reaching a maximum within 24 h. The latter time course was coincident with that of the repair process of UV-damaged DNA, as judged from unscheduled DNA synthesis.

1202 BOLT, H. M.*, H. KAPPUS, A. BUCHTER and W. BOLT. (Inst. Toxikol., Univ. Wilhelmstr. 56, D-7400 Tuebingen, W. Ger.) **Disposition of [1,2-^{14}C] vinyl chloride in the rat.** ARCH TOXICOL 35(3): 153-162. 1976. [In Engl. with Engl. and Ger. summ.]—Rats were exposed to the carcinogen [1,2-^{14}C] vinyl chloride in a closed system at initial concentrations below 100 ppm. When the system was occupied by 3 rats, a half-life of vinyl chloride in the system's atmosphere of 1.13 ± 0.12 h was observed. The volume of the system was 10.3 l. Calculation of the clearance of vinyl chloride from the system revealed that about 40% of inspired vinyl chloride is absorbed by lung. Changes in respiration did not influence uptake of vinyl chloride. Uptake of vinyl chloride by the rats was completely blocked by acute pretreatment with potent inhibitors of cytochrome P-450 dependent microsomal drug metabolism (i.e., 35 mg/kg 3-bromophenyl-4(5)-imidazole or 50 mg/kg 6-nitro-1,2,3-benzothiadiazole in 0.6 ml/kg DMSO [dimethylsulfoxide]). A weaker inhibition was observed after SKF 525 A [2-dimethylaminoethyl-2,2-diphenyl valerate] or 5,6-dimethyl-1,2,3-benzothiadiazole (50 mg/kg in 0.6 ml/kg DMSO). Metyrapone did not cause inhibition. Uptake of vinyl chloride was increased by pretreatment with DDT and, to a lesser extent, clotrimazol. No significant stimulation of uptake was observed after pretreatment with phenobarbital, 3-methylcholanthrene, rifampicin or chronic ethanol treatment. Immediately after exposure, highest radioactivity levels were observed in liver and kidney. The radioactive metabolites of ^{14}C-vinyl chloride were rapidly excreted, largely by the kidneys. Excretion of radioactivity in the urine was 69.4 ± 2.6% within 24 h.

BIOSIS ABSTRACTS
February 1977
Volume 6 No. 2

...were injected s.c. with a range of doses of N-ethyl-N-nitrosourea (ENU). A high proportion of treated mice developed tumors, particularly hepatomas, pulmonary adenomas and carcinomas and malignant lymphomas of thymus and spleen. Liver tumors occurred most frequently in C57BL and DBAf mice, lung tumors in A mice and lymphomas in A and DBAf mice. A small proportion of C57BL, DBAf and IF mice developed tumors of the nervous system. The results are discussed with reference to the ready induction of nervous system tumors in similarly treated rats, and their relevance for human cancer.

1204 WONG, JEFFREY J. and DENNIS P. H. HSIEH. (Dep. Environ. Toxicol., Univ. Calif., Davis, Calif. 95616, USA.) Mutagenicity of aflatoxins