## Reducing HIV replication with CRISPR could lead to effective cure

Scientists in Japan have used CRISPR-Cas9 technology to stop human immunodeficiency virus type 1 (HIV-1) replication in latently infected T cells that can't be controlled using existing drug treatments. The gene-editing approach effectively disrupts two regulatory HIV-1 genes, tat and rev, which are essential for viral replication.

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[W]hile lifelong antiretroviral therapy (ART) can help convert what is otherwise a deadly infection into a more "manageable chronic disease," current treatments are not a cure because they can't completely eradicate the virus, which inserts its genes into the host cells' DNA, the authors explain. Despite treatment using ART, HIV-1 continues to replicate at a very low level in some latently infected immune system cell types, such as CD4+ cells, macrophages, and follicular dendritic cells."

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The scientists generated six guide RNAs (gRNAs)—three targeting tat and three targeting rev—to direct the DNA-cleaving Cas9 enzyme to the relevant sites in the proviral DNA. They packaged gRNAs and the Cas9 enzyme system into a lentiviral vector, which they could then introduce into cultured cells.

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They claim that the ability to transduce nondividing cells, such as resting CD4+ T cells, using lentiviral constructs and achieve long-term Cas9 transgene expression supports the feasibility of using the system to eradicate cells that act as latent reservoirs of HIV-1.

Editor's note: Read full study

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