for 30 days. In these conditions, PBL proliferated but did not develop cytotoxic activity against Auto-Me and K562 cells. The phenotype of PBL at the 30th day was 95% CD3, 95% CD4, 1% CD8 and 0% CD16. Clones were then derived at 1 cell/well in the presence of irradiated Auto-Me, RIL-2 (25 U/ml) and Daudi cells as feeder. The 81 clones were screened growing for cytotoxicity and proliferating activity in the presence of Auto-Me. Twelve clones were cytotoxic for Auto-Me and 22 clones showed significant proliferation with Auto-Me; 67 clones exhibited both cytotoxic and proliferating activity. Preliminary results of the specificity analysis showed that one clone which proliferated to Auto-Me expressed cytotoxicity on Auto-Me but not on 12 different targets including autologous EBV-B cells and fibroblasts, 6 allogeneic melanomas, 2 lymphoblastoid lines, Daudi and

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BIOCHEMICAL EVALUATION OF HYPERCALCAEMIA ASSOCIATED WITH BRONCHOGENIC CANCER

G.Francini and C.Gennari

Institute of Medical Semeiotics, Division of Clinical Oncology, University of Siena, P.zza Duomo, 2-53100 Siena, Italy

Bronchogenic carcinoma (BC) may be frequently associated with hypercalcaemia, due to direct bone resorption by tumour cells, or alternatively by osteoclastic bone resorption stimulated by humoral factors (i.e. parathyroid hormone (PTH)-like substances and prostaglandins (PGE)).

In 160 patients with BC, hypercalcaemia was found in 19 cases (11.8%). By measuring the biological parameters of PTH activity and the circulating levels of PTH and PGE the hypercalcaemic patients were divided into three groups. The first group (n=6) presented the typical biochemical pattern of hyperparathyroidism, the second group (n=10) was characterized by high circulating levels of PGE; in the third group (n=3) all the parameters considered were normal. A metastatic bone involvement as evidenced by radiologic and scintigraphic means, was documented only in patients of the first and second group. These data further emphasize the importance of humoral factors in the pathogenesis of hypercalcaemia associated with BC.

NUCLEIC ACID BINDING OF TRANS-4-AMINOSTILBENE DERIVATIVES IN VITRO AND IN VIVO

R.Franz and H.-G.Neumann

Institute of Toxicology, University of Würzburg, Versbacherstr. 9, Würzburg, F.R.G.

Trans-4-acetylaminostilbene (AAS) is a strong tumour initiator in rat liver. The model ultimate carcinogen, N-acetoxy-AAS, reacts with nucleic acids in vitro and predominantly with guanine. The major adducts are four cyclic isomers in which guanine is substituted at N2 and N3 as shown by reactions with Guo and d-Guo. addition, two minor guanine adduct fractions were identified and shown to consist also of sets of isomers. After oral administration of trans-4-dimethylaminostilbene (DAS) and AAS, the cyclic adducts and presumably also the minor adducts are formed in rat liver DNA. Substitution of guanine at N2 and N3 labilizes the glycosidic bond, which results in depurinization of DNA, and may impair nuclease activity, which could explain the observed incomplete hydrolysis of modified DNA. Experiments with AAS labelled in the acetyl group indicate that non-acetylated adducts are also generated in liver RNA and DNA to some extent. Among these non-acetylated adducts the cyclic guanine adducts are also present. A number of persistent adducts could be demonstrated 28 days after oral administration of DAS.

CORRELATION OF ELASTOSIS WITH SOME MORPHOPATHOLOGICAL PROGNOSTIC FACTORS IN EREAST CARCINOMAS

V.Gădăleanu, A.Ursan and Milena Duma

Pathology Department, Oncological Institute Cluj-Napoca, Romania

The increase of elastic tissue was found on haematoxylin-eosin stained tumour sections in 54 (22%) from 245 invasive ductal carcinomas of the breast treated by radical mastectomy, as a pink, homogeneous or finely fibrillar sheaths around carcinomatous ducts and focal deposits in contact with tumour parenchyma. elastosis was assessed subjectively in three degrees and analysed in relation to the tumour size, histological pattern, grade of malignancy, lymph node metastases and age of patients. The percentage of elastin positive tumours increased in parallel with their histological differentiation from trabecular carcinomas to pure glandular carcinomas. Elastosis correlated to some extent with the grade of malignancy - a higher proportion of elastin positive tumours was found in low grade group and conversely, a higher proportion of cases without elastosis in high grade tumours. No relation was established between elastosis