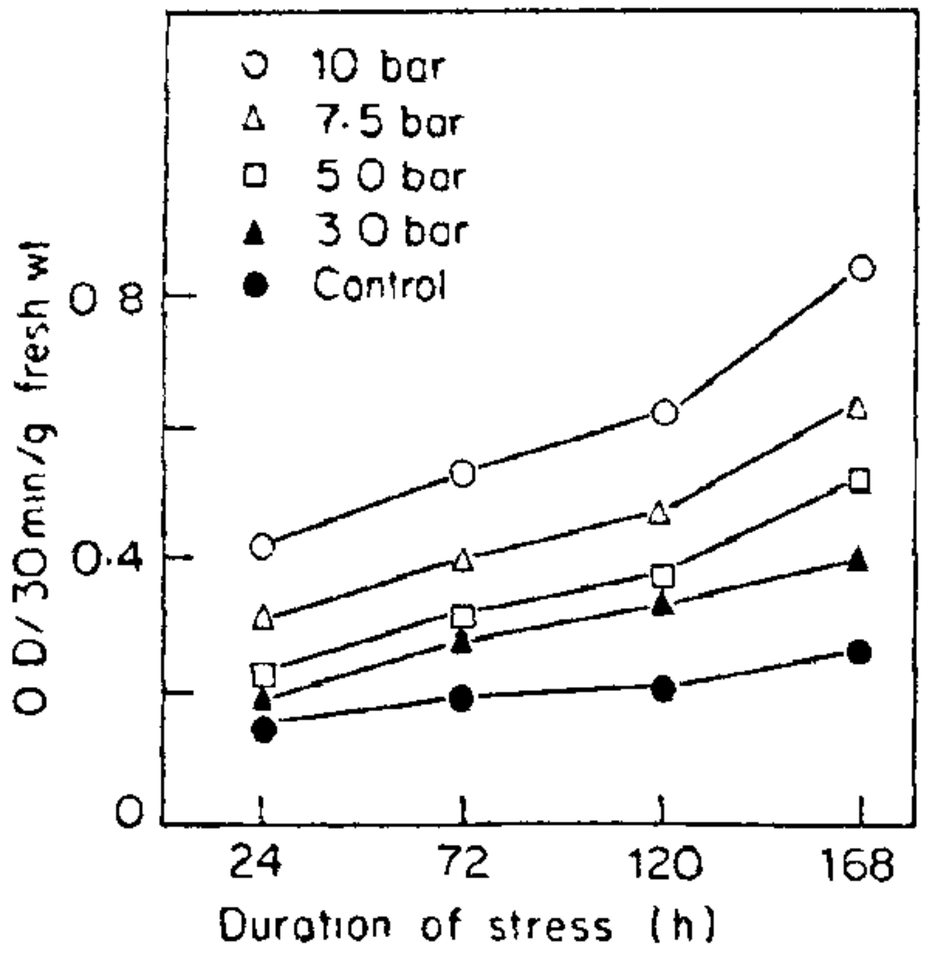

Figure 2. Effect of water stress on IAA oxidase activity.

The results obtained for the IAA oxidase activity for the various groups are shown in figure 2. The IAA oxidase activity in leaves of seedlings belonging to the control group shows a marginal increase from day 1 to day 7. The same pattern is seen in the test groups under water stress, the increase being steeper than in the control group. The IAA activity in leaves of the test group was considerably higher than that for the control group. The pattern was,

Control < -3 bar < -5 bar < -7.5 bar < -10 bar.

Thus it is clear that during water stress, the peroxidase activity declines while the IAA oxidase activity increases. Maintenance of normal auxin levels is important for the well-being of the plant tissues. Considering that both IAA oxidase and peroxidase might be involved in preventing deleterious accumulation of auxins, it is likely that during water stress there is a fall in peroxidase level and this is compensated by an accompanying increase in IAA oxidase activities. The results also suggest that the two enzymes are separate and individual entities.

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1. Ahuja, B. S. and Kaur, K., *Indian J. Exp. Biol.*, 1985, **1**, 57.
2. Balasimha, D., *Plant Physiol. Biochem.*, 1982, **9**, 130.
3. Hinman, R. L. and Lang, J., *Biochemistry*, 1965, **4**, 144.
4. Hoyle, M. C., *Plant Physiol.*, 1972, **50**, 15.
5. Hadas, A., *J. Exp. Bot.*, 1976, **27**, 480.
6. Machly, A. C., In: *Methods of biochemical analysis*, Interscience, New York, 1954.
7. Darbyshire, B., *Plant Physiol.*, 1971, **47**, 65.

A REPORT ON THE LEVEL OF PROTEIN CATABOLISM IN DOXORUBICIN-TREATED RATS

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THE anthracyclin glycoside doxorubicin (former generic name, adriamycin) is a widely used anticancer drug. In addition to its well-known cytotoxicity[1], doxorubicin has cardiotoxic[2] and nephrotoxic[3] effects. Considerable evidence from *in vitro* and *in vivo* studies suggests that activation of the drug to the semiquinone-free radical is an important step in the cascade of doxorubicin-induced events[4]. Some activated oxygen species formed during the reoxidation of semiquinone radicals can damage a variety of cellular macromolecules, including enzymes[5]. Since liver is the major organ involved in detoxification and degradation, an attempt has been made to study the level of protein catabolism in liver. Here, we report the activities of a few enzymes viz. transaminases, arginase, ornithine transcarbamylase and the levels of blood urea, serum and liver protein.

Doxorubicin was bought from Sigma Chemicals, USA. Weanling male albino rats derived from Wistar strain, weighing from 50 to 60 g were selected for the study. The animals were from the Institute of Forensic Science, Madras.

All the animals were given commercial pelleted feed (Hindustan Levers, Bombay). Food and water were given *ad libitum*. The rats were divided into 2 groups. Group I served as control. Group II animals were treated with doxorubicin (7.5 mg/kg body wt) intravenously for 2 days in 0.2 ml of sterile saline[3]. The control animals were injected with 0.2 ml of sterile saline. The animals were killed after 4–5 days, by cardiac puncture and 1 ml of blood was collected in potassium oxalate and the remaining blood was collected without any anticoagulant and the serum was separated. The method of Geyer and Dabich[6] was adopted for urea estimation.

Table 1 *Levels of blood urea, serum and liver protein in control and doxorubicin-treated rats*

	Control	Test
Blood urea mg/dl	30.1 ± 1.10	21.9 ± 0.92*
Serum protein g/dl	4.21 ± 0.21	3.05 ± 0.15*
Liver protein mg/g wet wt of the tissue	200 ± 10.1	240.4 ± 12.9*

*$P < 0.01$; Values are expressed as mean ± SD from 6 animals in each group.

The liver dissected out was immediately washed in ice-cold saline and 0.1% homogenate was prepared in 0.1 M tris-HCl buffer (pH 7.4). The homogenate was centrifuged at 2500 rpm for 5 min. The supernatants were used for enzyme assay. Aspartate aminotransferase (E.C. 2.6.1.1) and alanine aminotransferase (E.C. 2.6.1.2) were assayed[7]. The method adopted for arginase (E.C. 3.5.3.1) was that of Herzfeld and Raper[8]. Ornithine transcarbamylase (E.C. 2.1.3.3) was assayed[9] and the protein was estimated[10].

The levels of blood urea, serum and liver protein are shown in table 1. The levels of transaminases, arginase and ornithine transcarbamylase are decreased (table 2) due perhaps to the decreased level of protein catabolism. This is also supported by the decrease observed in blood urea and the increase in liver protein levels. It has been reported that exposure of cells to doxorubicin leads to more rapid rate of protein synthesis relative to the rate of degradation[11]. Arena *et al*[12] pointed out that doxorubicin stimulates protein synthesis in the liver. Our results confirm that the rate of protein catabolism is less when compared to protein synthesis.

Table 2 *Levels of liver transaminases, arginase and ornithine transcarbamylase in control and doxorubicin-treated rats*

	Control	Test
GOT	101.1 ± 5.10	72.75 ± 2.70*
GPT	243.0 ± 10.01	200.00 ± 8.12*
Arginase	1207.0 ± 20.50	800.00 ± 16.15*
Ornithine transcarbamylase	558.0 ± 15.15	348.00 ± 11.15*

*$P < 0.01$; GOT, GPT–n mol of pyruvate liberated per min per mg protein; Arginase–n mol of urea liberated per min per mg protein; OT case–n mol of citrullin formed per min per mg protein; Values are expressed as mean ± SD for 6 animals in each group.

The serum protein levels decrease significantly in doxorubicin-treated rats. This could be due to the nephropathy and proteinurea[3,13] in doxorubicin-treated animals. It is therefore tentatively suggested that the decrease in the activity of transaminases, arginase and OT case may lead to a decrease in protein catabolism in doxorubicin-treated rats. Further studies on the chronic effect of doxorubicin on protein catabolism are being carried out following a long-term treatment.

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1. Gary, M. D., Barlogie, B., Johnston, D., Mettard, D. and Freireich, J. E., *Eur. J. Cancer*, 1981, **17,** 549.
2. Bertazzoli, C. and Ghione, M., *Pharmacol. Res. Commun.*, 1977, **9,** 235.
3. Tullio, B., Poggi, A., Pozzoni, R., Delaini, F., Mecca, G., Remuzzi, G. and Donati, M.B., *Lab. Invest.*, 1982, **46,** 16.
4. Goormaghtigh, E. and Ruysschaert, J. M., *Biochem. Biophys. Acta*, 1984, **779,** 271.
5. Kim, K., Rhee, S. G. and Stadtman, E. R., *J. Biol. Chem.*, 1985, **260,** 15394.
6. Geyer, J. W. and Dabich, G., *Anal. Biochem.*, 1971, **39,** 412.
7. King, J., *Practical clinical enzymology*, D. Van Nostrand, London, 1965, p. 121.
8. Herzfeld, A. and Raper, S. M., *Biochem. J.*, 1976, **153,** 469.
9. Weber, G., Queener, S. F. and Morris, H. P., *Cancer Res.*, 1972, **32,** 1933.
10. Lowry, O. H., Rosebrough, N. J., Farr, A. L. and Randal, R. J., *J. Biol. Chem.*, 1951, **193,** 265.
11. Dennis Warren, R., *Blood cells*, 1983, **9,** 57.
12. Arena, E., Bionda, F., D' Alessandro, N., Dusonchet, L., Gebbia, N., Gerbasi, F., Rausa, L. and Sanguedolce, R., *IRCS. Libr. Compend.*, 1974, **2,** 1053.
13. Fajardo, Luis, F., Eltringham, J. R., Stewart, J. R. and Klauber, M. R., *Lab. Invest.*, 1980, **43,** 242.