Land Letter
THE NATURAL RESOURCES WEEKLY REPORT

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1. INVASIVE SPECIES: Despite gains in science, origins of Sudden Oak Death still a mystery

Arthur O'Donnell, *Land Letter* editor

SANTA ROSA, Calif. -- After years of investigation, forest biologists have developed a deeper understanding of the cause and effects of "Sudden Oak Death." Still, they cannot fully explain how a fungal pathogen, *Phytophthora ramorum*, developed into such a fatal agent, responsible for killing as many as 1 million oak trees in California while currently infecting an equal number with no hope of preventing their eventual mortality. And with more recent confirmation of similar symptoms -- from a different strain of the fungus -- affecting oaks and beech trees in the United Kingdom and parts of Europe, the mystery remains as to the origins of this disease.

During a scientific symposium this week devoted to the latest research into Sudden Oak Death, researchers offered more information about the extent of the problem, but few potential solutions beyond continued quarantines of host plants and mixed success in attempts to eradicate the problem at the local level.

As Sudden Oak Death spreads, its impacts are plainly visible along the U.S. Highway 101 corridor in Sonoma County, Calif. Photo by Arthur O'Donnell.

The epidemic, first detected in 1995, affects several species of oak trees most severely -- coast live oak, California black oak and Shreve oak -- along with the tanoak that proliferates along the state's coastal ranges.

However, some two dozen other plant species ranging from the rhododendron to the California bay laurel can carry non-fatal leaf or stem infections and have been identified as hosts of strains of the *Phytophthora* (literally, "plant destroyer") fungus. Though the pathogen was reported as a likely cause of disease in nursery rhododendrons as early as 1993, *P. ramorum* was not isolated as the cause of SOD until 2000 by researchers at the University of California.

As scientists sort through discoveries of additional strains of the fungus, including *P. kernoviae* in the United Kingdom and likely avenues of transmission, they have reached something of a consensus about how the disease was introduced. But they are still working out theories about how it has spread so quickly.

"For a recently discovered organism, our understanding is relatively good," said Matteo Garbelotto, a forest pathologist at the University of California, Berkeley. "In four years, it's amazing to see how far we've come."

While a single imported rhododendron is generally considered to be the original host that brought the pathogen into California, it is the bay laurel that has now become the "number one culprit" in spreading the disease in the wild, Garbelotto said.

Studies have also provided better understanding of necessary conditions for transmission. "Water is essential and temperatures that are relatively warm. But this is a disease that can survive a pretty wide spread in cold or hot. It's one of the most heat resistant in the genus, and so eradication is very difficult," he explained.

Also, the pathogen presents a great deal of diversity, with at least four separate strains now characterized, all leading to the same symptoms, although with possible variations in species affected and optimal conditions for infections. "What we have in California is different than in Europe," Garbelotto explained. "It looks very different under the microscope."

Joan Webber of the U.K. Forest Research Agency concurred. "In Europe, invasive *ramorum* colonizes slightly different plants. There are quite distinct differences."

Stephen Hunter of the U.K.'s Department of Environment, Food and Rural Affairs, reported, "The reality is, there are no imports

from the West Coast of the States of host material. I can say we did not get the disease from you and you did not give the disease to us."

The big question remains, said Webber: "Where does it come from?"

Aerial transmission confirmed, forest impacts quantified

Among reports presented during this week's symposium was a mapping study in the United Kingdom that appeared to confirm previous reports that the spores of the fungus can be spread through the air -- as much as 50 meters away from the original site.

"It's aerial," Garbelotto said. "That's relatively new. That it can move through the air rings warning bells, even though its movement ability in the air is limited. What we do to contain soil is not going to be effective."

Funding has also been devoted to monitoring how broadly the disease has spread and how deeply it affects lands that have been infected.

According to Dave Rizzo, professor of plant pathology at U.C. Davis, "The pathogen has continued to expand its range. It used to be patchy. Now we're seeing bigger patches and more trees affected."

The effect of two consecutive wet springs has caused a "dramatic expansion on how much it's reproducing and the number of dead trees visible in the landscape," he said.

As of January 2001, SOD appeared confined to an approximately 100-mile-long stretch from Santa Cruz to Sonoma County. Currently, the disease has been identified as far south as San Luis Obispo, further west in Mondocino County to within four miles of the Pacific Ocean, and just north of the California/Oregon border into Curry County.

The Oregon location was first identified in 2001 but thought to be eradicated. "Because it was an isolated problem, they started an eradication effort to try to get rid of the pathogen completely," Rizzo explained. Part of the strategy was to create a buffer zone around the oaks by removing potential host species and burning material. However, this year, *P. ramorum* was again identified in 139 tanoak trees at 35 sites, at least two of which were outside the eradication quarantine area. The largest site was 10 acres and included 40 infected trees. The quarantine area will be expanded, federal officials said.

"*Ramorum* is fairly resilient," Rizzo said. "You can knock it way back, but it's probably impossible to eradicate. You'd have to clear cut Marin County and Sonoma County."

Burning has also been tried on 45-acres of public and private lands in Humboldt County under a silvicultural treatment program conducted by the U.C. Cooperative Extension.

New research has quantified the impacts of Sudden Oak Death in the Big Sur ecoregion. More than 200,000 trees have been killed, covering about 17 percent of the study area. Photo courtesy of Dave Rizzo, University of California, Davis.

In the United Kingdom, an eradication program in England has, so far, been a success. "Where eradication has been undertaken quickly and thoroughly there were no further infections," reported Judith Turner of the Central Science Laboratory in York. Still, the group has detected spores of the pathogen, although evidently not enough to cause new outbreaks. "This is all about thresholds," she said.

A death-pocked landscape

Rizzo and colleagues presented new research into quantifying SOD mortality in an 80,000-hectare area of Big Sur, south of Monterey. Using satellite imaging and ground level surveys, the group concluded that more than 214,000 tanoak trees have been killed. About two-thirds were located in mixed redwood/tanoak forests at the highest elevations, while the other third were located in mixed broadleaf forests at mid-level elevations. There were no significant tree deaths in the lower grassy areas.

Overall, 17 percent of the susceptible forest had been infected as of 2005, reported Doug Shoemaker of the Center for Applied GIS at the University of North Carolina, and up to 60 percent of some stands of redwood/tanoak forest were killed. The group hopes to continue monitoring the area to see if the infection deepens among surrounding trees. "This ends up being a snap shot of what happened up to 2005," Shoemaker said.