persisted after fractionation of the crude extracts and revealed two molecular forms of the enzyme activity with a net charge difference in stationary and growing cell cultures. These findings indicate the role of mRNA plyadenylation in cell transitional states and further support its regulatory role in differentiation and transformation.

CHARACTERIZATION OF CHROMOSOME 7 LONG ARM DELETIONS BY DNA PROBES IN MYELODYSPLASTIC SYNDROME

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Partial deletion of the long arm of chromosome 7 is a common abnormality in the bone marrow of patients with myelodysplastic syndrome (MDS) or acute non-lymphocytic leukaemia (ANLL). We have used chromosome 7-specific DNA probes in restriction fragment length polymorphism studies to characterize these deletions in molecular terms. Four patients with a deletion of 7q and MDS or ANLL were studied. Three closely linked loci in band 7q22 (detected by the probes MetH, J3.11, B79a) were deleted in all patients. The proalpha2(I)collagen gene (probe NJ-3, band 7q21-22) was present in the deleted chromosome in one patient. In one patient the deletion was interstitial since the T cell receptor beta chain locus was present in the deleted chromosome. Our results suggest that the proximal breakpoint is in a narrow region in 7q22, that a common region deleted in all patients is 7q22-32, and that the distal breakpoint is variable, at least one deletion being interstitial rather than terminal.

TRANSFORMING GROWTH FACTOR-β: A MAJOR REGULATOR OF CELL GROWTH AND PROTEOLYTIC ACTIVITY

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The effects of TGFβ on the growth and plasminogen activator (PA) activity of normal and malignant human cells were studied in culture. A549 cells are very sensitive to TGFβ growth inhibition. In these cells TGFβ enhanced the mRNA levels and secretion of urokinase (u-PA). Increased proteolytic activity appeared to correlate with TGFβ inhibition of growth in A549 cells. In WI-38 lung fibroblasts the expression of u-PA and t-PA were decreased by TGFβ. Induction of PA-inhibitor-1 and fibronectin were observed in both cell lines. In an NRK cell line exogenous u-PA was able to revert TGFβ inhibition of anchorage-independent growth. Altered responsiveness to TGFβ growth inhibition correlated with decreased ability to produce PAI-1 in transformed NRK cells. Inactive forms of TGF are secreted by most cultured cells, a fraction of which is activatable by plasmin. Local PA-mediated activation of TGF may be a way for the cells to regulate the proteolytic balance in their vicinity. This may offer a physiological feed-back mechanism for TGFβ activation. Divergent effects on normal and malignant cell suggest that TGFβ may participate in the regulation of the invasive, proteolytically active phenotype of cancer cells.

TESTICULAR CANCER - INFLUENCE OF THE MAJOR HISTOCOMPATIBILITY COMPLEX (MHC) ON PURE SEMINOMA

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There is some evidence that genetic factors might play an important role in the pathogenesis of germ-cell tumours. Analyses of the MHC antigens have so far yielded inconsistent results, which might be due to the heterogeneity of the disease. We have therefore conducted a multicenter study on HLA antigens and testicular cancer. 13 HLA-A, 23 HLA-B, 7HLA-C and 10 HLA-DR antigens were tested by the standard microlymphocytotoxicity technique in 44 patients and 257 unrelated healthy controls from the same geographical area. Statistical analysis was carried out using the Chi-squre test with Yates' correction. The data obtained were as follows: in the group of 22 patients with pure seminoma, a statistically significant increase in HLA-B 44 (12) was found (4.3% compared to 21% in the control group, Chi-square 4.11, $p<0.05$). There was a minor, not significant increase in DR 7 (37% compared to 20%), together with a complete absence of DR 1. These preliminary data suggest that susceptibility or resistance to seminoma could be HLA-associated. More prospective studies on HLA antigens and seminoma are needed, which might also yield additional information on correlations with the development of tumour dissemination.