

reactions. This is further supported by comparable studies in athymic ("nude") mice, where admixture with BCG prevented subcutaneous development of rat tumour cell inocula.

The implication from these findings is that localized BCG treatment may still be applicable clinically, even when immunosuppression has resulted from chemotherapy or radiotherapy.

**TUMOUR ANTIGEN IN HUMAN CANCER PATIENTS' SERA.** J. G. BOWEN, Cancer Research Campaign Laboratories, University of Nottingham.

Previous studies (Baldwin, Bowen and Price, *Br. J. Cancer*, 1973, **28**, 16) have shown that tumour specific antigen can be isolated from the serum of rats bearing a transplanted aminoazo dye-induced hepatoma. The present study was undertaken to determine if the sera of human bladder cancer patients contained a tumour specific antigen. Sera were fractionated by Sephadex G-150 gel chromatography and material with a molecular weight less than 150,000 Daltons examined for the presence of tumour specific antigen by the leucocyte migration inhibition test using leucocytes from bladder cancer patients, other cancer patients and non-cancer hospital patients. Antigen could be detected in the sera of patients bearing tumour *in situ* and those within one month of tumour elimination. Patients tumour free for longer than 3-4 years lacked reactivity against the tumour bearer serum fraction.

**CELL MEDIATED CYTOTOXICITY (CMC) IN PATIENTS WITH ACUTE MYELOBLASTIC LEUKAEMIA RECEIVING IMMUNOTHERAPY.** G. M. TAYLOR, R. HARRIS and C. B. FREEMAN, Department of Medical Genetics, St Mary's Hospital, Manchester.

The effect of immunotherapy with allogeneic leukaemic blasts in patients with acute myeloid leukaemia (AML) was assessed by stimulating lymphocytes from these patients when in remission and also lymphocytes from normal donors with immunotherapy blasts (I blasts), Burkitt's lymphoma (RAJI) cells and normal lymphoblastoid cells (LCL). After 6 days in culture, stimulated lymphocytes were cross-tested for cell mediated cytotoxicity (CMC) against <sup>51</sup>Cr

-labelled target cells of the same origin as those used to stimulate the lymphocytes. Patients on immunotherapy, and stimulated *in vitro* with I blasts invariably showed a higher level of line directed CMC compared with normals whereas cross tests of the same lymphocytes against RAJI and LCL cells revealed lower, though reproducible, cytotoxicity. RAJI generally failed to produce cytotoxicity either in patients or normals, whilst LCL induced cytotoxicity in both sources of lymphocytes, with some evidence of cross-reactivity. Immunotherapy patients seem to possess a pool of lymphocytes capable of restimulation *in vitro* to give high levels of CMC, a property which could be useful for evaluating the response to immunotherapy of AML patients and which could aid the search for tumour associated antigens on autochthonous blasts. Moreover, CMC response is direct evidence that the measures being used at present to induce cell mediated immunity in these patients do in fact work and may be of value in selecting cells for use in immunotherapy.

**ACTIVE IMMUNOTHERAPY IN ACUTE MYELOID LEUKAEMIA.** C. B. FREEMAN, G. M. TAYLOR and R. HARRIS, St Mary's Hospital, Manchester, and C. G. GEARY, J. E. MACIVER and I. W. DELAMORE, Manchester Royal Infirmary.

The duration of first remission in a small group (7) of acute myeloid leukaemia (AML) patients maintained with irradiated allogeneic leukaemia cells and BCG, after initial consolidation chemotherapy, was similar to that achieved in comparable trials using conventional chemotherapy (Freeman *et al.*, *Br. med. J.*, 1973, *iv*, 571). In a second series of 20 patients in the MRC 6th AML Trial, to whom the same immunotherapy was given but without prior consolidation chemotherapy, the median duration of first remission was considerably shorter.

Compared with patients receiving other therapeutic protocols, there was a remarkably high reinduction rate following relapse in both series of patients maintained with immunotherapy alone (second remissions in group 1: 5/6, group 2: 12/18 with four third remissions) and the overall survival figures are very encouraging (group 1: 4/7 alive, current mean survival 115 weeks, group 2: 13/20, current mean survival 50 weeks).

The high reinduction rate may result from (1) avoidance of drug resistance, (2) early diagnosis of relapse or (3) positive effect of immunotherapy or a combination of these factors.

**IMMUNE COMPETENCE IN COLON CANCER: RELATIONSHIP OF PRE-TREATMENT TESTS TO DIAGNOSIS AND TUMOUR STAGE.** A. M. MANDER, P. M. BOLTON, R. H. WHITEHEAD, R. G. NEWCOMBE and L. E. HUGHES, Department of Surgery, Welsh National School of Medicine, Cardiff.

A spectrum of immunological tests was performed to assess cellular and humoral immunity in 40 patients with colon cancer. Controls had suspected cancer but proved to have benign disease.

The tests employed were measurements of peripheral white cell count, lymphocyte count, serum immunoglobulin levels, lymphocyte response to PHA, DNCB response, and the Montoux test. Results were correlated with diagnosis and tumour stage. The colon cancer patients had both absolute and relative lymphocytopenia. Serum IgM and IgA levels were significantly raised in the cancer group and were highest in the patients with distant spread. No difference in lymphocyte response to PHA was observed. DNCB and Mantoux responses were markedly depressed in the cancer group even in patients with early cancer. Discrimination between benign and malignant conditions was not greatly improved by using the tests in combination.

**HOST TUMOUR RELATIONSHIP IN STOMACH CANCER—CORRELATION OF HISTOLOGICAL CRITERIA WITH TESTS OF IMMUNE COMPETENCE.** A. M. MANDER, C. A. MORGAN, E. W. OWEN, J. ZLOSICK and L. E. HUGHES, Welsh National School of Medicine, Cardiff.

The current prospective study involves multifactorial computer analysis of 50 stomach cancer patients who have been assessed from two separate approaches: the first by preoperative estimation of host immune competence, measuring lymphocyte response to phytohaemagglutinin, peripheral lymphocyte count, serum immunoglobulin levels, Mantoux response and dinitrochlorobenzene skin testing, and the second approach

by histological examination of tumour and lymph nodes for evidence of cellular and humoral immune response.

Correlation of the two sets of data with each other and with clinical tumour staging has given a broad view of host tumour interaction in stomach cancer. The results will allow an assessment of whether histological parameters of cellular and humoral immunity correlate with results obtained by immunosurveillance tests, and which of these is most relevant to the clinical outcome of the disease.

**HUMORAL IMMUNITY IN HUMAN LUNG NEOPLASIA.** M. DAWSON and M. MOORE, Paterson Laboratories, Manchester.

Sera from patients with carcinoma of the lung were examined for evidence of humoral immunity towards allogeneic lung carcinoma cells in short-term culture.

Microcytotoxicity assays were used to investigate: (a) complement dependent cytotoxicity; (b) serum mediated cellular cytotoxicity and (c) "blocking" activity of sera towards leucocytes obtained from patients with lung carcinoma.

The incidence of complement dependent cytotoxic antibody to the sera was low, only 3/18 (17%) sera giving positive reactions. A higher proportion 8/18 (44%) of the same sera were found to induce cellular cytotoxicity against lung carcinoma cells in leucocytes obtained from healthy donors, while "blocking" activity towards lung cancer patients' leucocytes was found in 10/18 (56%) sera. The latter phenomenon was not specific since 5/7 (71%) sera from patients with unrelated cancers also reduced the cytotoxicity of leucocytes from lung cancer patients for cultured lung carcinoma cells. The implications of these findings for the interpretation of *in vitro* cytotoxicity tests in the human allogeneic context are to be examined.

**MACROPHAGE MIGRATION INHIBITION AND IMMUNOGLOBULIN PRODUCTION BY HODGKIN'S DISEASE (HD) BIOPSY SPECIMENS *IN VITRO*.** D. B. JONES, S. V. PAYNE, J. L. SMITH, and D. H. WRIGHT, Department of Experimental Pathology, Southampton University.