RETROVIROLOGY

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Potential inflammatory consequences in HIV controllers

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Aim

Most HIV controllers have strong HIV-specific T cell responses that likely contribute to viral control. However, there may be negative inflammatory consequences to the immunologic control of viral replication in these individuals.

Materials and methods

We compared the frequency of activated (CD38+ HLA-DR+) T cells and carotid intima-media thickness (a measure of atherosclerosis) between untreated HIV controllers maintaining plasma HIV RNA levels <75 copies/ml and HIV-infected ART-suppressed, untreated HIV-infected "non-controllers," and HIV-uninfected controls. We also assessed the relationships between the frequencies of Gag-specific and activated T cells, and cell-associated HIV RNA and DNA levels in HIV controllers.

Results

The 52 HIV controllers had higher frequencies of activated CD4+ and CD8+ T cells than HIV-uninfected controls ($P < 0.001$ for both) and higher CD8+ T cell activation than the ART-suppressed ($P = 0.017$). HIV controllers also had higher carotid intima-media thickness than HIV-uninfected individuals even after adjustment for traditional cardiac risk factors ($P = 0.003$). In HIV controllers, higher CD4+ and CD8+ T cell activation was associated with lower CD4 counts in HIV controllers ($P < 0.001$ for both). While HIV controllers had the highest frequencies of Gag-specific CD4+ T cells of any group, suggesting a role in the control of viral replication, higher Gag-specific CD4+ (but not CD8+) T cells were associated with both higher CD8+ T cell activation ($P < 0.001$) and higher cell-associated HIV DNA levels ($P = 0.019$).

Discussion

Strong HIV-specific CD4+ T cell responses in HIV controllers may assist in the control of viral replication, but may also contribute to viral persistence and generalized immune activation, which may drive both CD4+ T cell depletion and subclinical cardiovascular disease even in the absence of clinically detectable viremia.

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