

New knowledge on how HIV beats the body's early immune response

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Dr Najla Nasr with senior author Professor Tony Cunningham. Credit: Westmead Institute for Medical Research

In an important step towards eradicating HIV-associated viral reservoirs, researchers at Sydney's Westmead Institute for Medical Research have identified how the HIV virus hijacks the innate immune system to facilitate its replication and spread, thus gaining a foothold infection in the body.

The findings, published in the *Journal of Virology*, show how HIV induces antiviral interferon stimulated genes in the absence of [interferon production](#).

Lead author of the study, Dr Najla Nasr said it's like a prize fight, with both HIV and the body's immune response both fighting for supremacy.

"When a person is infected with HIV, the virus infects immune cells and knocks out the body's interferon production; the first line of defence in our bodies.

"When interferon production is inhibited, the virus infects adjacent cells and spreads throughout the body.

"Surprisingly, we found that although HIV inhibits interferon production it also stimulates more than twenty antiviral interferon-stimulated genes—or ISGs—in its key target cells. This is the largest effect yet shown by any individual virus.

"ISGs are critical for controlling [virus infections](#). They provide the earliest protective response to counter invading pathogens, but paradoxically they can also contribute to virus persistence.

"For its own survival advantage, HIV induces these genes in order to survive and spread throughout the body," Dr Nasr explained.

Dr Nasr said the ultimate goal of this research was to understand how these antiviral genes are induced and thus coming up with strategies to boost their expression so that the [virus](#) replication is not just slowed but completely stopped.

More information: Najla Nasr et al, Mechanism of Interferon Stimulated Gene Induction in HIV-1 Infected Macrophages, *Journal of*

Virology (2017). [DOI: 10.1128/JVI.00744-17](https://doi.org/10.1128/JVI.00744-17)

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