successively washed with water, ethanol and ether and was finally dried in vacuo at 50°C over P_2O_5 for 10 hr. The exchange capacity was determined by passing aqueous 1 M sodium chloride solution (100 ml) through the resin (0.3 g) in a column. The amount of sulphinate in the eluent was titrated with 0.01 N hydrochloric acid using methly orange as indicator.

A typical procedure for synthesis of methyl phenyl sulphone: Amberlite IRA 400 *p*-toluenesulphinate form 5 g; (capacity 1 mmol sulphinate anion/g of dry resin was stirred with alkyl halide (4.9 mmol) in refluxing benzene (15 ml) for 6 hr. The resin was then filtered off, washed with dichloromethane and the solvent was removed in vacuo. Distillation of the crude product gives the sulphone.

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1. Bauser, H., *J. Am. Chem. Soc.*, 1939, **61,** 617.
2. Suter, C. H., *The organic chemistry of sulphur*, John Wiley, New York, 1948, p. 667.
3. Meek, J. S. and Fowler, J. S., *J. Org. Chem.*, 1968, **33,** 3422.
4. Veenstra, G. E. and Zwanenbury, B., *Synthesis*, 1975, p. 519.
5. Sande, A. R., Jagdale, M. H., Mane, R. B. and Salunkhe, M. M., *Tetrahedron Lett.*, 1984, 3501.
6. Deshmukh, J. G., Jagdale, M. H., Mane, R. B. and Salunkhe, M. M., *Synthetic Commun.*, 1986, **16,** 479.
7. Deshmukh, J. G., Jagdale, M. H., Mane, R. B. and Salunkhe, M. M., *Chem. Ind.*, 1986, 179.
8. Field, L. and Clark, R. D., *J. Org. Chem.*, 1957, **22,** 1129.

GLYCOGEN METABOLISM IN LIVER IN PERCHLORATE-TREATED RATS

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PERCHLORATE is one of the toxic effluents of Space Research Centre. Perchlorate as potassium or ammonium salts caused decreased food intake and loss of weights in rats[1-3]. It was observed in our laboratory[4] that the blood glucose level decreased significantly in perchlorate-treated rats when compared to control rats. It was also observed that the specific activities of aldolase and lactate dehydrogenase were increased whereas glucose-6-phosphatase activity decreased in perchlorate-treated rats[4]. Subsequent to these findings it is proposed to study the glycogen metabolism in liver of rats treated with perchlorate. The muscle glycogen synthetase and muscle phosphorylase have also been estimated, both in the control and in the perchlorate-treated rats to find out the alteration if any in these parameters.

Weanling male albino rats derived from the Wistar strain were purchased from Veterinary College, Madras. Rabbit liver glycogen, phosphoenolpyrruvate, uridine 5'diphosphoglucose, uridine 5'diphosphate were obtained from Sigma Chemical Company, St. Louis, MO, USA, Glucose-1-phosphate and glucose-6-phosphate were purchased from centre for biochemicals (CSIR), V. P. Chest Institute, Delhi. Bovine serum albumin was the product of Fluka Buchs, SG, Switzerland, Cysteine hydrochloride, dinitrophenyl hydrazine, ethylene diamine tetra acetic acid were obtained from British Drug House, Poole, England. All other chemicals used were of analytical grade. Pyruvate kinase used in the present investigation was prepared from rabbit muscle according to the method of Davidson[5].

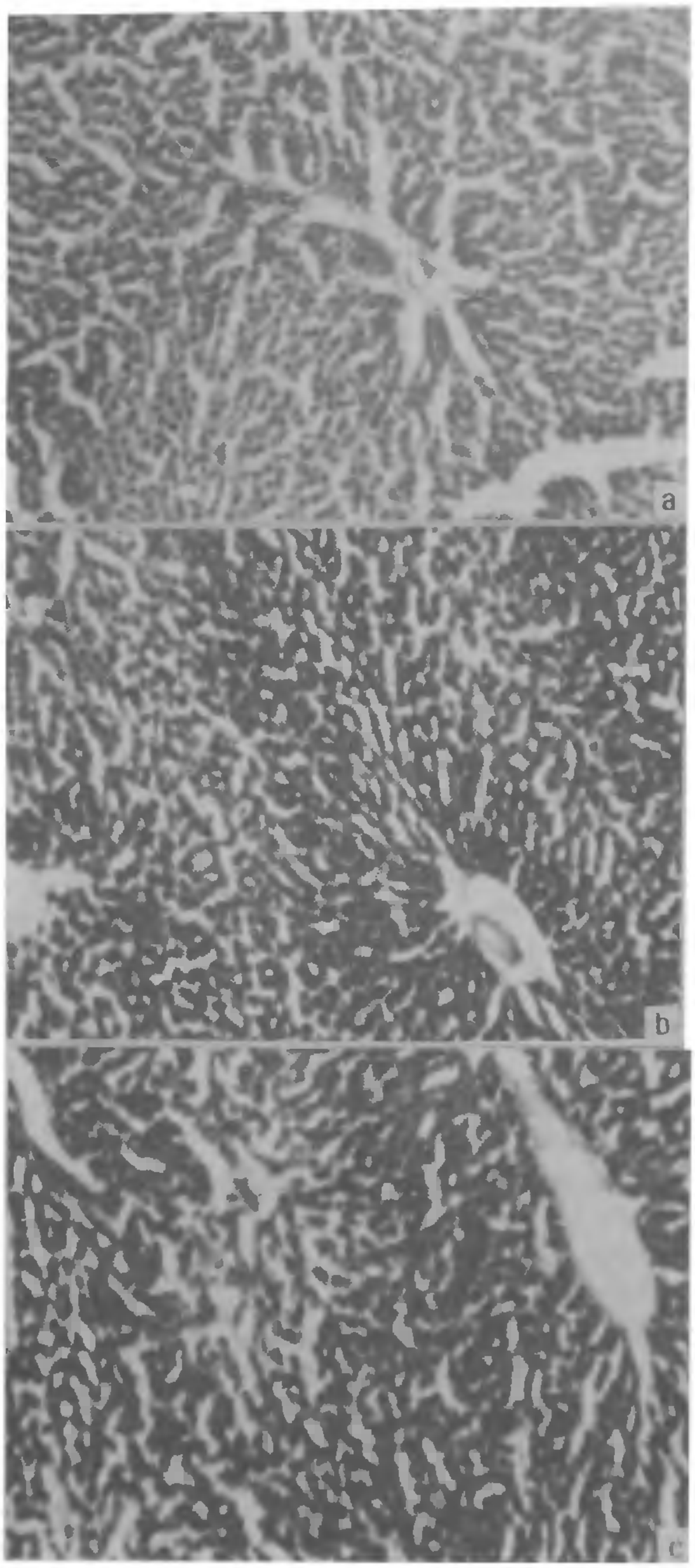
The animals were fed with commercial rat feed with paired feeding and water *ad libitum* along with an oral administration of a chronic dosage (500 mg/kg body weight/day) of potassium or ammonium perchlorate for 45 days. The dosage was selected based on the report of Spreca *et al* [6]. The animals were then sacrificed by stunning and decapitating. The liver, kidney and muscle were dissected out immediately and a portion of the liver was kept in Rossman's fluid[7] for histochemical studies of glycogen with periodic acid Schiff's reagent[8]. Another portion of the liver and the kidney were used for the estimation of glycogen according to the method of Morales *et al* [9]. The rest of the liver and a portion of muscle were washed with ice cold saline and appropriate amounts of the tissues were homogenized in 0.1 M Tris-HCl buffer pH 7.4. The homogenates were centrifuged at 2500 rpm for 10 minutes at 4°C. The supernatants were used to

assay the enzymes. Glycogen synthetase and glycogen phosphorylase were assayed according to the methods of Leloir and Goldemberg[10] and Cornblath[11] respectively. The method of Lowry *et al*[12] was adopted for the estimation of protein.

Glycogen contents of liver and kidney of control and perchlorate-treated rats are given in table 1. A significant increase in glycogen content is observed in the liver and kidney of perchlorate-treated rats when compared to that of the control. Liver sections of control, potassium perchlorate-treated and ammonium perchlorate-treated rats stained with periodic acid for glycogen are shown in figure 1. A moderate amount of glycogen was observed in the control liver cells whereas a heavy accumulation of glycogen throughout the liver cells of perchlorate-treated rats was observed.

Table 2 shows the activity of glycogen synthetase and phosphorylase in liver and muscle of the control and perchlorate-treated rats. A significant increase ($P < 0.05$) in glycogen synthetase activity and a significant decrease ($P < 0.001$) in glycogen phosphorylase activity were observed in the liver and muscle of perchlorate-treated rats when compared to control rats.

Perchlorate was reported to increase the glycogen content in heart and thigh muscles in rats[2]. The activity of glycogen phosphorylase in liver is the major factor that controls glycogen metabolism in liver[13]. In the present investigations the observed rise in glycogen content and decrease in liver glycogen phosphorylase activity in perchlorate-treated rats are similar to these observations. The reduced activity of liver phosphorylase indicates the decreased formation of glucose-6-phosphate from glycogen. The three main pathways for the metabolism of glucose-6-phosphate are (i) conversion to glucose by phosphatase, (ii) isomerization to fructose-6-phosphate in the process of glycolysis, and



Figures 1a–c. Liver tissue sections stained for glycogen. **a.** Control; **b.** Potassium perchlorate-treated, and **c.** Ammonium perchlorate-treated rats.

Table 1 *Liver and kidney glycogen contents in control and perchlorate-treated rats. The values are expressed as mean ± SD for six animals in each group (mg/gm of wet tissue)*

Tissues	Control	$KClO_4$ treated	NH_4ClO_4 treated
Liver	36.0 ± 3.21	60.8 ± 5.0*	61.20 ± 4.8*
Kidney	3.6 ± 0.4	5.1 ± 0.8*	4.9 ± 0.7**

* $P < 0.001$; ** $P < 0.01$.

(iii) conversion to glycogen. Increased activities of enzymes in glycolysis[4] and increased activity of liver glycogen synthetase in the present studies suggest the utilization of glucose-6-phosphate in the two pathways. However, the decrease in the activity of glucose-6-phosphatase in liver reduces the amount

Table 2 *Activities of glycogen synthetase and phosphorylase in control and perchlorate-treated rats. Glycogen synthetase activity is expressed as nanomoles of UDP formed/mg of protein, glycogen phosphorylase is expressed as nanomoles of Pi liberated/mg of protein under the conditions of incubation. The values are given as mean ± SD for six animals in each group*

Tissues	Enzymes	Control	$KClO_4$ treated	NH_4ClO_4 treated
Liver	Glycogen synthetase	410 ± 40	481 ± 36*	493 ± 45*
	Glycogen phosphorylase	350 ± 25	210 ± 7**	177 ± 18.6**
Muscle	Glycogen synthetase	621 ± 37	696 ± 49*	687 ± 50*
	Glycogen phosphorylase	500 ± 60	290 ± 43**	306 ± 46**

* $P < 0.05$; ** $P < 0.001$.

of glucose formed from glucose-6-phosphate and hence the lowering of the blood glucose level as *reported earlier*[4].

Hepatic glycogen accumulation is due to the decrease in the activity of glycogen phosphorylase along with the increase in the activity of glycogen synthetase in perchlorate *administered rats.*

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1. Makarevich-Galperin, L. M., Ushenko, S. N. and Breslowskii, R., *Ukrain Biokhimzhur.*, 1958, **30**, 678.
2. Makarevich-Galperin, L. M. and Ushenko, S. N., *Patol Fiziol ieksptl Terapiya.*, 1959, **3**, 63.
3. Gauss, W. and Rrosk, Z. M., *Anat. Forsch.*, 1972, **85**, 469.
4. Sangan, P. and Motlag, D. B., *Curr. Sci.* (In press).
5. Davidson, E. A., *Biochem. Biophys. Acta*, 1959, **33**, 238.
6. Spreca, A., Laszlo, M. and Musy, J. P., *Pharm. Acta Helv.*, 1973, **48**, 297.
7. Smitherman, M. L., Lazarow, A. and Sorensen, R. L., *J. Histochem. Cytochem.*, 1972, **20**, 463.
8. Bedi, K. S. and Horobin, R. W., *Histochemistry*, 1976, **48**, 153.
9. Morales, M. A., Jabbagy, A. J. and Terensi, N. P., *Neurospora Newslett.*, 1973, **20**, 24.
10. Leloir, L. F. and Goldenberg, S. H., *Methods Enzymol.*, 1962, **5**, 145.
11. Cornblath, M., Randle, P. J., Parmeggiani, A. and Morgan, M. E., *J. Biol. Chem.*, 1963, **238**, 1592.
12. Lowry, O. H., Rosenbrough, N. J., Farr, A. L. and Randall, R. J., *J. Biol. Chem.*, 1951, **193**, 265.
13. Hers, H. G., *Annu. Rev. Biochem.*, 1976, **45**, 167.

FIRST REPORT OF CONODONTS FROM THE INFRA-TRAPPEAN LIMESTONES AT DUDDUKURU, ANDHRA PRADESH, INDIA

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THE light yellowish limestones outcropping south of Devarapalli, Duddukuru and Pangidi (figure 1) in coastal Andhra Pradesh, west of the Godavari River, have attracted the attention of several workers[1-10], for their fossil content, which formed here the basis of bio-stratigraphic correlation. The stratigraphic succession is as follows:

Rajahmundry Sandstones	:	Miocene
Deccan Traps	}	Cretaceous-Eocene
Inter-trappean limestones		
Deccan Traps		
Unconformity		
Infra-trappean limestones		
Unconformity	:	Lower Cretaceous
Tirupati Sandstones		(Gondwana)