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HIV: Current Research

Perspective

# HIV-2 Tropism and Disease: Molecular Interactions between Lentiviruses and Macrophages

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## DESCRIPTION

Human Immunodeficiency Virus type 2 (HIV-2), while less prevalent globally compared to HIV-1, poses significant clinical challenges, particularly in its interaction with host cells and its role in disease progression. HIV-2, a lentivirus, exhibits distinct tropism and pathogenic mechanisms compared to HIV-1, especially concerning its interactions with macrophages. This article delves into the molecular interactions between HIV-2 and macrophages, examining the implications for disease progression and potential therapeutic strategies. HIV-2 primarily infects $CD4^{+}$ T cells and macrophages. Unlike HIV-1, HIV-2 has a broader range of co-receptors, including CCR5 and CXCR4, which influence its tropism. The differential utilization of co-receptors by HIV-2 impacts its tissue distribution and pathogenic potential. HIV-2 shows a preference for CCR5, though some strains also utilize CXCR4. The preference for CCR5 can influence the virus's ability to infect various tissues and contribute to its overall pathogenicity. CCR5-tropic strains are associated with macrophage infection, while CXCR4-tropic strains can be more pathogenic in the later stages of infection, affecting CD4+ T cells more significantly. Macrophages are crucial target cells for HIV-2. The virus's ability to infect macrophages is facilitated by the interaction between viral envelope proteins and cellular receptors. HIV-2's envelope glycoprotein (gp120) binds to CD4 and co-receptors on macrophages, leading to viral entry and subsequent replication. The interaction between HIV-2 and macrophages is complex and involves several key molecular mechanisms:

The process begins with the binding of HIV-2 gp120 to the CD4 receptor on macrophages. This interaction induces conformational changes in the gp120 protein, facilitating the binding to the CCR5 co-receptor. The subsequent interaction between the viral envelope and the macrophage membrane enables fusion, allowing viral entry. Upon entry, HIV-2 activates several cellular signaling pathways within macrophages. This includes the activation of Nuclear Factor-kappa B (NF-κB), which promotes the transcription of inflammatory cytokines and facilitates viral replication. HIV-2 also induces the expression of Matrix Metallo Proteinases (MMPs) in macrophages, which can contribute to tissue damage and inflammation.

HIV-2-infected macrophages produce various cytokines and chemokines, including Tumor Necrosis Factor-alpha (TNF-α) and Inter Leukin-6 (IL-6), which contribute to chronic inflammation and tissue damage. HIV-2 can impair macrophage antigen presentation by affecting Major Histocompatibility Complex (MHC) class II expression, leading to reduced T cell activation and immune response. HIV-2 can establish a persistent infection in macrophages, contributing to viral reservoirs that are difficult to eradicate. The virus uses mechanisms such as downregulation of CD4 and co-receptors to evade immune detection. Additionally, HIV-2's ability to replicate within macrophages contributes to viral persistence and the establishment of a chronic infection.

HIV-2 infection of macrophages contributes to disease progression by promoting inflammation and tissue damage. The persistent activation of macrophages and the production of pro-inflammatory cytokines can lead to chronic immune activation, contributing to disease progression and the development of AIDS. HIV-2-induced inflammation and activation of macrophages can result in tissue damage, particularly in the lymph nodes and other sites of viral replication. The production of matrix metalloproteinases and cytokines can lead to fibrosis and disruption of tissue architecture. HIV-2 infection affects the overall function of the immune system by impairing macrophage-mediated antigen presentation and cytokine production. This can lead to a weakened immune response and increased susceptibility to opportunistic infections.

Developing inhibitors that block the interaction between HIV-2 gp120 and CD4 or co-receptors could prevent viral entry into macrophages. Entry inhibitors that specifically target the HIV-2 envelope may provide an effective means to reduce viral replication and infection. Therapeutic approaches aimed at modulating macrophage activation and reducing inflammation could mitigate the pathogenic effects of HIV-2. Anti-inflammatory agents and cytokine inhibitors may help reduce tissue damage and improve disease outcomes. Strategies to target

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and eliminate viral reservoirs within macrophages are crucial for achieving a functional cure. Approaches such as shock-and-kill strategies, which involve activating latent virus and subsequently targeting it with antiretroviral therapy, may hold promise. Boosting the immune response through immunotherapy or vaccines could improve the body's ability to control HIV-2 infection and reduce the impact of macrophage-associated inflammation.

## CONCLUSION

Unspliced HIV-2's interaction with macrophages plays a critical role in disease progression and pathogenesis. Understanding the molecular mechanisms underlying HIV-2 tropism and macrophage infection provides valuable insights into potential therapeutic strategies. By targeting viral entry, modulating macrophage activation, eradicating viral reservoirs, and enhancing immune responses, researchers and clinicians can work towards more effective treatments and ultimately, better management of HIV-2 infection. Continued research is essential to resolve the complexities of HIV-2 interactions with host cells and to develop innovative approaches for combating this challenging virus.