PROLACTIN IN SERUM IN PATIENTS WITH PULMONARY TUMOURS
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The serum prolactin level was radioimmunologically measured in 103 male patients (average age 47.9 ± 12.5 years) with different pulmonary diseases; 28 of these patients suffered from histologically proven, untreated bronchogenic carcinoma. 13 healthy persons served as controls. In 20 patients with bronchogenic carcinoma, we found a significantly reduced mean prolactin level (4.51 ± 2.5 ng/ml; n=20) compared to the other patients with non malignant pulmonary disorders (8.5 ± 2.7 ng/ml; n=75) ($p<0.001$) and to healthy controls (8.47 ± 2.66 ng/ml; n=13). Additionally, we performed TRH-stimulation tests in 8 patients with histologically proven untreated bronchogenic carcinoma in which 6 patients showed a decreased basis prolactin level of 4.23 ± 0.93 ng/ml; 2 patients, on the other hand, had an increased basis prolactin level. The results of these TRH-Tests were considered normal, suggesting hypothalamic control. The cause of the decrease of basis prolactin concentration in lung carcinoma is not known; perhaps the tumour cells produce an inhibitor which hinders the release of prolactin from the front lobe of the hypophysis.

MONITORING OF WORKERS IN INDUSTRIES FOR EXPOSURE TO CARCINOGENS
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Occupational exposure to chemical carcinogens may be monitored by measuring the chemical interaction which occurs between the exposing agent and biological macromolecules. Such interaction is detectable in the body fluids as adducts of, for example, DNA or glutathione. The mutagenic compounds or their metabolites can also be measured from urine samples, using bacterial mutagenicity assays. At present, cytogenetic approaches appear to be nearest to routine surveillance in detecting early biological effects in exposed humans. Studies on structural chromosome aberrations or sister chromatid exchanges (SCEs) in peripheral blood lymphocytes obviously detect different molecular injuries, and the results from in vivo occupational exposures do not necessarily correlate. Somatic chromosome damage should always be considered a warning sign of potentially adverse effects, and such damage should lead to decreased exposure to the causative agent.

EFFECT OF DIETARY BUTYLATED HYDROXYANISOLE ON THE HEPATIC MONOOXYGENASE SYSTEM OF NUCLEAR AND MICROSOMAL FRACTIONS IN MICE. E.E.Hennig, K.Demkowicz-Dobrzański, J.T.Sawicki and H.Mojska. Department of Environmental Health Science, Warsaw, Poland.

The effect of butylated hydroxyanisole (BHA) administration on the hepatic monooxygenase system of nuclear and microsomal fractions was investigated in male mice. Addition of BHA to the diet significantly lowered the content of cytochrome P-450 in liver nuclei and increased the specific activity of NADPH-cytochrome c reductase and the content of cytochrome b_5 in liver microsomes. Incubation of benzo(a)pyrene (BP) with liver nuclei from BHA fed mice resulted in inhibition of binding of BP metabolites to nuclear macromolecules by 50% compared to the control. However, there was no effect on BHA on the binding of BP metabolites to macromolecules when BP was incubated with added DNA and liver microsomes from BHA-fed mice. It has been postulated that modification of nuclear monooxygenases by BHA may play a role in the inhibitory effect of BHA on BP carcinogenesis.