Researchers discover how HIV hides from treatment

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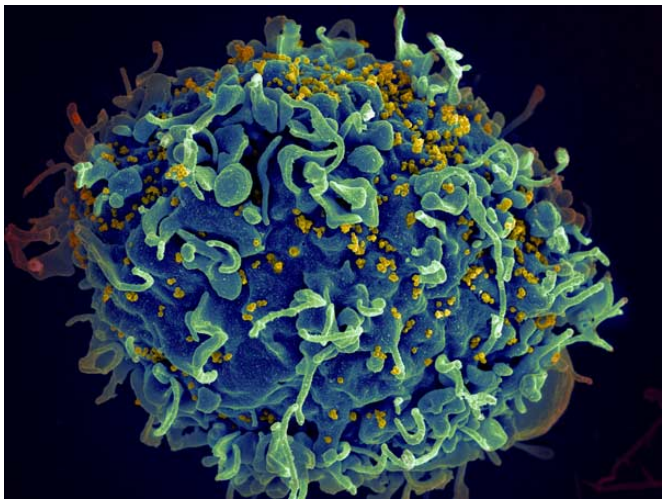

HIV (yellow) infecting a human immune cell. Credit: Seth Pincus, Elizabeth Fischer and Austin Athman, National Institute of Allergy and Infectious Diseases, National Institutes of Health

Even after successful antiretroviral therapy, HIV can hide dormant in a tiny number of immune system cells for decades and re-emerge to threaten the life of its host. Now Yale University researchers have discovered a molecular explanation for how the virus accomplishes this insidious trick, they report May 13 in the journal *Science Translational Medicine*.

Long-living CD4 T cells, which act as the immune system's memory of past infections, are HIV's safe harbor. The virus actually intertwines itself in the DNA of T cells, but because the virus is inactive it leaves nothing to mark its location.

"HIV integrates itself into the human DNA so that antiretroviral therapy cannot find and kill it," said senior author Ya-Chi Ho, assistant professor of microbial pathogenesis and medicine (infectious diseases).

"It is very hard to study these cells—only one in a million CD4 T cells have infectious HIV," said Ho. "They are like dandelions hidden in a lawn—impossible to find in the grass but can suddenly pop out as yellow flowers."

The Yale team developed a way to find these hidden infected cells and also a potential way to control viral spread.

Ho's team took cells from HIV patients who had undergone antiretroviral therapy. The scientists activated the virus in T cells in a lab dish and used florescent probes to label the viral RNA that identifies infected cells. They then removed these rare HIV-infected cells and studied individual cells at high resolution. This method, called HIV SortSeq, allowed researchers to track the interplay between the RNA of the host and the virus within single cells.

They found that HIV essentially hijacks genes that allow T cells to survive. Intriguingly, said the researchers, HIV works to promote the expression of genes implicated in cancer. Essentially, HIV wrests control of activation of these cancer genes from the host cells. Although this process does not cause cancer in T cells, the researchers said, the process may help these HIV-harboring T cells to proliferate.

Understanding these processes could help scientists develop new ways to control HIV infection, the researchers said.

More information: R. Liu el al., "Single-cell transcriptional landscapes reveal HIV-1–driven aberrant host gene transcription as a potential therapeutic target," *Science Translational Medicine* (2020). stm.sciencemag.org/lookup/doi/ … scitranslmed.aaz0802

Provided by Yale University

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