higher trend to develop secondary changes.

RELATIONSHIP BETWEEN AUTOPHOSPHORYLATION AND KINASE ACTIVITY OF P56LCK

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A novel putative oncogene has been described: lck is a member of the Tyrosine Kinase (TPK) family, it shares 70% homology with src. We have described the TPK (P56) coded for by lck in LSTRA, a murine lymphoma induced by MoMuLV. P56 is highly expressed in LSTRA, in several human lymphomas, in one case of acute myeloblastic leukaemia, it has been detected in normal and mitogen stimulated T lymphocytes. It is expressed at a very low level in B lymphocytes and is thought to be lymphatic specific. We have studied P56 both in crude membrane preparations and with immunopurified P56 using a specific antibody prepared by immunizing rabbits against a peptide from the N-terminal region of P56, a region sharing no homology with other known TPKs (in particular P60src). In the two systems, we observed that P56 autophosphorylation leads to an increased TPK activity towards exogenous substrates. Chemicals that change the autophosphorylation of P56 have identical effects on the TPK activity. From these data, it appears that autophosphorylation is an important step of the activation of P56lck.

THE THERAPEUTIC USE OF RADIOACTIVE C215 IN MURINE TRANSPLANTED TUMOURS

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The therapeutic role of monoclonal antibody C215 labelled with ^{131}I was investigated in transplantd murine mammary carcinomas. Fragments (approximately 10mg of mammary tumour from (P x Pc) F1 hybrid mice) were implanted subcutaneously in 15 mice of the same strain. Eight mice were injected with ^{131}I-C215 starting from day 12 following tumour implantation and these survived subsequently. In contrast, all 7 control mice died within 35 days. Therefore this study has shown a beneficial anti-cancer effect of radiolabelled C215 in improving survival in the treated mice.

STEARIC ACID AND CARCINOGENESIS

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Decreased membrane rigidity is one of the characteristics of malignant cells, resulting in part from the desaturation of stearic acid into oleic acid. In this study, we investigated the influences of stearic acid in tumour cell inhibition in vitro and tumour development in vivo. Stearic acid inhibited the colony-forming ability of four out of five rat and two human tumour continuous cell lines in vitro. In contrast, the colony-forming ability of rat fibroblasts was not inhibited. Using a model of rat mammary carcinoma induced by nitrosomethyl urea (MNU), the subcutaneous injection of stearic acid at weekly intervals prevented tumour development in 5 of 10 rats. Using iodostearic acid twice weekly, 11 of 19 rats were alive and tumour free at week 22 whilst all of 14 animals injected with NMU alone had died of tumour by the sixteeenth week. The ratio of stearic to oleic acids in erythrocyte membranes was significantly reduced in the tumour-bearing rats, but was normal in tumour-free animals treated with stearic or iodostearic acid. These preliminary data indicate that stearic acid kills human tumour cells in vitro and inhibits tumour development in rats.

GROWTH INHIBITORY ACTIVITY OF HUMAN COLONIC ADENOCARCINOMA CELL LINES IN VITRO

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Under competitive culture conditions cells with growth-inhibitory activity should, if themselves refractory, be among