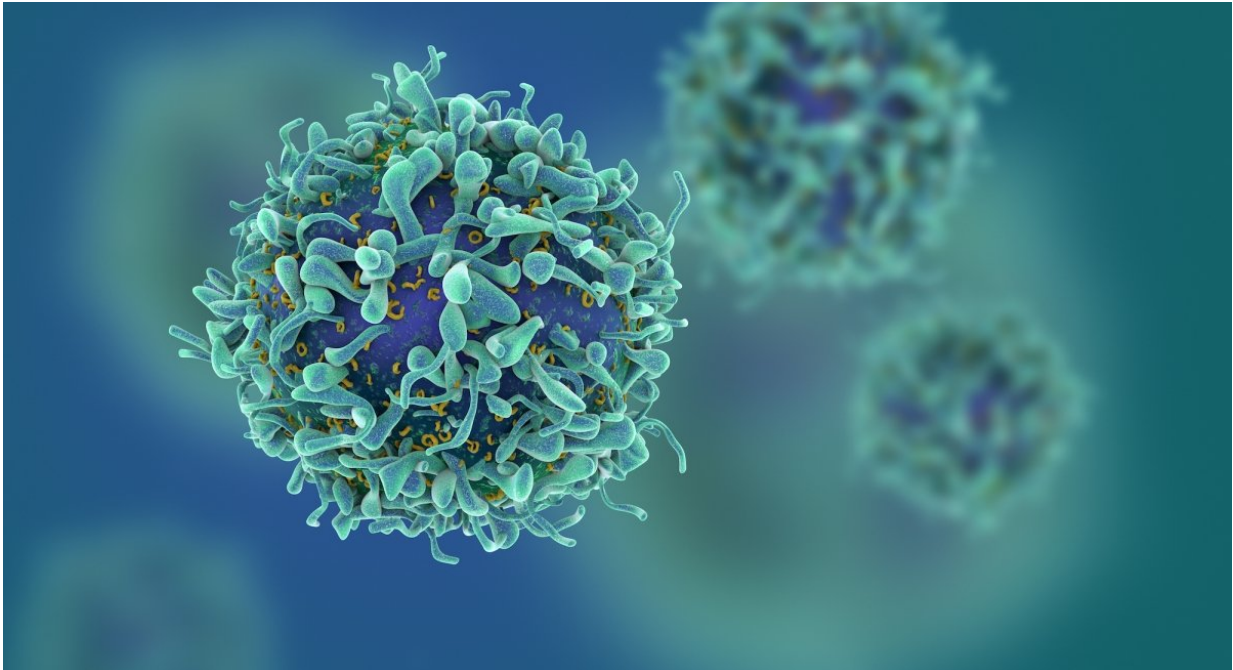


How viruses disarm the immune system

February 5 2018



Credit: McGill University

How do viruses that cause chronic infections, such as HIV or hepatitis c virus, manage to outsmart their hosts' immune systems?

The answer to that question has long eluded scientists, but new research from McGill University has uncovered a [molecular mechanism](#) that may be a key piece of the puzzle. The discovery could provide new targets for treating a wide range of diseases.

Fighting off infections depends largely on our bodies' capacity to quickly recognize [infected cells](#) and destroy them, a job carried out by a class of immune cells known as CD8+ T cells. These soldiers get some of their orders from chemical mediators known as cytokines that make them more or less responsive to outside threats. In most cases, CD8+ T cells quickly recognize and destroy infected cells to prevent the infection from spreading.

"When it comes to viruses that lead to chronic infection, [immune cells](#) receive the wrong set of marching orders, which makes them less responsive," says Martin Richer, an assistant professor at McGill's Department of Microbiology & Immunology and senior author of the study, published recently in the journal *Immunity*.

The research, conducted in Richer's lab by graduate student Logan Smith, revealed that certain viruses persist by driving the production of a cytokine that leads to modification of glycoproteins on the surface of the CD8+ T cells, making the cells less functional. That maneuver buys time for the pathogen to outpace the [immune response](#) and establish a chronic infection. Importantly, this pathway can be targeted to restore some functionality to the T cells and enhance the capacity to control infection.

The discovery of this regulatory pathway could help identify new therapeutic targets for a variety of diseases. "We might be able to take advantage of the pathways induced by these signals to fight [chronic viral infections](#) by making the [immune system](#) more responsive," Richer says. "The findings might also prove useful for diseases like cancer and autoimmunity, where T cells function is poorly regulated."

Interleukin-10 Directly Inhibits CD8+ T Cell Function by Enhancing N-Glycan Branching to Decrease Antigen Sensitivity was published in *Immunity*.

More information: Logan K. Smith et al. Interleukin-10 Directly Inhibits CD8 + T Cell Function by Enhancing N-Glycan Branching to Decrease Antigen Sensitivity, *Immunity* (2018). [DOI: 10.1016/j.immuni.2018.01.006](https://doi.org/10.1016/j.immuni.2018.01.006)

Provided by McGill University

Citation: How viruses disarm the immune system (2018, February 5) retrieved 21 July 2024 from <https://medicalxpress.com/news/2018-02-viruses-immune.html>

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