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.

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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

pregnancy, and the foetal plasma concentration is considerably higher than the maternal. In contrast, the pregnant rhesus monkey was found to increase its plasma concentration of cystine from 4.7 ± 0.5 to $8.1 \pm 0.5 \mu\text{mol}/100\text{ml}$; the foetal plasma cystine concentration was intermediate.

Four pregnant rhesus monkeys were injected with 5mCi (0.25mmol) of L-[^{35}S]methionine or L-[^{35}S]cystine into the cubital veins of the mothers or into the umbilical veins for the foetuses, under Sernylan anaesthesia, and blood samples were withdrawn from indwelling catheters in the femoral veins of mother and foetus at 0, 15, 30, 45 and 60 min after injection. Amino acids and radioactive compounds were measured with an automatic amino acid analyser coupled with a flow-cell scintillation counter (Sturman, Cohen & Gaull, 1970).

When methionine was injected into the mother, it was accumulated by the foetus, which maintained its usual threefold higher concentration. The specific radioactivity of methionine in foetal plasma exceeded that in maternal plasma for much of the experiment. When methionine was injected into the foetus, the concentration of methionine in foetal plasma, and its specific radioactivity, remained higher than in maternal plasma.

When cystine was injected, in each case the circulation injected had the highest concentrations and highest specific radioactivities of cystine for the duration of the experiments, the concentration of cystine in the secondary circulation being increased relatively little.

These results indicate that the foetus can quickly accumulate methionine from the maternal circulation against a concentration gradient, as it can for other amino acids; however, it cannot similarly accumulate cystine from the maternal circulation. A load of methionine or cystine in the foetal circulation was dissipated into the maternal circulation very slowly. This inability of the foetal placenta to concentrate cystine against a gradient may account for the anomalous behaviour of cystine during pregnancy in the rhesus monkey.

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Peptides from Human Blood-Group-Specific Glycoproteins

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Human blood-group-specific glycoproteins isolated from ovarian-cyst fluids were subjected to degradation with 0.25M-sulphuric acid in acetic acid (Donald, Creeth, Morgan & Watkins, 1969). The products were considerably enriched in peptide compared with the original glycoprotein, and the amino acid composition was largely unchanged. Most of the remaining sugar was *N*-acetylgalactosamine, but small amounts of galactose and *N*-acetylglucosamine were also present even after 24 h treatment. Periodate oxidation, followed by reduction with sodium borohydride and acid hydrolysis of the resulting polyalcohols (Abdel-Akher, Hamilton, Montgomery & Smith, 1952), was used in attempts to remove the residual carbohydrate. The oxidized and reduced fragment of *N*-acetylgalactosamine glycosidically linked to the hydroxy amino acids in the peptide was resistant to hydrolysis under the mild acid conditions normally employed in the Smith degradation; this fragment was liberated by treatment with 0.2M-hydrochloric acid for 1.5 h at 100°C. After three cycles of oxidation, reduction and hydrolysis, small yields of non-diffusible peptide were obtained that were free from sugar. The amino acid compositions of these materials were closely similar to that of the parent glycoproteins, but paper electrophoresis at pH 1.9 revealed them to be mixtures of many different peptides.

The peptides from the degradation products of a blood-group A substance (no. 401) were separated by column chromatography on Sephadex G-100, G-50 and G-25 and by preparative electrophoresis at pH 1.9. Nineteen peptides, judged to be pure on the basis of (1) their electrophoretic behaviour, (2) the molar proportions of the component amino acids and (3) the fact that they gave only one *N*-terminal acid by the dansyl procedure (Gray, 1967), were isolated in sufficient yield for partial sequence analysis by the dansyl-Edman method (Gray, 1967). From their behaviour on Sephadex columns and their amino acid composition these peptides contained 10–20 amino acid residues. Four peptides, differing in overall composition, had the *N*-terminal sequence Ala-Gly-Ser, three had Ala-Gly-Gly, two had Asp-Gly-Gly, two had Ser-Gly-Gly and two had Thr-Pro-Gly. The sequences of the other six peptides were Gly-Ala-Gly, Gly-Gly-Ser, Asp-Asp-Gly, Glu-Gly-Ala, Ala-Gly-Ala and Gly-Pro-Ala-Gly. Three peptides obtained from a blood-group B glycoprotein (no. 531) had the