value as authentic 2-deoxy-2-sulphoamino-D-glucose. The electrophoretic mobility of this ^{35}S -labelled product also coincided with that of 2-deoxy-2-sulphoamino-D-glucose.

The enzymically prepared heparan [^{35}S]sulphate was degraded with nitrous acid (Lagunoff & Warren, 1962) and the products were fractionated on a Sephadex G-15 column. Approx. 75% of the radioactivity was eluted with inorganic sulphate. These results suggest that predominantly N-sulphate groups are synthesized in the presence of the ox lung enzyme.

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Investigation of the Metabolic Oxidation of Acenaphthen-1-ol

By C. J. SIMMONS, R. P. HOPKINS and P. CALLAGHAN. (*Department of Biochemistry, St Thomas's Hospital Medical School, London S.E.1, U.K.*)

After the administration of acenaphthene, acenaphthylene and *cis*- and *trans*-acenaphthene-1,2-diol to rats, 1,8-naphthalic acid was isolated, as its anhydride, from the acidified urine of the dosed animals (Chang & Young, 1943; Hopkins, Brooks & Young, 1962; Hopkins & Young, 1966). Following the suggestion of Hopkins (1968) that oxidation of the acenaphthene-1,2-diols to 1,8-naphthalic acid might involve an initial dehydrogenation step, acenaphthenequinone was isolated from a microsomal incubation of the acenaphthene-1,2-diols in the presence of NAD^+ (Drummond & Hopkins, 1969). Drummond, Callaghan & Hopkins (1971) have demonstrated that there are at least two systems in rat liver that dehydrogenate acenaphthene-1,2-diol, one in the microsomal fraction and another in the soluble fraction. These findings and the work of Sims (1966), on the structurally related 3-methylcholanthrene, led us to investigate the metabolism of acenaphthen-1-ol in order to determine whether it proceeds by a pathway involving an initial dehydrogenation reaction.

Rats were injected subcutaneously with acenaphthen-1-ol and the urine was acidified to pH 2 and extracted with ether. Examination of the extract by t.l.c. indicated the presence of acenaphthen-1-one and 1,8-naphthalic anhydride, which were isolated and characterized. 1,8-Naphthalic anhydride was also isolated and characterized from urine obtained from rats that had been dosed with

acenaphthen-1-one. Hydrolysis of the extracted urines with 6M-hydrochloric acid or β -glucuronidase gave rise to further quantities of acenaphthen-1-one and 1,8-naphthalic anhydride.

The oxidation of acenaphthen-1-ol was also investigated by incubation at 37°C with a rat liver microsomal preparation and NAD^+ at pH 7.4. Ether extracts of the incubation system, after acidification to pH 2, were examined by t.l.c. and found to contain acenaphthen-1-one, which was isolated and characterized. This reaction was followed in a similar incubation system by measuring the increase in E_{340} due to accumulation of acenaphthen-1-one. The microsomal dehydrogenase system was found to use either NAD^+ or NADP^+ as cofactor.

It was also found that a rat liver microsomal fraction incubated at 37°C with an NADPH-generating system at pH 7.4 converted acenaphthen-1-one into acenaphthenequinone, which was identified by t.l.c. and g.l.c. of ether extracts of the incubation medium after acidification to pH 2. Acenaphthenequinone was isolated and characterized.

These observations will be discussed and a metabolic sequence suggested for the conversion of acenaphthen-1-ol into 1,8-naphthalic acid.

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Investigation on the Effects of Cigarette Smoke on Rabbit Alveolar Macrophages

By G. M. POWELL* and G. M. GREEN. (*Department of Medicine, University of Vermont, Burlington, Vt. 05401, U.S.A.*)

Alveolar macrophages occupy a functional position of primary importance in pulmonary clearance, since they are capable of the uptake, transport and elimination of particles, micro-organisms and fluids through the processes of phagocytosis, pinocytosis and digestion. Green & Carolin (1967) have shown that the presence of the filtered gas phase of cigarette smoke has a very marked inhibitory effect with respect to the phagocytic function of rabbit alveolar macrophages. Green (1968) has also shown that the cells could be protected from the effects of

* Present address: Department of Biochemistry, University College, P.O. Box 78, Cardiff CF1 1XL, U.K.