human urine, and to a lesser extent by AFB and its primary metabolites. The results suggest that carcinogen-albumin adducts formed after carcinogen exposure can act as an immunogen.

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SUSCEPTIBILITY TO LUNG CANCER

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Lung cancer is the most prevalent form of cancer in the industrialised world, and smoking of tobacco products is considered the single most important etiological factor. One of the plausible host factors is the genes controlling both the oxidative metabolism and deactivation of the tobacco carcinogens. A wide inter-individual variation in the primary metabolism of tobacco associated carcinogens, e.g. benzo(a)pyrene, in the target tissue has been reported. Glutathione transferases are involved in the secondary metabolism, and the genes expressing the isozyme(s) is another host genetic determinant. The initial damage introduced in cellular DNA by the ultimate form of the tobacco smoke carcinogens can be repaired by DNA repair enzymes. One of these, O-6 alkyltransferase, shows a wide inter-individual variation in human bronchial epithelial cells.

Other acquired or inherited diseases do influence an individual's risk of developing lung cancer, i.e. sarcoidosis and scar (tuberculosis).

THE EFFECT OF PHENOLIC ANTIOXIDANT, OCTYL GALLATE ON THE BINDING OF BENZO(A)PYRENE METABOLITES TO NUCLEAR DNA

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The effect of intraperitoneal (ip) administration of octyl gallate on the formation of benzo(a)pyrene (BP) metabolites-DNA adducts in vitro in rat liver, kidney and lung nuclei was investigated. Male Wistar rats received 2 ip injections per week for 2 weeks. Nuclei were incubated with [^{3}H]BP and DNA was purified and enzymically hydrolysed to deoxyribonucleosides. The BP-DNA adducts were resolved by HPLC. In the nuclei of all tissues the nature of adducts was identical but the level of BP binding was different - the highest in the liver and the lowest in the lung nuclei. The major adducts identified were the BP-4,5-oxide-DNA and the BP-trans-7,8-diol-9,10-epoxide deoxyguanosine adduct. Treatment of animals with octyl gallate decreased the total binding level of BP to DNA of the liver nuclei and the formation of all adducts by 40%. In the kidney nuclei the total binding level and formation of adducts were slightly elevated and in the lung nuclei unchanged. These results indicate that octyl gallate may play a certain role in the inhibition of BP-induced carcinogenesis but this effect is tissue specific.

EFFECTS OF ASCORBIC ACID (AA) ON GENOTOXIC ACTIVITY OF CHEMICAL CARCINOGENS

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The influence of AA on mutagenic and carcinogenic activity of benzo(a)pyrene (BP), tobacco smoke (TS), urethane (U) and diethylnitrosamine (DEN) were investigated in vitro and in vivo.

AA added to the top agar (0.2 to 2.0mg/plate) suppressed in about 50% the mutagenic activity of BP in S. typhimurium TA98 but not in TA100 and failed to influence the mutagenic effect of TS. AA (0.3%, 1.0%, 1.5%, with the drinking water) depressed the clastogenic activity of BP (2.0mg/mouse) and TS without influencing the effect of U (1.0g/kg). When applied after the U administration but not given together with the carcinogen, AA inhibited the lung carcinogenesis in mice. An inhibition of liver carcinogenesis was also established in rats treated with DEN (80mg/kg) and AA.

COMBINATION TREATMENT OF 4-EPI-DOXORUBICIN AND RADIATION ON HAMSTER LUNG CELLS

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The purpose of this study was directed to test the type and the degree of cytotoxic effects of epirubicin and radiation in combined treatment on Chinese hamster lung cells in vitro. Experiments were performed with proliferating tissue culture cells. Cell killing was determined by colony-forming ability. The maximum killing effects were obtained when simultaneous action of drug treatment and irradiation occurred. Their interaction was synergistic. Synergism depended on time of drug incubation (epirubicin present for 1