60% was absorbed within 90 min. Any unabsorbed ethionine was later excreted in the feces. During the passage through the gastrointestinal tract, a portion of ethionine was metabolized. The fate of absorbed ethionine was investigated in the small intestine, liver, blood, kidney, and urine as a function of time after application. A great part of ethionine was quickly oxidized to ethionine sulfoxide. In liver and kidney, the concentration of ethionine sulfoxide was higher than that of free ethionine. In all organs, the presence of *N*-acetylethionine sulfoxide was also demonstrated. Ethionine sulfoxide can be reduced, and *N*-acetylethionine can be deacetylated *in vivo* as demonstrated by the formation of *S*-adenosylethionine from ethionine sulfoxide and *N*-acetylethionine. In urine, four

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6X (3686) MUTAGENICITY *IN VITRO* AND POTENTIAL CARCINOGENICITY OF CHLORINATED ETHYLENES AS A FUNCTION OF METABOLIC OXIRANE FORMATION. (Eng.) Greim, H. (Dept. Toxicology of Gesellschaft fur Strahlenund Umweltforschung, 8042 Munchen-Neuherberg, West Germany); Bonse, G.; Radwan, Z.; Reichert, D.; Henschler, D. *Biochem. Pharmacol.* 24(21): 2013-2017; 1975.

The mutagenicity of six chlorinated ethylenes formed during microsomal activation was tested in a metabolizing *in vitro* system with *Escherichia coli* K_{12}. Mutation rates were determined for three back mutation systems gal+, arg+, and nad+, and for the MTR system where forward mutation leads to resistance to 5-methyl-DL-tryptophan. Cells were incubated

(3687-3690)

① K-2521 ② K-2520 ③ K-2197 ④ K-9388
⑤ K-9387 ⑥ K-1711

for two hours with microsomal protein from mouse liver, an NADPH-generating system with 5 μM $MgCl_2$, and various concentrations of the test compounds: tetrachloroethylene, trichloroethylene (TRI), 1,1-dichloroethylene (1,1-DCE), cis-1,2-dichloroethylene, trans-1,2-dichloroethylene, and vinyl chloride (VCM). Only those compounds (VCM, 1,1-DCE, and TRI) that form very unstable oxiranes induced mutations in the test system. The highest mutation rates were detected in the arg+ system; the other systems were less sensitive to the mutagenic effects of the metabolites. The mutagenicity of TRI was unexpected, and further study of the mutagenic and potentially carcinogenic properties of this compound are recommended.

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55(1):35-36; 1975.

Five groups of 68 male and 68 female Charles River rats, approximately five weeks old, were fed ethylene thiourea (ETU) for two years at levels of 0, 5, 25, 125, 250 or 500 ppm in the diet. Body weights, the weights of the thyroid and other organs, thyroidal uptake of ^{131}I, hematology, and histology were the criteria studied. ETU was found to be a thyroid carcinogen for the rat at the 250 and 500 ppm dietary levels and a thyroid tumorigen at the 125 ppm level; it caused a slight thyroid hyperplasia at the 5 and 25 ppm feeding levels. Thyroid hyperplasia was not noticeably reversible in those rats in each group that were changed to control diet after 66 wk on ETU diets. ETU ingestion at the 5 and 25 ppm levels was not biologically deleterious to the rat. The data support the view that ETU-induced thyroid neoplasia in the rat are the result of excessive pharmacological stimulation, i.e., they occur because of excessive endocrine organ stimulation and not because of the reaction of one molecule of carcinogen with one cell to initiate neoplasia.

The growth effects
human prolactin on
tissue from 30 wom
used at 5 μg/ml.
chicine (0.1 μg/ml
to each culture, a
were counted in hi
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little mitosis or
hormones *in vitro*.
activity of cultur
of human prolactin
tivity over that o
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crease in the insu
results suggest th
breast tissue can
further studies to
of prolactin *in vi*
man prolactin.