Results. Wet weights of testes and prostates decreased markedly, whereas acid and alkaline phosphatases were significantly elevated following treatment with embelin and *Vinca* extract (tables 1 and 2).

Discussion. The effect of embelin and *Vinca rosea* extract appear to be dose- and duration-dependent. Reduction in weights of testes and prostates indicates impairment in the function of these organs.

Alkaline phosphatase is associated with the transport of metabolites, differentiation of cells and synthesis of testicular hormones[8-10]. Increased alkaline phosphatase activity in testes and prostates identifies these as the sites of tissue damage and sperm resorption[11,12]. Acid phosphatase, a lysosomal enzyme is present in all the germinal cells of the testis. Increase in acid phosphatase activity coincides with a decrease in the spermatocyte count[13,14]. It has also been associated with the disposal of dead germinal elements and spermatozoa[15] and cell disintegration[16]. However, we observed a significant fall in acid phosphatase levels of prostate at almost all the doses on treatment with *Vinca rosea* extract. Whether this is due to some metabolic alterations in prostate itself has to be ascertained.

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2 N. Radhakrishnan and M. Alam, Indian J. exp. Biol. *13*, 70 (1975).
3 A.O. Prakash, R.B. Gupta and R. Mathur, Probe *17*, 315 (1978).
4 I.S. Johnson, J.G. Armstrong, M. Gorman and J.P. Burnett, Cancer Res. *23*, 1390 (1963).
5 M.S. Joshi and R.Y. Ambaye, Indian J. exp. Biol. *6*, 256 (1968).
6 T.V. Purandare, S.R. Munshi, S. Jayaraman and X.R. Swami in: Abstracts of Symp. Gen. comp. Endocr. p. 139. Endocr. Soc. India, Dharwar.
7 C.H. Fiske and Y. Subba Row, J. biol. Chem. *66*, 375 (1925).
8 J.M. Allen and J.J. Slater, Anat. Rec. *129*, 255 (1957).
9 B.L. Lobel and F. Levey, Acta Endocr. *59*, 1 (1968).
10 J.P. Arzae, Excerpta Med. *7*, 226 (1954).
11 L.J. Linnetz and R.P.J. Amann, J. Reprod. Fert. *16*, 343 (1968).
12 M.O. Pulkkinen and K. Willman, Acta. obstet. gynec. scand. *46*, 525 (1967).
13 J.S.H. Eckington, A.W. Blackshaw and B. Dejong, Aust. J. biol. Sci. *26*, 491 (1972).
14 A.W. Blackshaw, J. Reprod. Fert. 18, suppl. 77 (1973).
15 L. Nicander, J. Ultrastruct. Res. *14*. 424 (1966).
16 De Duve, in: Lysosomes in Biology and Pathology. Ed. J.T. Dingle and H.B. Fell. Elsevier, New York 1969.

Water regulation in Barmer goat of the Rajasthan desert

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Summary. The different body fluid compartments in normally watered and 4-day water-deprived goats of Rajasthan desert, India, were measured in autumn. The goats maintained plasma volume and extracellular fluid volume, but lost gut and cell water considerably under the experimental conditions; indicating that the maintenance of the fluidity of the blood has priority over the body's other fluid requirements in this desert-adapted species during water deprivation.

Maintenance of homeostasis is of prime importance for survival in desert-dwelling mammals. The goat occupies a dominant position in all desert biomes. It is only recently, however, that some quantified information on the desert goat's physiological characteristics have been recorded[2-4]. The Rajasthan desert goat has been reported to be a particularly hardy animal[4]. A measure of this animal's ability to maintain its various body fluid compartments under conditions of water stress has been made and the results are reported in this note.

5 3-year-old castrated desert goats of similar body weight were used in this study which was conducted during September-October, 1978 in Jodhpur (26°05'N; 73°01'E) in the Rajasthan desert. Throughout the experimental period, the animals were housed individually in metabolic cages kept inside a well ventilated hall. The environmental conditions prevailing inside the hall in the immediate vicinity of the animals were recorded daily at 7.00 h and at 15.00 h. For the experimental period, the average daily mean maximum and minimum temperatures and the relative humidity of the air were 35.16±0.38 °C, 23.06±0.38 °C and 60.90±0.40% respectively.

The experimental feed, given adlibitum, comprised a 50:50 mixture of *Cenchrus ciliaris* (winter cut) hay and

Body fluid compartments in normally watered and water deprived Barmer goat

Characters	Before water restriction mean±SE	Day 4 of water restriction mean±SE*	Change of complete water restriction from adlibitum value (%)	Average amount of water lost	Percent of the total body water lost
Body weight (kg)	40.10±0.75	35.30±1.08	−11.97	-	-
Total body water (l)	24.46±1.53	18.21±1.09	−25.55	6.24±2.11	-
Plasma volume (l)	1.47±0.03	1.28±0.05	−12.92	0.19±0.07	3.04
Blood volume (l)	2.03±0.07	1.93±0.06	− 4.92	0.14±0.05	2.24
Extracellular fluid volume (l) (thiocyanate space)	11.49±0.22	10.62±0.32	− 7.57	0.90±0.28	14.42
Cell and gut water (l)**	12.96±1.52	7.59±1.03	−41.43	5.58±1.80	89.42
Interstitial fluid volume (l)	10.02±0.19	9.33±0.31	− 6.88	0.71±0.27	11.37

* Significantly different from control animals ($p < 0.05$). ** Cell and gut water = total body water-thiocyanate space.

Zizyphus nummularia leaves (average dry matter 97%). Initially, the animals were allowed free access to water for a week. The volume of urine, voided by each animal and collected under liquid paraffin, was measured at 24-h intervals during this pre-experimental period. The different body fluid compartments (plasma volume, blood volume, extracellular fluid volume and total body water) of the animals, under normal and water deprived conditions, were measured (table). Plasma volume was obtained from the dilution of Evans blue (T1824) in plasma[5]. The total blood volume was calculated from the plasma volume and the packed cell volume. Extracellular fluid volume (ECF) (Thiocyanate space) was determined by Bowler's[6] method. The total body water (urea space) was calculated from the dilution of urea in the plasma[7]. The intracellular fluid volume was obtained by subtracting ECF from total body water. The interstitial fluid volume was obtained by difference from ECF and plasma volumes.

A 43% cell and gut water loss accounted for nearly 90% of the total loss in body water brought about by water restriction. Apparently, these animals tend to maintain the fluidity of the blood at the cost of intracellular and gut water when faced with acute water stress. This may be a part of the desert goat's strategy for adaptation which is akin to that in the camel[8], but which appears somewhat different from that apparently operating in the Merino[9] and the Marwari sheep[10]. There was only a minor reduction in plasma volume in the water-deprived goat while a 40–50% reduction in this parameter has been reported in sheep[9,10] of different breeds under more or less similar environmental conditions. Interestingly, the camel reportedly loses less than 10% of its normal plasma volume at a body weight loss of 20% due to dehydration[11]. Under approximately similar environmental and experimental conditions, plasma water loss in the Rajasthan desert sheep[10] has been reported to constitute about 8% of the total body water loss, i.e. 3 times more than in the desert goat, as shown in table. Thus, the desert goat's physiological ability to maintain normal haemodynamic conditions during acute water stress would give it a greater chance of survival than the sheep during prolonged drought spells. This finds corroboration in the almost 70% increase in the goat population of the Rajasthan desert during the drought-stricken decade 1961–71, when the sheep population registered a mere 18.5% increase[12].

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2 A. Shkolnik, A. Borut and J. Choshniak, Symp. Zool. Soc. Lond. *31*, 229 (1972).
3 G.M.O. Maloiy and C.R. Taylor, J. Agric. sci. *77*, 203 (1971).
4 M.S. Khan, T.O. Sasidharan and P.K. Ghosh, J. Arid Envir. *1*, 351 (1978).
5 P.S. Hawk, B.L. Oser and W.H. Summerson, in: Practical Physiological Chemistry. 14th ed. Blakiston, New York 1965.
6 R.G. Bowler, Biochem. J. *38*, 385 (1944).
7 S. Natelson, in: Microtechniques of Clinical Chemistry for the Routine Laboratory, p.381. C.C. Thomas, Springfield, Illinois 1957.
8 W.V. Macfarlane, R.J.H. Morris and B. Howard, Nature *197*, 271 (1963).
9 W.V. Macfarlane, R.J.H. Morris, B. Howard, J. McDonald and O.E. Budtz-Olsen, Aust. J. agric. Res. *12*, 889 (1961).
10 G.R. Purohit, P.K. Ghosh and G.C. Taneja, Aust. J. agric. Res. *23*, 685 (1972).
11 K. Schmidt-Nielsen, in: Desert animals: Physiological problems of heat and water. Clarendon, Oxford 1964.
12 Statistical Abstract, Rajasthan (1961, 1972). Directorate of Economics & Statistics, Rajasthan, Jaipur.

Inhibition of hepatic Na^+, K^+-adenosinetriphosphatase in taurolithocholate-induced cholestasis in the rat[1]

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Summary. Na^+, K^+-adenosinetriphosphatase (Na^+, K^+-ATPase) activity was decreased in liver plasma membranes from rats in which cholestasis had been induced by i.v. administration of sodium taurolithocholate (5 µmoles/100 g b. wt). Incubation of liver plasma membranes with taurolithocholate (10–1300 µM) caused significant and dose dependent reductions of Na^+, K^+-ATPase activity at taurolithocholate concentrations above 100 µM. These findings lend support to the hypothesis that cholestasis induced by monohydroxy bile acids is at least partially the result of an inhibition of hepatic Na^+, K^+-ATPase activity.

The potential role of monohydroxy bile acids in various cholestatic conditions has attracted increasing interest in recent years[3–7]. Taurolithocholate-induced cholestasis has been shown to be a useful experimental model for studies of the mechanism by which monohydroxy bile acids induce cholestasis[8–13]. Besides precipitation of taurolithocholate within the bile canaliculi[9], alteration of canalicular membrane function[11,13,14], and increased biliary membrane permeability to solutes[12], inhibition of bile acid-independent bile formation has been suggested as cause of taurolithocholate-induced cholestasis[10]. This component of bile flow appears to be related to the activity of Na^+, K^+-adenosinetriphosphatase (Na^+, K^+-ATPase) in liver plasma membranes[15–19]. The present study was conducted to investigate the in vivo and in vitro effects of taurolithocholate on the activity of this enzyme in rat liver plasma membranes.

Methods. Male Sprague-Dawley rats (Tierfarm Hartmut and Vos, Tuttlingen, FRG) weighing 278 ± 19 g were maintained on a standard rat diet (Altromin 300R, Altromin GmbH, Lage, FRG) and tap water ad libitum. In the in vivo experiments, the animals were anesthetized with i.p. sodium pentobarbital (5 mg/100 g b. wt) and the common bile duct was cannulated with PE 10 tubing. Body temperature was monitored with a rectal thermometer and maintained at 37.5 ± 0.5 °C. Groups of 5 animals each received 5 µmoles/100 g b. wt of sodium taurolithocholate (Calbiochem, San Diego, California, USA) or the solvent (0.15 M NaCl containing 10% w/v bovine serum albumin) into the inferior vena cava. This dose of taurolithocholate has been shown to produce cholestasis within 20 min after i.v. injection[3]. Bile was collected for a period of 20 min before and after the administration of the bile acid solution or the solvent. Immediately after completion of bile collection, heparin (250 IU/100 g b. wt) was injected into the inferior vena cava and the animal was sacrificed by severing the thoracic aorta and vena cava. The liver was perfused with