

HIV pays a price for invisibility

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Mutations that help HIV hide from the immune system undermine the virus's ability to replicate, show an international team of researchers in the April 13 issue of the *Journal of Experimental Medicine*. The study was published online on March 23.

When HIV infects a cell, a complex of human immune proteins called HLA (short for human leukocyte antigen) alert killer <u>T cells</u> by displaying bits of the virus on the surface of the cell. The T cells recognize these HIV fragments and mobilize an attack.

Individuals who have certain types of HLA proteins control infection better than others. In people with HLA-B*5703, for example, the virus multiplies less than in people with some other HLA variants—likely because killer T cells in these individuals are quick to attack infected cells. But <u>HIV</u> is tricky. To get around HLA-B*5703, the virus mutates three amino acids that T cells need to recognize the infected cells, causing the killers to pass by the infected cell unnoticed. Thus by mutating, the virus becomes invisible to the <u>immune system</u>.

In the new report, Hayley Crawford at the University of Oxford and colleagues show that the virus pays a price for its invisibility. The triple mutant replicated 20 times slower than normal in cell culture.

The researchers went on to study Zambian couples in which one HLA-B*5703-expressing person infected with triple-mutant virus passed the infection to a partner who either did or didn't have the same HLA variant. When transmitted to a person without HLAB*5703, the virus



changed its mutated amino acids back to their original sequence, most likely because the benefit of avoiding killer T cells no longer outweighed the cost of reduced <u>replication</u>. However, when transmitted to another HLA-B*5703-expressing person, the triple-mutated virus came out on top despite its reduced replication. In these individuals, the avoidance of killer T cells allowed the infection to rapidly proceed to clinical illness. This study suggests that vaccines should be designed to produce a T cell response against a number of different viral peptides - something that experimental human vaccines so far have yet to achieve.

Source: Rockefeller University (<u>news</u> : <u>web</u>)

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