EuroSciCon

Infectious Diseases and STD-AIDS

April 26,27 2018
Rome, Italy

J Transm Dis Immun 2018 Volume 2
DOI: 10.21767/2573-0320-C1-003

A NEW UNDERSTANDING OF HIV/AIDS IMMUNOBIOLOGY BY THE ADAPTATION MODEL OF IMMUNITY

Masoud H Manjili[1, 2]

[1]Virginia Commonwealth University
[2]Massey Cancer Center, USA

The immune system has a major role in limiting HIV infection by facilitating HIV dormancy rather than eliminating the infection. Dormant HIV could eventually resume replication and establish AIDS. A theoretical gap in our understanding of the immunobiology of HIV infection has hindered the development of effective immunotherapeutic for patients with AIDS. Current immunotherapeutic strategies are inspired by two schools of thought in immunology which include the self-nonself (SNS) model and the danger model. The SNS model solely emphasizes the foreignness of the antigen (signal I) or pathogen-associated molecular pattern, PAMP (signal II) inducing the immune response. The danger model emphasizes the damage-associated molecular pattern (DAMP) that induces co-stimulation of lymphocytes or signal II. These models explain how an immune response is induced but they fail to predict whether an immune response succeeds or fails. To this end, the adaptation model of immunity proposes that molecular crosstalk between the adaptation receptors (AR) and adaptation ligands (AL) in the immunological synapse determine the success or failure of the immune response. In fact, target cells determine the outcome of the immune response. Latency or dormancy of the HIV infection is perhaps because of the expression of AR on the infected cells. The engagement of AR facilitates survival of infected cells while inhibiting viral replication in the presence of the HIV-specific CD8+ T cells induced during a transient acute phase of the infection. However, immunoediting of the infected cells by chronic CD8+ T cell responses facilitates downregulation of AR and subsequent elimination of HIV-infected cells and progression to AIDS. On the other hand, anti-viral immune responses induced by the HIV infection fail to alter the AR and as a result do not progress to AIDS. This concept is supported by recent findings demonstrating that factors independent of antigen specificity determine the efficacy of the immune response against HIV.

Masmasoud.manjili@vcuhealth.org