mg	Olive oil/ intraperitoneal	Alternate to each other regularly for 30 days.
8	Hexachlorobenzene	5(M)+5(F)	70 mg	Gavage	Each alternate day for 30 days
9.	Hexachlorobenzene + testosterone propionate	10(M)	70 mg + 0 25 mg	Gavage/ intraperitoneal	Alternate to each other regularly for 30 days.
10	Hexachlorobenzene + progesterone	10(F)	70 mg/ 0 25 mg	Gavage/ intraperitoneal	Alternate to each other regularly for 30 days.

Carbon tetrachloride was procured from E. Merck A G , Darmstadt, FRG; trichloroethylene from BDH (India), hexachlorobenzene from Fluka, Switzerland; testosterone propionate and progesterone from German Remedies., Bombay.

Table 2 *Effect of hepatotoxins and hepatotoxin-hormone combinations on serum levels of GOT and GPT in rat*

Treatment	GOT	GPT
Control	29 76 ± 2 57	20 00 ± 2 08
CCl_4	82.03 ± 0 50*	27 60 ± 2.66
CCl_4 + testosterone	80 35 ± 0 61 '	28 26 ± 1 06 ' '
CCl_4 + progesterone	^{b}75.04 ± 0 38 ' ' '	^{a}32.26 ± 0 64 ' ' '
TCE	74.58 ± 0 37*	38 68 ± 0 44*
TCE + testosterone	70.90 ± 1 90	28.83 ± 0.22 ' ' '
TCE + progesterone	75 19 ± 0 82	^{b}39 24 ± 0.54
HCB	79.17 ± 0.30*	42.52 ± 0.79*
HCB + testosterone	77 09 ± 0 64 ' '	42 20 ± 0.67
HCB + progesterone	76.93 ± 0 46	41 84 ± 0 57

Enzyme levels are expressed as Kramen units.
Results are presented as ± SE for five observations. Asterisk have been used to indicate the significant difference between the control and halogenalkane(s) treatments. ' denotes the level of difference between the hormone supplemented and halogenalkane treatments. *a* and *b* have been superscribed to signify intergroup comparisons. * $P < 0.01$; ' $P < 0.05$, ' ' $P < 0.01$, ' ' ' $P < 0.0001$, $^a P < 0.05$, $^b P < 0.001$

cularly that of progresterone are against trichloroethylene. Metabolic studies of sex hormones have revealed that specific steroid dehydrogenases handle testosterone and progesterone in the liver[13]. Introduction of "free radical" into a "steroid–protein" molecule potentiates hormonal activity that inhibits further generation of free radicals in hepatic parenchyma. Probably it is this complex that plays a vital role in enrouting toxic manifestations. Nevertheless, the present results do not strictly support earlier observations on lipid peroxidation and morphological lesions[5]. Transaminases, it seems, behave independently under different hormonal milieu but under the influence of biochemical lesions.

Financial help from CSIR, New Delhi, is gratefully acknowledged.

14 December 1987; Revised 27 February 1988

1. Schwarzmann, V., *Rev. Int. Hepatol.*, 1957, **7**, 387.
2. Rees, K. P. and Sinha, K. P., *J. Pathol. Bacteriol.*, 1960, **80**, 297.
3. Griffiths, D. B., Rees, K. R. and Sinha, K. P., *J. Pathol. Bacteriol.*, 1961, **82**, 109.
4. Boivin, P., Auzepy Ph. and Fauvert, R., *Ann. Biol. Clien (Paris)*, 1961, **19**, 793.
5. Kumar, S. and Rana, S. V. S., *Gagenbaurs Morphol. Jahrb.*, 1987, **133**, 657.
6. Reitman, S. and Frankel, S., *Am. J. Clin. Pathol.*, 1957, **28**, 56.
7. Fisher, R. A., *Statistical methods for research workers*, 11th edn, Oliver and Boyd, London, 1950.
8. Gyorygy, P., Scifter, J., Tomarelli, R. M. and Goldblatt, H., *J. Exp. Med.*, 1946, **83**, 449.
9. Tronche, P., Bressolette, M. H. and Laroussinie, C., *C. R. Soc. Biol.*, 1962, **156**, 1298.
10. Teutsch, H. F., *Chemomorphology of liver parenchyma*, Gustav Fischer Verlag, Stuttgart, New York, 1981.
11. Desai, S., Sanna, A., Mascia, A. and Congin, L., *Riv. Farmacol Ter.*, 1974, **5**, 149.
12. Carlson, G. P., *Res. Commun. Chem. Pathol. Pharmacol.*, 1974, **7**, 637.
13. Genot, A., Loras, B., Monbon, M. and Bertrand, J., *Steroid Biochem.*, 1975, **6**, 1247.

NEWS

ACID RAIN CLOUD INCREASE THE POWER OF MOSSES TO KILL FORESTS

According to Lee Klinger of the National Center for Atmospheric Research (Boulder, CO). Mosses injure trees by producing organic acids that combine with and activate aluminum in soil and air. The aluminium is conducted to the roots of trees where it displaces the calcium that is essential to the health of the trees' fine roots. These roots die, and the trees cannot absorb nutrients. Klinger has found that acid rain stimulates the growth of mosses and exacerbates their effects in forests. Moreover, mosses absorb water, thereby converting dying forests to bogs. Bogs, in turn, emit gases that form acid rain. Widespread bog formation also can reduce nutrient levels on land and in waters and cause mass extinctions of plants and animals according to Klinger. (*Environmental Science and Technology*, Vol. 22, No. 11, November 1988, p. 1248; Published by: The American Chemical Society, 1155, 16th Street, N.W., Washington D.C.)