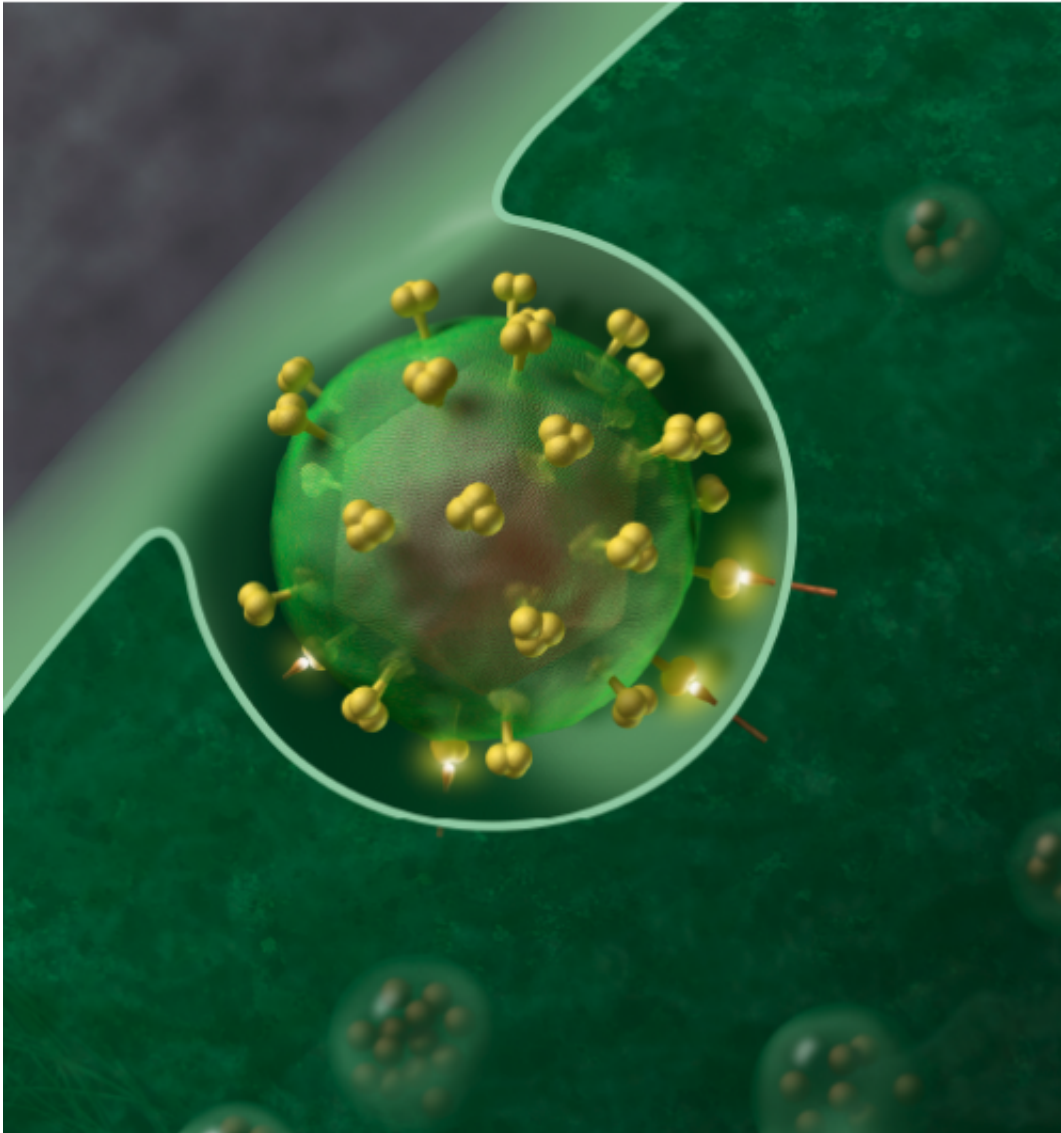


# Study suggests a way to stop HIV in its tracks

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HIV-1 Virus. Credit: J Roberto Trujillo/Wikipedia

When HIV-1 infects an immune cell, the virus travels to the nucleus so quickly there's not enough time to set off the cell's alarm system.

Now, a Loyola University Chicago study has discovered the protein that helps the virus travel so fast. Researchers found that without this protein, the virus became stranded in the cytoplasm, where it was detected by the viral defense system. (The cytoplasm is the portion of the cell outside the nucleus.)

"By preventing its normal movement, we essentially turned HIV-1 into a sitting duck for cellular sensors," said Edward M. Campbell, PhD, corresponding author of the study, published in the *Proceedings of the National Academy of Sciences*. Campbell is an associate professor in the Department of Microbiology and Immunology of Loyola University Chicago Stritch School of Medicine.

HIV-1 infects and kills immune system cells, including T cells and macrophages that were used in the study. This cripples the immune system, making the patient vulnerable to common bacteria, viruses and other pathogens that are usually harmless in people with healthy immune systems.

After HIV-1 enters a cell, it has to work its way through the cytoplasm to the nucleus. Once inside the nucleus, HIV-1 takes control of the cell and makes additional HIV-1 copies. But getting through the cytoplasm is not easy. Cytoplasm consists of fluid that is thick with proteins and structures such as mitochondria. "Something the size of a virus cannot just diffuse through the cytoplasm," Campbell said. "It would be like trying to float to the bathroom in a very crowded bar. You need to have a plan."

HIV-1 is able to get to the nucleus quickly via tubular tracks called microtubules. The virus attaches itself to a molecular motor called

dynein, which moves down the microtubule like a train car on tracks.

Campbell and colleagues discovered the "ticket" HIV-1 needs to get on the train—a protein called bicaudal D2. HIV-1 binds to bicaudal D2, which recruits the dynein [molecular motor](#). The dynein then transports HIV-1 towards the [nucleus](#).

The finding raises the possibility of developing a drug that would prevent HIV-1 from binding to bicaudal D2, thus stranding the [virus](#) in the [cytoplasm](#). This would not only prevent infection, but also give the cell time to turn on antiviral genes that would protect it and neighboring [cells](#) from infection.

The study is titled "Bicaudal D2 facilitates the cytoplasmic trafficking and nuclear import of HIV-1 genomes during infection."

**More information:** Adarsh Dharan et al, Bicaudal D2 facilitates the cytoplasmic trafficking and nuclear import of HIV-1 genomes during infection, *Proceedings of the National Academy of Sciences* (2017). [DOI: 10.1073/pnas.1712033114](#)

Provided by Loyola University Health System

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