rh-GM-CSF and rh-IFN-γ resulted in an augmentation of cytotoxicity. Furthermore, cell-bound TNF-α was detectable by immunohistochemistry and correlated with mRNA (in situ hybridisation). In contrast, GM-CSF, as well as IFN-γ reduced the production of TGF-β by TAM, verified by ELISA assay as well as by in situ hybridisation. Our studies show that TAM obtained from malignant effusions of cancer patients can be stimulated by GM-CSF and IFN-γ for cytotoxicity and cytokine production. Though, TGF-β release was reduced. Whether this observation is of therapeutical relevance has to be determined by further studies.

588 POSTER

TUMOR NESCROSIS FACTORα PRODUCTION BY MONOCYTES FROM LUNG AND COLORECTAL CANCER PATIENTS

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Tumor Necrosis Factor (TNFα) is a monocyte (MO) derived cytokine which plays an essential role in host defense mechanisms against cancer. In the present work we evaluate levels of TNFα (by ELISA Test), in culture supernatants of MO from untreated lung (L) and colorectal (C) cancer (C) patients and healthy controls (HC) spontaneously (SP) or LPS stimulated.

Results expressed as pg/ml (range) were LCP: Sp = 450 $\pm$ 113 (53–1000) LPS = 588 $\pm$ 134 (59–1000); CCP; Sp = 84 $\pm$ 30, (36–304). LPS = 430 $\pm$ 110 (61–1000); HC: Sp = 72 $\pm$ 20 (10–189) LPS = 573 $\pm$ 82 (168–1000).

Conclusions: No significant differences were observed in CCP but MO from LCP release high levels of TNFα Sp. ($P < 0.01$) vs HC.

589 POSTER

DO PATIENTS WITH CANCER TREATED WITH INTERFERON PRODUCE ANTIBODIES?

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Production of antibodies to Interferon-a (INF-a) has been described in certain cases. In the present study the evaluation of antibody production was tested in cancer patients treated with INF-a.

Material-Methods: Twenty five cancer patients that have been on therapy with interferon-a were included. The type of cancers were multiple myeloma, chronic myelogenous leukaemia hypernephroma, melanoma and colon carcinoma. All the patients had been on INF-a treatment for over 6 months. 9 other patients with malignancies that had not been on INF-a were also tested as control. The dose of INF-a varied from 5 mU to 9 mU per time given every 2d day subcutaneously. Elisa technique was used for the antibody demonstration.

Results: No patient with INF-a therapy nor the control produced antibodies to INF-a.

Comment: The lack of antibody production is discussed on whether it is due to immunosuppression of cancer patients, to the insensitivity of the technique or to the real lack of production.

590 POSTER

SOLUBLE IMMUNOLOGICAL PARAMETERS IN PATIENTS TREATED WITH INTERLEUKIN-2 (IL-2) PLUS INTERFERON-α (IFN)

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We evaluated the haematological and immunological changes in 4 patients (pts) with advanced melanoma and 6 pts with advanced renal cell carcinoma treated with IL-2 (s.c.) and IFN-α-2b. Serum samples taken before and during six weeks courses of IL-2 plus IFN-α were assayed for the presence of IL-2, soluble IL-2-receptor (sIL-2R), IFN-α, tumor necrosis factor (TNF)-α, soluble intercellular adhesion molecule-1 (sICAM-1), IL-6 and IL-8. Whole blood counts were taken weekly during treatment. Eosinophilia occurred in all pts, lymphocytosis in 8 pts. The median survival of the pts with higher eosinophil and lymphocyte counts tended to be short. The higher maximum level of IL-2 during treatment was connected to longer survival: it was a median of 578 pg/ml in the pts with a median survival of 7 months, and 1025 pg/ml in the pts who survived a median of 15 months. Conversely, an increase in sIL-2R was an unfavourable sign: it was a median of 8-fold and 3-fold, respectively, in the pts with a median survival of y and 16 months, respectively. The sICAM-1 level was a median of 296 ng/ml before treatment; during treatment sICAM-1 levels paralleled with those of sIL-2R. Serum TNF-α and IFN-α concentrations were measured only occasionally; mostly they remained low. There was major intraindividual and interindividual variation in serum IL-6 and IL-8 levels. It can be concluded that treatment with IL-2 plus IFN-α caused remarkable changes in immunological mediators; their prognostic value should be further evaluated in future trials.

591 POSTER

GRANULOCYTE-COLONY STIMULATING FACTOR (G-CSF) ADMINISTRATION FOR CHEMOTHERAPY-INDUCED NEUTROPENIA

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Neutropenia due to cytotoxic chemotherapy causes significant morbidity and mortality. In this study G-CSF (Granulocyte colony stimulating factor) was given to 37 patients treated with intensive combined chemotherapy, who developed absolute neutropenia after 51 chemotherapy cycles were evaluated. The patients were also analysed in two groups as for solid (ifosfamide and etoposide combined regimen) and hematological (mitoxantrone—cytarabine regimen) malignancies. G-CSF was initiated following the onset of neutropenia. Solid tumor patients included total 12 patients and hematologic malignancies consisted of 9 patients. Control group was available for the leukemia group and included 31 acute myeloid leukemia patients. G-CSF was started on the first day on absolute neutropenia and was given for one hour intravenous infusion in 100 cc %5 dextrose solution at the dose of 5 μg/kg/day until absolute neutrophil count was above 1000/ml for two consecutive days. G-CSF was found to be effective for the early recovery of neutrophil count. Expected response was achieved within 14 days in 91.5% of course on median fifth (range 2–14 days) day of G-CSF treatment. Duration of neutropenia was significantly shorter in the study group. The incidence of febril episodes and documented infection rates were not found to be significantly decreased. The incidence of fungal infections was not found to be decreased as well when compared to the control group. In conclusion, administration of G-CSF patients receiving intensive combination chemotherapy at the onset of neutropenia was found to be effective in shortening the period of neutropenia but did not reduce the incidence of febrile episodes and the rate of documented infections.

592 PUBLICATION

IN VITRO CD2 EXPRESSION MODULATION BY IL-1 IN CANCER PATIENTS

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In this study results are presented showing that interleukin-1 (IL-1) modulates the expression of CD2 molecule tested by using sheep erythrocyte roseting assay.

Lymphocytes from 110 patients with solid malignant tumors (breast cancers, melanomas, colonic, carcinomas), leukemias and lymphomas were analyzed for its CD2 antigen expression after 60 min. incubation, at 37°C, with IL-1 in comparison with lymphocytes from patients with clinical evidences of immunodeficiency, autoimmune diseases and normal controls. *In vitro* exposure of lymphocytes from healthy subjects to IL-1 significantly depressed their ability to form active E-rosettes. Results are presented showing that the same monokine significantly enhanced CD2 antigen expression in immunodeficient cancer patients (% Δ = 198.04 $\pm$ 29.13) and in active stages of immune diseases (% Δ = 199.81 $\pm$ 35.92).

The correlation of regulatory effect of IL-1 to the prognosis of the cancer patients is examined.

Functional investigation of CD2 modulation by IL-1 could provide perspectives for future pharmacological interventions into the immune system.