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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