History is key factor in plant disease, study finds

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The sudden oak death pathogen infects a bay leaf tree. Credit: Doug Schmidt, Garbelotto Lab, UC Berkeley.

(Phys.org) -- The virulence of plant-borne diseases depends on not just the particular strain of a pathogen, but on where the pathogen has been before landing in its host, according to a new study from researchers at the University of California and the United States Department of Agriculture's Agricultural Research Service (USDA ARS).

The study demonstrates that the pattern of gene regulation—how a cell

determines which genes it will express and how it will express them—rather than gene make-up alone affects how aggressively a microbe will behave in a plant host. The pattern of gene regulation is formed by past environments, or by an original host plant from which the pathogen is transmitted.

“If confirmed, this finding could add a key new dimension to how we look at microbes because their history is going to matter and their history may be hard to reconstruct,” said Matteo Garbelotto, an adjunct professor of environmental science, policy and management at UC Berkeley and corresponding author of the study.

Epigenetic factors—for example, gene regulation mechanisms controlled by diet or exposure to extreme environments—are well known to affect the susceptibility of humans to some diseases. The new study, published in the journal *PLoS ONE* on April 18, is the first to show a similar process for plant pathogens. Garbelotto said other scientists have hypothesized that gene regulation has an effect on plant pathogens, based on the evolutionary rates of portions of the genome that are known to have an effect on gene regulation. “Our work provides the concrete evidence those hypotheses were correct,” he said.

Researchers showed that genetically identical strains of the sudden oak death pathogen isolated from different plant hosts were strikingly different in their virulence and their ability to proliferate, and showed that these traits were maintained long after they had been isolated from their hosts.

An oak recently killed by sudden oak death. Credit: Doug Schmidt, Garbelotto Lab, UC Berkeley.

“We show that an identical strain placed in two different plant hosts will undergo distinct changes that will persistently affect the strain’s virulence and fitness,” said Takao Kasuga, a molecular geneticist with the USDA ARS, and the lead author on the study.

The implications for disease control are significant. Researchers say that it may not be enough to know what strain of pathogens they are dealing with in order to make treatment decisions; it also may be necessary to know how the pathogen’s genes are being regulated. This study shows that gene regulation may be the result of the environments the strain inhabited before being identified.

Garbelotto used a parallel example of a well-known human pathogen: particular strains of the H1N1 flu virus have been identified as highly virulent, so a diagnosis of one of these strains indicates to doctors that they should treat that flu aggressively.

“But, hypothetically, if you caught one of these aggressive strains of H1N1 from a guy that went to, for example, Paris, it could be 10 times more dangerous, and you may never know from whom you got it, and it’s even less likely that you’ll be able to learn where your infector visited before passing the germ on to you,” Garbelotto said.

In plants, Garbelotto said, tracking a pathogen’s history may prove even more difficult, but correct information could give scientists a new weapon to use against virulent strains of diseases like sudden oak death, which can devastate forests and the ecosystems that depend on them.

The researchers also identified two groups of genes that are capable of affecting virulence and whose expression patterns are indicative of the previous host species they inhabited. Over-expression of transposons—mobile genetic elements—combined with under-expression of crinkler genes—genes involved in host-pathogen interactions—is consistently associated with lowered fitness of the pathogen. Understanding the regulation of these genes may provide scientists with some future approaches to control the disease, such as manipulating the gene expression to artificially reduce the aggressiveness of plant pathogens.

While Garbelotto stresses that more study is needed, he says if the paper’s findings are confirmed, it could influence not just treatment but policymaking as well. “Most countries impose regulations on microbes based on their genetic make up—which ones can and can’t cross state and international lines and how they must be transported,” he said. “Our findings suggest that when making regulatory policy, we may also need to identify gene expression levels and take into account the history of a microbe.”

Coauthors on the study include Melina Kozanitas and Daniel Huberli also of UC Berkeley, Mai Bui of the USDA ARS and David M. Rizzo, a

professor of Plant Pathology at UC Davis. The National Science Foundation's Ecology and Evolution of Infectious Diseases program funded the study. The US Department of Agriculture-Forest Service's Pacific Southwestern Research Station Sudden Oak Death research program was also a funder.

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