

# Scientists discover one of the ways the influenza virus disarms host cells

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(Medical Xpress)—When you are hit with the flu, you know it immediately—fever, chills, sore throat, aching muscles, fatigue. This is your body mounting an immune response to the invading virus. But less is known about what is happening on the molecular level.

Now Northwestern University scientists have discovered one of the ways the [influenza virus](#) disarms our natural defense system. The virus decreases the production of key immune system-regulating proteins in [human cells](#) that help fight the invader. The virus does this by turning on the microRNAs—little snippets of RNA—that regulate these proteins.

The researchers, led by [molecular biologist](#) Curt M. Horvath, are among the first to show the influenza virus can change the expression of microRNA to control immune responses in [human lung](#) cells.

The findings reveal a new aspect of the interaction between the influenza virus and its host. Knowing how viruses disable the immune system to wreak havoc in the body will help researchers design therapeutics to preserve the immune response and keep people healthy. The knowledge also may be valuable for future diagnostics.

The study is published by the [Journal of Biological Chemistry](#). The paper will appear in its final form in September.

"It's a battle of supremacy between virus and host," said Horvath, the senior author of the paper. "Our goal is to understand how the flu

replicates in the host. Now we've discovered a new pathway in which the flu controls the immune response, by shutting down vital [protein production](#). With better understanding of this mechanism, one day we may be able to customize therapeutics to target individual [flu strains](#)."

Horvath is the Soretta and Henry Shapiro Research Professor in [Molecular Biology](#) and professor of [molecular biosciences](#) in the Weinberg College of Arts and Sciences. He also is professor of microbiology-immunology and medicine at the Feinberg School of Medicine.

A microRNA has only 17 to 24 nucleotides, and its function is to dampen or shut down the production of proteins in the body. (Proteins are the workhorses of the cell.) There are hundreds of different types of microRNAs in animals.

It's been known for many years that when a virus such as influenza infects respiratory cells there is an immediate antiviral response at the cellular level—the first barrier for protecting the body from the virus. Most of the changes that occur are a result of antiviral gene expression.

About 10 years ago, scientists first learned about small RNA pathways called microRNAs, which regulate gene expression. This led Horvath to want to investigate the role of microRNAs in influenza virus infection and determine what they are contributing to the antiviral response. Exactly which genes might the microRNAs be targeting?

In their current study, Horvath and his team used human [lung cells](#), infected them with the influenza A virus and looked to see which microRNAs were activated in response to the virus. They focused on six microRNAs that were found to increase in abundance during flu infection.

The researchers found the virus activated two microRNAs that turned on the genes IRAK1 and MAPK3. This resulted in a decrease in the amount of proteins that help turn on the immune response.

Essentially, the virus uses the cell mechanisms to its advantage, disarming parts of the natural antiviral system. The flu takes over the expression of microRNAs for its own purposes. The flu increases the expression of [microRNA](#), which decreases the amount of protein and diminishes the immune response.

Having identified a specific set of microRNAs whose expression in host respiratory cells is changed by the influenza virus, Horvath next is interested at looking at the clinical outcomes. He is working with Pedro C. Avila, M.D., professor of medicine-allergy-immunology at the Feinberg School to see if the microRNAs are dysregulated in patients with influenza.

Provided by Northwestern University

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