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Understanding the Progression of HIV Infection Using Molecular Biological Evaluation Approaches

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Abstract

Just about two decades have passed since the first discovery of severe immunodeficiency in previously healthy homosexual men due to the discovery of the causing virus, to the current state of knowledge about the HIV virus and the pathogenic mechanisms induced by the virus, which is extensive but still incomplete. Furthermore, better treatments have been gradually introduced at a rate that is likely unique. Various molecular biological approaches have supported these processes. Estimating viremia and sequencing viruses, and thus describing the evolution of the virus, are important methods for understanding pathogenic processes. Some of the findings are described in the current thesis. While it was once considered that the virological profile reflected the clinical profile, with an acute infection followed by clinical latency for years and only indications of severe immunodeficiency after on average ten years, this understanding has been modified.

Keywords: HIV replication; DNA; RNA; CD4 cells

Description

Entry andattachment

When HIV infects a cell, the process of creating new viruses begins. Attachment and fusion are the two stages of this process.

HIV infects cells in the immune system that have a CD4 receptor on their surface. T-lymphocytes(also known as t cells), monocytes, macrophages, and dendritic cells are examples of cells. The CD4 receptor is employed by the cell to signal the presence of antigens to other components of the immune system. When HIV comes into touch with a CD4 cell, its gp 120 spikes bind the CD4 receptor as well as another co-receptor, either CCR5 or CXCR4. The HIV envelopeis fused to the cell wall using the gp41 protein. The HIV capsid is able to penetrate the CD4 cell through fusion process. Antiretroviral drugs have been developed to prevent various phases of the attachment process.

Transcribing in reverse

Before HIV RNA can be integrated into the DNA of the host cell, it must first be 'reverse transcribed' into proviral DNA. Insidethe cell, HIV's reverse transcriptaseenzyme converts RNA to proviral DNA.

To halt the action of reverse transcriptaseand the synthesis of proviral DNA, two types of antiretroviral drugs have been developed:

NRTIs and NtRTIs (nucleoside and nucleotide reverse transcriptase inhibitors) stop HIV from being produced by inserting a nucleoside or nucleotide into the HIV DNA chain while it is being formed, thus ending the chain.

NNRTIs (non nucleoside reverse transcriptase inhibitors) HIV from being produced by binding to the reverse transcriptase enzyme directly.

Incorporation

HIV's integrase enzyme attaches itself to the end of the proviral DNA strands and passes through the cell nucleus wall once HIV RNA is transformed into DNA. After the proviral DNA connects to host DNA in the cell nucleus, the HIV DNA strand is introduced into host cell DNA. HIV integrase inhibitors have been created to prevent the HIV DNA strand from being transferred into the DNA of the host cell.

HIV remains dormant within the cellular DNA after the proviral DNA is incorporated into the host cell'sDNA. Latencyis the term for this stage, and the cell is defined as 'latently infected.'Even with the most sensitive testing, detecting these latently infected cells can be difficult.

Transcription and translation are the fourth and fifth steps in the process.

If the cell receives a signal to become active, it will create HIV RNA. When CD4 cells come into contact with an infectious pathogen, they become activated. When the cell becomes active, HIV makes messenger RNA with the help of the host enzyme RNA polymerase. The instructions for producing new viral proteins in lengthy chains are contained in this messenger RNA HIVs protease enzyme cuts large strand of HIV proteins into smaller ones.

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