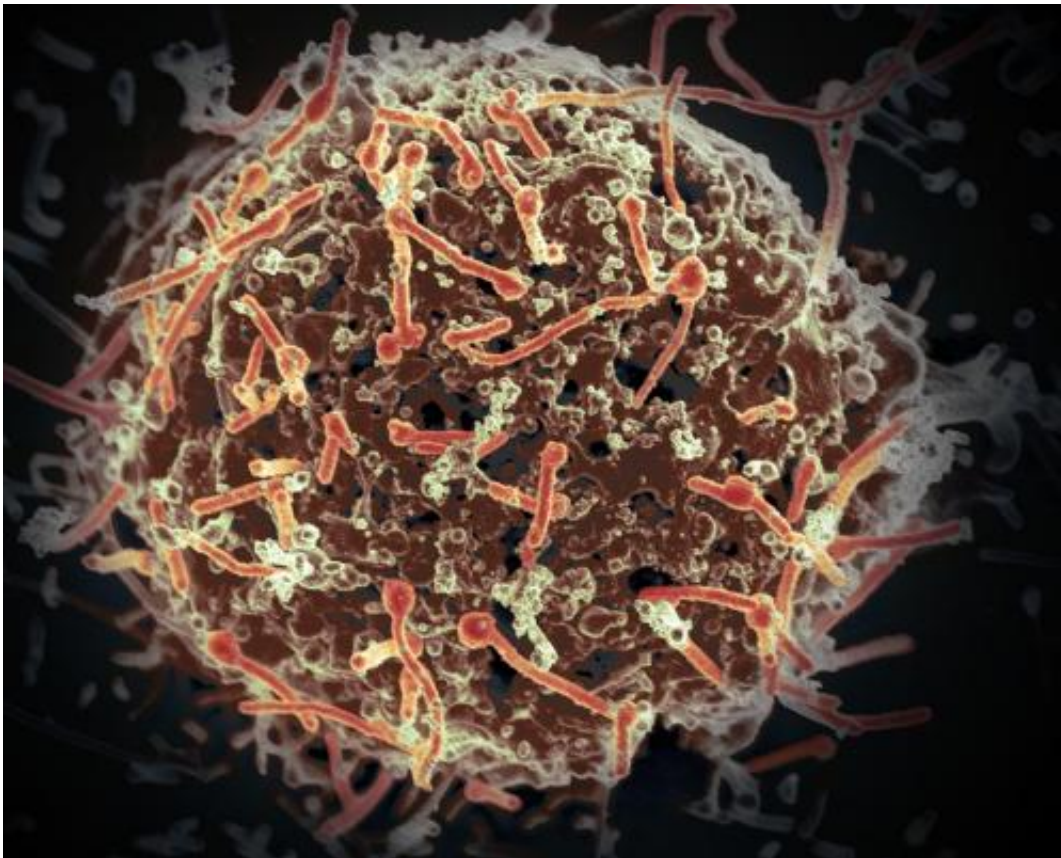


# Scientists reveal potential treatment for life-threatening viral infections

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The Ebola virus, isolated in November 2014 from patient blood samples obtained in Mali. The virus was isolated on Vero cells in a BSL-4 suite at Rocky Mountain Laboratories. Credit: NIAID

Scientists from the Florida campus of The Scripps Research Institute (TSRI) have shown for the first time how a previously unknown process

works to promote infection in a number of dangerous viruses, including dengue, West Nile and Ebola.

The new study also points to a potential treatment, an experimental antibiotic that appears to inhibit infection by these deadly viruses, all of which lack vaccines and treatments.

The study, which was published recently by the journal *Proceedings of the National Academy of Sciences (PNAS)*, was led by TSRI Associate Professor Hyeryun Choe.

"Most of these viruses use a specific molecule to enter cells," Choe said. "In the new study, we were able to show how a second molecule plays a major and previously unknown role in that process. We also show an antibiotic called duramycin inhibits the actions of this molecule. This looks to be a promising broad-spectrum antiviral strategy and deepens our understanding of the entire infection process."

## Emerging Health Concern

The viruses in question belong to several families, including the flavivirus and filovirus families. Flaviviruses like dengue and West Nile viruses cause tens of thousands of deaths each year. Filoviruses like Ebola have emerged as major health concerns, particularly in tropical and subtropical areas such as the recent highly publicized Ebola outbreak in West Africa. Perhaps the greatest concern is dengue virus. More than one third of the world's population is estimated to be at risk for dengue and more than 100 million people are estimated to be infected annually, according to recent studies.

The viruses take advantage of the process that normally occurs during [programmed cell death](#) or apoptosis. During this process, a lipid usually found on the inner side of the cell membranes, specifically

phosphatidylserine (PS), shifts to the surface. Apoptosing cells are then recognized by PS receptors on phagocytes—cells that devour invading pathogens and dying cells—and engulfed by them.

When cells are dying from a virus infection, their freshly exposed PS is grabbed by the exiting virus and phagocytes engulf the virus. Once engulfed, the virus quickly turns the cell's own biology on its head, forcing it to produce copies of the virus.

## New Insights

In the new study, Choe and her colleagues showed how another lipid known as phosphatidylethanolamine (PE), which is present on the viral surface, also contributes to the viral entry process.

"Despite the name, we found that PS receptors also detect PE, and [viruses](#) are able to take advantage of the abundance of PE on their surface," said Audrey Stéphanie Richard, the first author of the study and a research associate in the Choe lab. "Through their PE, they latch onto the PS receptors on the host cell, taking control of the process and insuring entry and replication."

Duramycin blocks viral entry into the cells by binding to the virus's PE, preventing the virus from using it to latch onto the PS receptors on the cell. Duramycin, which is currently used as an imaging agent, binds specifically to PE.

Disrupting the relationship between these two molecules could open the door to new and novel antiviral strategies, potentially including duramycin and similar PE-inhibitors.

"This new study goes a long way in helping us understand how so-called PS receptors contribute to flavivirus and filovirus infections and how we

can block them through the PE-binding compounds," Choe said.

The study also shows that PE is exposed on the surface of apoptotic cells and promotes their uptake by phagocytes.

**More information:** Audrey Stéphanie Richard et al. Virion-associated phosphatidylethanolamine promotes TIM1-mediated infection by Ebola, dengue, and West Nile viruses, *Proceedings of the National Academy of Sciences* (2015). [DOI: 10.1073/pnas.1508095112](https://doi.org/10.1073/pnas.1508095112)

Provided by The Scripps Research Institute

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