Scientists discover way to potentially track and stop human and agricultural viruses

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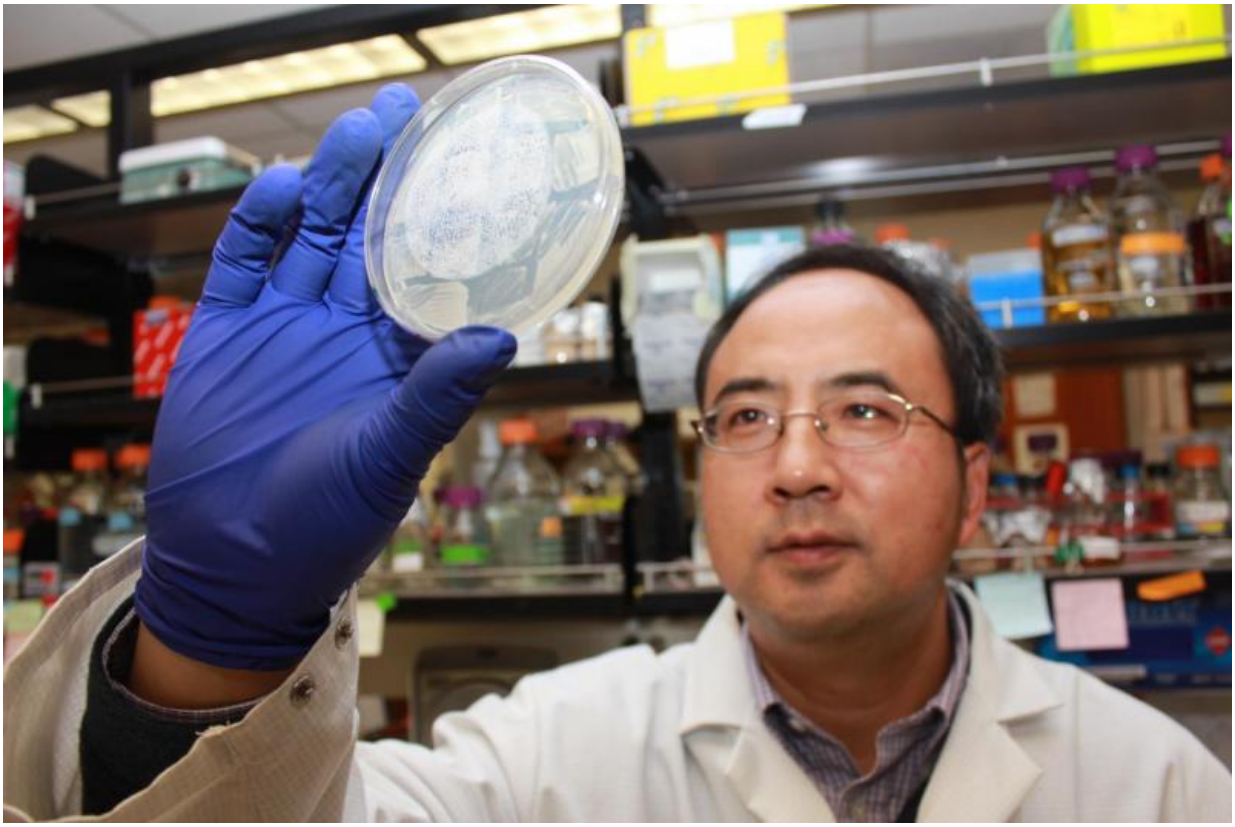

Xiaofeng Wang, an assistant professor in the Virginia Tech College of Agriculture and Life Sciences, has discovered a way to track and potentially stop viruses. Credit: Virginia Tech

Viruses are molecular thieves that take from their hosts under the cloak

of darkness. But now a Virginia Tech scientist has found a way to not only track viral hijackers, but also potentially stop them from replicating.

The discovery has broad ranging applications in stopping viral outbreaks such as Hepatitis C in humans and a number of viruses in plants and animals because it applies to many viruses in the largest category of viral classes—positive-strand RNA viruses.

The findings were recently published in the *Proceedings of the National Academy of Sciences*.

"Even though these viruses infect very different hosts, they all replicate similarly across the board, so what we learn from one virus can potentially be translated to control viruses in agricultural production as well as human health," said Xiaofeng Wang, an assistant professor of plant pathology, physiology, and weed science in the College of Agriculture and Life Sciences.

Wang's findings could target any number of plant viruses. One virus Wang has studied—the cucumber mosaic virus—affects pumpkin, squash and gourds in 1,200 species in over 100 plant families.

Sprays could be developed to halt the virus on plants, saving millions of dollars in agricultural sectors.

Wang, who is a Fralin Life Science-associated faculty member, used brome mosaic virus to study how viral infections start. He found that the brome mosaic virus stimulates synthesis of host lipid cells called phosphatidylcholine at the sites where viral replication occurs, and that by inhibiting its synthesis, the viral replication stopped.

Wang also collaborated with researchers to study how human viruses like

Hepatitis C virus and poliovirus regulate host lipid synthesis and found that viral replication behaved in the same way as using plant viruses. The ramifications for human health mean that developing a drug delivery system to combat the Hepatitis C virus would be much more nimble in treating viral outbreaks than slow-moving vaccines, and could play a crucial role in halting the debilitating infection which affects 3.5 million people in the U.S. according to the Centers for Disease Control.

Viruses can't replicate by themselves. They are essentially thieves that break into cells and multiply by hijacking the machinery of the host cells and proliferating and remodeling lipid-containing membranes such as phosphatidylcholine—one the most prominent lipids in host membranes. Wang and his collaborators were able to see where exactly the virus replications started and how they managed their hosts to meet their needs. Based on the finding of Wang and his collaborators, new ways can be developed to stop phosphatidylcholine synthesis for viral replication, but leave the host undamaged.

"The better we understand the mechanisms of a biological process, be it virus replication or cell division, the better are our options to rationally design tools that can control it," said George Belov, a collaborator of Wang's and an assistant professor of virology at the University of Maryland. "In the case of viral replication it may provide us with novel ways to control infection without causing host toxicity and a generation of viral-resistant mutants."

More information: Jiantao Zhang et al. Positive-strand RNA viruses stimulate host phosphatidylcholine synthesis at viral replication sites, *Proceedings of the National Academy of Sciences* (2016). DOI: 10.1073/pnas.1519730113

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