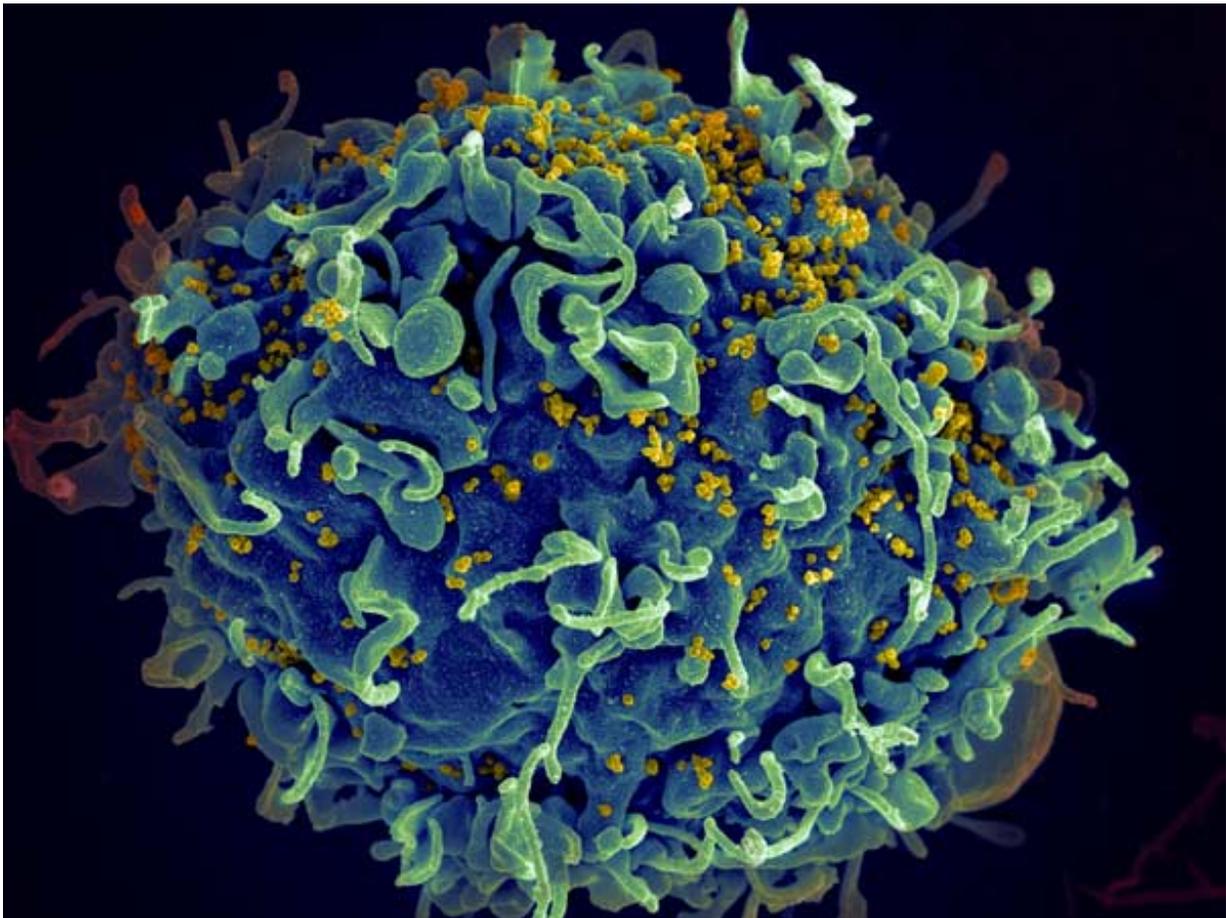


# Adenosine deaminase may help the immune system fight HIV on its own

February 2 2016

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HIV (yellow) infecting a human immune cell. Credit: Seth Pincus, Elizabeth Fischer and Austin Athman, National Institute of Allergy and Infectious Diseases, National Institutes of Health

New research findings published in the February 2016 issue of the *Journal of Leukocyte Biology*, suggest that a new therapeutic strategy for HIV may already be available by repurposing an existing prescription drug. The drug, an enzyme called adenosine deaminase, or ADA, ultimately may be able to activate the immune system against HIV and to help the immune system "remember" the virus to prevent or quickly eliminate future infection.

"We hope this study puts ADA in the spotlight as a powerful immune modulator in vaccine strategies enhancing anti-HIV immune responses and limiting the need for life-lasting treatments," said Núria Climent, Ph.D., a researcher involved in the work from the Retrovirology and Viral Immunopathology Laboratory, at AIDS Research Group from IDIBAPS in Barcelona, Spain.

To make their discovery, scientists studied the effects of ADA in [cells](#) from HIV patients and non-infected individuals. Dendritic cells derived from blood cells were exposed to HIV-1 proteins or whole inactivated HIV-1 virus in the presence or absence of ADA. The degree of lymphocyte proliferation, regulatory T-cell generation and cytokine secretion were measured. ADA addition resulted in adenosine degradation leading to a reduction of regulatory T-cell mediated suppression. Notably, the presence of ADA produced an increase in CD4+ responder T cells, in CD8+ T cell proliferation and in T cell memory generation. An increase in the secretion of immunologically relevant Th1 cytokines was also seen.

"We need to find new strategies that will empower the [immune system](#) towards long-term control of HIV infection," said Luis J. Montaner, D.V.M., M.Sc., D.Phil., Editor-in-Chief of the *Journal of Leukocyte Biology*. "The availability of an approved drug that already targets the mechanisms described here ensures the quick translation of this work from the bench to the clinical."

Provided by Federation of American Societies for Experimental Biology

Citation: Adenosine deaminase may help the immune system fight HIV on its own (2016, February 2) retrieved 3 May 2024 from <https://medicalxpress.com/news/2016-02-adenosine-deaminase-immune-hiv.html>

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