the findings of Sydow (1969). The activity of pyruvate kinase, the other glycolytic enzyme studied, changed very little during the period investigated. Glucose 6-phosphate dehydrogenase activity fell slightly after birth to a minimum at 20 days of age and then rose gradually to reach adult values at 50 days of age. Isocitrate dehydrogenase and 'malic' enzyme exhibited similar developmental patterns, the activities of both enzymes rising steadily throughout postnatal development. The activity of kidney 'malic' enzyme was 50% higher in female rats than in male rats.

Extramitochondrial aconitate hydratase activity increased after birth, reaching adult values by 20 days of age. ATP citrate lyase activity, which was very low throughout development, fell after birth to a minimum at 20 days of age and then rose steadily to adult values. [U-¹⁴C]Acetate incorporation into lipid rose after birth to a maximum at 20 days of age and then fell to a minimum at 50 days of age, thereafter rising to adult values. Fructose 1,6-diphosphatase activity rose after birth, reaching adult values at about 30 days of age, thus exhibiting a similar developmental pattern to that of glucose 6-phosphatase and gluconeogenesis reported by Zorzoli *et al.* (1969).

We thank the Medical Research Council for grants to C. A. H. and E. A. L.

Greengard, O. (1971) *Essays Biochem.* 7, 159–205

Sydow, G. (1969) *Hoppe-Seyler's Z. Physiol. Chem.* 350, 263–268

Zorzoli, A., Turkenkopf, I. J. & Mueller, V. L. (1969) *Biochem. J.* 111, 181–185

Effects of Protein Deficiency on Uridine Diphosphate Glucuronyltransferase Activity and Phospholipid Composition of Rat Liver Microsomal Fraction

By ALLAN B. GRAHAM, GEOFFREY C. WOOD and BARRY G. WOODCOCK (*Drug Metabolism Research Unit, Department of Pharmaceutical Chemistry, University of Strathclyde, Glasgow G1 1XW, U.K.*)

Protein deficiency markedly increases the activity of rat liver microsomal UDP-glucuronyltransferase with *o*-aminophenol or *p*-nitrophenol as acceptor (Wood & Woodcock, 1970; Woodcock & Wood, 1971). Since the enzyme's activity is affected by treatment of microsomal preparations with agents perturbing the phospholipids of the microsomal membrane (Graham & Wood, 1969; Attwood *et al.*, 1971; Hänninen & Puukka, 1971), it was suggested (Woodcock & Wood, 1971) that protein deficiency might affect enzyme activity by altering the structure of the microsomal membrane. We have therefore determined the phospholipid composition of liver microsomal preparations from normal and protein-deficient rats.

Male Wistar rats were force-fed on a protein-free purified diet for 5–7 days. In agreement with our previous work, the activity of microsomal UDP-glucuronyltransferase in livers from these animals was substantially higher than in those from control animals fed on the same diet supplemented with 18% casein (protein-deficient, 148.7 ± 13.4 nmol of glucuronide formed/h per mg of microsomal protein; controls, 63.2 ± 6.8).

Although the weights of microsomal protein and phospholipid per g of liver were both 30% lower in protein-deficient animals than in control animals, there was no significant difference in the ratio of phospholipid to protein between the groups (protein-deficient, 1.07 ± 0.03 g/g; controls, 1.00 ± 0.01 g/g).

The microsomal phospholipid compositions were similar in the two groups, but there were significant differences in the content of phosphatidylinositol (protein-deficient, $6.1 \pm 0.5\%$ of total phospholipid; controls $8.7 \pm 0.3\%$) and sphingomyelin (protein-deficient, $7.0 \pm 0.6\%$; controls, $4.2 \pm 0.3\%$). Moreover, lysophosphatidylcholine ($7.4 \pm 0.5\%$) and lysophosphatidylethanolamine ($3.1 \pm 0.9\%$) were found with protein-deficient rats whereas no lysophosphatides were detected with control animals.

Hänninen & Puukka (1971) report that addition of lysophosphatides to rat liver microsomal fractions or their treatment with phospholipase A increases UDP-glucuronyltransferase activity. Treatment of our control microsomal preparations with phospholipase A showed that a degree of phospholipid hydrolysis similar to that observed in preparations from protein-deficient rats increased enzyme activity 2–3-fold.

We therefore suggest that the elevated enzyme activity in microsomal fractions from protein-deficient rats is probably due to the presence of lysophosphatides. The involvement of other factors such as the alterations of phosphatidylinositol and sphingomyelin contents cannot, however, be excluded.

We gratefully acknowledge support for this work from the Medical Research Council and the Science Research Council.

Attwood, D., Graham, A. B. & Wood, G. C. (1971) *Biochem. J.* 123, 875–882

Graham, A. B. & Wood, G. C. (1969) *Biochem. Biophys. Res. Commun.* 37, 567–575

Hänninen, O. & Puukka, R. (1971) *Chem.-Biol. Interact.* 3, 282–284

Wood, G. C. & Woodcock, B. G. (1970) *J. Pharm. Pharmacol.* 22, 60S–63S

Woodcock, B. G. & Wood, G. C. (1971) *Biochem. Pharmacol.* 20, 2703–2713