

those most likely to survive. Five human cancer cell lines (HT29, EC, GER, GYL, MDA-MB-157) were cultured individually and as a mixture for sixteen weeks without subculture. Conditioned media from these cultures were tested at intervals for their effect on the growth of freshly subcultured aliquots of the same cell lines by Neutral Red assay (Fiennes *et al.*, 1984). Although HT29 and EC (colonic adenocarcinoma) were the slowest growing, they came to dominate the mixed culture. Over time, conditioned medium from this culture inhibited the growth of the remaining three cell types. Conditioned medium from mono-cultures of HT29 and EC had the same effect. These findings are compatible with the activity of a cell-line-specific growth inhibitory factor in the HT29 and EC conditioned media.

ALTERED CELL SURFACE CARBOHYDRATES AND METASTATIC POTENTIAL IN VARIANTS OF B16 MOUSE MELANOMA

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Variants of B16 mouse melanoma selected for wheat germ agglutinin (WGA) resistance show reduced metastatic potential as compared to the wild type F1 cells. The variant cells express a 60 to 70x increase in a specific fucosyltransferase activity, increased fucosylation of cell surface glycoproteins including expression of the SSEA-1 antigen, and (secondarily) decreased sialylation and WGA-binding of the glycoproteins. Variants selected from two WGA-resistant clones by two different lectins show reversion of the fucosyltransferase activity, the glycosylation changes and wheat germ agglutinin resistance to the original F1 phenotype. In order to assess the relationship of the glycosylation change to the metastatic potential, the cell lines were injected intramuscularly into syngeneic mice. Of the seven revertant lines isolated and tested, seven showed an increase in metastatic potential as compared to the WGA-resistant lines. The results suggest a possible relationship between the properties of cell surface carbohydrates and metastatic potential.

EXPERIMENTAL TUMOUR MODELS FOR AN ASSESSMENT OF THE THERAPEUTIC POTENTIAL OF BIOLOGICAL RESPONSE MODIFIERS (BRM)

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Various attempts for the non-cytotoxic treatment of neoplastic disease are based on the concept of stimulation of host biological responses against its own neoplasm. However, there are distinct differences between therapeutic results achieved in animal tumour systems and in clinical trials. These disappointments are suggested to be the result of the disparity between experimental models and clinical neoplasms. To study the role of immunogenicity of a tumour model in respect to "its sensitivity" to BRM, several new transplantable lines have been established from mouse tumours of spontaneous origin suggested to be non-immunogenic: rhabdomyosarcoma and mammary carcinoma of Balb/c mice, mammary adenocarcinoma of DBA/2 mice and two adenocarcinomas of CBA mice. Characterization of these tumours include the following: growth curve *in vivo*, TD50, immunogenicity and the ability to metastasize. The immunotherapeutic potential of BRM has been studied against primary tumours and when applicable against metastases. In general, these tumours do not respond to pustulan (glucan with immunomodulating activity). In some models an inhibition of the primary tumour has been observed but at the same time the number of spontaneous metastases have increased.

MODULATION OF SARCOMA Sa1828 GROWTH IN RATS BY HISTAMINE AND ITS H2 AGONIST AND ANTAGONIST

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This study was conducted in transplantable methylcholanthrene induced sarcoma-bearing rats to determine if tumour growth is affected after the administration of histamine (1mg/kg), dimaprit (H2 agonist, 2mg/kg) or ranitidine (H2 antagonist, 3mg/kg). Sa1828 was induced by s.c. inoculation of 2 million tumour cells/animal. All compounds and 0.9% saline (control) were given by i.p. injection 5 times weekly for 3 weeks. Body weight was monitored regularly. Of the drugs studied, ranitidine prevented the body weight loss associated with tumour growth. Likewise the tumour incidence and mean tumour mass tended to be much lower in the ranitidine treated group. Histaminics and antihistaminic normalized body histamine level and