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HIV: Current Research

Perspective

Establishment of Innovative HIV Reservoir-Elimination Methods

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DESCRIPTION

Antiretroviral treatment for Human Immunodeficiency Virus (HIV) is efficient in controlling viral replication but cannot totally remove HIV due to the HIV reservoir's durability. It has been claimed that innate and adaptive immune responses can help prevent HIV acquisition, limit HIV replication, and eliminate HIV-infected cells. However, spontaneously produced immune responses in HIV-infected patients seldom eliminate HIV infection, which may be due to immunological escape, insufficient amplitude and breadth of immune responses, and immune fatigue. Optimizing these immune responses may be able to address the issues of epitope escape and inadequate persistent memory responses. Furthermore, immunological therapies focused at enhancing host immune response can lower HIV reservoirs, which has been one in the development of novel HIV reservoir-eradication techniques. The immune system's reaction to HIV and how antiviral immune responses impact HIV reservoirs are the subject of this work. It also discusses about developing novel techniques to eradicate HIV reservoirs and promote functional cure of HIV infection. Despite the fact that effective antiretroviral medication can lower HIV replication, viremia returns within weeks following Antiretroviral Therapy (ART) discontinuation. This viral rebound might be powered by reservoirs that form early in HIV infection and are regulated by a variety of virological and immunological variables. The revival of transcriptionally quiet but replication-competent viruses harboured in latently infected cells may be a cause of viremia.

Furthermore, earlier research has revealed that HIV reservoirs degrade relatively slowly, having a half-life of about 44 months. As a result, individuals living with HIV cannot be cured by ART alone and must undergo ART for the rest of their lives.

A replication-competent variant of HIV lives longer in a specific cell type or anatomical region than in the main pool of actively replicating viruses, generating HIV reservoirs. Despite the fact that there are various reservoirs, the persistence of replication-competent HIV in resting memory CD4+ T cells remains a key barrier to HIV elimination. Furthermore, HIV latency can be established in resting CD4+ T cells. In addition to generating reservoirs in all CD4+ T cell subsets, HIV survives in macrophages, Dendritic Cells (DCs), and astrocytes, all of which can produce HIV reservoirs. The removal of these dormantly contaminated cells is crucial for HIV cure. However, identifying indicators of the HIV reservoir that aid in the removal of these latently infected cells is difficult. Multiple investigations have shown that the immunological checkpoint protein programmed death-1 might be possible biomarkers expressed on latently infected cells, although this is not the only possibility. Additional indicators will need to be found in order to enhance HIV reservoir targeting.

HIV and eradicating HIV in the vast majority of HIV-infected persons. As a result, combining therapies aimed at inducing an HIV-specific immune response as well as preventing HIV infection and dissemination might be a helpful strategy for regulating viral reservoirs and attaining a functional HIV cure. This study describes how antiviral immune responses impact the HIV reservoir, in addition to the implications for immunological therapies aimed at curing HIV.

Innate immunity is the initial line of defense towards infection by viruses, and innate immune cells play a critical role in the initiation of HIV infection. During acute HIV infection, viral pathogen-associated molecular patterns are recognized by receptors that recognize patterns produced by infected cells, triggering intracellular innate immune reactions that offer antiviral defences and try to achieve viral limitation. These reactions also cause the creation of cytokines and chemokines, which can recruit and activate innate immune cells such as macrophages, Natural Killer (NK) cells, and DCs, resulting in viral control and the activation of the adaptive immune response. This innate immunity might be employed to improve HIV cure techniques by increasing the clearance of early infected cells by cytokines or immune cells.

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