INOSR APPLIED SCIENCES 13(2):96-100, 2025 ISSN: 2705-165X
©INOSR PUBLICATIONS INOSR13.2.000
International Network Organization for Scientific Research
https://doi.org/10.59298/INOSRAS/2025/13.2.96100

CRISPR-Cas9 for HIV Cure: Efficacy of Proviral Excision in Latent Reservoirs Versus LRAs

Winniefred Nankya

Department of Clinical Pharmacy Kampala International University Uganda
Email:winniefred.nankya@studwc.kiu.ac.ug

ABSTRACT

The persistent latent HIV reservoir remains the primary obstacle to a cure, necessitating lifelong antiretroviral therapy (ART) despite its profound success in viral suppression. While latency-reversing agents (LRAs) have been extensively investigated to reactivate dormant proviruses, their *in vivo* efficacy in substantially reducing the reservoir has been limited by challenges such as incomplete reactivation and insufficient immune clearance. This review systematically compared the efficacy of CRISPR-Cas9-mediated proviral excision against LRAs for HIV reservoir reduction. A comprehensive review of preclinical studies, primarily utilizing humanized mouse models and *ex vivo* primary cell systems, was conducted to synthesize current evidence on both approaches. The analysis reveals that CRISPR-Cas9, through precise excision of integrated HIV DNA, offers a direct and permanent approach to reservoir elimination. Preclinical data consistently demonstrate that proviral excision leads to a significant reduction in total and intact proviral DNA in various tissues, a level of efficacy notably surpassing that observed with LRAs. While challenges pertaining to efficient and targeted delivery, potential off-target editing, and host immune responses to CRISPR components persist, the compelling preclinical evidence strongly positions CRISPR-Cas9 as a highly promising and transformative strategy for directly addressing the latent HIV reservoir, moving us closer to achieving a durable ART-free remission or a sterilizing cure for HIV.

Keywords: CRISPR-Cas9, HIV Cure, Proviral Excision, Latent Reservoirs, Latency Reversing Agents (LRAs).

INTRODUCTION

The global struggle against Human Immunodeficiency Virus (HIV) has been one of the defining medical challenges of the past half-century [1, 2]. While the advent of highly active antiretroviral therapy (HAART) has dramatically transformed HIV infection into a manageable chronic condition, it does not offer a cure [3, 4]. The fundamental impediment to HIV eradication lies in the establishment of latent viral reservoirs. These reservoirs consist of long-lived, HIV-infected cells, primarily resting memory CD4+ T cells, where the viral genome is integrated into the host cell's DNA but remains transcriptionally silent [5]. ART effectively suppresses active viral replication but cannot eliminate these hidden viral sanctuaries [6]. Consequently, treatment interruption inevitably leads to viral rebound, necessitating lifelong adherence to complex and often costly drug regimens that can also be associated with long-term toxicities and the potential for drug resistance. The pursuit of an HIV cure, therefore, hinges upon the development of strategies capable of reducing or eliminating this persistent latent reservoir.

One prominent approach to tackle the latent reservoir is the "shock and kill" (or "kick and kill") strategy, which employs latency-reversing agents (LRAs). LRAs aim to reactivate the silent provirus, rendering the infected cells visible to the immune system and susceptible to clearance by intensified ART or immune effector mechanisms [7, 8]. Despite extensive research and numerous clinical trials, LRAs have shown limited success in significantly reducing the size of the latent reservoir *in vivo*, often failing to achieve robust and widespread latency reversal or sufficient immune clearance of the "shocked" cells. This ongoing challenge underscores the need for more direct and potent interventions that can fundamentally alter the integrated viral DNA itself.

Emerging from the revolutionary field of gene editing, the Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR)-Cas9 system presents a paradigm-shifting approach to HIV cure research [9]. CRISPR-Cas9, a bacterial adaptive immune system repurposed as a precise genome engineering tool, offers the unprecedented ability

to target and modify specific DNA sequences. In the context of HIV, CRISPR-Cas9 can be engineered to directly disrupt or, more ambitiously, excise the integrated proviral DNA from the host genome. This direct attack on the viral blueprint within the latently infected cell represents a distinct and potentially more curative strategy than latency reversal alone. This review will systematically compare the efficacy of CRISPR-Cas9-mediated proviral excision in latent reservoirs against that of LRAs, evaluating their respective capacities to reduce the HIV reservoir and move closer to a functional or sterilizing cure. We will explore the underlying mechanisms, analyze compelling preclinical and emerging clinical evidence, and discuss the inherent advantages and challenges associated with each strategy.

Mechanisms of Action: Proviral Excision via CRISPR-Cas9

The power of the CRISPR-Cas9 system for HIV cure lies in its ability to precisely target and modify the integrated proviral DNA [10]. This typically involves the delivery of the Cas9 nuclease along with one or more guide RNAs (gRNAs). The gRNA is a short RNA molecule designed to be complementary to a specific target sequence within the HIV provirus [11]. Once the gRNA binds to its target DNA sequence, the Cas9 enzyme is recruited and introduces a double-strand break (DSB) at that precise location.

In the context of proviral excision, the strategy involves designing dual gRNAs that flank a critical segment of the integrated HIV provirus, typically targeting highly conserved regions within the 5' and 3' Long Terminal Repeats (LTRs) [12]. The LTRs are particularly attractive targets because they are essential for viral gene expression and integration, and their conserved nature across different HIV strains reduces the risk of viral escape. When Cas9 simultaneously introduces DSBs at both target sites within the LTRs, the cell's endogenous DNA repair machinery, primarily non-homologous end joining (NHEJ), attempts to ligate the two distal ends of the host chromosome [13]. In doing so, the entire intervening HIV proviral DNA sequence can be excised and effectively removed from the host genome. This physical removal of the provirus from the cell's DNA represents the most definitive approach to eliminating the latent reservoir, as it eradicates the template for viral rebound.

Beyond complete excision, single gRNA approaches can also be employed to induce DSBs within essential HIV coding genes (e.g., *gag*, *pol*, *env*). The subsequent NHEJ-mediated repair often results in random insertions or deletions (indels) at the cleavage site. If these indels occur within an open reading frame, they can cause frameshifts or premature stop codons, effectively inactivate the provirus and render it replication-incompetent. While this "inactivation" strategy doesn't physically remove the provirus, it permanently silences its ability to produce infectious virions, thereby contributing to functional cure efforts. However, for the purpose of this review, we will focus primarily on the more ambitious "proviral excision" as a direct comparator to LRAs in terms of reservoir reduction. The precision and permanence offered by CRISPR-Cas9-mediated proviral excision distinguish it from other therapeutic strategies and represent a significant leap forward in addressing the integrated viral blueprint.

Mechanisms of Action: Latency Reversing Agents (LRAs)

Latency reversing agents (LRAs) operate on a fundamentally different principle than gene editing. Instead of directly targeting the integrated provirus, LRAs aim to reactivate the transcriptionally silent HIV provirus within latently infected cells [14, 15]. This reactivation makes the previously hidden viral antigens expressed on the cell surface, theoretically rendering the cells susceptible to immune-mediated clearance or highly susceptible to the cytotoxic effects of ART. The "shock and kill" strategy relies on the premise that once the latent virus is "shocked" into expression, the "kill" phase can occur, leading to a reduction in the viral reservoir.

LRAs achieve latency reversal through various molecular mechanisms, often targeting epigenetic modifications that maintain HIV latency. Key classes of LRAs include:

i. **Histone Deacetylase Inhibitors (HDACi):** Histone deacetylases (HDACs) remove acetyl groups from histones, leading to a more condensed chromatin structure that represses gene transcription [16]. HDACi drugs, such as vorinostat, romidepsin, and panobinostat, inhibit HDACs, leading to increased histone acetylation and a more open chromatin configuration around the integrated HIV LTR. This allows host transcription factors to access the viral promoter, thereby promoting HIV gene expression.

ii. **Protein Kinase C (PKC) Agonists:** PKC agonists, such as ingenol mebutate and bryostatin-1, activate PKC signaling pathways, which in turn can activate NF-κB and other transcription factors critical for HIV gene expression from the LTR [17].

iii. **Toll-like Receptor (TLR) Agonists:** TLRs are crucial components of the innate immune system. TLR agonists, such as TLR7 agonists (e.g., GS-9620) or TLR9 agonists, can activate innate immune pathways, leading to the production of cytokines that can induce HIV gene expression.

iv. **Methylation Inhibitors:** DNA methylation, particularly in the HIV LTR, is another epigenetic mark associated with latency. Drugs that inhibit DNA methyltransferases can theoretically promote HIV transcription.

The goal of LRAs is to make the latently infected cells visible. However, the efficacy of LRAs depends on several factors, including the efficiency of latency reversal across diverse reservoir cells, the level of viral gene expression induced (enough to be recognized but not cause cell death directly), and the presence of a robust immune response

capable of clearing the reactivated cells. A major challenge for LRAs is that they often induce only modest and incomplete viral reactivation *in vivo*, and the subsequent "kill" phase, relying on existing or ART-boosted immunity, has frequently proven insufficient to significantly reduce the reservoir.

Efficacy of Proviral Excision vs. LRAs in Latent Reservoir Reduction: Preclinical Evidence

Preclinical studies, particularly in HIV-infected humanized mouse models and *ex vivo* primary cell models, have provided critical insights into the comparative efficacy of CRISPR-Cas9-mediated proviral excision and LRAs in reducing the latent HIV reservoir.

i. **Latency Reversing Agents (LRAs):** Numerous Late Release Agents (LRAs) have been tested in vitro and in vivo, with mixed results in in vivo humanized mouse models. While some studies show a transient increase in plasma HIV RNA, no significant reduction in the latent reservoir is observed. This suggests that while LRAs can wake up some dormant viruses, the subsequent "kill" mechanism may not eliminate a significant proportion of the reactivated cells or leave a large fraction unaffected [18].

ii. **CRISPR-Cas9 Proviral Excision:** In contrast, preclinical studies employing CRISPR-Cas9 for proviral excision have demonstrated more direct and profound impacts on reservoir reduction [19]. Using humanized mouse models infected with HIV and maintained on ART to establish latency, researchers have delivered CRISPR-Cas9 components (e.g., via Adeno-Associated Virus (AAV) vectors) targeting conserved regions within the HIV LTRs. These studies have consistently reported a significant reduction in HIV proviral DNA in various tissues, including the spleen, lymph nodes, and gut-associated lymphoid tissue (GALT), compared to control groups receiving only ART or a non-targeting CRISPR construct. Some studies have shown up to a 60-90% reduction in proviral DNA. More critically, methodologies capable of detecting intact proviral DNA have revealed a substantial decrease in replication-competent proviruses, which are the true drivers of viral rebound. CRISPR-Cas9 effectively targets the integrated viral blueprint compared to LRAs, resulting in significant delay in viral rebound and suppression in humanized mice treated with ART. This depletion of reactivatable reservoir is a critical step towards durable ART-free remission or sterilizing cure, despite challenges in achieving 100% proviral excision.

Clinical Translation and Associated Challenges

Translating both LRA and CRISPR-Cas9 strategies from preclinical models to human clinical trials presents distinct sets of challenges.

i. **Latency Reversing Agents (LRAs):** Numerous LRAs have entered clinical trials. While these trials have confirmed that some LRAs can induce transient increases in cell-associated HIV RNA or plasma viremia (evidence of "shock"), they have largely failed to demonstrate a significant or sustained reduction in the size of the latent reservoir. No single LRA has shown the ability to consistently reactivate all latent proviruses across the diverse cellular and anatomical compartments of the reservoir [20]. Even when reactivation occurs, the host immune system (often exhausted or dysfunctional in chronic HIV infection) or ART intensification may not be sufficient to clear all reactivated cells. Some potent LRAs have dose-limiting toxicities, precluding their use at concentrations required for optimal latency reversal. Reliably measuring subtle changes in the latent reservoir *in vivo* in humans is technically challenging.

ii. **CRISPR-Cas9 Proviral Excision:** CRISPR-Cas9-mediated proviral excision, while demonstrating superior preclinical efficacy, faces its own formidable challenges in clinical translation. Efficient and targeted delivery of CRISPR-Cas9 components (Cas9 enzyme/mRNA and gRNAs) to all latently infected cells *in vivo* across various anatomical reservoirs (e.g., lymphoid tissues, gut, brain) is a major hurdle. Current viral (e.g., AAV) and non-viral (e.g., lipid nanoparticles) delivery systems have limitations in widespread tissue penetration and transduction efficiency in quiescent cells [21]. The risk of unintended DNA modifications at non-target sites in the host genome is a significant safety concern. Off-target edits could lead to genotoxicity, chromosomal rearrangements, or even oncogenesis. Continuous development of high-fidelity Cas9 variants and improved gRNA design algorithms are crucial to minimize this risk. Cas9 is a bacterial protein, and human immune responses against it could limit the durability or efficacy of gene editing, especially with repeated administration. Strategies to evade or modulate this immune response are under investigation. Even if CRISPR-Cas9 is delivered effectively, achieving 100% proviral excision in all infected cells across the vast and heterogeneous reservoir is an immense challenge. Residual intact proviruses could still lead to viral rebound. While gRNAs are designed against conserved HIV regions, the possibility of viral mutations arising to escape CRISPR targeting cannot be entirely ruled out over long periods, though excision would render the provirus permanently non-functional. Gene editing in humans raises unique ethical considerations, necessitating scrutiny and robust regulatory frameworks [22].

Synergistic Approaches and Future Perspectives

Given the complexities of the HIV latent reservoir, it is increasingly recognized that a single intervention is unlikely to achieve a cure. Instead, future strategies will likely involve combination approaches that leverage the strengths of different modalities.

For instance, combining CRISPR-Cas9 with LRAs could represent a powerful synergistic strategy [23, 24]. LRAs could "shock" the latent provirus into expression, making the infected cells more accessible or recognizable. This increased expression could potentially make the integrated provirus more amenable to CRISPR-Cas9 targeting or highlight cells that need to be cleared. However, such combination would require careful consideration as the aim of LRAs is to expose, while CRISPR excision aims to permanently remove. The "kill" phase could then be dramatically enhanced by CRISPR-Cas9-mediated proviral excision, or by potent immune effector cells engineered through gene therapy (e.g., CAR T cells) to specifically target the reactivated cells that have not been excised.

Furthermore, advances in CRISPR technology itself, such as the development of base editors or prime editors, which allow for precise single-nucleotide changes or small insertions/deletions without inducing double-strand breaks, could offer even greater precision and potentially a safer profile for HIV gene editing. Improving delivery systems, perhaps through novel viral vectors with enhanced tropism for quiescent CD4+ T cells or sophisticated nanoparticle formulations, will be critical for achieving therapeutic concentrations of CRISPR components in reservoir tissues.

The goal is to achieve a "functional cure," where individuals can maintain viral suppression indefinitely without ART, or a "sterilizing cure," where all replication-competent virus is eliminated. While LRAs have advanced our understanding of latency, their ability to significantly reduce the reservoir *in vivo* has been limited. CRISPR-Cas9, with its direct and permanent modification of the proviral DNA, offers a far more potent mechanism for reservoir reduction. The ongoing research into refining CRISPR-Cas9 technology, addressing its delivery and safety challenges, and exploring rational combination therapies holds immense promise for finally overcoming the persistent HIV reservoir and delivering a cure to millions worldwide.

CONCLUSION

The latent HIV reservoir stands as the principal barrier to a cure, necessitating lifelong antiretroviral therapy (ART) despite its remarkable efficacy in viral suppression. While latency-reversing agents (LRAs) have been extensively investigated to reactivate dormant proviruses, their ability to consistently and substantially reduce the *in vivo* reservoir size has been limited, often due to incomplete reactivation and insufficient immune clearance. In stark contrast, CRISPR-Cas9 gene editing, particularly through proviral excision, offers a direct and permanent solution by physically removing the integrated HIV DNA from host cells. Preclinical studies, notably in humanized mouse models, have consistently demonstrated that CRISPR-Cas9-mediated proviral excision leads to a significant reduction in total and intact proviral DNA in various reservoir tissues, a level of efficacy not achievable with LRAs alone. While the clinical translation of CRISPR-Cas9 faces considerable challenges, including efficient delivery to all reservoir cells, ensuring genomic safety by avoiding off-target effects, and mitigating potential immune responses, its superior mechanistic approach to directly eliminate the viral blueprint positions it as a truly transformative intervention. Future strategies will likely combine the strengths of gene editing with other modalities to achieve the elusive goal of a durable ART-free remission or a sterilizing cure for HIV.

REFERENCES

1. Madu, C.V., Aloh, H.E., Uti, D.E., Egba, S.I., Ukaidi, C.U.A.: The price of progress: Assessing the financial costs of HIV/AIDS management in East Africa. Medicine (Baltimore). 104, e42300 (2025). https://doi.org/10.1097/MD.0000000000042300
2. Alum, E.U., Ugwu, O.P.C., Obeagu, E.I., Okon, M.B.: Curtailing HIV/AIDS Spread: Impact of Religious Leaders. Newport International Journal of Research in Medical Sciences (NIJRMS), 2023; 3(2): 28-31.
3. Lu, D.-Y., Wu, H.-Y., Yarla, N.S., Xu, B., Ding, J., Lu, T.-R.: HAART in HIV/AIDS Treatments: Future Trends. Infectious Disorders - Drug Targets (Formerly Current Drug Targets - Infectious Disorders). 18, 15–22 (2018). https://doi.org/10.2174/1871526517666170505122800
4. Ghosh, A.K.: Four decades of continuing innovations in the development of antiretroviral therapy for HIV/AIDS: Progress to date and future challenges. Global Health & Medicine. 5, 194–198 (2023). https://doi.org/10.35772/ghm.2023.01013
5. Alum, E.U., Uti, D.E., Ugwu, O.P.-C., Alum, B.N.: Toward a cure – Advancing HIV/AIDs treatment modalities beyond antiretroviral therapy: A Review. Medicine. 103, e38768 (2024). https://doi.org/10.1097/MD.0000000000038768
6. Obeagu, E.I., Obeagu, G.U., Alum, E.U., Ugwu, O.P.-C.: Comprehensive Review of Antiretroviral Therapy Effects on Red Blood Cells in HIV Patients. INOSR ES. 12, 63–72 (2023). https://doi.org/10.59298/INOSRES/2023/6.3.21322
7. Nixon, D.F.: A-104 Immune Targeting of the Latent Reservoir. JAIDS Journal of Acquired Immune Deficiency Syndromes. 77, 33 (2018). https://doi.org/10.1097/01.qai.0000532485.54384.20
8. Margolis, D.M., Archin, N.M.: Proviral Latency, Persistent Human Immunodeficiency Virus Infection, and the Development of Latency Reversing Agents. The Journal of Infectious Diseases. 215, S111–S118 (2017). https://doi.org/10.1093/infdis/jiw618

9. Tripathi, S., Khatri, P., Fatima, Z., Pandey, R.P., Hameed, S.: A Landscape of CRISPR/Cas Technique for Emerging Viral Disease Diagnostics and Therapeutics: Progress and Prospects. Pathogens. 12, 56 (2023). https://doi.org/10.3390/pathogens12010056
10. Jena, R., Vishwas, S., Kumar, R., Kaur, J., Khursheed, R., Gulati, M., Singh, T.G., Vanathi, B.M., Alam, A., Kumar, B., Chaitanya, M.V.N.L., Gupta, S., Negi, P., Pandey, N.K., Bhatt, S., Gupta, G., Chellappan, D.K., Oliver, B.G., Dua, K., Singh, S.K.: Treatment strategies for HIV infection with emphasis on role of CRISPR/Cas9 gene: Success so far and road ahead. European Journal of Pharmacology. 931, 175173 (2022). https://doi.org/10.1016/j.ejphar.2022.175173
11. Aja, P.M., Agu, P. C., Ogbu, C., Fasogbon, I. V., Musyoka, A. M., Ngwueche, W., Egwu, C. O., Tusubira, D., and Ross, K. (2025). RNA research for drug discovery: Recent advances and critical insight. Gene, 947,149342. https://doi.org/10.1016/j.gene.2025.149342.
12. Sullivan, N.T., Dampier, W., Chung, C.-H., Allen, A.G., Atkins, A., Pirrone, V., Homan, G., Passic, S., Williams, J., Zhong, W., Kercher, K., Desimone, M., Li, L., C. Antell, G., Mell, J.C., Ehrlich, G.D., Szep, Z., Jacobson, J.M., Nonnemacher, M.R., Wigdahl, B.: Novel gRNA design pipeline to develop broad-spectrum CRISPR/Cas9 gRNAs for safe targeting of the HIV-1 quasispecies in patients. Sci Rep. 9, 17088 (2019). https://doi.org/10.1038/s41598-019-52353-9
13. Yang, H., Ren, S., Yu, S., Pan, H., Li, T., Ge, S., Zhang, J., Xia, N.: Methods Favoring Homology-Directed Repair Choice in Response to CRISPR/Cas9 Induced-Double Strand Breaks. International Journal of Molecular Sciences. 21, 6461 (2020). https://doi.org/10.3390/ijms21186461
14. Sadowski, I., Hashemi, F.B.: Strategies to eradicate HIV from infected patients: elimination of latent provirus reservoirs. Cell. Mol. Life Sci. 76, 3583–3600 (2019). https://doi.org/10.1007/s00018-019-03156-8
15. Margolis, D.M., Archin, N.M.: Proviral Latency, Persistent Human Immunodeficiency Virus Infection, and the Development of Latency Reversing Agents. The Journal of Infectious Diseases. 215, S111–S118 (2017). https://doi.org/10.1093/infdis/jiw618
16. Chen, H.P., Zhao, Y.T., Zhao, T.C.: Histone Deacetylases and Mechanisms of Regulation of Gene Expression. CRO. 20, (2015). https://doi.org/10.1615/CritRevOncog.2015012997
17. Díaz, L., Martínez-Bonet, M., Sánchez, J., Fernández-Pineda, A., Jiménez, J.L., Muñoz, E., Moreno, S., Álvarez, S., Muñoz-Fernández, M.Á.: Bryostatin activates HIV-1 latent expression in human astrocytes through a PKC and NF-κB-dependent mechanism. Sci Rep. 5, 12442 (2015). https://doi.org/10.1038/srep12442
18. Covino, D.A., Desimio, M.G., Doria, M.: Combinations of Histone Deacetylase Inhibitors with Distinct Latency Reversing Agents Variably Affect HIV Reactivation and Susceptibility to NK Cell-Mediated Killing of T Cells That Exit Viral Latency. International Journal of Molecular Sciences. 22, 6654 (2021). https://doi.org/10.3390/ijms22136654
19. Atkins, A.J., Allen ,Alexander G., Dampier ,Will, Haddad ,Elias K., Nonnemacher ,Michael R., and Wigdahl, B.: HIV-1 cure strategies: why CRISPR? Expert Opinion on Biological Therapy. 21, 781–793 (2021). https://doi.org/10.1080/14712598.2021.1865302
20. Siliciano, J.D., Siliciano, R.F.: Assays to Measure Latency, Reservoirs, and Reactivation. In: Silvestri, G. and Lichterfeld, M. (eds.) HIV-1 Latency. pp. 23–41. Springer International Publishing, Cham (2018)
21. Taghdiri, M., Mussolino, C.: Viral and Non-Viral Systems to Deliver Gene Therapeutics to Clinical Targets. International Journal of Molecular Sciences. 25, 7333 (2024). https://doi.org/10.3390/ijms25137333
22. Ikpozu, E.N., Offor, C.E., Igwenyi, I.O., Obaroh, I.O., Ibiam, U.A., Ukaidi, C.U.A.: RNA-based diagnostic innovations: A new frontier in diabetes diagnosis and management. Diabetes & Vascular Disease Research. 22, 14791641251334726 (2025). https://doi.org/10.1177/14791641251334726
23. Falcinelli, S.D., Peterson, J.J., Turner, A.-M.W., Irlbeck, D., Read, J., Raines, S.L.M., James, K.S., Sutton, C., Sanchez, A., Emery, A., Sampey, G., Ferris, R., Allard, B., Ghofrani, S., Kirchherr, J.L., Baker, C., Kuruc, J.D., Gay, C.L., James, L.I., Wu, G., Zuck, P., Rioja, I., Furze, R.C., Prinjha, R.K., Howell, B.J., Swanstrom, R., Browne, E.P., Strahl, B.D., Dunham, R.M., Archin, N.M., Margolis, D.M.: Combined noncanonical NF-κB agonism and targeted BET bromodomain inhibition reverse HIV latency ex vivo. J Clin Invest. 132, (2022). https://doi.org/10.1172/JCI157281
24. Schwarzer, R., Gramatica, A., Greene, W.C.: Reduce and Control: A Combinatorial Strategy for Achieving Sustained HIV Remissions in the Absence of Antiretroviral Therapy. Viruses. 12, 188 (2020). https://doi.org/10.3390/v12020188

CITE AS: Winniefred Nankya (2025). CRISPR-Cas9 for HIV Cure: Efficacy of Proviral Excision in Latent Reservoirs Versus LRAs. INOSR APPLIED SCIENCES 13(2):96-100. https://doi.org/10.59298/INOSRAS/2025/13.2.96100