Sudden Oak Death in California

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Abstract

A new canker disease of *Lithocarpus densiflorus*, *Quercus agrifolia*, *Q. kellogii*, and *Q. parvula* var. *shrevei* in California has been shown to be caused by *Phytophthora ramorum*. The pathogen is a recently described species that was previously known only from Germany and The Netherlands on *Rhododendron* and *Viburnum*. This disease has reached epidemic proportions in mixed evergreen and redwood forests over an area approximately 300 km long along the central coast of California. The most consistent and diagnostic symptoms on larger trees are the cankers that develop before foliage symptoms become evident. Cankers have brown or black discolored bark, seep dark red sap and occur on the trunk at the root crown up to 20 m above the ground. Cankers do not enlarge below the soil line into the roots. Cankers can be over 2 m in length and are delimited by thin black zone lines in the inner bark. Foliage on affected trees often turns from a healthy green color to brown over a period of several weeks. In *L. densiflorus* saplings, *P. ramorum* was isolated from branches as small as 5 mm diameter. Lithocarpus densiflorus and *Q. agrifolia* inoculated with *P. ramorum* in the field and greenhouse developed symptoms similar to those of natural infections. The pathogen was re-isolated from inoculated plants, thereby confirming pathogenicity. The host range in California has been greatly expanded and now includes *Rhododendron* spp., madrone (*Arbutus menziesii*), huckleberry (*Vaccinium ovatum*), manzanita (*Arctostaphylos* spp.), California bay laurel (*Umbellularia californica*), buckeye (*Aesculus californica*), bigleaf maple (*Acer macrophyllum*), toyon (*Heteromeles arbutifolia*), coffeeberry (*Rhamnus californica*) and honeysuckle (*Lonicera hispidula*). On these hosts, *P. ramorum* causes a variety of foliar and branch symptoms. Significant dieback and plant death has been recorded on the ericaceous hosts (e.g., rhododendron, madrone). On hosts such as bay laurel and buckeye, only leaf symptoms have been noted. However, disease progression is poorly understood on most non-oak species.

In some mixed-evergreen forests nearly all woody plants can serve as host for *P. ramorum*. For example, at China Camp State Park in Marin County, the overstory consists of coast live oak, black oak, bay laurel, and madrone. Toyon is the primary understory shrub species. Buckeye and manzanita are found at the margins of closed canopy. Valley oak (in riparian areas and at the edge of the closed canopy) appears to be the only woody plant species unaffected by *P. ramorum* at this location. There are many other plant species that co-occur with oaks and tanoak that have not been tested for susceptibility to *P. ramorum*. This includes additional species in the Ericaceae (e.g., salal). As the geographic range of *P. ramorum* changes, it will potentially encounter other hosts that are not found in forests in the current range.

The most encouraging result from our inoculation studies was the finding that *P. ramorum* did not cause lesions on the white oaks, valley oak (*Q. lobata*) and blue oak (*Q. douglasii*) that differed significantly from the control wounds. However, the pathogen did survive in the discolored tissue for at least 6 weeks following inoculation. It is unknown if the pathogen can continue to survive for long periods in white oak tissue and eventually cause mortality of plants. To date, no species in the white oak group has been diagnosed with the disease in the field.

We have conducted a preliminary study comparing the susceptibility of California oaks and eastern North American oak species to *P. ramorum*. Challenging seedlings of northern red oak (*Quercus rubra*) and pin oak (*Quercus palustris*) with *P. ramorum* resulted in longer lesions in the bark than those that developed in the coast live oak and California black oak. Lesions in northern red oak were nearly twice as long as those observed in coast live oak and about the same length as those observed in tanoak. Tanoak is considered to be the most susceptible California tree species to *P. ramorum*. Extrapolation of results from seedling experiments to the potential effects on mature trees must be done cautiously. However, because lesion sizes in red oak and pin oak seedlings were much larger than in coast live oak seedlings (a species in which the adults are very susceptible), we suggest that it is likely that mature trees of northern red oak and pin oak will be susceptible to infection by *P. ramorum*. Therefore, efforts to prevent spread of *P. ramorum* to eastern North American forests are critical.

An important step in controlling *P. ramorum* involves understanding how it is spread. The presence of diseased oaks at all elevations on hillsides and the above-ground nature of the disease suggest wind-blown rain or rain splash as a common mechanism for movement of spores. Although viable spores have yet to be found on infected oak tissue, other hosts may serve as sources of rain-dispersed inoculum. In the laboratory, abundant sporangia form on moistened leaves of infected bay laurel and rhododendron within 72 hours. These sporangia break off and easily disperse in water. Chlamydospores are also observed on the surface of moistened bay leaves. Consistent with these results, *P. ramorum* has been recovered from rain, soil, litter and stream water from woodlands with infected oak and bay trees. Spores of *P. ramorum* do not survive drying, but in moist conditions can survive for at least one month.

The broad host range of *P. ramorum* that we have discovered in California forests suggest that this pathogen has the potential to cause similar, long-term landscape level changes in these forests. Loss of oaks and other overstory trees and shrubs will have cascading effects on these ecosystems including increased fire hazards, soil erosion, and loss of habitat for wildlife. Developing restoration plans for these ecosystems will represent a significant future challenge for forest managers. An understanding of the host range and spatial distribution of the pathogen is pivotal in formulating hypotheses for further epidemiological research, developing monitoring strategies and management guidelines that may either prevent further spread of the disease or ameliorate disease conditions where the disease may be only recently present.

Literature Cited

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