

Aging impairs innate immune response to flu

December 13 2017, by Ziba Kashef



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Aging impairs the immune system's response to the flu virus in multiple ways, weakening resistance in older adults, according to a Yale study. The research reveals why older people are at increased risk of illness and death from flu, the researchers said.

The study is published in *Science Signaling*.

"Influenza virus mortality is the highest in [older adults](#). This study sheds light on a mechanism that underlies this impaired antiviral response," said senior author Akiko Iwasaki, the Waldemar Von Zedtwitz Professor of Immunobiology and Howard Hughes Medical Institute investigator at Yale School of Medicine.

Iwasaki and her colleagues investigated the innate, or inborn, [immune response](#) to the flu virus. The Yale team collected blood samples from healthy young adults and older adults aged 65 and above. They isolated monocytes, a type of white blood cell, from the samples and stimulated the monocytes with either flu virus or a mimic of the virus.

The immune response in cells from older adults was severely impaired in critical ways, the researchers found. To fight the [flu virus](#), the body needs to activate potent antiviral proteins called interferons. But in older adults, this response is weakened by age-related damage to a molecule, TRAF3, that signals immune cells to make interferon. Without that signal, and another involving antiviral genes, resistance to flu falls short, they noted.

The finding offers new insight into why flu disease is severe in older adults, said Iwasaki, and points to a potential strategy for reducing flu-related mortality in that age group.

"In older adults, we might have to use a different strategy to treat and immunize against flu," she said. "We need to find a way to boost antiviral defense that does not rely on interferon production. For vaccines, we need to find an adjuvant—a component added to a vaccine—that would still stimulate the [innate immune response](#) in older adults."

More information: Ryan D. Molony et al. Aging impairs both primary and secondary RIG-I signaling for interferon induction in human

monocytes, *Science Signaling* (2017). [DOI: 10.1126/scisignal.aan2392](https://doi.org/10.1126/scisignal.aan2392)

Provided by Yale University

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