

Researchers See Dramatic Drop in HIV-Infected Immune Cells After Cancer Treatment

Study by Johns Hopkins Medicine-led team suggests finding may be an early step toward a potential HIV cure.

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Advancements in HIV/AIDS research, drug development and clinical practice since the 1980s have made it possible for people living with HIV to lead long, productive lives and keep the virus in check at undetectable levels and nontransmissible as long as therapy is maintained. However, a cure—completely ridding the body of the virus—has only been documented in a handful of patients who underwent complex and high-risk bone marrow transplants for life-threatening blood cancers such as leukemia or lymphoma.

[In a paper published today in the Journal of Clinical Investigation \(JCI\)](#), researchers at Johns Hopkins Medicine report they may have taken an early step toward a more practical HIV cure. The researchers—in a study done largely with federal funding—focused on a patient undergoing cancer treatment and also living with HIV, who after receiving chemotherapy, had a significant reduction in the number of CD4+ T immune cells that contained an HIV provirus—a key player in HIV’s ability to persist in the body.

In a person living with HIV, proviruses—strands of HIV DNA—are typically integrated into the T cell genome and become a permanent part of the cell’s genetic makeup. This integration enables the provirus to be passed on to daughter T cells when the parent cell divides—a process known as clonal expansion of HIV-infected T cells.

Over time, clonal expansion leads to an increase in the frequency of infected cells in a patient. The proviruses in daughter T cells can remain dormant or become active and begin producing new HIV particles, especially if antiretroviral therapy is stopped.

CD4+ T cells (also known as helper T cells) are immune cells that recognize antigens, the body’s foreign invaders such as bacteria and viruses; stimulate antibody production by another type of immune cell, the B cell; and help a third type of immune cell, the CD8+ T cell (also known as a killer T cell), target and remove antigens from the body.

“CD4+ T cells with dormant HIV proviruses make it difficult to eradicate the virus from the body, because the potential is always there for a renewed HIV infection,” says study co-senior author [Joel Blankson, MD, PhD](#), professor of medicine at the Johns Hopkins University School of Medicine. “It’s vitally important for us to learn why there were significantly fewer clonally expanded, infected CD4+ T cells in the patient who received chemotherapy. If we can understand the mechanism by which that happened, perhaps it can be translated into a means of curing HIV.”

Blankson says that the patient in the JCI study received two chemotherapeutic drugs for metastatic lung cancer: paclitaxel and carboplatin. “We suspect that the HIV-infected CD4+ T cells might have been highly susceptible to these drugs and were kept from proliferating in the patient,” says Blankson. “Our experiment was designed to learn if that’s what really occurred.”

In the JCI study, the researchers studied an HIV-infected CD4+ T cell clone from the patient, a cell with an active, replication-competent (able to infect other T cells) provirus integrated into its genome. They stimulated the clones with the T cell’s cognate peptide, a portion of HIV protein that activates the infected T cell and enables proliferation.

“We treated the stimulated T cells with paclitaxel and carboplatin in one experimental group, and an antiproliferative drug, mycophenolate mofetil, in another experimental group while leaving the stimulated clones in the control group untreated,” says Blankson. “The untreated infected clones continued to proliferate, but the treated infected ones did not. This was a significant finding, because it suggests a means by which infected cells could be selectively eliminated.”

As this phenomenon was only seen in T cell clones from one patient, Blankson says his team plans to look at the HIV elimination ability of CD4+ T cells from other people living with HIV.

“We suspect the reason that the infected T cell clones we studied were so susceptible to chemotherapy and the antiproliferative drug is because they rely on frequent proliferation to persist in the body,” says study co-senior author [Francesco Simonetti, MBChB, PhD](#), assistant professor of medicine at the Johns Hopkins University School of Medicine. “Showing this happens in other people living with HIV will provide evidence that this suspicion is correct, and in turn, direct future research toward HIV cure strategies.”

“A key advantage of such an approach is that it can eliminate infected T cells without having to deal with other mechanisms that enable HIV to persist in the body,” says Simonetti.

Along with Blankson, the members of the research team from the Johns Hopkins University School of Medicine are Tyler Beckley, study lead author Filippo Dragoni, Isha Gurusamy, Kellie Smith and Joel Sop.

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Simonetti received payments from Gilead Sciences for participation at scientific meetings. Smith is an inventor of a subset of technologies related to the FEST assay described in the paper, receives research support from AbbVie and Bristol-Myers Squibb, and owns founder's equity in Clasp Therapeutics.

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