Agent that triggers immune response in plants is uncovered

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Although plants lack humans' T cells and other immune-function cells to signal and fight infection, scientists have known for more than 100 years that plants still somehow signal that they have been attacked in order to trigger a plantwide resistance. Now, researchers at the Boyce Thompson Institute for Plant Research (BTI) on the Cornell campus have identified the elusive signal in the process: methyl salicylate, an aspirin-like compound that alerts a plant's immune system to shift into high gear.

This phenomenon is called systemic acquired resistance and is known to require movement of a signal from the site of infection to uninfected parts of the plant.

The findings are published in the Oct. 5 issue of *Science*.

"By finally identifying a signal that moves from an infection site to activate defenses throughout the plant, as well as the enzymes that regulate the level of this signal, we may be in a position to alter the signal in a way that enhances a plant's ability to defend itself," said BTI senior scientist Daniel F. Klessig, an adjunct professor in plant pathology at Cornell, who conducted the work with Sang-Wook Park and other BTI colleagues.

Their approach, using gene technology to enhance plant immunity, could have wide consequences, boosting crop production and reducing pesticide use.

Methyl salicylate is a modified form of salicylic acid (SA), which has been used for centuries to relieve fever, pain and inflammation, first through the use of willow bark and, since 1889, with aspirin, still the most widely used drug worldwide.

In the 1990s, Klessig's research group reported that SA and nitric oxide are two critical defense-signaling molecules in plants, as well as playing important roles in human health. Then, in 2003 and 2005, the group reported in the Proceedings of the National Academy of Sciences that an enzyme, salicylic acid-binding protein 2 (SABP2), is required for systemic acquired resistance and converts methyl salicylate (which is biologically inactive as it fails to induce immune responses) into SA, which is biologically active.

After plants are attacked by a pathogen, the researchers had previously found, they produce SA at the infection site to activate their defenses. Some of the SA is converted into methyl salicylate, which can be converted back into SA by SABP2.

Using plants in which SABP2 function was either normal, turned off or mutated in the infected leaves or the upper, uninfected leaves, Klessig's group showed that SABP2 must be active in the upper, uninfected leaves for systemic acquired resistance to develop properly. By contrast, SABP2 must be inactivated in the infected leaves by binding to SA.

"This inactivation allows methyl salicylate to build up," explained Klessig. "It then flows through the phloem (or food-conducting "tubes") to the uninfected tissue, where SABP2 converts it back into active SA, which can now turn on the plant's defenses."

Klessig said that it is unclear why plants send this hormone to uninfected tissue in an inactive form, which then must be activated by removal of the methyl group.

"This research also provides insight into how a hormone like SA can actively regulate its own structure -- and thereby determine its own activity -- by controlling the responsible enzyme," noted Park, the lead author of the paper.

Source: Cornell University News Service