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Interfering with interferon boosts antiretroviral efficacy in HIV-infected mice

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Although combined anti-retroviral therapy (cART) can help HIV-1-positive patients effectively manage their infection, some individuals experience ongoing activation of the immune system that can exacerbate disease progression. This chronic activation has been attributed to persistent signaling by type I interferons (IFN-I), proteins that bind to IFN receptors to regulate the immune system.

Two studies published this week in the *JCI* have demonstrated that antibodies targeting IFN-I signaling can enhance the effectiveness of cART in humanized mouse models of HIV-1 infection.

In work led by Scott Kitchen at UCLA, researchers treated HIV-1-infected mice with an antibody to block human IFN receptor 2. They observed reduced signs of T cell exhaustion and viral load, indicating a decrease in chronic immune activation and improved infection management, respectively.

Lishan Su's lab at the University of North Carolina developed a different antibody targeting the human IFNα/β receptor and observed that it, too, reversed immune hyperactivation in the mouse model.

In both studies, blockade of IFN-I signaling synergized with cART treatment, leading to better outcomes compared to HIV-1-infected mice treated with cART alone. The results of these two studies provide strong support for further evaluation of IFN-I blockade as a supplement to cART.

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More information: Anjie Zhen et al, Targeting type I interferon–mediated activation restores immune function in chronic HIV infection, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI89488

Steven G. Deeks et al. The interferon paradox: can inhibiting an antiviral mechanism advance an HIV cure?, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI91916

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