Scientists map attack tactics of plant pathogens

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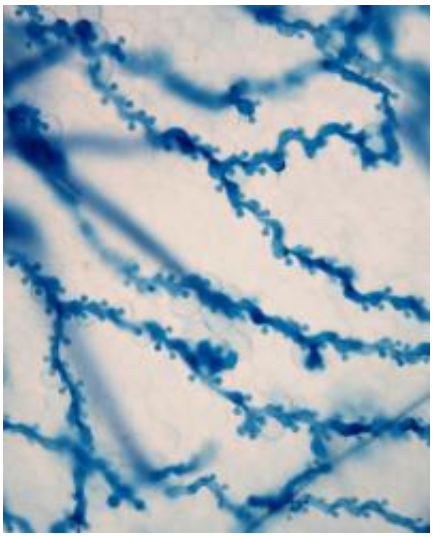
A leaf infected by a pathogen called an oomycete. The oomycete gains entry into the leaf's intracellular spaces through natural openings and then grows by extending hyphae (filaments) between cells. When the hyphae fill up the leaf, the oomycete releases the next generation of infectious spores (the white tree-like structures emerging from the leaf surface. Oomycetes cause downy mildew diseases of many plants; one was responsible for the Irish Potato Famine and another for Sudden Oak Death Syndrome. Credit: Petra Epple, Dangl Lab, UNC-Chapel Hill.

Every year, plant diseases wipe out millions of tons of crops, lead to the waste of valuable water resources and cause farmers to spend tens of

billions of dollars battling them.

Now a new discovery from a University of North Carolina at Chapel Hill-led research team may help tip the war between plants and pathogens in favor of flora.

The finding – published in the July 29, 2011, issue of the journal *Science* – suggests that while pathogens employ a diverse arsenal of weapons, they use these to attack plants by honing in on a surprisingly limited number of cellular targets.



A leaf infected by an oomycete pathogen. Special feeding structures, called haustoria, bulge from the pathogen's hyphae (filaments) into the inside of the plant cells (the purple balloon-like structures inside the clear-colored individual cells). Credit: Petra Epple, Dangl Lab, UNC-Chapel Hill.

"This is a major advance in understanding the mechanisms involved in the ongoing evolutionary battle between plants and pathogens," said Jeff Dangl, Ph.D., the study's lead author and John N. Couch Professor of

Biology in the College of Arts and Sciences.

The new finding is one of two studies published concurrently in *Science* related to the first comprehensive plant "interactomes" – maps of the tens of thousands of interactions that link a cell's proteins. Those connections govern how proteins assemble into complex functional machines that dictate the tasks a cell can perform, such as growth, division and response to light, water and nutrients. And these same machines are often recruited into the battle against infectious agents.

One of the new studies mapped the interactome for about a third of the proteins encoded by the genome of the plant Arabidopsis thaliana, or thale cress. Arabidopsis is widely used for research purposes as a model organism – similar to the way mice are used in medical research – because of traits that make it useful for understanding the workings of many other plant species.

Dangl's group led an additional study incorporating that interactome data with the construction of a second interactome. The second map focused on understanding how two very different pathogens (the bacteria Pseudomonas syringae and the oomycete parasite Hyaloperonospora arabidopsidis) infect plants and how plants fight back.

One method that these pathogens, which live in between cells, use for successful infection is to deploy virulence proteins (known as effectors) into the plant cell. The effectors muzzle the host's defenses and allow the pathogen to hijack the plant's cellular machinery.

In the new study, Dangl and his collaborators at institutions including Harvard University, the Salk Institute in La Jolla, Calif., and the University of Warwick, U.K., found that these two pathogens have evolved to focus their effectors onto a limited set of roughly 165 interconnected proteins that act in cellular machines in Arabidopsis cells

– despite the fact that they last shared a common ancestor over 2 billion years ago, and use vastly different mechanisms to colonize plants.

"This likely means that to suppress host plants' defenses, all plant pathogens have evolved weapons that focus on a relatively small group of cellular machines," Dangl said. "Knowing this should facilitate faster breeding for disease resistance and development of environmentally sustainable treatments for many devastating plant diseases."

He said that neither interactome is a complete map, and more work needs to be done fully identify which protein networks are targeted by pathogens.

"We've found the needles in the haystack, but we still have to comb through another two-thirds of the hay," said Dangl. "Our data suggest that there will be only a few hundred targets for effectors from all pathogens, out of the roughly 27,000 proteins encoded in the whole Arabidopsis genome."

Academic scientists, seed breeders and biotech companies interested in these proteins will benefit from freely available data from both interactomes. The findings also could have implications for human health research.

"Professor Dangl and colleagues have used a powerful combination of network theory and laboratory experimentation to develop an approach to understanding the evolutionary logic by which pathogens and their hosts interact," said James Anderson, Ph.D., who oversees regulatory biology grants at the National Institutes of Health. "While this study focused on plants, the results illustrate the value of model organisms in revealing fundamental principles that help us understand human responses to infectious diseases and provide the basis for devising new therapeutic strategies."

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