

animal model system with well-established tumour inheritance, such as the hereditary melanoma formation in small tropical fish, platyfish and swordtails. In this system, a 1:1 Mendelian segregation of benign and malignant melanomas is observed. As suggested by Vielkind (1976) and Ahuja, Schwab and Anders (1980), this is due to a single regulatory locus. Studies on the differentiated state of the pigment cells in the two melanoma types show almost completely differentiated cells in the benign and very poorly differentiated cells in the malignant type. We have now mapped this locus, and can show a recessive, perhaps deletogenic, mode of melanoma inheritance. Thus the regulatory locus, termed *Diff*, presumably codes for information necessary in differentiation; it does not influence the severity of the melanoma in a dominant fashion.

[99m]Tc AND [111]In LABELLING OF MONOCLONAL F(ab')<sub>2</sub>-FRAGMENTS AGAINST PROSTATIC ACID PHOSPHATASE FOR RADIOIMAGING OF PROSTATIC CANCER

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Tumour detection by external imaging following administration of radiolabelled monoclonal antibodies specific for tumour-associated antigens has drawn considerable attention in the diagnosis of cancer. Although radioisotopes of iodine have been employed for labelling antibodies, these labels are not stable, and significant deiodination may take place rapidly *in vivo*. An alternate approach to attach [99m]Tc or [111]In to the antibody using the anhydride of DTPA as a bifunctional chelate.

Monoclonal F(ab')<sub>2</sub>-fragments (1 to 10 mg/ml) against prostatic acid phosphatase were derivatized with cDTPAA (molar ratio of cDTPAA/F(ab')<sub>2</sub> = 1:1, 5:1, 10:1, 20:1). The best labelling efficiencies (90 to 95% or 70 to 80%) using [111]In or [99m]Tc were obtained with molar ratio of cDTPAA/F(ab')<sub>2</sub> of 5:1 and with protein concentrations of 10 mg/ml. Under these conditions the antibodies retained their immunoreactivity totally and had no aggregation formation when studied by SDS-PAGE. A successful purification process for [99m]Tc labelled antibodies was developed to increase specific activity of labelled antibody. The radioactive antibody derivatives synthesized revealed metastases of prostatic cancer when used in radioimaging studies.

URACIL-DNA GLYCOSYLASE IN NORMAL AND MALIGNANT HAEMATOPOIESIS

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The activity of uracil-DNA glycosylase, a repair enzyme for the excision of uracil from DNA, was studied systematically in different types of blood and bone marrow cells in normal individuals, in haematological malignancies, and in established leukaemia cell lines. The patients represented a wide range of acute and chronic leukaemias.

The highest uracil-DNA glycosylase activities were found in primitive cells of normal and malignant haematopoiesis, although considerable variation was noted in blastic leukaemias. The expression of uracil-DNA glycosylase gradually diminished towards the more mature cells. This was observed in normal bone marrow, in chronic granulocytic leukaemia, and in TPA-induced malignant histiocytes. Blood lymphocytes in healthy individuals and in chronic lymphoproliferative disorders had stronger uracil-DNA glycosylase expression than the other mature cells, such as erythrocytes, granulocytes, and platelets.

INVESTIGATION OF CALCITONIN (CT) BINDING TO ITS RECEPTORS (CTR) IN MCF-7 HUMAN BREAST CANCER CELL LINE

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The CTR content of 10 breast cancer tissues was measured by a single-point inhibition technique using human [125]-I-CT as labelled ligand and salmon CT (sCT) as inhibitor. Six cases out of 10 proved to be CTR-positive, the CTR content (mean  $\pm$  SD) was 620 $\pm$ 298 femtomol/mg protein and all the normal samples were CTR-negative. In the CTR-positive cancer tissue the cytosol estradiol receptor (ER) level was 154 $\pm$ 90 femtomol/mg protein. On the basis of our human study the CT binding to MCF-7 human ER-positive breast cancer cell line was investigated. The CTR content of the intact cells was 5.3 femtomol/10<sup>6</sup> cells and the K<sub>d</sub> value was 0.413 $\times$ 10<sup>9</sup> M, indicating that the binding was very specific to the CTR. According to an exchange assay, the CTR binding sites were already occupied by the sCT within 10 min. A 3 hr exposure of the