Retrovirology

Poster presentation

Clonal Selection and Population Dynamics of Vγ2/Vδ2 T Cells in Macaca Fascicularis

Cristiana Cairo*‡[1,2], Andrew Hebbeler[1], Nadia Propp[1], Vittorio Colizzi[2], Joseph L Bryant[1] and C David Pauza[1]

Address: [1]Institute of Human Virology, Baltimore, MD, 212101 and [2]University of Rome, Tor Vergata, Rome, Italy
* Corresponding author ‡Presenting author

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HIV infection increases the susceptibility to new M. tuberculosis (Mtb) infections, the risk of reactivating latent infections and the risk of rapid TB progression. γδ T cells, in particular the Vγ2Jγ1.2 subset, are thought to be part of the innate immune response to both HIV and Mtb. Importantly, both HIV and Mtb perturb gd T cells homeostasis, causing a profound and highly specific depletion of the Vγ2Jγ1.2 subset.

We used a primate model (M. fascicularis) to investigate the Vg2 response to mycobacterial infections and we followed Vγ2 population dynamics at the clonal level after infection with attenuated Bacille Calmette-Guerin (BCG). There was a modest increase of circulating Vγ2 T cell and changes in the Vγ2 repertoire following BCG inoculation. The increase of circulating Vγ2 T cell frequency correlated with an increase in Vγ2 responsiveness to secondary stimulation *in vitro*, both in terms of proliferation capacity and IFNγ production. CDR3 sequence analysis showed the existence of discrete clones that were selected after BCG exposure. Two CDR3 sequences were found frequently in all of the four animals analyzed and both were encoded by multiple nucleotide sequences converging on the same amino-acid sequence. Few other CDR3 sequences were found in more than one animal. A second BCG inoculation caused a dramatic contraction of the Vγ2Jγ1.2 population and specific deletion of the responsive clones, likely as a result of activation induced cell death.

These results show that the Vγ2 T cell response to live BCG tends to be clonal in M. fascicularis. The presence of a few preferred CDR3 sequences used frequently in different animals strongly suggests that, if any presenting molecule is involved in Vγ2 antigen recognition, it is not highly polymorphic.

Our established M fascicularis model provides important information about Vγ2 clonal deletion induced by mycobacterial infection and is a model for the impact of pathogens including HIV and P falciparum on the Vγ2 population.