and in pathology of malignant melanoma.

GENOTOXICITY OF PRISTANE AND OTHER ALKANES BY THE SOS CHROMOTEST

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The most extensively studied model of plasmacytoma genesis is by induction of BALB/c mice with i.p. injections of mineral oil or, chemically more defined, by several branched alkanes, such as pristane (2,6,10,14-tetramethylpentadecane), phytane (2,6,10,14-tetramethylhexadecane), and 7-n-hexyloctadecane. The available evidence suggests that the primary action of these plasmacytomagenic agents is to induce the formation of a chronic granulomatous tissue, the histological matrix of plasmacytoma development. However, certain genotoxic (mutagenic) effects caused by these substances can not be ruled out a priori. Pristane, 2-methyldodecane, and 1,3-di-tert-butyl-5-methylcyclohexane as well as hexahydrodibenzauberane and perhydroanthracose were shown to be potential genotoxic agents using the SOS Chromotest, a quantitative bacterial colorimetric assay for genotoxicity. The tested substances, which widely differed in their toxicity, did not provide any evidence for mutagenicity.

ACTIVATION OF MACROPHAGES IN HODGKIN'S DISEASE

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We describe the results of an investigation in frozen sections of 80 cases of Hodgkin's (HD) and non-Hodgkin's lymphoma (NHL) with a panel of monoclonal antibodies directed to human macrophage subsets. A variety of macrophage patterns were observed with the antibodies Ki-M6, Ki-M8, UCHM1 and 44. The greatest frequency of macrophages in all cases was demonstrated with antibodies directed to the alpha-chain of the p150:95 complex (CD11c). The antibody, 10.1, putatively directed to a high-affinity Fc receptor, absent in NHL, was strong in HD and in certain large cell lymphomas positive for Ki-1. In a separate series of experiments we have shown that gamma interferon and the supernatants of Hodgkin's lymph nodes, in short-term culture, are capable of inducing 10.1 positivity on blood monocytes. This suggests that the presence of this marker in HD and Ki-1 lymphomas is due to the local production of high levels of lymphokine.

CARCINOGEN-DNA ADDUCTS AS PROBES FOR THE MECHANISMS OF CHEMICAL MUTAGENESIS AND CARCINOGENESIS

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The formation of specific carcinogen-DNA adducts, their relative persistence in the target tissues of experimental animals, and their demonstated mutagenicity in both microbial and mammalian test systems have provided strong evidence for their role in the initiation of the neoplastic process. For several classes of chemical carcinogens including aromatic amines, polycyclic aromatic hydrocarbons and their nitroaromatic derivatives, metabolic activation pathways leading to DNA adduct formation have been elucidated and found to be quite comparable in tissues of humans and experimental animals. Structure-activity studies have indicated that DNA adducts can induce specific chemical or conformational changes that, upon cellular replication, can lead to specific base transitions or transversions. These same mutations hae also been implicated in the activation of certain cellular proto-oncogenes in both human and animal tumours. Consequently, biochemical methods, which are now being developed to quantify carcinogen-DNA adducts in human tissues, may provide not only an estimate of exposure to occupational and environmental carcinogens but also a reasonable assessment of cancer risk.

ACTION MECHANISMS AND ANTI-LYMPHOMA PROPERTIES OF NEPLANOCIN A

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We have analysed the antineoplastic activity of Neplanocin A (NA), a carbocyclic adenosine analog, against several cultured cell lines. NA was cytostatic and cytotoxic against human and murine T and B Lymphoma cell lines. 50% growth inhibition was brought about at 1 to 10 nM drug levels in 3-day toxicity tests. Several non-lymphoid cell lines were about 1000-fold resistant to NA. Normal peripheral blood lymphocytes