

Male albino rats (200 to 220 g) were exposed 4 h to CS<sub>2</sub> (2.0 mg/l.) and urine collected in the subsequent 17 h. The animals were then decapitated and the liver taken for histology.

Urine samples were analysed for bivalent sulphur by the catalytic iodine azide reaction which is used in industry to detect excessive exposure (Djuric, Surducki & Berkes, 1965). The net reaction on which the method is based is  $I_2 + 2 N_3^- \rightarrow 3 N_2 + 2 I^-$ . The spectrophotometric method of Strickland, Mack & Childs (1960) was adapted to the bivalent sulphur metabolite(s) of CS<sub>2</sub>. The calibration curve was prepared with diethyldithiocarbamate, the reaction product of CS<sub>2</sub> with diethylamine.

Rats were exposed to CS<sub>2</sub> after feeding or after a 24 h fast with or without treatment by intraperitoneal injection of 80 mg/kg or 50 mg/kg phenobarbitone given 24 and 18 h respectively before exposure.

In rats fasted for 24 h before exposure to CS<sub>2</sub> the excretion of bivalent sulphur due to CS<sub>2</sub> exposure fell from 65  $\mu$ mol/kg found in fed rats to 44  $\mu$ mol/kg whether or not there had been pretreatment with phenobarbitone. However, in almost all fasted animals pretreated with phenobarbitone the same degree of liver damage was seen as that observed by Bond, Butler, DeMatteis & Barnes (1969), who gave 1.0 ml/kg CS<sub>2</sub> *per os* to fasted phenobarbitone-treated animals. This oral dose was approximately 15 times more than the calculated (72 mg/kg = 0.95 mmol/kg) CS<sub>2</sub> retained by the rats during the 4 h exposure to CS<sub>2</sub> vapour. Feeding before exposure decreased or completely prevented liver necrosis.

The observations that phenobarbitone had no effect on the LD50 of CS<sub>2</sub> (Bond *et al.*, 1969) and failed to affect the excretion of the CS<sub>2</sub> metabolite, which accounts for approximately 6% of the body burden, suggests that phenobarbitone may not interfere with the metabolism of CS<sub>2</sub>. As CS<sub>2</sub> inhibits at least some of the enzymes which are induced by phenobarbitone (Bond & DeMatteis, 1969) and the extent of microsomal changes depends on the activity of the drug metabolizing enzymes at the time when CS<sub>2</sub> is administered (Bond & DeMatteis, 1969), it is possible that some imbalance in the catabolic and anabolic processes induced by phenobarbitone and intensified by fasting renders the liver cells more sensitive to CS<sub>2</sub>.

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#### Effects of pargyline on body temperature and on hypothalamic levels of monoamines in the rabbit (T)

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#### Effect of amylobarbitone, dextro-amphetamine and a mixture of these on performance and learning in man (T)

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