

# RISPR-Based Gene Editing in HIV/AIDS Treatment: Recent Developments

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## Abstract

Despite significant efforts to prevent and treat HIV-1 infection, HIV/AIDS continues to pose a significant threat to human health worldwide. Even though combination antiretroviral therapy (cART) can stop HIV-1 from reproducing, it can't get rid of the proviral DNA that is embedded in the human genome. Because of this, it needs to be taken for the rest of one's life and may cause side effects. Cas9-related gene-editing systems, which are clustered regularly interspaced short palindromic repeat (CRISPR)-associated nuclease 9 (Cas9) related, have been developed and designed as effective treatments for HIV-1 infection in recent years. However, new gene-targeting tools, such as base editor, prime editor, SHERLOCK, DETECTR, PAC-MAN, ABACAS, and pfAGO, have been developed and improved for the purpose of detecting pathogens and correcting diseases. These tools are derived from or function in the same way as CRISPR/Cas9. On the basis of research on the molecular basis of HIV-1 infection, we provide additional gene-editing targets and a summary of recent research on HIV-1/AIDS gene therapy. Additionally, we identify the strategies and potential applications of these brand-new gene-editing technologies for future HIV/AIDS treatment. In addition, we discuss the limitations and issues that need to be resolved prior to the clinical application of these adaptable CRISPR-based gene targeting tools. In conclusion, we present alternative strategies for enhancing gene targeting in HIV/AIDS gene therapy.

**Keywords:** Gene therapy • Gene editing • Effective treatments for HIV

## Introduction

Since it was first reported by the Centers for Disease Control and Prevention (CDC) in the United States in the early 1980s, acquired immunodeficiency syndrome (AIDS), which is caused by HIV-1 infection, has posed a significant threat to human health worldwide. HIV-1 is a retrovirus with two copies of approximately 9.8 kb of full-length genomic RNA that contains two long terminal repeat (LTR) sequences and genes encoding ten viral proteins, including antisense protein (ASP), gag, pol, vif, vpr, vpu, env, tat, rev, and nef (Liu et al., 2019). After binding to the CD4 main receptor on the membrane of the target cell with its envelope surface protein gp120, HIV-1 invades the cell and interacts with the chemokine co-receptors CCR5 or CXCR4. According to CCR5 is the primary co-receptor for CCR5 (R5)-tropic HIV-1 entry into CD4+ T cells, monocytes, and lymphocytes. 2008), CXCR4 serves as a co-receptor for CXCR4 (X4)-tropic viral strains. HIV-1 has evolved to use CXCR4 as an alternative co-receptor for viral entry during the late stage of R5-tropic infection or when CCR5 is destroyed -1 enters cells through membrane fusion and binds to the receptors. RNA reverse transcriptase transforms the viral genomic RNA into double-stranded DNA (dsDNA). After that, the dsDNA fuses with the host genome to form a provirus that can either enter a latent state without producing viruses or actively transcribe RNA to make new viruses. During the earliest stages of HIV infection, resting CD4+ T cells become latent HIV reservoirs. Chun and others, Macrophages, microglial cells and astrocytes and as a result, evade the body's defenses and antiviral medications. New viruses can be produced to infect adjacent cells and new latent reservoirs can be established once the latent provirus is reactivated by stimuli. Additionally, it has been

demonstrated that HIV-1 can enter cells through endocytosis; however, this is a contentious claim that requires additional evidence. HIV-1 requires the use of essential host factors like transportin-3 and lens epithelium-derived growth factor in order to complete the replication, packaging, and budding processes. Primary CD4+ T cells are the primary targets of HIV-1, which increases the likelihood of opportunistic infections, other infectious diseases, and some types of cancer. In the central nervous system, HIV-1 can also infect monocytes, dendritic cells, microglia, astrocytes, and perivascular macrophages.

## Discussion

Currently, combined antiretroviral therapy (cART), a combination of multiple drugs that effectively inhibit the functions of various viral proteins during HIV-1 replication, is the primary treatment for HIV/AIDS. However, cART has many disadvantages, such as lifelong treatment, high cost, chronic liver disease, damage to the cardiovascular and cerebrovascular systems, complications in aging, and neurological disorders. As a result, more efficient approaches to treating HIV/AIDS patients and removing latent HIV-1 provirus need to be developed. The "Berlin patient" with AIDS and acute myelocytic leukemia (AML) was functionally cured after accepting a CCR5 32 genotype of bone marrow transplantation, according to clinical research. [1,2].

## Conclusion

More recently, a clinical report on the "London patient" with Hodgkin's lymphoma and AIDS provided similar evidence of curative treatment. In addition, reported the case of a "Beijing patient" with AIDS and AML who was transplanted with CRISPR/Cas9-disrupted hematopoietic stem cells. The AML got better; However, due to the low efficiency of gene editing, HIV-1 replication was only suppressed, and an AIDS cure was not achieved. These clinical cases suggest that, at this time, gene editing treatment is not appropriate for all AIDS patients and should only be considered as a last resort for patients who also have hematological malignancies that pose a threat to their life. Gene therapy, on the other hand, has the potential to be an effective treatment for HIV/AIDS in the future if we are able to overcome all of its limitations and guarantee that it will be effective and safe [3-5].

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## Conflict of Interest

None.

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