THE FIBRINOLYSIS OF TUMOUR CELLS IN CULTURE AND ITS INHIBITION BY HUMAN SERA.
B.Nagy, J.Ban and B.Brdar.
Central Institute for Tumours and Allied Diseases, Zagreb, Yugoslavia.

The time-related release of the plasminogen activator in several tumour cell cultures which are derived from histologically examined, fresh surgical specimens of human tumours was studied. The assay for plasminogen-dependent fibrinolysis was based on the release of ^{125}I-labelled fibrinopeptides from ^{125}I-fibrin coated petri dishes. The fibrinolytic activity in early primary cultures was similar to the activity of corresponding tumour tissue. In the later cultures, this activity was much lower and corresponded to the activity of HeLa cells. The inhibition of this activity in early culture was observed with increasing concentration of human sera. The human sera were obtained from healthy donors and tumour patients and the inhibitory effect in the serum from tumour patients was less pronounced.

THE PERITONEAL CELL CARCINOGENICITY TEST: A PROMISING APPROACH FOR ASSESSING THE HUMAN CANCER RISK. N. Nashed. Institute of Microbiology, Frankfurt University, D-6000 Frankfurt, F.R.G.

The Peritoneal Cell Carcinogenicity Test is a new short term in vivo - in vitro test in rats. Both application and activation occur in vivo, after which the peritoneal target cells are collected and scored for suspended colony formation. So far, 17 known chemicals belonging to 8 different classes have been successfully tested. The assay evaluates more than one parameter in assessing the agent under examination and could thus serve as a confirmatory test for microbial test systems. By using freshly collected cells exposed for only one week to the anomalous culture conditions, the new test avoids a major source of error, namely the appearance of a high rate of false-positives in the controls.

ENDOGENOUS ECOTROPIC MuLV OF DBA/2 MICE. ACTIVATION AND PATHOGENICITY:
B.A.Nexø[1], N.Jenkins[2], N.Copeland[2] and K.Ulrich[1]. [1]The Fibiger Laboratory, Copenhagen, Denmark; [2]Jackson Laboratory, Bar Harbor, Maine, U.S.A.

Evidence for pathogenicity of inherited C-type retroviruses has been limited to studies of a few strains of mice that have exceptionally high incidences of leukaemia. We have searched for a pathogenic role of endogenous virus in the mouse strain DBA/2, which has a fairly low incidence of leukaemia. It appears that expression of virus, evaluated as the level of the core protein p30 in the blood, is associated with a reduced lifespan, early death in many instances occurring without signs of malignancy.

Two types of AKR-MuLV related ecotropic viruses, called E_a and E_b, have been isolated from splenocytes of DBA/2 mice. Presence of E_b viruses is selectively associated with viraemia in the animals. When injected into C3H mice, E_a viruses appeared relatively innocuous, while E_b viruses reduced the average lifespan and caused a moderate incidence of late leukaemias.

DBA/2 mice carry one ecotropic proviral locus homologous to AKR-MuLV. We have analyzed the structural proteins of E_a and E_b viruses by peptide mapping and compared them to xenotropic DBA/2 isolates and to viruses obtained by transfection of NIH cells with a molecular clone of the endogenous DBA/2 locus. A possible mechanism for generating the E_a vs. E_b polymorphism has been developed.