How pathogens hijack host plants

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Infestation by bacteria and other pathogens result in global crop losses of over $500 billion annually. A research team led by the Carnegie Institution's Department of Plant Biology developed a novel trick for identifying how pathogens hijack plant nutrients to take over the organism. They discovered a novel family of pores that transport sugar out of the plant. Bacteria and fungi hijack the pores to access the plant sugar for food. The first goal of any pathogen is to access the host's food supply to allow them to reproduce in large numbers. This is the first time scientists have a direct handle on controlling the food supply to pathogens and thus a new means to prevent a wide range of crop diseases and losses.

Mutation of the pore-protein genes prevents pathogen infection, such as blight in rice plants. In the absence of a pathogen, the pore proteins, called SWEETs, supply sugars to the developing pollen and may even be the long sought suppliers of nectar in flowers. The researchers found that humans and animals make a similar pore protein, which may play a role in the release of sugars from cells of the intestine, liver, testes, and mammary cells in animals including humans. Studies describing the fluorescence resonance energy transfer (FRET) sensor-based technology for identifying the pore proteins appeared in *FASEB Journal* (April 2010) and the *Biochemical Journal* (Sept 2010). The identification of the novel transporters is published in the November 25, 2010, issue of *Nature*.

Li-Qing Chen, the lead author of the study, explains the pathogenic process: "The primary goal of a pathogen is to tap the plants nutrient

resources. They enter the spaces between the cells where they camp out, feed, and reproduce. Pathogenic bacteria inject activator proteins into the cell that directly induce expression of what we call sugar efflux transporters. Our novel sugar transporters—little flood gates that release sugar from the cell—at the cells' plasma membrane, turn out to be essential for bacterial reproduction. Without food, bacteria can't divide and amplify and thus cannot infect other plants."

Previous to this study, the same lab had identified import mechanisms that drive sugar into cells, and has been searching for the elusive a sugar-pumping mechanism that exports sugar out of cells.

Plants convert energy from sunlight into sugar to nurture pollen, seeds, and nectar and cultivate beneficial microbes in the soil. The researchers work with plants containing FRET sugar nanosensors led them to the hypothesis that yet unknown sugar pores must be present in plants. The researchers looked for potential sugar transporters by screening for genes that might create pore-like activities in the cell membrane in Arabidopsis, a relative to mustard that is widely used for research.

They expressed these genes together with their FRET sensors in human cells that are very inefficient in taking up sugars and thus the nanosensors did not report sugar transport. However, when sugar pore genes are expressed with the nanosensors, the researchers obtain an optical report of sugar transport into the cell. The novel pore proteins in Arabidopsis turned out to have counterparts in rice, the worm C. elegans, and humans.

The scientists then found that pathogenic bacteria and fungi causing powdery mildew disease hijacked different SWEET family members to access to the plant's nutrient resources. The SWEET genes are the cousins of one of the most important rice blight resistance genes used widely all across Asia to prevent blight infections. The researchers

showed that the rice resistance gene Xa13, which had originally been identified by Frank White's group from Kansas and Shiping Wang's lab from Huazhong Agricultural University, China, also functions as a sugar pore. When production of the pore is suppressed, the plant becomes resistant to a blight bacteria. This result suggests to the researchers that the protein supplies sugar to the bug during infection. Since different pathogens try to hijack different pore genes, a drug that can block the activity of all SWEET cousins at the same time would turn off sugar supply to a pathogen that requires its host to live. Such a drug might be a powerful new way to reduce crop losses to a wide spectrum of pathogens. The research changes the perspective of plant-pathogen researchers by getting to the root of the process of infection. Understanding this process will advance infection prevention research against a wide variety of pathogens—bacterial and fungal.

Sugar efflux has been a mystery in human and animal systems as well. It is required in our intestine to transfer food-derived sugars to our blood stream and from the liver during fasting to keep glucose levels in the blood constant. The fact that the researchers also found a similar gene HsSWEET1 in humans that mediates sugar outflow in the liver and intestine suggests that study of this gene could open up a new avenue for diabetes research.

"These discoveries, made in the model plant Arabidopsis, have opened up an entirely new line of investigation for better crop protection techniques. Specifically, plant breeding and genetic changes that prevent hijacking of the pore function may serve to provide resistance to some problematic plant diseases," remarked Frank White from Kansas University, a coauthor of this study. "Since the same genes are shared in many different organisms, including people, these genes could also be important to medicine."

Provided by Carnegie Institution

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