Retrovirology

Oral presentation

Mucosal Pathogenesis of HIV Infection

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Like HIV-infected humans, simian immunodeficiency virus rapidly and selectively infects, replicates in, and destroys memory CD4+ T cells co-expressing CCR5 (viral "target"cells) resulting in loss of the majority of the bodies CD4+ T cell pool within 21 days of infection. The vast majority of these cells reside in the intestinal tract and other mucosal tissues but selective loss of these target cells is detectable throughout the lymphoid system. Restoration of memory CD4+CCR5+ T cells directly correlates with improved clinical course and lower viremia, but these cells are never restored in macaques that progress to AIDS. Continuous and effective antiviral treatment initiated within days of SIV infection can rescue mucosal CD4+ T cells, but delaying therapy for a couple of weeks does not restore these vital helper memory cells, despite effective control of viremia. Similarly, monkeys that "appear" protected in vaccine challenge studies may in fact harbor smouldering infection in the intestine with continuous CD4+ T cell loss, despite undetectable plasma viremia. The rapidity and severity of the loss of memory CD4+ T cell function is likely the major reason no cure or vaccine is in sight. In fact, converging evidence suggests that other primate species changed fundamental properties of their immune system, such as eliminating the need for CD4+CCR5+ T cells (yet maintaining CD8+CCR5+ T cells), rather than cope with this subversive infection. This and other data suggest that conventional immune responses simply may not be adequate to control or prevent HIV infection.