Infectious bacteria force host plants to feed them, study finds

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Sweet corn showing symptoms of Stewart's wilt. Credit: C. Welty, College of Food, Agricultural, and Environmental Sciences

A species of bacteria that infect corn crops compel their hosts to

produce a feast of nutrients that keeps the pathogens alive and thriving long before they start to kill the plant's cells, new research shows.

The study in young maize plants reveals that these bacteria not only generate food for themselves in crops they inhabit, but also coax life-sustaining water from the plants.

Though the lab conditions don't represent exactly what happens in the field, the research provides insights into fundamental processes central to the ability of a pathogen to cause a corn disease prevalent in the central and northeastern U.S. called Stewart's wilt. These bacteria are also creating problems for rice and jackfruit crops in parts of the Eastern hemisphere.

By confirming that a bacterial virulence factor, a protein called WtsE, initiates the mobilization of food and water into spaces where the bacteria reside, the study lays groundwork for future breeding of plants that can put the brakes on these bacterial survivalist tactics. Current breeding practices in corn are based on previous research that focused on boosting the plants' immune response to these infectious bacteria, a species known as Pantoea stewartii.

"No one has shown before that a dynamic flow of nutrients from plant to bacteria supports proliferation of the bacteria during the initial stages of infection. Our findings reveal a bacterial feeding frenzy," said lead study author David Mackey, professor of horticulture and crop science at The Ohio State University.

"There have been no targeted efforts to control nutrient availability as a means to control Pantoea stewartii, or other plant pathogenic bacteria that rely on proteins similar to WtsE for their virulence. This opens up an opportunity for us to look at the mechanism of how WtsE accomplishes this. How is it manipulating the plant cells?"

The research is published in *Cell Host & Microbe*.

The research focuses on a phase called biotrophy: After infecting a plant, the bacteria initially parasitize living host cells and multiply dramatically. Only later do the bacteria start killing plant cells to release further nutrients and cause disease.

In the field, corn flea beetles carry Pantoea stewartii and deposit the bacteria in wound sites the beetles create when feeding on crop stems and leaves. Infections then spread unevenly from these sites.

For this study, Mackey and colleagues infiltrated maize seedlings with a powerful dose of the infectious bacteria, creating a series of uniformly infected leaves. This model system made it possible for the researchers to determine that the release of nutrients and water preceded the death of plant cells.

The team zeroed in on observing the actions of WtsE, one of a class of proteins in pathogenic bacteria known as type III effectors. These proteins are transported from the bacteria into infected plant cells to both suppress plant immunity and, as discovered in the case of Pantoea stewartii, promote availability of water and food.

All of this activity takes place in the apoplast, a relatively dry compartment on the interior of a plant tissue but outside of the plant cells. That dryness is relevant, because one of WtsE's tricks is promoting the availability of water in this space. A leading hypothesis has been that this condition, called "water soaking," results from dying plant cells spilling their contents into the apoplast when bacteria begin their lethal attack.

"This is one of the main points we showed: Infection causes water to accumulate in the apoplast well in advance of killing the plant cells. It's

an active process and it's dependent on the WtsE effector," said Mackey, also a professor of molecular genetics.

And then, once hydrated, the apoplast begins to fill with nutrients that function as sources of nitrogen and carbon for the bacteria—sugars, amino acids and organic acids that are generated and consumed in much higher quantities than exist in the apoplast of a healthy plant.

The researchers confirmed the massive size of the feast by removing the bacteria from the plants and measuring how much carbon and nitrogen they had taken up in a specific period of time—which was 6 and 30 times higher, respectively, than that present in the apoplast of an uninfected plant.

"It's not like the bacteria arrived and ate what was already available," Mackey said. "Plants are relinquishing sources of carbon and nitrogen into the apoplast, where they are assimilated by the bacteria. Additionally, the plant metabolic networks respond to the depletion by making more of those compounds. It's a really dynamic process and the WtsE effector drives that process."

The role of the effector was confirmed genetically—mutant bacteria lacking WtsE were unable to accomplish these same tasks.

With these findings, Mackey's lab is now positioned to pinpoint how WtsE is able to coerce maize into doing its bidding—and specifically, which plant proteins the effector hijacks for help—which could inform future resistant breeding practices.

More information: Irene Gentzel et al, Dynamic nutrient acquisition from a hydrated apoplast supports biotrophic proliferation of a bacterial pathogen of maize, *Cell Host & Microbe* (2022). DOI: 10.1016/j.chom.2022.03.017

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