Revealing how a potato disease takes hold

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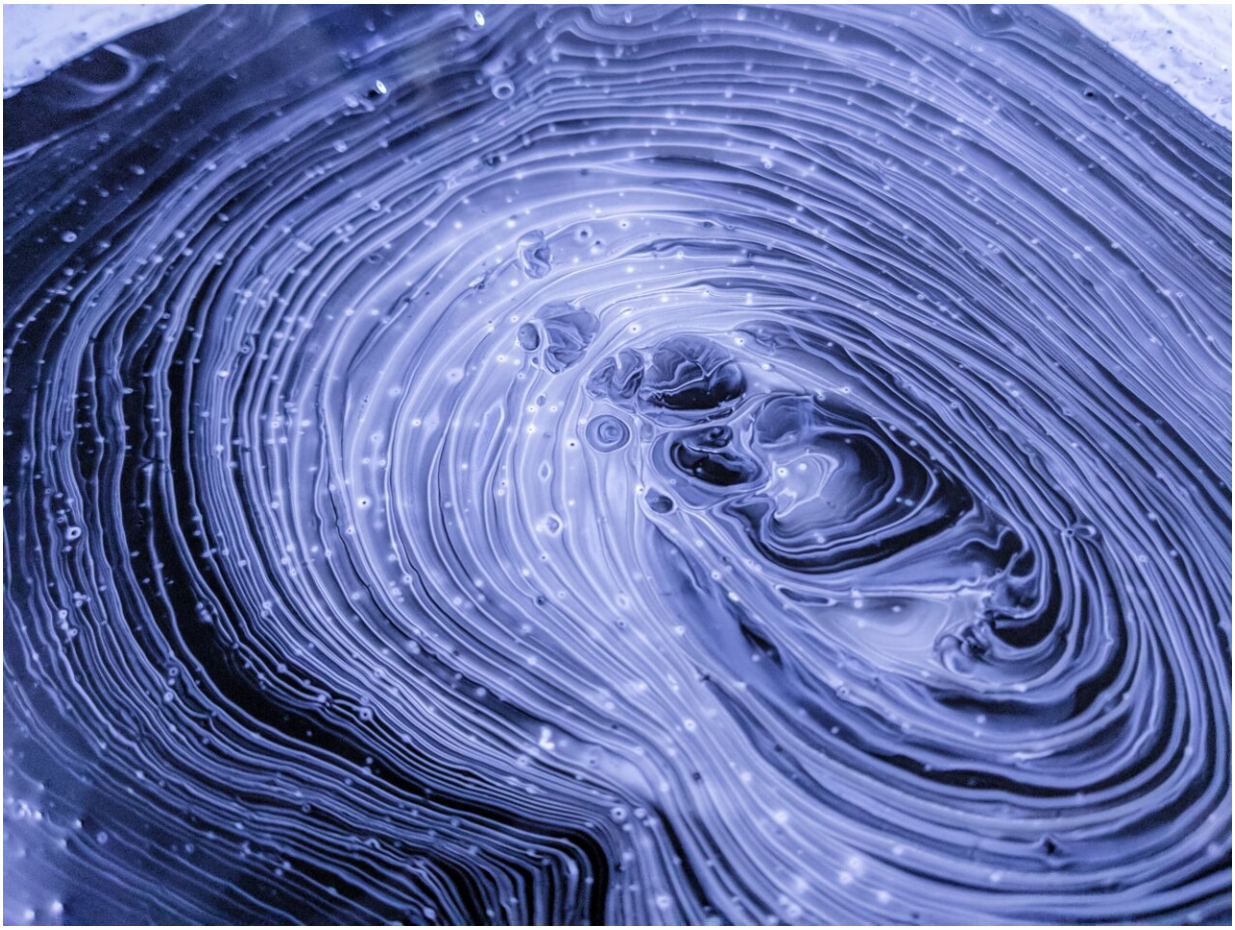

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Late blight is an economically devastating disease for potato farmers worldwide, causing tens of billions of dollars worth of damage each year. Phytophthora infestans, the causal agent of late blight, has evolved to

overcome fungicides and major resistance genes that have been bred into commercial potato cultivars. In order to dampen the immune response of its host, P. infestans secretes molecules called disease effectors at the site of infection.

Now, a research team from Japan and the UK has determined the molecular structure of the disease effector called avirulence protein 3a (AVR3a), which is known to inhibit disease resistance in potato plants. Understanding the molecular structure and function of AVR3a will help plant biologists to elucidate how P. infestans causes infection so that they can develop better control measures.

Using nuclear magnetic resonance spectroscopy, a technique employed to study the configuration of molecules, the research team—led by Ken Shirasu from the RIKEN Plant Science Center in Yokohama—identified a patch of positively charged amino acids in the structure of AVR3a. The team found the positively charged section of amino acids is conserved among different versions of AVR3a from P. infestans and the model pathogen P. sojae, indicating that this part of the overall structure could be important to AVR3a's role in the infection process.

By examining potato plants to determine the final destination of AVR3a within the infected host, the researchers found that the disease effector binds to a lipid molecule called phosphatidylinositol phosphate (PIP), which forms part of the structure of the potato cell membrane.

To determine whether the newly identified section of amino acids is essential to PIP-binding, Shirasu and his colleagues generated AVR3a mutants by substituting the positively charged amino acids found in the newly identified section with negatively charged amino acids. Using a binding assay to investigate the interactions between molecules, the researchers showed that the AVR3a mutants are unable to join to PIP and hence attach to the potato cell membrane. They therefore proposed

that AVR3a may bind to PIPs in the cell membrane in order to remain undetected by the immune system of the potato.

The team's next challenge is to determine how AVR3a molecules and other disease effectors from P. infestans translocate into the host from the site of infection. "Developing ways to block the action of AVR3a and other disease effectors will provide means to control this damaging crop disease," says Shirasu.

More information: Yaeno, T., et al. Phosphatidylinositol monophosphate-binding interface in the oomycete RXLR effector AVR3a is required for its stability in host cells to modulate plant immunity. Proceedings of the National Academy of Sciences USA 108, 14682–14687 (2011).

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