shown to be present in the urine of rats injected with *S*-allylglutathione and *S*-allyl-L-cysteine. The animals dosed with allyl chloride also excreted 3-hydroxypropylmercapturic acid.

The reactions of a number of foreign compounds with glutathione are known to be catalysed by glutathione *S*-transferases. These include glutathione *S*-alkyltransferases (Johnson, 1966) and glutathione *S*-alkenyltransferases (Boyland & Chasseaud, 1967, 1968), which catalyse the reaction of glutathione with alkyl compounds and with $\alpha\beta$ -unsaturated compounds respectively. The observations made in the present work on the metabolism of allyl compounds were considered in the light of knowledge concerning the glutathione *S*-transferases.

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The Effect of Phenobarbitone on the Incorporation of δ -Aminolaevulinic Acid into Rat Liver Microsomes

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Phenobarbitone given in the drinking water causes an increase in the microsomal drug-metabolizing enzyme system including cytochrome *P*-450. Values of cytochrome *P*-450 content rise from 20 ± 3 nmoles/g. in controls to 135 ± 3 nmoles/g. in treated rats. This high value is maintained as long as phenobarbitone is given (Marshall & McLean, 1969). The increased content of cytochrome *P*-450 could be due to increased synthesis (Conney & Gilman, 1963), decreased breakdown (Holtzmann & Gillette, 1968), or a combination of both. In order to study the synthesis and breakdown of microsomal haem, we have given ^{14}C -labelled δ -aminolaevulinic acid (δ -ALA) and measured radioactivity in various liver components as a percentage of the dose given.

Two groups of 150–200g. male rats, one group

having been pretreated with oral phenobarbitone (1mg./ml.) for 20 days, were given $2\mu\text{C}$ of [^{14}C] δ -ALA (specific radioactivity 19mc/m-mole)/100g. by intraperitoneal injection. Rats were killed 4, 8, 24 and 48hr. after injection, and liver microsomes were prepared. Haem was extracted from the microsomal suspension with acid-acetone and its specific radioactivity found.

Of the administered dose of δ -ALA, 16% was incorporated into the livers of both groups by 4hr. After 48hr. the activity had fallen to 6% and 8% for control and phenobarbitone-pretreated groups respectively. In the phenobarbitone-pretreated group, essentially all the activity was found to be in the microsomal haem. In the control group, however, the radioactivity in the microsomes after 4hr. was 10%, and only half of this was ^{14}C -labelled haem.

Regression analysis of the fall in radioactivity of microsomal haem from 8 to 48hr. gave a half-life of microsomal haem in control animals of 42hr., compared with 45hr. in phenobarbitone-pretreated rats. The contribution to the total microsomal haem radioactivity by cytochrome *b*₅ was not significant. There does not appear to be any gross difference in the rate of breakdown of microsomal haem between control and phenobarbitone-pretreated groups. This suggests that the inductive effect of phenobarbitone is not mediated by a decrease in the rate of breakdown.

This work has been supported by Grants from the British Empire Cancer Campaign and the U.S. Public Health Service.

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Destruction of Reduced Nicotinamide-Adenine Dinucleotide Phosphate by Bromotrichloromethane and by Carbon Tetrachloride *in vitro* and *in vivo*

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The administration of CCl_4 to rats results in severe centrilobular necrosis and fatty degeneration of the liver. Numerous biochemical changes occur during the first 1hr. of poisoning (for references see Slater, 1966), and some of these are related to the development of necrosis (Slater & Sawyer, 1969). One such early change is the decrease in total liver