in bile (up to 58% of the dose) was diethylstilboestrol monosulphate monoglucuronide, and only traces (up to 2%) of parent ester were detected. Between 20 and 30% of the injected radioactivity was present in the urine after 6h as inorganic [^{35}S]sulphate.

Experiments with isolated perfused rat livers identified the liver as the major site of metabolism of both diethylstilboestrol disulphate and diethylstilboestrol monosulphate. Other experiments *in vitro* showed that both esters are desulphated by microsomal sulphohydrolase activity. Thus, in spite of the apparent suitability of diethylstilboestrol disulphate for elimination via the biliary route, this does not occur to any significant extent, perhaps owing to the activity of the desulphating system of the liver. Although diethylstilboestrol monosulphate is desulphated to some extent in the liver it would appear that the availability of a free phenolic hydroxyl group, for conjugation with glucuronic acid, allows the preferential formation of a double conjugate. The considerable biliary excretion of radioactivity after the administration of diethylstilboestrol [^{35}S]monosulphate is therefore explicable in these terms.

Although both diethylstilboestrol monosulphate and diethylstilboestrol disulphate possess physicochemical properties that, on theoretical grounds, render them suitable for biliary elimination, only traces of unchanged parent ester appear in the bile in either case.

P. A. G. is grateful to the Medical Research Council for a Research Studentship.

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Mercapturic Acid Formation from Sodium Alkyl Sulphates in the Rat

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The injection of sodium benzyl sulphate and sodium 1-menaphthyl sulphate (sodium naphth-1-ylmethyl sulphate) into rats is followed by the excretion of benzylmercapturic acid and 1-menaphthylmercapturic acid respectively in the urine of the dosed animals (Clapp & Young, 1970). Similarly, sodium allyl sulphate gives rise to the excretion of allylmercapturic acid (Kaye & Young, 1970). In the present work the possibility that sodium

alkyl sulphates also give rise to formation of mercapturic acids in the rat has been investigated.

The acid sulphate esters of the alcohols in the series methanol to decan-1-ol were prepared by the action of concentrated sulphuric acid on the alcohol at -5°C (Lloyd, Tudball & Dodgson, 1961) and were separated as their sodium salts. These, and also sodium *n*-dodecyl sulphate, were injected subcutaneously into male rats, and urine extracts were examined chromatographically for the corresponding alkylmercapturic acids. No such metabolites were detected for sodium methyl sulphate and sodium ethyl sulphate, whereas sodium *n*-propyl sulphate appeared to give rise to traces of *n*-propylmercapturic acid. Significant amounts of *n*-butyl-, *n*-pentyl- and *n*-hexyl-mercapturic acid were detected as metabolites of the corresponding sulphates, but sodium *n*-heptyl sulphate gave rise to only a very small amount of *n*-heptylmercapturic acid. Very little of the corresponding alkylmercapturic acid was detected after administration of sodium *n*-octyl, *n*-nonyl, *n*-decyl and *n*-dodecyl sulphate.

After these preliminary studies, *n*-butylmercapturic acid, as the free acid and as the dicyclohexylammonium salt, was isolated from the urine of rats dosed with sodium *n*-butyl sulphate. The amount isolated corresponded to 0.8% of the administered compound. After injection of sodium *n*-pentyl sulphate and sodium *n*-hexyl sulphate into rats, *n*-pentyl- and *n*-hexyl-mercapturic acid were isolated from the urine in amounts corresponding to 1.0% and 1.1% respectively of the sulphate administered. No mercapturic acid was detected in the urine after the administration of butan-1-ol, pentan-1-ol or hexan-1-ol to rats, and it appears unlikely that these compounds are intermediates in the formation of mercapturic acids from their acid sulphate esters.

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Maternal-Foetal Relationship of Methionine and Cystine in the Pregnant Rhesus Monkey

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We have found that, in the pregnant human, cystine behaves like most plasma amino acids: the maternal plasma concentration decreased during