

- SHA CHANGE IN THE EXPRESSION OF THE C-MYC ONCOGENE DURING CELL TRANSFORMATION
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We have determined the steady-state level of c-myc mRNA during cell transformation in primary rodent cell cultures. We have demonstrated that there is an alteration in the regulation of the expression of this oncogene during cell transformation. By contrast, the expression of B-actin, histone and several other cellular oncogenes is not altered. This work also confirms that alteration in oncogene expression during tumorigenesis depends on the stage that is being investigated.

- SHE REPAIR OF RADIATION AND BLEOMYCIN-INDUCED DNA DAMAGE IN METASTATIC VARIANTS OF THE BL6 MURINE MELANOMA
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The cytotoxic potential of DNA-damaging agents is often reduced because mammalian cells can repair the DNA damage. We have investigated the repair of DNA strand breaks produced in F1 and BL6 variants of the BL6 murine melanoma, upon exposure of the cells to γ -radiation and bleomycin (BLM) and have related it to the sensitivity of the variants to the treatments.

BL6 cells, a high metastasis variant, repaired γ -radiation-induced strand breaks twice as efficiently as the F1 variant cells, which have virtually no metastasizing ability. BLM-induced damage was repaired at similar rates, but BL6 were twice as resistant to BLM as F1 cells. 3-aminobenzamide (3AB), an inhibitor of poly ADP-ribosyl transferase (pADPRT), which is implicated in the repair of single strand breaks, had no effect on strand rejoining in either cell type if 3AB treatment followed exposure to BLM. Pretreatment with 3AB increased DNA repair in BL6 cells but F1 were unaffected. Basal levels of pADPRT activity were similar and were not increased by DNA damage. The data suggest that the metastatic variants differ considerably in their sensitivity to DNA damaging agents and in their ability to repair strand breaks. (This work was supported by the North of England Cancer Research Campaign).

- SIK TUMOUR LOCALIZATION BY HUMAN MONOCLONAL ANTIBODIES
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We have derived sets of human monoclonal antibodies by fusing lymphocytes from cancer patients with a human lymphoid line, LICR-LON/HMy2. Two antibodies, LGL1.1D6 and LLU6.3A4, derived from patients with glioma and bronchial carcinoma respectively, were selected for clinical study on the basis of binding patterns in radioimmunoassays with tumour cell lines and localisation of human tumour xenografts. Highly purified monoclonal antibodies were prepared using bulk supernatants from hybridomas grown in serum free medium. After radiolabelling with ^{131}I , 1 mg antibody was injected intravenously into patients with advanced glioma and carcinoma of the bronchus. Good localisation was obtained in six out of ten patients with glioma and eight out of ten patients with carcinoma of the bronchus. Despite the technical difficulties inherent in the production of human monoclonal antibodies this study demonstrates their potential for the clinical localisation of solid tumours.
