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A cell line derived from a MSV-M induced sarcoma spontaneously lost tubourigenicity after approximately 40 in vitro passages. At the same time these cells began to produce a "factor" inhibiting colony growth in soft agar. This factor is not cell or species-specific because it is active on cells from different tumours, both of human (6/9 lines tested) and murine (6/8 lines) origin. The inhibiting factor is not produced by another cell line (MS-2) derived from the same tumour, which maintains its tumourigenicity. The inhibiting factor has little or no activity on normal cells. The activity is resistant to acid treatment (0.01 N HCl), to heat (4 min at 100° C) and to lyophilization. It is not due to a polyamine and it has no antiviral effect if tested for interferon activities. It inhibits thymidine incorporation, in tumour cells after a treatment of 48 to 72 hr, but it has no activity against DNA or RNA or protein synthesis if tested in cell-free systems. The inhibiting activity appears to be linked to a hydrophilic molecule of low molecular weight.

EXAMINATION OF THE STRUCTURE AND BIOSYNTHESIS OF THE HUMAN PDGF RECEPTOR

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The sequence of a 2.8 kb cDNA clone, corresponding to most of the translated part of the human platelet derived growth factor (PDGF) receptor was determined. The homology between the murine (Yarden et al., Nature 323, 266-232) and human nucleotide sequence is 80 to 85%. The information on the primary structure of the PDGF receptor, deduced from the nucleotide sequence, was correlated with an examination of the biosynthesis and processing of the receptor. It is synthesized as a 145 kD precursor, which carries about ten N-linked oligosaccharide groups, and is chased to a 165 kD molecule within 15 min in the absence of PDGF and even more rapidly in its presence. After additional modifications, for example addition of phosphate, the receptor reaches a final size of 170 to 175 kD.

EWING'S TUMOUR: PHENOTYPIC CHARACTERIZATION AND LONG RANGE MOLECULAR ANALYSIS AROUND THE CHROMOSOMAL BREAKPOINTS

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Ewing cells have been demonstrated to express antigens associated with the neuroectoderm lineage, including the neural cell adhesion molecule NCAM and the receptor for the nerve growth factor. In addition, Ewing and neuroepithelioma cells display the same cytogenetic abnormality, a chromosomal translocation t(11;22) (q23-24;q11-12) which suggests that both tumours are derived from closely related neuroectodermal cells. Several genes could be implicated in the molecular mechanism of malignant transformation. Genes located on chromosome 11 encode NCAM, the delta subunit of the T lymphocyte T3 antigenic complex and Thy-1. In addition, the proto-oncogene c-ets also maps to this chromosomal region. On chromosome 22, the bcr gene maps to band q11. None of these genes was found rearranged when DNA from a variety of Ewing cell-lines was analysed by hybridization of Southern blots obtained by conventional methods. Using recently described techniques and pulse field gel electrophoresis, we have now explored a significant portion of Ewing genomic DNA in the region of the chromosomal breakpoints.

IDENTIFICATION OF A LEUKOCYTE ANTIGEN WITH A HIGH FREQUENCY EXPRESSION IN LEUKAEMIA PATIENTS

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In this report we describe the production and characterisation of a monoclonal antibody to HL-60 cells (a human promyelocytic leukaemia cell line). The antibody, termed NC-2, did not react with any other human cell lines tested. NC-2 precipitated a 50 and 57 kD protein from ^{125}I labelled HL-60 cells. Cell distribution and molecular weight studies indicated that the protein was not an HLA antigen. When NC-2 was screened for reactivity against human peripheral blood cells, 7 individuals from a population of 130 showed a reaction. Blood and bone marrow cells from leukaemia patients (n=50) exhibited a much higher level of reactivity, with cells from 20 individuals showing a reaction with NC-2. In these patients the antigen was expressed on both leukaemic and normal cells. The association of the antigen identified by NC-2 with leukaemia has been evaluated.

ROLE OF CHROMOSOME TRANSLOCATIONS IN HUMAN NEOPLASIA

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