ON THE MECHANISM OF TUMOUR PROMOTION IN SKIN
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Tumour promotion in skin can be accomplished by wounding or local application of certain agents such as the phorbol esters. Promotion is always accompanied by inflammatory and hyperproliferative processes which are thought to be necessary but not sufficient conditions of promotion. Employing "incomplete promoters", i.e. phorbol esters which contain conjugated double bonds in their long-chain fatty acid residue, the chronic process of promotion can be subdivided into two stages. Stage 1 which is achieved by wounding or a single promoter treatment includes an almost irreversible change in cell function which is assumed to consist of an expression of the neoplastic phenotype. During stage 2 the formation of visible skin papillomas is brought about by continuous mitogenic stimulation. Whether formation of carcinomas (5-10% of the tumours produced) is entirely the result of initiation or, in addition, due to selective pressure exerted in the course of promotion, is still an open question.

In both stages of promotion a crucial role seems to be played by endogenously produced arachidonic acid metabolites such as prostaglandins. Activated oxygen species may, in addition, be involved in stage 1. There are striking relationships between promotion and the wound response which may lead to the proposal that "wound hormones" could act as endogenous promoters.

THE PERITONEAL CELL TEST: EVALUATION OF THE SENSITIVITY OF VARIOUS PARAMETERS
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The peritoneal cell test is a new short term in vivo-in vitro carcinogenicity test in rats with colony growth in soft agar. Using dimethylnitrosamine at 20 mg/kg applied orally to 3 month old male and female Wistar rats, the effect of varying the parameters of the standard test was investigated. Mitogenic medium containing aluminium hydroxide from 0 to 4 mg/rat and agar from 0 to 15 mg/rat was given, and the effect of timing assessed at 15 hours before to 9 hours after the carcinogen application. Maximal scores for colony growth in soft agar were obtained using $Al(OH)_3$ at 1 mg/rat and agar at 10 mg/rat when given 6 hours after the carcinogen. These results suggest that these parameters of the test as published in 1980 (N.Nashed and P.Chandra, Cancer Letters 10, 95-107, 1980) are optimal.

ANALYSIS OF FACTORS AFFECTING LYMPHOGENOUS METASTASIS OF CLONED RAT TUMOUR CELLS.
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Cloned variants of the BSp73 rat tumour show either a high (ASML) or a low (AS) capacity of lymphatic metastasis irrespective of the route of inocculation. The non-metastatic clones exhibited high susceptibility to natural cytotoxicity in vitro as in vivo, pronounced adherence to various types of coated surfaces, and a fibroblast-like arrangement of long filopodia at the outer cell surface. Metastatic clones on the other hand showed low susceptibility to natural cytotoxicity in vitro (but sizeable susceptibility in vivo), low capacity of adherence to surfaces coated with fibronectin and laminin, and a dense coat of microvilli at the cell surface. The karyotope of both lines was highly irregular, non-metastatic clones having a near-diploid set of chromosomes, and metastatic clones having a near-tetraploid set. From modulation experiments it is concluded that metastatic spread results from genuine potencies of the tumour cell, while the host response (via natural defense) can only reduce and retard, but never abolish metastasis of BSp73ASML.