PATTERN OF TISSUE HISTAMINE AND HISTAMINE METABOLISM IN SARCOMA Sal828

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Histamine has been implicated in various growth processes. The aim of this study was to determine if the growth of the transplantable methylcholanthrene induced sarcoma, Sal828 in rats is associated with changes in body histamine. Sal828 was induced in both syngenic and allogenic rats by s.c. inoculation of $2x10^6$ tumour cells/animal. Histamine content in the liver, lung, small intestine and tumour tissue which was significantly increased during active tumour growth, and evidently decreased during the phase of tumour regression in allogenic rats and after tumour extirpation in syngenic rats, suggesting the existence of a causal relationship between histamine and tumour. High histamine in the intestine coincided with a marked enhancement of oxidative deamination route. Thus, increased amine content in tissues of tumour-bearing rats is not due to an inhibition of its catabolism but rather to its overproduction.

INFLUENCE OF TPA ON THE RECOVERY OF DRUG RESISTANT COLONIES

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The effect of the tumour promoter TPA on the frequency of mouse and hamster cells resistant to methotrexate (MTX), N-phosphonacetyl-L-aspartate (PALA) and cadmium has been examined. TPA was shown to enhance manifold the recovery of mouse cell clones resistant to all three drugs. Detailed examination of the recovery of MTX-resistant 3T6 mouse cell clones has shown TPA to enhance recovery in both single-step and multi-step selection protocols. However, quantitation of the levels of the dihydrofolate reductase gene product in these clones by dot blot hybridisation and FACS analysis showed that TPA-induced enhancement of MTX-resistant colony recovery was not due to gene amplification in this sytem. Our observations suggest that tumour promoters do not facilitate gene amplification *per se*, but in some way increase the probability of colony formation by pre-existing or newly formed mutants.

PROTEIN PATTERNS OF A METASTATIC AND A RELATED NON-METASTATIC RAT TUMOUR CELL LINE

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We used the lymphatically metastasizing tumour cell line BSp73 ASML (=ASML) and BSp73 AS (=AS) a non-metastatic line, both descendants of the rat adenocarcinoma BSp73 (1). *In vitro*, ASML- and AS-cells display different surface and adhesion characteristics and invasive behaviours that do not correlate with their respective metastatic potentials (2,3). In order to clarify this contradiction we isolated native plasma membranes (PMs) of ASML and AS and analysed the proteins on SDS-gels: there are several quantitative differences but, except for a 42 kD band, no clear qualitative differences could be detected. Cell surface iodination revealed a protein of about 43 kD that is expressed only by ASML-cells. By staining the PM proteins with Ricinus lectin I (RCA I) we found a 30 kD glycoprotein in the ASML-PM that is not visible among AS-PM proteins. This difference cannot be shown with other lectins. Thus, the RCA I binding 30 kD glycoprotein of ASML-cells seems to be of importance and requires further study.

(1) Matzku *et al*, Invas. Metast., 6: 109-123, 1983; (2) Paku *et al*, Anticancer Res., 6: 957-966, 1986; (3) Knierim *et al*, Anticancer Res., 6: 669-682, 1986.

MAC (MORPHOLOGY-ANTIBODY-CHROMOSOME) METHOD IN CYTOGENETIC STUDY OF LYMPHOMAS

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A new MAC (Morphology-Antibody--Chromosome) method, which allows the study of karyotype, surface markers and cell morphology on the same metaphase, has been employed in our laboratory in the study of lymphoproliferative malignancies. The method enables one to distinguish which cells are truly neoplastic and which cells belong to the reactive cells essential for the body's defense system. The usefulness of the new technique has been evaluated with examples of Hodgkin's disease and T and B cell lymphomas.