Change in temperature uncovers genetic cross talk in plant immunity

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Like us, plants rely on an immune system to fight off disease. Proteins that scout out malicious bacterial invaders in the cell and communicate their presence to the nucleus are important weapons in the plant's disease resistance strategy. Researchers at the University of Missouri recently "tapped" into two proteins' communications with the nucleus and discovered a previously unknown level of cross talk. The discovery adds important new information about how plant proteins mediate resistance to bacteria that cause disease and may ultimately lead to novel strategies for boosting a plant's immune system.

Special proteins in the plant, called resistance proteins, can recognize highly specific features of proteins from pathogen, called effector proteins. When a pathogen is detected, a resistance protein triggers an "alarm" that communicates the danger to the cell's nucleus. The communication between the resistance protein and nucleus occurs through a mechanism called a signaling pathway.

"The signaling pathway is like a telephone wire that stretches between each resistance protein all the way to the nucleus," said Walter Gassmann, senior author of the study and associate professor of plant sciences in the Christopher S. Bond Life Sciences Center at the University. "Until now, evidence suggested that, among certain classes of resistance proteins, these wires don't cross -- one resistance protein can't hear what another one is saying."

But in a recent study, Gassmann and his MU colleagues -- post-doctoral

researchers Sang Hee Kim and Saikat Bhattacharjee, graduate students Fei Gao and Ji Chul Nam, and former undergraduate student Joe Adiasor -- "tapped" into these lines and found evidence for cross talk between two different resistance proteins.

The discovery was made while studying another plant protein, SRFR1, which helps to moderate the immune response of the wild mustard plant Arabidopsis thaliana to the bacterial pathogen Pseudomonas syringae. The researchers were interested in why removal of the SRFR1 gene resulted in a plant with an immune system that was always activated. They traced the effect back to expression of the resistance protein, SNC1.

"The connection between SRFR1 and SNC1 was somewhat surprising," said Gassmann. "We identified SRFR1 based on its effect on the plant immune response to the bacterial effector protein AvrRps4, which is usually detected by the resistance protein RPS4, not SNC1."

This class of plant resistance proteins has been thought to be highly specific detectors, meaning each member responds to a different effector protein.

"Based on our work, we think part of the answer is that both SNC1 and RPS4 physically associate with SRFR1. In other words, SRFR1 is where the SNC1 and RPS4 telephone wires get crossed."

The researchers tapped into this cross talk while studying temperature effects on resistance. They found that both proteins, SNC1 and RPS4, contribute to detection of AvrRps4 at 22 degrees Celsius, but only RPS4 does so at 24 degrees Celsius. Gassmann speculated that the temperature dependence may explain why this cross talk had not been previously observed.

"The discovery adds important new knowledge about the underlying mechanism of how plants fight off bacterial infection," said Gassmann, who is also a member of the University's Interdisciplinary Plant Group.

More information: The new research is reported in the November 4 issue of *PLoS Pathogens*.

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