

Lack of Sleep May Worsen Inflammation in People With HIV

A mechanism that reduces inflammation and induces sleep may not work well in HIV-positive people.

April 24, 2024 By [Liz Highleyman](#)

Inadequate sleep can contribute to increased inflammation in people living with HIV, and a compensatory mechanism that normally counteracts this process and encourages sleep may be ineffective, according to research presented at the [Conference on Retroviruses and Opportunistic Infections \(CROI 2024\)](#) in Denver.

Studies have shown that many people with HIV—perhaps as many as 70%—have trouble getting enough sleep. Side effects of certain medications, comorbidities and stress can all lead to poor sleep. Inadequate sleep has been linked to a wide range of health problems, including [cardiovascular disease](#).

Bernard Macatangay, MD, and colleagues at the University of Pittsburgh, looked at the effects of short-term sleep deprivation on levels of immune activation and inflammation in HIV-positive people. They also assessed the function of the adenosine pathway, a compensatory mechanism that reduces inflammation and [increases the urge to sleep](#). Prior research has shown that this pathway is dysregulated in people with HIV.

This analysis included 20 people with HIV, mostly men, who were on stable antiretroviral therapy with viral suppression for at least one year. The median age was approximately 60 years, and the median CD4 T-cell count was high. They first had one week of regularized sleep, with at least eight hours of opportunity to sleep each night, then stayed awake for 24 hours.

The researchers collected blood samples before and after sleep deprivation to measure biomarkers of T-cell, monocyte and macrophage activation, cell cycling, T-cell ectoenzyme expression of CD39 and CD73 (markers of adenosine pathway activity) and inflammation markers, including interleukin 6 (IL-6), TNF-alpha, sCD14 and sCD163.

They found that CD8 “killer” T-cell immune activation levels increased significantly after sleep deprivation, though there was no difference in CD4 “helper” T-cell activation. There was a trend toward greater monocyte and macrophage activation, but these differences did not reach statistical significance. Cell cycling decreased for both CD8 and CD4 T cells. There was a trend

toward an increase in sCD163, but no differences for IL-6, TNF-alpha or sCD14.

Despite the increase in CD8 activation and inflammation, they did not see a compensatory increase in the expression of CD39 or CD73 on T cells. Plasma adenosine levels were similar before and after sleep deprivation, indicating that the compensatory pathway did not kick in.

“Our results suggest that among virally suppressed people with HIV, sleep deprivation could impact systemic inflammation by activation of CD8 T cells and macrophages,” the researchers concluded. “However, there is no compensatory increase in ectoenzyme expression that can increase extracellular adenosine and counteract the inflammatory process.”

These findings suggest that inadequate sleep may be particularly detrimental for people living with HIV and that interventions to improve sleep should be part of HIV care.

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