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T-cell subsets in HIV infection

The human immunodeficiency virus (HIV) is a virus that assaults the body's immunologic system. If HIV is not tackled, it can eventually drive the organism to AIDS, which a dreadful condition known as acquired immunodeficiency syndrome. In both animal models and humans some phenotypic and practical heterogeneity of the T-lymphocyte responses have been revealed during chronic virus infections. Initial studies displayed that the virus-specific CD4+ and CD8+ T lymphocytic response during primary HIV infection was characteristically an effector response, entailing of single IFN-γ secreting CD4+ T-lymphocytes, which is now known as a monofunctional response. If we keep in mind that T- lymphocytes are in a dynamic state of differentiation, we must emphasize that T- lymphocytes expressing the CD8 antigen may be subdivided into naïve, "effector" memory, "central" memory, and "terminally differentiated" effector memory (TEMRA) cells. The link between phenotypic expression indicators on CD8+ T- lymphocytes with functional properties is key. It includes an *ex-vivo* effector function. In the absence of automatic approaches, traditional ways of data analysis entailing CD57, CD62L, CD27, and CD127 should not be used as solitary primary staging markers. Also, the antiviral retrovirus therapy (ART) can decrease the quantity of HIV in the blood to an untraceable level, in most chronically infected people. However, it does not fully restore health. ART cannot eliminate reservoirs of HIV. This virus can persist in latently infected immunologic cells. Reservoir cells comprise latently infected resting, T follicular helper cells (Tfh) or T stem cell-like memory (TSCM), memory CD4+ T-cells, and other cells. In line with this topic of study, Meraviglia et al. (2019) carried out a phenotypic and functional investigation of T-lymphocytes expressing CD4, CD8, and plasma cytokine pattern levels in a cohort of HIV-1 infected patients, (both highly active antiretroviral treated patients and untreated patients). The aim was to investigate the association of T cell immunologic responses with the control of HIV infection.

In this study, Meraviglia et al. recognized the patterns of circulating T-lymphocytes. They performed phenotypic analysis in naïve patients with highly active antiretroviral therapy. The serum cytokine profiles of HIV infected patients with varying levels of viremia was also studied, on and off ART therapy. These authors examined the ability of CD4+ and CD8+ T-lymphocytes to secrete interferon-gamma (IFN-γ) and interleukin 2 (IL-2) following antigenic specific stimulation. The use of specific (Gag-region) T-cell responses were founded on investigations that have exposed it to possess a more defensive role than responses directed against other regions (non-Gag regions). The authors reported a higher rate of T-lymphocytes expressing CD4 and CD8 producing single IFN-γ in non-controllers and non-responders. This data was statistically significant. In particular, the significance was key to find when the authors compared the patients within the two groups: controllers versus non-controllers and responders versus non-responders. They displayed the involvement of both CD4+ and CD8+ T-lymphocytes over time. This data seem to endorse that IFN-γ is detected in chronic HIV infection. It seems that it has an influence on the viral load set point. The levels of CD4 and CD8 IFN-γ in vitro assembly could reflect the aspect of complex connections between natural killer (NK) lymphocytes, macrophages, and dendritic cells (DCs) to govern HIV replication during chronic HIV infection. It seems that it is crucial that *in vitro* tests presented that DCs stirred HIV Gag-virus-like particles. These particles induced vigorous antigen-specific NK cell production, IFN-γ making, and energetic cytotoxicity against HIV-infected CD4+ T-lymphocytes. The pathways demonstrated by Meraviglia et al. (2019) can provide significant understandings into mechanisms of virulence of HIV. They lead to new approaches of immunological phenotype differentiation within these cells, particularly in the chronic phase of persistent HIV infection. In the future, the authors promised to examine the role of NK cells, which might reveal some cracks in the

current HIV research. A better understanding of T-cell subsets may be key not only for HIV infection, but also in the study of the adaptive response seen during the infection of SARS-CoV-2 (severe acute respiratory syndrome–related coronavirus 2) variants. These studies may be fundamental in expanding our knowledge and expand personalized care and precision medicine strategies within the infected human population.

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