

EPO and COL1A2. However, direct evidence of the involvement of dysfunctioning PAI-1 in cancer has as yet not been shown.

A NOVEL SYSTEM FOR *IN VIVO* ANALYSIS OF THE EFFECT OF TUMOUR PROMOTERS ON DNA TUMOUR VIRUSES

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An animal system has been developed that allows the analysis of the interaction of persisting hamster papovavirus (HaPV) genomes with the tumour-promoting phorbol ester, TPA. In a colony of HaPV-bearing Syrian hamsters developing spontaneously skin epitheliomas at higher ages, extrachromosomal HaPV-genomes were detected by Southern blot hybridization in skin DNA. Chronic topical treatment of young hamsters with TPA resulted in a dramatic increase of viral DNA in skin cells at the site of TPA application. After finishing the TPA treatment, the amount of extrachromosomal viral DNA declined but was still enhanced more than three months thereafter. This model offers the possibility of investigating effects of TPA and a variety of drugs on the activation of endogenous virus genomes.

VIRAL CARCINOGENESIS - ILLUSTRATED INTRODUCTORY THOUGHTS

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Virus infection of an animal may lead to neoplasia of uninfected cells (indirect mechanisms) or infected cells (direct mechanisms). In the latter case, neoplasia results from the activity of a viral gene or insertional mutagenesis by the viral genome. In either case, a restriction of virus expression in the host may modify pathogenesis. Experimental examples from our laboratory have been obtained that support different facets of this general scheme. In particular, a detailed dissection of the v-src oncogene and host-imposed restrictions on the transcription of retroviral proviruses has been considered and investigated.

ACTIVATION OF ONCOGENE EXPRESSION IN PSORIATIC LESIONS

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Psoriasis is characterized by hyperproliferation and alterations in differentiation programme of epidermal cells. The reason for abnormal behaviour of psoriatic keratinocytes is unknown. There are hypotheses connecting the nature of the disease with the genetic predisposition, with the involvement of retroviruses and mobile genetic elements. It is known now that cellular proliferation is accompanied or caused by expression of proto-oncogenes. We analysed by dot-blot (1) and Northern blot hybridization techniques the expression of proto-oncogenes in the epidermis of psoriatic patients. Cloned [32]P-labelled retroviral oncogenes were used as probes. Elevated levels of *Ki-ras*, *myc*, *fos* and *abl* specific were detected in RNAs from psoriatic lesions whereas expression of other oncogenes like *src*, *yes*, *fes*, *sis*, *erbB* and *mos*, was not observed. Southern analysis of DNAs isolated from leukocytes of psoriatic patients revealed structural alterations in some proto-oncogenes. These DNAs were tested in transfection assay of mouse fibroblasts NIH 3T3. One of two samples were active in transformation. All the data are consistent with the suggestion that genome of psoriatic cells is involved in pathogenesis of the disease. The resemblance of the neoplastic and psoriatic cells in oncogene expression opens new avenues in studying the etiology of this ancient disease and its cure.

(1)Zabarovsky, E.R., Starkov, I.V., Mordovtsev, V.N. and Kisselev, L.L., Biopolymery i Kletkam, 2: N4, 212-215, 1986.

EGF-DOWN REGULATION OF EGF-R IN TUMOUR CELL LINES OF GYNAECOLOGICAL CARCINOMAS

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EGF-receptor levels were assayed in two mammary (T47D and MDA-MB-231), one ovarian (SK-OV-3) and two endometrium (HEC-I-A and HEC-I-B) carcinoma cell lines by binding of [125]I labelled EGF (specified activity 160µCi/µg) and Scatchard plot analysis. Numbers of binding sites varied between 1.7