Retrovirology

Poster presentation

Rapid Activation of an Effector Phenotype in Human Vγ2/Vδ2 T Cells Stimulated With a Toll-Like Receptor 2 (Tlr2) Agonist

Carl Deetz*‡, Andrew Hebbeler, Ilia Tikhonov, Cristiana Cairo, Jean-Saville Cummings and C David Pauza

Address: Institute of Human Virology, University of Maryland Biotechnology Institute, Baltimore, MD 21201
* Corresponding author ‡Presenting author

from 2005 International Meeting of The Institute of Human Virology
Baltimore, USA, 29 August – 2 September 2005

Published: 8 December 2005

Retrovirology 2005, **2**(Suppl 1):P148 doi:10.1186/1742-4690-2-S1-P148

Approximately 1–10% of circulating $CD3^+$ cells in the blood express the gamma delta (γδ) T cell receptor (TCR) and, of these γδ T cells, the majority express the Vγ2/Vδ2 receptor. In HIV-infection, there is a targeted destruction of Vγ2-Jγ1.2/$V\delta2^+$ T cells. This is the only TCR-specific change common to all individuals infected with HIV. Because Vγ2/Vδ2 are potently cytotoxic for tumor cells, loss of these cells may be part of the mechanism that promotes AIDs-related malignancies. Tumor recognition by γδ T cells may require a 60 kDa heat shock protein (HSP60) on Daudi Burkitt's lymphoma cells. However, HSP60 recognition may not be mediated by the TCR but by another receptor on γδ T cells. Since γδ T cells also respond to microbial infection, this additional activatory receptor may be in the toll-like family of receptors that recognize pathogen-associated molecular patterns. To explore this recognition, we treated isopentenyl pyrophosphate (IPP) expanded Vγ2/Vδ2 with the TLR2 agonist PAM3Cys and analyzed the activation of an effector phenotype by measuring IFN-γ secretion and cell killing. Daudi cell killing appears to be enhanced by the addition of the TLR2 agonist. Intracellular staining of γδ T cells after a two-hour incubation with PAM3Cys and anti-γδ TCR antibody revealed as much as a ten-fold increase in IFN-g over that produced by anti-γδ TCR antibody stimulation alone. Therefore, it seems that TLR2 does play a role in Daudi cell killing and signaling through this receptor works synergistically with TCR signaling to induce an early T_H1-type immune response by IFN-γ production.