Plants teach humans a thing or two about fighting diseases

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Avoiding germs to prevent sickness is commonplace for people. Wash hands often. Sneeze into your elbow. Those are among the tips humans learn.

But plants, which are also vulnerable to pathogens, have to fend it alone. They grow where planted, in an environment teeming with microbes and other substances ready to attack, scientists note.

Now, researchers are learning from plants' immune response new information that could help them understand more about humans' ability to ward off sickness and avoid autoimmune diseases.

This week's journal *Science* reports findings by Texas AgriLife Research scientists of a "unique regulatory circuit" that controls how a plant turns on and off its immune sensor.

"Plants and animals live out their lives mostly in good health, though they may have been subjected to a lot of pathogenic microbes," said Dr. Libo Shan, AgriLife Research plant molecular biologist and lead author for the journal article. "Scientists all around the world have been interested in how a healthy host can fend off invasions of pathogens and turn off the defense responses promptly once the intruder risk factors are decreasing."

The research team found a "unique regulatory circuit" in which BAK1, a protein involved with cell death control and growth hormone regulation,

recruits two enzymes -- PUB12 and PUB13 -- to the immune sensory complex and fine-tunes immune responses.

Basically, the surface of plant cells has sensors that sense microbial invasion. One of the best understood plant receptors is FLS2, found in the common laboratory plant Arabidopsis.

FLS2 could sense the bacterial flagellin, which is a part of the flagellum, or tail-like projection on cells which help it to move. When FLS2 perceives flagellin, a series of "evolutionary conserved immune responses" is activated to fend off bacterial attack, Shan said.

But the immune response can not stay activated or the plant will stop growing and producing.

"To avoid detrimental effects of long-lasting immune activation, plant and animal hosts need a way to switch the activation off," she noted. "How that can be has been a mystery to scientists."

The team discovered that the flagellin perception recruited PUB12 and PUB13 to the receptor FLS2 complex.

Those two enzymes could add a biochemical signature tag, ubiquitin, to the FLS2 receptors which inform sells to degrade the immune senors, she added.

As a result of these actions, immune signaling decreased.

Knowing how immune signaling works may help researchers devise ways to help plants and animals – including humans – regulate their immune systems.

Shan said the mechanism her lab discovered is very broad in that it can

be found in both plants and animals.

"We needed to understand the mechanism so that we can regulate it better," she said. "The host needs to know when the signal is triggered (to fight off a pathogen). Then the immune response needs to go quickly up and then back down when it is no longer needed."

Shan believes that this ability could lead to cures, rather than medical relief, from an assortment of ailments including allergies and autoimmune diseases.

"Plants have figured out how to survive in terms of disease and pest resistance," she added. "And what we learn from them at the molecular level might help us understand animal pathogens better."

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