Under-Celling PWAs

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It’s the virus, stupid. Still true, but HIV’s main effect may not be what we’d thought. Using a new cell-labeling system that employs sugar molecules to track the life and death of CD4 and CD8 cells, researchers at the University of California (at San Francisco and Berkeley) have found evidence that may reshape our understanding of how HIV causes immune destruction. The old view—first advanced by the prince of HIV eradication, David Ho, MD—is that of the immune system’s ultimate exhaustion and collapse after years of feverishly churning out millions of cells doomed to quick infection and death. The new California research—comparing HAART-takers, untreated HIVers and HIV negatives—shows that although the virus does indeed destroy cells, the primary cause of T-cell decline is a greatly shortened lifespan, not compensated by an increased cell-production rate. Compared to those not on therapy, HAART-takers had markedly higher cell-production rates—the explanation for their elevated T-cell counts—but those cells’ lifespans didn’t increase. Researchers have yet to figure out why the body’s cell production fails to counter the effects of the shortened lifespan, but a good guess is HIV’s effects on the thymus and bone marrow. While some top scientists consider these findings very controversial, others believe they will add an important dimension to our understanding of the disease—and, most important, focus attention on therapies to boost cell production.