

By complement fixation, murine oncornavirus group specific antigen was identified in crude extracts of MSV-FBJ sarcomata together with soluble antigens of type specificity.

The significance of these antigens in relation to those previously defined for "wild" type murine leukaemias will be discussed.

HOST IMMUNE RESPONSES IN B.C.G. THERAPY OF A RAT OSTEOSARCOMA, N. Lawrence and M. Moore, Charles Salt Research Centre, The Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry.

The growth of syngeneic grafts of tumour cells is suppressed when cells are mixed with B.C.G. before inoculation, or if clinically established nodules are infiltrated with microorganisms. Theoretically, three types of processes operating individually or in combination might account for this phenomenon: (i) non-immune effects; (ii) B.C.G. immunity and (iii) tumour immunity.

In experiments performed with an immunogenic, chemically induced osteosarcoma, growth inhibition by B.C.G. was less effective in rats immunosuppressed by thymectomy and irradiation than in their immunocompetent counterparts. In the study of the respective roles of immunity to tumour and B.C.G., the differential radiosensitivity of primary and secondary immunity permitted the evaluation of the effect of immunity to one antigen on a second antigen, to which primary immunity had been prevented by interim irradiation.

Evidence will be presented to show that the host response to B.C.G. is essentially local and immunological. Tumour outgrowth from B.C.G. tumour cell inocula occurred with greater frequency in rats prevented from responding to tumour antigen than in normal recipients, suggesting that while local immune reactivity to B.C.G. is a necessary component of successful tumour growth inhibition, it is not invariably sufficient.

TUMOUR THERAPY IN DOGS USING B.C.G., L. N. Owen and D. E. Bostock, Department of Animal Pathology, School of Veterinary Medicine, University of Cambridge.

Experimental dogs have been injected

intradermally, intravenously, intrathoracically and intraperitoneally with percutaneous B.C.G. vaccine (Glaxo).

Following i.v. injection a patchy interstitial pneumonia has been found with small granulomata in lungs and liver. There is lymph node hyperplasia. A small and transient rise in temperature occurs.

Dogs with osteosarcoma treated surgically or by x-irradiation have been injected i.v. with B.C.G. alone or B.C.G. and autologous tumour cells. Results are encouraging. Dogs with lymphosarcoma treated by chemotherapy and followed by intravenous B.C.G. alone have not responded well.

Two dogs, one lymphosarcoma and one osteosarcoma, had anaphylactic shock after the second B.C.G. injection and an anti-histamine drug is now routinely given before the B.C.G.

IMMUNOLOGICAL MECHANISMS IN CONTROL OF MALIGNANT DISEASE, C. Bone and R. S. Camplejohn, Departments of Surgery and Pathology, University of Newcastle upon Tyne.

Cellular immune mechanisms have often been implicated as important factors in the restriction and control of neoplastic proliferation. A study was planned to investigate the relationship between cellular immunity, the rate of malignant cell proliferation and prognosis in 40 patients with carcinoma of the rectum.

Cellular immunity was measured by assessing the patients' delayed hypersensitivity responses to 2-4 dinitrochlorobenzene (DNCEB). The proliferation rates of the rectal carcinomata and the mucosa from which they arose were measured using an *in vivo* stathmokinetic technique (Refsum and Berdal, *Tidsskr. norske Laegeforen.*, 1968, 126, 1224).

Each tumour was staged according to its size and evidence of lymphatic involvement or metastases. The patients' clinical progress was followed.

It was found that there was highly significant ($P > 0.001$) relationship between competent cellular immunity and favourable prognosis. It was also found that the proliferation rate of the rectal carcinomata was only half that of the mucosa from which they arose and growth was due to a diminished cell loss.

There was no correlation between the rates at which the tumours were proliferating and cellular immune competence.

CELL MEDIATED IMMUNITY IN CHILDHOOD TUMOURS, S. Kumar, Christie Hospital, Manchester.

Lymphocytotoxicity tests using the Hellströms' technique were carried out to study cell mediated and humoral immunity in several histological types of childhood tumour. Tumour type specific lymphocyte reactivity was detected in most of these patients; lymphocytes from children with renal tumours showed a significant cross reactivity. The degree of lymphocyte mediated tumour cell killing appeared to be independent of the stage of progression of the tumour. Sera from children with clinically active tumours blocked the lymphocytotoxicity.

A NEW LOOK AT THE EB VIRUS AND ONCOGENESIS, R. N. P. Sutton, Department of Microbiology, King's College Hospital Medical School, London.

The EB virus, a herpes-like DNA virus, has been associated with malignant disease (Burkitt's lymphoma, nasopharyngeal carcinoma) and with infectious mononucleosis and asymptomatic infections. Spiegelman and his colleagues (*Proc. natn. Acad. Sci. U.S.A.*, 1973, **70**, 5) have recently detected nucleic acid homologous with that of the RNA Rauscher murine leukaemia virus in Burkitt's lymphoma and nasopharyngeal carcinoma cells.

This apparent conflict may be resolved by the assumption that the temporary depression of cell mediated immunity which occurs with EB infection in infectious mononucleosis also occurs in asymptomatic infections and is the necessary stimulus which activates an otherwise dormant RNA virus genome.

Sixty-eight children with acute lymphoblastic leukaemia were tested for antibodies to commonly encountered viruses, including the EB virus; their antibody responses were normal, indicating intact humoral immunity. In the case of the EB virus, the antibody pattern in children tested during the first month of their leukaemic illness suggested that a high proportion had experienced infection with this virus. In view of the

potential immunosuppressive properties of the EB virus, it is suggested that these findings may be related to the leukaemogenic process.

INDEPENDENT RESPONSES OF DIFFERENT BINDING SITES ON EHRlich ASCITES TUMOUR CELLS TO TRYPSIN AND TRYPSIN INHIBITOR, P. Whur, Noreen E. Payne and H. Koppel, The Marie Curie Memorial Foundation, Oxted.

We previously reported (Whur, Robson and Payne, *Br. J. Cancer*, 1973, **28**, 80) that Ehrlich ascites tumour cells do not adhere to mouse host intraperitoneal surfaces unless treated with soybean trypsin inhibitor.

We have subsequently found that the binding of these cells to NCTC clone 929 mouse cell monolayers *in vitro* is significantly enhanced in the presence of trypsin inhibitor. However, under the same conditions, adhesion to plastic surfaces is inhibited by trypsin inhibitor. Furthermore, incubation of tumour cells in very low concentrations of trypsin significantly enhances their adhesion to plastic surfaces.

These findings suggest that the degree to which binding sites are expressed is altered by increasing or decreasing proteolysis of cell surface components, and that altered proteolysis may suppress the expression of one site while simultaneously enhancing another.

ULTRASTRUCTURAL AND BIO-CHEMICAL STUDIES OF THE EFFECTS OF CONCAVALIN A ON LANDSCHUTZ ASCITES TUMOUR (LAT) CELLS, R. G. P. Pugh-Humphreys and D. Lawson, Department of Zoology, University of Aberdeen.

Concanavalin A, a protein which binds terminal non-reducing α -methyl-D-mannopyranosides and α -methyl-D-glucopyranosides (Goldstein, Lucy and Yang, *J. Immun.*, 1969, **103**, 695), agglutinates and induces functional changes in a variety of cells (Sharon and Lis, *Science, N.Y.*, 1972, **177**, 949) and is cytotoxic to ascites tumour cells (Inbar, Ben-Basset and Sachs, *Int. J. Cancer*, 1972, **9**, 143).

Concanavalin A treatment of LAT cells resulted in marked cell agglutination, stimulated O₂ uptake and pinocytotic activity (including pinocytosis of concanavalin A),