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Commentary

Effect of HIV on Host Immune Cell Activity

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DESCRIPTION

HIV is a spectrum of conditions caused by infection with the Human Immuno Deficiency Virus (HIV), a retrovirus. The infection progresses, and it inhibi more with the immune system, increasing the risk of developing common infections such as tuberculosis, as well as other opportunistic infections, and tumours that have normal immune function. HIV is the cause of the spectrum of disease known as HIV/AIDS. HIV is a retrovirus that primarily infects components of the human immune system such as CD4+ T-cells, macrophages and dendritic cells. It destroys CD4+ T-cells directly and indirectly.

HIV-1 infection affects the morphology and functioning of cellular immune responses, with the majority of capability recovering after Anti-Retroviral Therapy (ART). ART does not completely restore normal immune cell function, inflammatory changes that can result in co-morbidities and mortality following HIV (Human Immuno Deficiency Virus). In order to identify cellular dysfunction that occurs during HIV-1 infection and persists during therapy, it is crucial to comprehend the dynamics of the immune cell landscape during HIV-1 infection and ART. Acute Immunodeficiency Syndrome is carried upon by the progressive depletion of CD4+ T-cells caused by HIV-1 infection (AIDS). Anti-Retroviral Therapy (ART) can be initiated and used regularly to achieve viral suppression, which lowers HIV-1 mortality and reduces the risk of transmitting the virus to others. It is also apparent that HIV-1 impairs the host immune system and that ART may only partially repair the damage and the amount of immune system activation because the chronic inflammation brought on by the virus has been associated to non-AIDS related morbidity and mortality. In order to recognize infection and improve therapy outcomes, it is critical to comprehend the effect HIV-1 has on host immune cell activity. These observations, along with the amount of new HIV-1 infections that continue to occur despite prevention and treatment. High-throughput Single-Cell RNA sequencing (scRNA-seq) has recently been recognized as a potent technique for decoding the transcriptional variations in cell populations at the single-cell level and has made significant contributions of the pathophysiology of human disease. The variability of the HIV-1 latent reservoir, HIV-1 replication and infection, and the cellular response to latency reversal medications have all been investigated using single-cell techniques. Numerous of these studies have concentrated on how HIV-1 affects CD4+ T-cells and have discovered significant pathways that are crucial for controlling HIV-1 infection and latency. Although non-CD4+ T-cell subsets shown how innate and adaptive immunity interact to control the viral replication and broadly neutralize antibody responses to HIV-1infection. The HIV-1 co-receptor CCR5 can bind to the protein MIP-1b, which is encoded by the CCL4 gene to prevent HIV infection. Following DNAJB1, which is a component of the HSP40 complex and has been demonstrated to play a mechanism in anti-inflammatory processes in autoimmunity, the next three elevated genes in HIV treated individuals were RGS1 and SOD1, which are essential for antiviral immune responses. The transcription factor ZNF90, NHSL2, which is unknown in function, and IL7R, which has been found to be involved in CD-4 T-cell depletion during HIV-1 infection.

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