Eighth International
Natural Killer Cell
Workshop and
First Meeting of
the Society for
Natural Immunity

Molecular and Cellular Aspects of
Natural Killer Cell Triggering and Signalling

Symposium Chairpersons:
Julie Y. Djeu, Tampa, FL, USA
John C. Ruckdeschel, Tampa, FL, USA

Abstracts

October 4-6, 1992
St. Petersburg Beach, FL, USA
Markers in Urology

Proceedings of the 2nd Mediterranean Congress of Urology
Rome, July 3–6, 1991

An extensive survey of the diagnostic and prognostic roles of tumor markers.

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Bladder Tumors

- J. A. Cortés: An Update on Urothelial Tumors
- A. J. H. Bond: The Use of Intravesical Therapy in the Management of Bladder Cancer

Prostate Tumors

- P. J. G. L. A. de Koning: Prostatic Cancer: A Diagnostic Challenge
- M. A. D. de Boer: Prostate-specific Antigen: Its Clinical Use and Limitations

Kidney Tumors

- R. J. Brown: Renal Cell Carcinoma: An Update
- A. J. K. Thong: Nephroblastoma: The Pediatric Urologist's Perspective

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NK Cells and Cytokines

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Abstracts

1 Regulation of Splenic Natural Killer Cell Responses During Viral Infections

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Natural killer (NK) cell activation and proliferation peak on day 3 and subside by day 7–10 post-infection of mice with either lymphocytic choriomeningitis virus (LCMV) or murine cytomegalovirus (MCMV). Our laboratory has been characterizing the positive and negative regulation of these NK cell responses. Cytokine studies have demonstrated that interferons (IFN) are potent positive regulators whereas transforming growth factors-ß (TGF-ß) are potent negative regulators of the in vivo NK cell responses. The work presented here was undertaken to evaluate cellular interactions contributing to in vivo NK cell regulation. Histological examination of sections isolated from LCMV-infected mice revealed dramatic changes in splenic architecture at times coinciding with IFN production and NK cell activation and proliferation. White pulp areas were increased but leukocyte concentrations in red pulp areas were decreased. To evaluate IFN expression and the contribution of IFN to the induction of the observed morphological changes, C57BL/6 mice were treated with the chemical inducer of IFN, poly-inosinic-cytidylic acid (poly-IC). As early as 3 h after poly-IC administration, biologically active IFN was present in spleen and serum. Neutralizing studies and Northern blot analyses demonstrated that IFN-ß was preferentially induced. Histological examination revealed poly-IC-induced splenic changes comparable to those observed during infection, i.e. dramatic increases in splenic white pulp area and decreases in red pulp leukocytes. The changes were observed at 6–36 h post-treatment. Spleen weights and splenic leukocyte yields remained relatively constant. Cell transfer experiments with Dil-labeled cells demonstrated that poly-IC enhanced the accumulation of cells migrating from blood to white pulp regions. The changes in splenic leukocyte distribution were shown to be a result of IFN induction as: (1) treatment with anti-IFN antibodies inhibited the poly IC-induced changes, and (2) administration of purified IFN-ß induced similar changes. In situ hybridization and immunohistochemical staining demonstrated that, at early times post-treatment, cells expressing high levels of IFN-ß were found in vasculature and dispersed in red pulp regions. By later times post-treatment, the intensely positive cells were localized deep within white pulp regions and in perivascular spaces. The results indicate that IFN induces a profound migration of leukocytes into white pulp regions of the spleen. The data also suggest that a minor sub-population of leukocytes is an especially potent and early producer of IFN-ß. Studies are underway to characterize the specific distribution of NK cells during the IFN-induced redistribution and to determine how the architectural changes may contribute to the regulation of NK cell responses during viral infections.

2 Natural Killer Cell/Neutrophil Interactions for Resistance against Opportunistic Fungi

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Candida albicans is an opportunistic fungal pathogen that causes persistent infection in immunocompromised hosts. To define if normal resistance might be controlled by natural killer cells in concert with neutrophils, we isolated large granular lymphocytes (LGL) from human peripheral blood by Percoll gradients and neutrophils after separation from mononuclear cells with Ficoll-Hypaque gradients. Human I.G.I. were found not to be able to inhibit Candida growth in a...
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