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Unconventional CD8+ T cells ($CD4^{dim}CD8^{bright}$ cells) in anti-HIV immunity and neuropathogenesis

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Conventional paradigm of T cell biology tells us that expression of the CD4 and CD8 molecules on mature T cells is mutually exclusive. However, my group and others have identified a unique subset of CD8+ T cells that co-express CD4 dimly on their surface. This subset is termed CD4dimCD8bright T cells or double positive (DP) T cells. DP cells represent a genuine phenotype of CD8+ T cells. They express αβTCR and αβCD8 and are not prematurely released from the thymus. Post TCR or superantigen stimulation, 15-60% of purified CD8+ T cells induce stable CD4 expression on their surface. DP cells constitute 3-5% of peripheral CD8+ T cells in healthy donors and are expanded to up to 15% among HIV long-term nonprogressors. DP cells are highly enriched in anti-viral (CMV and HIV) responses. In comparison to their CD8 single-positive T cell counterparts, DP cells constitute greater than 55% of anti-HIV and -CMV responses, as evaluated by MHC-I tetramer analysis, polyfunctional cytokine and effector molecule expression, and antigen-specific proliferation. DP cells are also highly enriched in β-catenin expression, a pro-survival transcriptional co-activator that drives CD4 induction on CD8+ T cells and may protect them from activation-induced cell death. DP cells are found in the CNS of HIV-infected NOD/SCID/IL-2rcγ-/- mice reconstituted with human peripheral blood lymphocytes and while they are susceptible to HIV infection, they are not depleted to the same extent as CD4+ T cells. Astrocyte-conditioned media also induces CD4 expression on CD8+ T cells. Our findings identify DP cells as a potent anti-viral T cell subset; however, their contributing role to HIV-associated neurocognitive impairment, whether protective or pathogenic, remains to be elucidated.

Biography

Dr. Al-Harthi is a Professor in the Department of Immunology/Microbiology at Rush University Medical Center in Chicago, IL. She has over 60 peer-reviewed publications and invited reviews/book chapters. Her research over the past 16 years has focused on HIV/host interactions, with a special emphasis on bridging basic and clinical science in the HIV/AIDS field. Because of her experience in HIV molecular biology, immunology, and neuroAIDS, she has been able to probe mechanistic questions that are clinically relevant to HIV/AIDS. She is actively investigating the molecular pathways by which Wnt/β-catenin signaling inhibits HIV replication, its impacts on HIV neuropathogenesis, and the role of host and viral factors in modulating β-catenin interaction with HIV.

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