

Information
Crossfile

Sudden oak death has reached epidemic levels in some coastal forests of central California. Identified in 2001, the pathogen *Phytophthora ramorum* causes the disease, which has spread rapidly along the Pacific Coast. The deadly fungus is not confined to oaks; investigators have confirmed bigleaf maple (*Acer macrophyllum*), wild rose (*Rosa gymnocarpa*), Douglas fir (*Pseudotsuga menziesii* var.

menziesii), coast redwood (*Sequoia sempervirens*), and dozens of other species, including rhododendron (*Rhododendron* spp.) and other popular horticultural plants, as hosts. (Further details about host plants are available at <http://www.suddenoakdeath.org>. The Web site also provides information about research, nursery updates, management recommendations, training, and regulations.)

Sudden oak death manifests itself as either lethal branch or stem infections, or nonlethal foliar and twig infections. The lethal form of the disease kills several ecologically important trees, including tanoak (*Lithocarpus densiflora*), coast live oak (*Quercus agrifolia*), California black oak (*Quercus kelloggii*), canyon live oak (*Quercus chrysolepis*), and Shreve's oak (*Quercus parvula* var. *shrevei*) (Rizzo et al. 2002). Except for tanoak, these oak species appear to be epidemiological dead-ends or "terminal hosts" because the pathogen does not form spores and spread from the trees.

Many governmental agencies are involved in finding solutions to this problem. For instance, the California Oak Mortality Task Force is composed of agencies such as the USDA Forest Service, National Park Service, and California Department of Forestry along with the University of California and many other local agencies and private organizations. The task force has established an extensive monitoring program for the disease. The program focuses on the early detection of

pathogen activity at isolated locations, where applying chemical treatments or attempting eradication may be possible. The considerable cost of monitoring necessitates careful targeting and prioritization and presents a significant challenge given California's extensive size, the diversity of host species, and the environmental variability in the state. Therefore, understanding where and when the risk of establishment of *P. ramorum* is elevated is essential in order to effectively monitor the disease and manage threatened forests.

Meentemeyer et al. (2004) presents a model for predicting the spread and establishment of sudden oak death in plant communities in California. The California Oak Mortality Task Force is already using this model to target early detection monitoring and predict oak and tanoak mortality. Based on the combined effects of spatial variability in climate (i.e., 30-year monthly averages [1961–1990]) and host vegetation (i.e., USDA CALVEG dataset) for each month of the pathogen's general reproductive season (December–May), the model



The Integrated Pest Management Program of the NPS Pacific West Region has designed a variety of devices for disinfecting park visitors and staff of the organism causing sudden oak death, which can be transported unwittingly to and from parks in soil clinging to anything from shoes to bicycle tires. This device is being tested for effectiveness and acceptance by bicyclists at a park administered by the Mid-Peninsula Open Space District in the San Francisco Bay area.

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predicts the risk of continued spread and establishment. The five predictor variables are a host species index and four temperature and moisture variables (i.e., precipitation, relative humidity, and minimum and maximum temperature). Investigators evaluated the model's performance by comparing its predictions to field observations of disease presence and absence.

The model mapped sites as very high risk where dense concentrations of host species (i.e., very high host index values) coincide with highly suitable climate conditions (e.g., mild temperatures [64°F–68°F {18°C–20°C}] and water existing on plant surfaces for at least 6–12 consecutive hours). Very high risk habitats occur in the coastal environments within 30 miles (50 km) of the Pacific Ocean. In addition, high risk areas form a nearly contiguous band through the coastal counties from the Oregon border to northwestern San Luis Obispo County. High risk habitats occur where moderately high host index values correspond with moderately to highly suitable climatic conditions.

To date, plants at Golden Gate National Recreation Area, Muir Woods National Monument, and Point Reyes National Seashore have been infected with the disease. Additional monitoring this year will be investigating whether or not the disease occurs at Santa Mountains National Recreation Area and Redwood National Park. The pathogen affects both urban and wildland forests and may spread via nursery plants and soil movement. Hikers and mountain bikers also commonly transport the pathogen (see photo, page 13). Although much remains to be learned about the ecology and epidemiology of sudden oak death, the model that Meentemeyer and others present serves as a seemingly simple yet effective management tool for targeting forests for early detection monitoring and protection. Based on the model's results, an alarming number of uninfected forest ecosystems in California face considerable risk of infection by *P. ramorum*. The risk maps of Meentemeyer et al. (2004) are available at <http://kellylab.berkeley.edu/SODmonitoring/SODmapsState.htm>.

More information about *P. ramorum* is also available at <http://www.aphis.usda.gov/ppq/ispm/pramorom> (U.S. Department of Agriculture) and <http://www.na.fs.fed.us/sod> (USDA Forest Service).

References

- Meentemeyer, R., D. Rizzo, W. Mark, and E. Lotz. 2004. Mapping the risk of establishment and spread of sudden oak death in California. *Forest Ecology and Management* 200:195–214.
- Rizzo, D. M., M. Garbelotto, J. M. Davidson, G. W. Slaughter, and S. T. Koike. 2002. *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp. and *Lithocarpus densiflorus* in California. *Plant Disease* 86:205–214.