OPEN ACCESS Freely available online

HIV: Current Research

ISSN: 2572-0805

Perspective

Virus Induced Cell Death in Pathogenic Process

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DESCRIPTION

An essential cell death process that aids in the preservation of equilibrium in living systems is the apoptotic pathway. Apoptosis variants have, however, been connected to a wide range of illnesses, including cancer and recurrent infections. Because of the steady depletion of CD4+ T cells caused by the HIV infection, mortality and morbidity have increased globally. Both the intrinsic and extrinsic apoptotic pathways' activation and mediation are essential for HIV pathogenesis and intracellular survival. Since this can open up new avenues for therapeutic intervention and control, a thorough molecular understanding of how apoptosis is triggered and controlled in HIV-mediated CD4 + T cell depletion is crucial.

One of the lentiviruses that mostly affects CD4+ T cells, macrophages, and dendritic cells, the Human Immunodeficiency Virus (HIV) is a single-stranded positive sense retrovirus. The gag, pol, and env genes, which respectively code for viral core proteins, reverse transcriptase/integrase, and viral envelop glycoproteins, are found in three open reading frames that make up the virus. The virus has regulatory and accessory genes that, when transcribed, produce the regulatory and accessory proteins Vif, Vpr, Vpu, and Nef. Long terminal repeats on each side of the genome play a key role in reverse transcription, viral integration, and viral transcription. The viral envelop protein gp120 binds to the CD4+ receptors on CD4+ T cells, macrophages, and dendritic cells to initiate the HIV infection.

Prophylactic HIV-1 vaccination would be the greatest option. However, there is currently no HIV vaccine that is 100 percent effective. gp41 of the virus interacts with co-receptors (CXCR4 in CD4+ T cells or CCR5 in monocytes/macrophages, dendritic cells, and activated T cells).

Untreated individuals' progressive chronic loss or depletion of CD4+ T cells is the pathogenicity of HIV infection, predisposing them to the opportunistic infections, chronic inflammation, and cancers that make up AIDS. Increased destruction, decreased generation, or relocation of CD4+ T cells are three common causes of the underlying mechanisms causing CD4+ T cell depletion. Studies have shown that HIV-infected people produce less thymic T cells than do people without the virus. This was attributable to both the death of uninfected thymocyte precursors and the direct cytopathic effect of the virus on HIV-infected thymocyte precursors. However, TREC investigations were able to validate the restoration of thymic function in kids following antiretroviral therapy.

These investigations supported the notion that thymic function and size are negatively impacted by HIV infection in a reversible manner. Further research was done to confirm the link between viral load and apoptosis in light of the impact of HIV on CD4+ T cells. On the other hand, it was established that there was no connection between viral load and the apoptosis of circulating CD4+ T cells. An investigation into this conundrum revealed that bystander non-infected CD4+ T cells in the lymph node underwent more apoptosis than infected cells. Additionally, additional investigations have shown that non-HIV CD4+ T cells die at a higher rate than HIV CD4+ T cells, proving that CD4+ T cells that are non-permissive undergo higher apoptosis.

Apoptosis is a type of controlled cell death where the death of the cell is programmed by the coordinated activation and performance of numerous controlled processes. Cell shrinkage, chromatin condensation, nuclear fragmentation, and plasma blabbing are its distinguishing features. The extrinsic and intrinsic pathways of apoptosis are separated. The activation of death receptors from the tumor necrosis factor receptor subfamily located on the cell surface starts the extrinsic pathway. It is made up of the well-known Fas/Apo/CD95 (TNFRSF6), DR3 (TNFRSF25), DR4 (TNFRSF10A), DR5 (TNFRSF10B), and DR6 (TNFRSF21) death receptors.

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Received: 21-Feb-2022, Manuscript No. HICR-22-18122; **Editor assigned:** 24-Feb-2022, Pre QC No. HICR-22-18122 (PQ); **Reviewed:** 10-Mar-2022, QC No. HICR-22-18122; **Revised:** 18-Mar-2022, Manuscript No. HICR-22-18122 (R); **Published:** 23-Mar-2022, DOI: 10.35248/2572-0805.22.7.208.

Citation: Sangalo P (2022) Virus Induced Cell Death in Pathogenic Process. HIV Curr Res. 7:208