High viral load in HIV-infected individuals underlies innate immune cell dysfunction

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Individuals infected with HIV exhibit both severe immune deficiency and aberrant inflammation, resulting in susceptibility to secondary infection as the disease progresses. HIV-associated deficiencies in adaptive immune responses have been well described; however, the effects of HIV on innate immune responses are not fully understood.

A new study in *JCI Insight* demonstrates that a high viral load associates with a dampened inflammatory response in innate immune cells from HIV-infected individuals. Eileen Scully, Marcus Altfeld, and colleagues from the Ragon Institute and the Heinrich Pette Institute evaluated multiple samples taken from a cohort of patients prior to and after initiation of antiretroviral therapy (ART).

Compared to cells collected prior to the initiation of ART, innate immune cells collected after patients started ART exhibited an increased response to inflammatory stimuli. Compared to other factors, patient viral load was the most predictive of the innate immune cell response. In monocytes, epigenetic modifications were observed at the locus of the gene encoding pro-inflammatory cytokine TNF? that associated with high levels of virus.

Together, the results of this study identify viral load as a driver of innate immune dysfunction in HIV-infected individuals.

More information: Eileen P. Scully et al. Innate immune reconstitution with suppression of HIV-1, *JCI Insight* (2016). DOI: 10.1172/jci.insight.85433