

ELEVATION OF CARBOXYPEPTIDASE N IN LUNG CANCER

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Serum carboxypeptidase N (3.4.17.3) was measured spectrophotometrically in 60 patients (untreated and treated) with histologically confirmed lung cancer. Hippuryl-L-arginine was used as enzyme substrate for carboxypeptidase N₁ (CN₁), hippuryl-L-lysine for carboxypeptidase N₂ (CN₂). The results were: squamous cell (untreated): CN₁ 37.4 ± 7.9 (± SD) U/ml, CN₂ 184.0 ± 34.8 (n = 10), treated by radiotherapy: CN₁ 61.9 ± 28.0, CN₂ 185.0 ± 47.4 (n = 10); oat cell (untreated): CN₁ 45.0 ± 26.7, CN₂ 168.7 ± 55.7 (n = 10), treated by chemotherapy (cisplatin, adriamycin, vindesine, VP-16): CN₁ 49.0 ± 36.9, CN₂ 171.9 ± 53.5 (n = 18); bronchioloalveolar: CN₁ 52.8 ± 3.8, CN₂ 181.5 ± 6.6 (n = 4); adeno: CN₁ 52.0 ± 29.0, CN₂ 192.4 ± 38.6 (n = 4); non classified 92.6 ± 58.8, CN₂ 209.5 ± 62.9 (n = 4) carcinomas. After treatment CN₁ and CN₂ slightly increased. Compared to 51 healthy controls CN₂ was significantly elevated (p<0.001), but CN₁ was not. In contrast to the reduced activity of the angiotensin converting enzyme, CN₂ is elevated in all types of lung cancer. Our results suggest that CN₂ could be used as a marker for lung cancer.

Supported by DFG (Schw 209/2-1).

BIOCHEMICAL AND MOLECULAR EPIDEMIOLOGY OF CANCER. A.Shamsuddin¹, H.Autrup, K.Vahakangas, N.Sinopoli, D.Mann and C.Harris. ¹Department of Pathology, University of Maryland School of Medicine, Baltimore, MD, USA; Laboratory of Human Carcinogenesis, National Cancer Institute, Bethesda, MD, USA.

The primary goal of biochemical and molecular epidemiology is to identify individuals at high cancer risk by obtaining evidence of (a) high exposure of target cells to carcinogens and/or (b) increased susceptibility due to inherited or acquired host factors. For example, laboratory methods have been recently developed to measure carcinogen bound to DNA isolated from cells from people exposed to environmental carcinogens. Our strategy uses complementary immunological and biophysical approaches.

IMMUNOHISTOCHEMICAL LOCALIZATION OF 48,000-M_r PLASMINOGEN ACTIVATOR IN MURINE TUMOURS. L.Skriver^{1,3}, L.-I.Larsson², P.Kristensen¹, L.S.Nielsen^{1,3} and K.Dang^{1,3}. ¹Laboratory of Tumor Biology, Institute of Pathology, University of Copenhagen; ²Institute of Medical Biochemistry, University of Aarhus; and ³Finsen Laboratory, Finsen Institute, Denmark.

The localization of a murine 48,000-M_r plasminogen activator (MPA48) in the Lewis lung tumour and a number of other transplantable and spontaneous murine tumours has been studied, using the peroxidase/antiperoxidase staining technique with polyclonal rabbit IgG antibodies against the enzyme. The presence of intense MPA48 immunoreactivity in the tumours was revealed. The specificity of the staining was controlled in a variety of ways, including the demonstration of only one immunoreactive band, corresponding in electrophoretic mobility to MPA48, among extracted tissue proteins separated by SDS-polyacrylamide gel electrophoresis and electrophoretically transferred to nitrocellulose paper. A pronounced variation in staining intensity was observed between different areas of the tumours, indicating cellular heterogeneity with respect to this characteristic. The enzyme was primarily located in areas where extensive degradation of normal tissue was observed. These findings support the hypothesis of a role of this enzyme in tissue degradation in cancer.