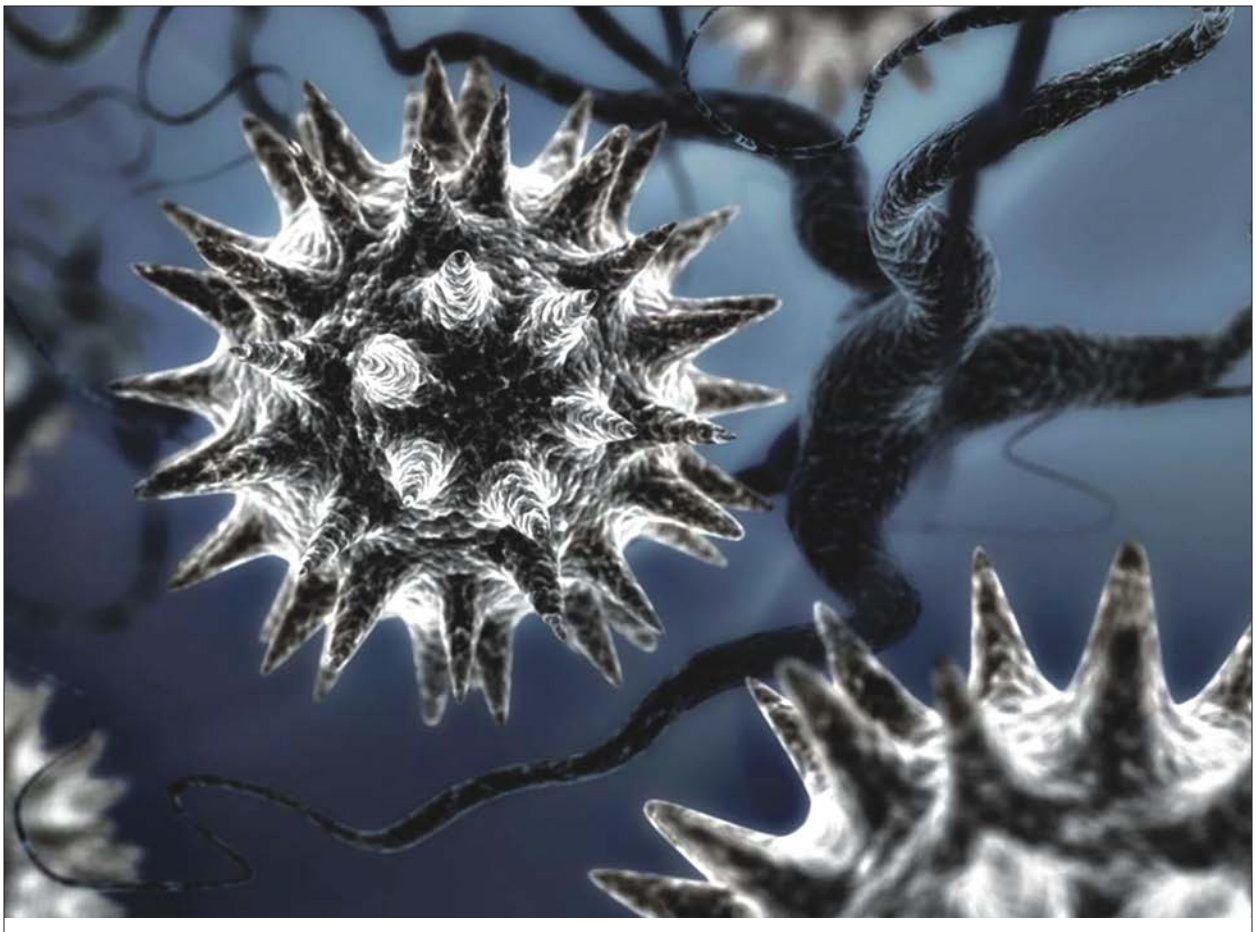

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New Discoveries Make It Harder for HIV to Hide from Drugs

The virus that causes AIDS is chameleon-like in its replication. As HIV copies itself in humans, it constantly mutates into forms that can evade even the best cocktail of current therapies. Understanding exactly how HIV cells change as they reproduce is key to developing better tests and treatments for patients.

In the Journal of Biological Chemistry and Nature Structural and Molecular Biology, MU Microbiologist and Biochemist Stefan Sarafianos, Ph.D, reveals new findings that shed light on how HIV eludes treatment by mutating. His discoveries provide clues into HIV's mechanisms for resisting two main families of drugs.

"These findings are important because identifying a new mutation that affects HIV drug resistance allows physicians to make better decisions and prescribe the proper drugs," Sarafianos said. "Without that knowledge, therapy can be suboptimal and lead to early failure."

Patients with HIV are routinely tested to track the levels of the virus and immune cells in their body. Results of the tests help physicians gauge the health of their patients and prescribe the right mix of antiviral drugs. The drugs help prevent the spread of HIV in patients by inhibiting the virus' ability to replicate.

Sarafianos' lab determined the biochemical properties that allow strains of HIV with a specific mutation -- the N348I mutation -- to escape inhibition despite treatment with Nevirapine. The drug is commonly used in combination with other antiviral medications to decrease the amount of HIV in the blood. As a result of Sarafianos' discovery, at least one major company that manufactures HIV mutation-testing kits has modified its test to detect the N348I mutation.

Sarafianos' recent findings resulted from research supported by five National Institutes of Health grants. He recently received another $417,000 award from the NIH to assist him in developing modified antibodies for HIV therapy.

"Our latest efforts to design broadly neutralizing antibodies against HIV will hopefully expand our toolbox against the virus, which remains a constantly moving target," Sarafianos said.

M. M. Schuckmann, B. Marchand, A. Hachiya, E. N. Kodama, K. A. Kirby, K. Singh, S. G. Sarafianos. The N348I Mutation at the Connection Subdomain of HIV-1 Reverse Transcriptase Decreases Binding to Nevirapine. Journal of Biological Chemistry, 2010; 285 (49): 38700 DOI: 10.1074/jbc.M110.153783