BIOCHEMICAL VARIATIONS IN CHEMICALLY INDUCED CIRRHOSIS FOLLOWING ADMINISTRATION OF NEW HEPATOPROTECTORS. C.Nistor[1], N.Mihail[2], Klara Makkay[3] and A.Cacoveanu[1]. [1]Oncological Institute, Cluj Napoca, Romania. [2]Centre for Biological Research, Cluj Napoca, Romania. [3]Faculty of Chemistry, Cluj Napoca, Romania.

In order to investigate the hepatoprotective effects of newly synthesised compounds, we determined the dynamic variations in several biochemical parameters following the administration of these compounds. These were as follows: the enzymes E.C. 3.1.3.1, E.C. 3.1.1.8, E.C. 2.6.1.1 and E.C. 2.6.1.2, bilirubin, total protein, calcium, phosphorus, magnesium, iron, copper, nitrogen and glucose in the liver and serum of white Wistar rats with chemically-induced cirrhosis. Animals were then treated with derivatives of choline hepatoprotectives. After administration of the choline derivatives, we observed the tendency of these biochemical parameters, which are modified in the course of cirrhosis (especially the enzyme and bilirubin levels), to return to normal values. All parameters normalized after 2-3 weeks of treatment.

GENETIC PREDISPOSITION TO LUNG CANCER. D.Nowak[1], P.Amstad[2], P.Cerutti[2], K.Hartmann[1] and H.W.Rüdiger[1]. [1]Unit of Hereditary and Constitutional Diseases, Department of Internal Medicine, University of Hamburg, F.R.G. [2]Swiss Institute for Experimental Cancer Research, Department of Carcinogenesis, Epalinges/Lausanne, Switzerland.

Epidemiologic findings have revealed that beside environmental factors genetic influences account for an enhanced lung cancer risk in individuals. A genetic predisposition might well act on the level of enzymatic activation or inactivation of carcinogenic compounds. We examined this hypothesis with blood monocytes and skin fibroblasts of lung cancer patients using benzo(a)pyrene as a model compound for tumour initiation by covalent binding to DNA: in patients we found an increased conversion to water-soluble products as well as an increase of DNA-bound material, as compared to normals. We have controlled these results by a separate evaluation of selected lung cancer patients which exhibit one of the following: positive family history of lung cancer, onset of disease below 45 years of age, negative history of smoking, multiple primaries. Again blood monocytes and fibrolasts in culture were evaluated from each patient and from 15 normal controls, including HPLC analysis of benzo(a) pyrene-DNA adducts.

CANCER INCIDENCE AMONG EMPLOYEES AT A MINERAL WOOL PRODUCTION PLANT IN DENMARK.
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Today the carcinogenic activity of asbestos fibres is beyond dispute. This fact has called attention to the possibility that a similar relationship between man-made mineral fibres and cancer may exist. The initiating hypothesis of this study has been that exposure to respirable fibres from mineral wool in concentrations seen around the production lines in the plant under study is carcinogenic to the respiratory system. The factory involved started the production of mineral wood in 1937. All persons employed from this date were followed-up in the Danish Cancer Registry, which started working on a nationwide scale in 1943. The endpoint of the investigation period was 1977. A comparison on the observed numbers of cancer cases was made with the expected numbers calculated on the basis of the age, sex and time specific cancer incidence rates for the Danish population. Within the study period 136 persons have been notified with one cancer and 4 persons with two primary cancers, i.e. a total of 144 cancer cases observed against 133.8 expected. Among workers with 20 or more years from first employment in the plant a significant excess of cancer cases in the lungs, bladder and skin were found (O/E = 9/4.3, 4/1.6 and 5/2.4 respectively). This study thus supports the working hypothesis of an association between lung cancer and mineral wool production, when considering the latency period for this type of cancer