carcinoma; (2)HPV involvement in precancer and cancer lesions has been demonstrated by morphological, immunohistochemical and DNA hybridization techniques; (3)natural history of cervical HPV lesions is equivalent to that of CIN, being potentially progressive to carcinoma in situ; (4)latent HPV infections exist in both sexes; (5)PVs induce malignant transformation in animal models; (6)PV-induced malignant transformation seems to depend on virus type, and physical state of its DNA, i.e. whether or not integrated in the host cell genome; (7)malignant transformation most probably requires synergistic actions between the PVs and chemical or physical carcinogens, or other infectious agents; (8) genetic disposition (at least in animals) significantly contributes to malignant transformation; (9)immunological defence mechanisms of the host probably are capable of modifying the course of PV infections although efficacy in man remains to be elucidated.

HUMAN PAPILLOMAVIRUS (HPV) DAN DETECTED IN BRONCHIAL SQUAMOUS CELL CARCINOMAS

S.Syrjänen, K.Syrjänen(1) and R.Mäntyjärvi(2)

Department of Oral Pathology; (1)Pathology; (2)Clinical Microbiology, University of Kuopio, Finland

The involvement of HPV in squamous cell carcinomas of the respiratory tract was recently suggested by the discovery of HPV 16 DNA sequences in carcinomas of the larynx, the nasal cavity/paranasal sinuses, and in an anaplastic lung cancer. In the present study, a systematic survey was made to assess the possibility that HPV could contribute to the development of bronchial cancer. Formalin-fixed, paraffin-embedded biopsies of 99 invasive bronchial squamous cell carcinomas were subjected to in situ DNA hybridization under stringent conditions (+42° C, 50% formamide; Tm -17), using a mixed probe of HPV types 6, 11, 16, 18, and 30 (provided by H.zur Hausen, DKFZ, Heidelberg, F.R.G.). HPV DNA sequences were disclosed in 5 (5.1%) of the 99 carcinomas, confined to nuclei of the squamous cells, both adjacent to and within the areas of frank invasion. This is the first occasion where HPV DNA sequences have been demonstrated in well characterized bronchial squamous cell carcinomas. The findings are in alignment with the recent theories emphasizing the mechanisms of potentiating and synergistic effects of physical and chemical agents (cigarette smoke among others) in HPV-induced carcinogenesis.

SPONTANEOUS AND SERUM-INDUCED CELLULAR REACTION IN RAT MAMMARY TUMOURS

B.Szende(1), B.Zbar(2), G.Glenn(2) and T.Borsos(2)

(1)1st Institute of Pathology and Experimental Cancer Research, Semmelweis University, Budapest, Hungary; (2)Laboratory of Immunobiology, NCI, Frederick, MD., U.S.A.

Mammary carcinomas were induced by a single MNU treatment in Buffalo rats. A strong mast-cell reaction was detected in the early phase of tumour growth in the connective tissue surrounding the tumour tissue nodules. After the tumours exceeded approximately one cm in diameter or one gram in weight a spontaneous inflammatory infiltration of the interstitial tissue appears parallel with the degranulation of the mast cells. The infiltration consists of neutrophil and eosinophil granulocytes, lymphocytes and in a low amount, plasma cells and macrophages.

A similar inflammatory reaction can be induced even in small tumours by the administration of rat serum absorbed against Protein A conjugated Sepharose. The absorbed serum contains products of the alternative pathway of complement degradation. It is supposed that both serum therapy and a factor released from the growing tumour can indicate mast cell degranulation leading to inflammatory reaction.

STRUCTURE AND EXPRESSION OF THE c-myc ONCOGENE IN MORTAL AND IMMORTAL, UNTRANSFORMED RODENT CELLS

Mahvash Tavassoli and Sydney Shall

University of Sussex, Brighton, Sussex, U.K.

We have analysed the role of the cellular oncogene, c-myc in the process of cellular ageing and cellular immortalization using rodent fibroblasts. The steady-state level of c-myc mRNA of mouse and rat fibroblasts does not change significantly during cellular ageing in vitro. By contrast, the steady state level of c-myc mRNA increases 3 to 5 fold upon spontaneous establishment of these rodent fibroblasts. The increase in the steady-state level of mRNA has a contribution both from an increase in the transcriptional rate as well as from a change in the stability of the mature message. The mRNA levels of both c-fos and c-Ki-ras do not alter; the mRNA of non-muscle actin also does not increase. The changes in the steady-state level of c-myc mRNA are not due to gene