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A Current Review of Molecular Mechanisms and Therapeutic Strategies for HIV-1 Latency

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Introduction

Human Immunodeficiency Virus Type remains one of the most significant global public health challenges. While antiretroviral therapyhas drastically improved the quality of life and life expectancy of individuals living with HIV-1, it does not cure the infection. This is largely due to the phenomenon of HIV-1 latency, where the virus persists in a dormant state in certain reservoirs within the body despite prolonged ART treatment. This latency is the major barrier to eradicating the virus. Understanding the molecular mechanisms behind HIV-1 latency and developing therapeutic approaches to overcome it are crucial areas of ongoing research. This review aims to explore the mechanisms underlying HIV-1 latency and examine the latest therapeutic strategies designed to target latent HIV-1 reservoirs, with the goal of achieving a functional cure or complete eradication of the virus. HIV-1 latency refers to the ability of the virus to persist in a quiescent, non-replicating state within the host's immune cells, particularly in T cells. These latently infected cells can survive for extended periods without actively producing new virions. The latent reservoir is a major challenge in the quest to cure HIV because even if the virus is undetectable in the plasma, it can rebound if ART is interrupted [1-3].

Description

HIV-1 latency presents a significant challenge to the eradication of the virus, as the latent reservoirs maintain persistent infection despite ART. The molecular mechanisms underlying HIV-1 latency are complex and involve interactions between viral and host factors, including chromatin structure, transcriptional regulation, immune modulation, and cell differentiation. Understanding these mechanisms is crucial for developing therapeutic strategies aimed at eliminating the latent reservoirs.

Current therapeutic approaches, such as latency-reversing agents, immune modulation, gene editing, and stem cell transplantation, offer promising avenues for addressing HIV-1 latency. However, significant hurdles remain, particularly in ensuring that reactivated virus is effectively cleared and that latent reservoirs are completely eradicated. Ongoing research and clinical trials will be essential in advancing these strategies toward the goal of a functional cure or complete eradication of HIV-1 [4,5].

Conclusion

As we continue to expand our knowledge of HIV-1 latency and refine therapeutic approaches, the possibility of achieving a cure for HIV-1 moves closer to reality. However, a more comprehensive understanding of the viral and host factors involved in latency, coupled with the development of innovative therapeutic strategies, will be key to overcoming this major barrier in the fight against HIV/AIDS. o Histone Deacetylase Inhibitors (HDACi): HDAC inhibitors, such as vorinostat and romidepsin, can induce the reactivation of latent HIV-1 by altering the chromatin structure surrounding the integrated provirus. These agents increase the acetylation of histones, thereby making the chromatin more accessible to the transcriptional machinery and promoting viral transcription. PKC activators, such as prostratin and bryostatin-1, can stimulate the activation of latent HIV-1 through the activation of the NF-κB pathway, which is essential for HIV transcription. Other compounds, such as the bromodomain and extraterminal domain inhibitors and Toll-like receptor agonists, have also been explored as potential LRAs. These agents work by targeting different pathways that control viral transcription.

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Conflict of Interest

None.

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