

Researchers identify potential new HIV vaccine/therapy target

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After being infected with simian immunodeficiency virus (SIV) in a laboratory study, rhesus macaques that had more of a certain type of immune cell in their gut than others had much lower levels of the virus in their blood, and for six months after infection were better able to control the virus.

SIV is a [retrovirus](#) that infects [primates](#). Strains of [SIV](#) that crossed over to humans resulted in the evolution of HIV. In [rhesus macaques](#), SIV causes simian AIDS (though in many primates it is harmless) and studying the virus in these animals offers crucial insights into how HIV acts in humans, the researchers said.

The discovery by researchers at UCSF may shed light on the mystery of why some people infected with HIV are better able to control the virus, live longer and have fewer associated [health problems](#) than others who have been infected as long, they said. It also provides a potential new [target](#) for developing therapies or vaccines.

The cells that have the protective effect, called Th17 (T helper 17) cells, are a subset of the type of disease-fighting immune cell targeted and killed by HIV and found in the gut of both primates and humans.

A prior study from the same UCSF team found that SIV infection causes a normally protective immune response to infection to go awry, leading to reduction in the protective activity in the gut of these Th17 cells and weakening of mucosal defenses against bacteria. Interestingly, in that study, Th17 cells were not affected by SIV in another primate, African [green monkeys](#), in which SIV infection is harmless and does not cause disease.

"Animals with more of these Th17 cells were better able to control SIV and this was due in part to macaques developing a more effective immune response by producing more SIV-specific

CD4-positive T-cells to fight the infection. Our next step is to see if we can augment the Th17 effect, perhaps by looking at [interleukin 17 \(IL-17\)](#), the [cytokine](#) released by these cells, and testing to see if it has an effect," said the study's primary investigator, Dennis Hartigan-O'Connor, MD, PhD, assistant professor of medicine at the UCSF Division of Experimental Medicine.

"Further, if a treatment can be developed to increase Th17 cells in the gut, it may allow for a more effective immune response after exposure to an HIV vaccine or the virus itself," he added.

The findings are being published in the May 30, 2012 issue of *Science Translational Medicine*.

In the new study, the investigators first determined the levels of Th17 cells in the gut of sixteen rhesus macaques and then infected them with SIV. They found that the animals with more Th17 cells to begin with were better able to control the virus. They then gave animals drugs that deplete Th17 cells and found that reducing the number of Th17 cells made controlling SIV more difficult for those animals.

"We found great variation in the levels of Th17 cells, with as much as a five-fold difference in numbers between animals. We are not sure why this is the case. It could be genetically determined or perhaps due to a previous exposure to a type of bacteria that stimulates production of Th17 cells," said Hartigan-O'Connor.

This study is part of a series of investigations undertaken by researchers at the UCSF Division of Experimental Medicine into how SIV, and by extension HIV, interacts with the immune system in the gut. The previous study was focused on chronic infection and persistent inflammation in the gut.

"The earlier study addressed the cause and consequence of inflammation after infection. We

found that inflammation induces an enzyme that knocks out Th17 cells, which normally help to keep the gut intact, and that disease progression was faster. Reciprocally, we have now found that animals do better if they have many Th17 cells at the outset of infection. We are gradually increasing our understanding of this important aspect of the immune system and we are working to translate this understanding into an approach that benefits patients," said study senior author, Joseph M. McCune, MD, PhD, chief of the UCSF Division of Experimental Medicine.

Study co-investigators include Bittoo Kanwar from UCSF Division of Experimental Medicine and Kristina Abel and Koen K. A. Van Rompay from the University of California, Davis.

Provided by University of California, San Francisco

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