G103 MHC Class I Presentation of an Exogenous Polypeptide Antigen Encoded by the Murine AIDS Defective Virus

Sung-Tae Yee*, Woen-Dong Han and Hyun-Wook Cho
Depart. of Biology, Sunchon National University

Peptides derived from endogenous proteins are presented by MHC class I molecules, whereas those derived from exogenous proteins are presented by MHC class II molecules. This strict segregation has been reconsidered in recent reports in which exogenous antigens are shown to be presented by MHC class I molecules in the phagocytic pathway. In this report, the presentation pathway of an exogenously added highly antigenic polypeptide encoded by the murine AIDS (MAIDS) defective virus gag p12 gene is investigated. A 25-mer polypeptide (p12-25) encoded within the gag p12 region of the MAIDS defective virus was found to be effective in stimulating unprimed B6 ($H-2^b$) $CD8^+$ T cells in vitro. The presentation of p12-25 is sensitive to cytochalasin B and D, brefeldin A and gelonin, a ribosome-inactivating protein synthesis inhibitor, but less sensitive or resistant to lactacystin, a highly specific inhibitor of the proteasome. Interestingly, CA-074, a selective inhibitor of cathepsin B, inhibited presentation of the polypeptide, indicating its involvement in the degradation of the p12-25 polypeptide. In fact, when p12-25 was digested with purified cathepsin B in vitro, a highly antigenic 11-mer peptide containing the class I ($H-2D^b$)-binding motif was obtained. Our results favor the phagosome / macropinosome - to - cytosol - to - endoplasmic reticulum(ER) - to - cell surface pathway for exogenous antigens presented by MHC class I molecules. These findings may be relevant to exploiting peptide vaccines that specifically elicit $CD8^+$ T cell immunity in vivo.

G301 Distributional Change of PMN in Thermally Injured Mice

Sang-Yun Nam
School of Life Science, Jeonju University

It has been well documented that global suppression of the immune responses occurs after thermal injury and these changes resulted in decreased resistance to infection, which is a leading cause of high mortality of burn patients. In this study, the distribution of PMN (polymorphonuclear leukocyte), which play a central role in primary host defense, and lymphocytes, for comparison, was investigated in thermally injured mice.

Mice (Balb/c) were anesthetized by intraperitoneal injection of 2,2,2-tribromoethanol and thermally injured by immersion of hair-removed dorsal skin (15% total body surface) in a boiling water bath (ca. 96 ℃) for 7 seconds.

Significant decrease of lymph node and spleen cell numbers was observed at day 2 and follwed by gradual increase over normal level unitil day 30 of injury. Such increase of cell number coincided with increased of PMN number, particularly in spleen (5% vs 30%). Reduction of lymphocyte proportion was due to relative increase of PMN number, not the decrease of absolute number of lymphocytes. In addition, the Th/Ts ratio of injured mice was comparative with control group.

These results shows that trafficking of PMN may be a critical factor related to alteration of immunity in thermal injury.