

UNC Charlotte scientist stalks the 'bird flu' of trees

UNC Charlotte scientist tracks the spread of Sudden Oak Death, caused by algae

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In 1995, oak trees began dying enmasse in some coastal California forests.

The disease, which causes oozing cankers and cuts off water and nutrition to oaks, was unknown to science but was soon dubbed Sudden Oak Death.

It took scientists five years to pinpoint a pathogenic fungus-like brown algae as the cause. In 2001, it received a scientific name, *Phytophthora ramorum*.

Sudden Oak Death spread north along the West Coast, and today it is established in forests from California to Washington. Nobody knows how much of North America's forests are at risk of infection, but in the decade and a half since it emerged, a scientist at UNC Charlotte has been one of the leaders in seeking to understand it - and find out if it might infect Eastern forests.

Ross Meentemeyer uses high-tech mapping technologies to examine how *P. ramorum* survives, reproduces and spreads.

"You might think of Sudden Oak Death as the bird flu of the plant world," Meentemeyer said. He began studying it a decade ago at ground zero of its emergence in California's Big Sur ecoregion, as a geography professor at Sonoma State University.

Today, Meentemeyer directs the Center for Applied Geographic Information Science at UNCC, a lab in the department of geography and earth sciences. CAGIS's approach to studying Sudden Oak Death cuts across academic boundaries and draws from epidemiology, plant pathology, molecular biology, land use planning and information sciences. Research at CAGIS is helping policymakers to better allocate resources to contain the disease's spread in the West, and to prepare for its possible occurrence in the East.

Their maps are not static, like the ones you may see on your car's GPS screen. Instead, they provide visual and spatial awareness for where the disease may occur. The data used to build them can also be manipulated to predict disease spread or test management policies.

Meentemeyer and his lab of 20 scientists and students can even predict how a host plant's genotype variability across a landscape might influence its susceptibility to the disease. This predictive power gives policy-makers visual information to figure out how best to fight it.

But the lab has struggled to quantify the total number of trees that have died, or are infected, because the data needed are so massive.

"Everyone is afraid to put a number on it," Meentemeyer said. "So we say 'potentially millions' of trees are infected, or have all ready died. It could be a very high number. And then the potential number of at-risk trees is going to be even larger."

Seemingly unrelated things such as fire suppression policies, hiking trails and urban sprawl have all found their way into Meentemeyer's data, offering surprising mechanisms for how Sudden Oak Death creeps and lurches across landscapes.



In one study, his team found that fire suppression policies in California's Big Sur led to an expansion of woodlands at the loss of shrubland and grassland habitat, which both depend upon fire. The newer woodlands were different than the old and were more likely to harbor *P.ramorum* because they contained comparatively more California bay laurel, which is a key host for transmitting the disease to oak trees, Meentemeyer said.

In another study, Meentemeyer and his colleague, J. Hall Cushman of Sonoma State University, found that human activities were strongly associated with *P.ramorum*'s presence. They found significant amounts of the organism in soil along hiking trails - but not off trail - and they found that it was more likely to be present near where wildlands and urban areas meet.

Under his guidance, UNCC graduate student Sarah Haas is investigating links between plant biodiversity and disease risk. She said their initial data show that in areas with a great variety of plant species, the presence of disease appears to decline.

"This pathogen is a generalist; it can live on many different hosts," Haas said. "But only two seem to drive its infectiousness: California bay laurel andtanoak, which produce about 90 percent of its reproductive spores."

When a tree succumbs, it often dies within a single growing season. But oaks are dead-end, accidental hosts for the pathogen. *P.ramorum* cannot complete its life cycle on them. For that, it needs woody plants called "foliar hosts," which do not die when infected.

More than 75 of those plant hosts have been identified, and all develop non lethal infections on their leaves. Spores are carried to new hosts by water droplets.

Interstate shipments of nursery plants are the most likely way *P.ramorum* might leap across the continent. The U.S. Department of Agriculture's Animal and Plant Health Inspection Service has regulated host plants shipped out of quarantined counties since 2002. But some plants slip by.

In March 2004, three Florida nurseries were found positive for *P.ramorum*. By the end of 2004, 177 nurseries in 22 states received infected shipments, resulting in the destruction of more than 787,840 plants and heightened inspections. In March 2009, *P.ramorum* was found near a nursery in Gadsden County, Fla. In June 2009, it was found on a rhododendron in a residential landscape in Greenville County, S.C.

Laboratory trials conducted by the U.S. Department of Agriculture have shown that live oaks and red oaks are susceptible. Meentemeyer's work shows that though Eastern forests contain dozens of common woody plants that act as hosts - rhododendron and camellias are like magnets for the pathogen - different weather patterns from the west might prevent it from reproducing.

"It's still considered a threat," said Kier Klepzig, assistant director for research at the Forest Service's Southern Research Station in Asheville, "but less so since it seems to have had several chances to establish here. But it has not."

Despite the low likelihood of *P.ramorum*'s establishing in Eastern forests, scientists don't like to say "never." If the pathogen were to mutate or adapt its reproductive cycle, Meentemeyer's work would come into play.

"We have the ability to develop models to predict the spread of the pathogen if it were to reach Eastern forests," Meentemeyer said. "And we'd be better prepared to contain it."