

SIII-4-1

Chemotherapy Resistance in Cancer: Expression of Chemoresistance Genes and its Possible Application to Gene Therapy

Young Do Yoo

Lab. of Experimental Therapeutics, Korean Cancer Center Hospital

Intrinsic or acquired resistance to chemotherapeutic drug is a major obstacle for effective chemotherapy of cancer. We have established 5-FU- and cisplatin-resistant cell lines from Korean gastric carcinoma cells to investigate the molecular mechanisms of chemoresistance. Differential expression between SNU638-FU2 and the parental cells was examined. Expression of 18 genes from 588 human genes was enhanced in SNU638-FU2, compared to the parental cells. In order to find the mechanisms of chemoresistance in stomach cancer, we tried to find and clone the unknown genes which might play an important role in chemoresistance by differential display-PCR method. Characterization of chemoresistant genes is under investigation. From this results, it might be possible to identify agents that specifically block chemoresistant gene expression. This chemogenotherapy will improve the therapeutic results in cancer therapy.

SIII-4-2

Tumor antigen presentation pathways in IL-12-mediated cancer therapy

Joo-Hung Park

Department of Biology, Changwon University

Two signals are required for proper T cell activation: signal one, delivered through the coupling of TCR and antigenic peptide bound to MHC class I molecules, and the other, through interactions between costimulatory molecules and their receptor molecules such as the coupled interaction of B-7 and CD28. However, that non-hematopoietic cells do not express co-stimulatory molecules such as B7-1 and B7-2 makes it likely that so-called professional antigen presenting cells of hematopoietic origin like dendritic cells, Langerhans cells and macrophages present antigenic peptides to T lymphocytes in mice carrying carcinomas and sarcomas. However, many tumor cells which are themselves tumorigenic in mice were rejected in vivo following MHC DNA transfection, suggesting that tumor cells themselves directly present antigens to T cells. In the present study, we have addressed the issue in a tumor model using M-MSV-BALB/3T3 cells, a sarcoma cell line, and collected some compelling evidence that tumor cell itself present MHC class I-restricted antigen to CTLs.