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In vitro restoration of Th17 response during HIV infection with an antiretroviral drug and Th17 differentiation cytokines

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HIV infection is marked by a profound loss of CD4 T cells and functions. The Th17 subset, which plays an important role in the control of extracellular bacteria and fungi, is particularly depleted as compared to the Th1 subset in chronically HIV-infected patients, even after sustained virus-suppressive anti-retroviral therapy. In this study, we have established an in vitro system utilizing primary human CD4 T cultures that recapitulates the dramatic loss of Th17 response upon HIV-1 infection with a less profound decrease of Th1 response. With this experimental system, we showed that active HIV-1 replication was needed for the depletion of Th17 response.

Blocking viral entry with CCR5 ligands or TAK779 reduced the infection and enhanced Th17 response but not Th1 response. The more potent anti-retroviral drug 3TC, given at the time of infection, prevented the loss of both Th17 and Th1 responses, but was not effective when given after infection was already established. Only when Th17 differentiation cytokines were given along with 3TC to the infected cultures, was Th17 cell response fully restored. Finally, up to 3 fold increase of Th17 response was achieved in PBMCs of patients on antiretroviral therapy after treatment with Th17 differentiation cytokines. These data demonstrate the presence of CD4 T cells remaining capable of mounting Th17 response during HIV infection and indicate the potential use of immunotherapeutic modalities to supplement anti-retroviral drugs for restoring Th17 response in chronically HIV infected patients.