



A Key to Long-Term HIV Control

Boosting the number and effectiveness of CD8 T cells with specific properties may help control HIV without the need for life-long treatment.

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- Two NIH-funded studies linked sustained suppression of HIV after experimental treatments to a certain type of immune cell.
- The findings suggest that boosting the number and effectiveness of those specific cells may help control HIV without the need for life-long treatment.

Roughly 40 million people around the world are living with HIV. More than 70% of them suppress the virus with antiretroviral therapy (ART). Unfortunately, when people stop ART, the virus usually resumes replicating and ravaging their immune systems. Thus people have to continue ART for life to keep the virus under control.

To overcome ART's limitations, many scientists are developing short-term HIV treatments that have long-lasting effects. One such treatment is broadly neutralizing antibodies (bnAbs). These antibodies can neutralize a wide range of HIV strains.

A recent NIH-funded study explored why bNABs have lasting effects in some people with HIV but not others. The study was led by Dr. David Collins at the Ragon Institute of Mass General Brigham, MIT, and Harvard University. Results were [published in Nature](#) on December 1, 2025.

The study analyzed blood samples from 12 people with HIV across four previous trials led by collaborators at Rockefeller University and Aarhus University. Participants stopped their ART regimens and received bnAbs. Among these 12 people, seven successfully suppressed the virus for up to 7 years after bnAb treatment. They were dubbed "controllers." In contrast, in the remaining five participants, viral levels rebounded soon after treatment.

Among the controllers, certain immune cells called CD8+ T cells multiplied much more than in non-controllers when the cells encountered HIV proteins. Surprisingly, they found that this difference in CD8+ T cell proliferation was present even before the bNAB treatment began. After treatment, proliferation of CD8+ T cells in response to HIV proteins increased in both groups. But it remained higher among the controllers. CD8+ T cells that multiplied more also did a better job at

killing HIV-infected cells.

Before the bnAb treatment, more of the controllers' HIV-specific CD8+ T cells were of a type known as stem cell-like memory T cells compared to the non-controllers. After treatment, CD8+ T cells in the controllers skewed even more heavily toward that type. The more stem cell-like memory T cells a participant had after treatment, the more their CD8+ T cells multiplied in response to HIV proteins.

Successful suppression of HIV was not related to T cells recognizing different targets on HIV after treatment. This suggests that the HIV-suppressing CD8+ T cells in the controllers were most likely present before the treatment began and that bnAb treatment helped these pre-existing cells to control HIV.

A second NIH-funded research team, led by Drs. Steven Deeks and Rachel Rutishauser at the University of California, San Francisco, bolstered and extended the first study's findings. Their study was [published in the same journal](#).

The team looked at T cells in 10 people with HIV who were treated with a combination therapy including a T cell-boosting vaccine and bnAbs before stopping ART. In seven of the participants, HIV replication remained controlled in the absence of ART for at least several months after bnAb levels declined in the blood. As the virus started to re-emerge, CD8+ T cells multiplied more in controllers than in non-controllers, which parallels the finding in the study described above. The responding CD8+ T cells in this study also had higher levels of a protein called TCF-1. TCF-1 marks cells with traits resembling those of stem cells, such as the ability to multiply and develop into specialized types.

Both studies suggest that HIV treatments designed to increase the number and effectiveness of CD8+ T cells with stem cell-like properties may be more likely to induce long-term HIV suppression in the absence of ART. Researchers are now working to develop treatments to boost these T cells.

"These studies offer important insights into how the body's natural defenses can be harnessed to achieve durable HIV remission," Collins says.

This [research summary](#) was published by the National Institutes of Health on December 16, 2025.