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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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Sequence analysis of the breakpoints involved in the translocations of non-Burkitt B-cell tumours has also provided evidence that in most cases the chromosome translocations occur at the pre-B-cell stage of differentiation during the process of VDJ joining and that the VDJ recombinase is responsible for the translocation by catalyzing the joining of the involved chromosomes. Three observations indicate that this is the case: (1) in the great majority of non-Burkitt lymphomas, the translocation breakpoints involve the 5' region of a J segment; (2) extra nucleotides (N regions) are detected at joining sites in both the t(11;14) and the t(14;18) chromosome translocations; and (3) heptamer and nonamer signal sequences, separated by a spacer of 12 nucleotides, that closely resemble those involved in physiologic VDJ joining, occur on chromosomes 11 and 18 near breakpoints. Thus one can speculate that in a rare B-cell, the recombinase mistakenly joins a heavy chain J segment to a cellular proto-oncogene instead of the proper immunoglobulin gene segment, leading to oncogene deregulation.

INHIBITION OF TUMOUR ANGIOGENESIS AND TUMOUR METASTASIS IN MICE DEFICIENT IN MAST CELLS

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Conflicting reports exist on the role of mast cells in neoplastic disease. We examined the growth, angiogenic response and spontaneous metastasis of B16BL6 melanoma cells, insensitive to *in vitro* killing by murine mast cells, in mast cell deficient W/Wv and control litter-mate mice. We inoculated 10^5 tumour cells subcutaneously into the external ears of 25 W/Wv and 25 control mice. Tumour latent periods, incidence, growth rates and the incidence of spread to draining lymph nodes were the same for both groups of mice. In contrast, the rate of neovascularization was slower, and the incidence and number of spontaneous pulmonary metastases was lower in W/Wv mice than in controls (26.5% and 1.9 *vs* 60.0% and 10). We conclude that host mast cells may facilitate early tumour angiogenesis and haematogenous metastasis. Experiments are in progress to confirm these interpretations using mast cell reconstituted W/Wv mice.

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ELEVATED EXPRESSION OF c-*myc* IN A HUMAN COLON CARCINOMA CELL LINE IS NEITHER ACCOMPANIED BY AMPLIFICATION NOR REARRANGEMENT OF THE GENE

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Over-expression of oncogenes is thought to be correlated to malignant progression of certain types of human tumours. In normal cells, c-*myc* expression is under stringent control, while regulation seems to be altered in a variety of tumours. Investigating a human cell line originating from a colon carcinoma, we found a four-fold elevated c-*myc* expression compared with β-actin. In spite of this, in a sarcoma derived cell line, the expression of both was equal. The over-expression was as high as in the preleukaemic cell line HL60, which is known to over-express c-*myc*. Northern blotting experiments showed in all samples a size of 2.0kb and 4.4kb for mRNA and pre-mRNA, respectively. DNA analysis revealed the absence of gene amplification and rearrangement of the c-*myc* locus. Since the colon cell line contains only one chromosomal translocation we are attempting to correlate the c-*myc* activation with this chromosomal abberration.

CARCINOEMBRYONIC ANTIGEN (CEA) DETERMINATIONS IN COLORECTAL CANCER

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In a programme of screening and follow-up for colorectal cancer (CC) in the city of Dunakeszi, Hungary, in 1983-86, the authors analysed the significance of serial CEA determinations in 68 patients and the findings were compared with those in the scientific literature. CEA levels of more than 30 µg/ml prior to surgery proved to indicate poor prognosis; in these cases, operation revealed advanced stage of disease. During follow-up, the CEA values increased following surgery and reached a level of more than 60 µg/ml in two patients. These patients died within a short time in spite of appropriate treatment. An increasing trend was observed in four patients; on the basis of additional investigations, suitable treatment was performed. In one case recurrence was