

Sudden oak death: endangering California and Oregon forest ecosystems

David M Rizzo¹ and Matteo Garbelotto²

Sudden oak death is a new disease affecting tanoak (*Lithocarpus densiflora*) and oaks (*Quercus* spp) in California and Oregon, caused by the recently described pathogen *Phytophthora ramorum*. It has reached epidemic proportions in several counties in central California, leading to the death of tens of thousands of trees. In addition to oaks and tanoak, *P ramorum* has been found in nearly all woody plant species in mixed evergreen and redwood forests from central California to southern Oregon. Plant species that are not killed appear to serve as a reservoir for the pathogen. The high susceptibility of tanoak to infection and death suggests that *P ramorum* is an exotic pathogen, but its origins, and most details of its biology and ecology, remain unknown. Our limited knowledge only compounds our concern over the long-term implications of this epidemic for the ecology of coastal forests.

Front Ecol Environ 2003; 1(5): 197–204

Over the past century, a number of plant diseases have had major impacts on forest ecosystems around the world. Classic examples include chestnut blight in North America, caused by *Cryphonectria parasitica*, and jarrah dieback in western Australia, caused by *Phytophthora cinnamomi* (Anagnostakis 1987; Weste and Marks 1987). Some exotic diseases, such as chestnut blight and white pine blister rust, have severely impacted populations of a single plant species, leading to a cascade of changes in forest ecosystems. In fewer cases, a pathogen with a broad host range has affected forest ecosystems directly. Since it was introduced in the 1920s, *P cinnamomi* has virtually eliminated most tree species over hundreds of thousands of hectares of the jarrah (*Eucalyptus marginata*) forests of western Australia, converting them to grassland or shrubland (Weste and Marks 1987; Erwin and Robiero 1996). The emergence and introduction of new plant diseases continues to this day (Baskin 2002; Campbell and Schlarbaum 2002).

In a nutshell:

- The newly discovered pathogen *Phytophthora ramorum* causes an emerging disease that has resulted in extensive mortality of tanoak and oak in California
- The pathogen has a broad host range that encompasses almost all woody plant species in coastal forests
- Pathways for the spread of *P ramorum* include rainsplash, infested soil, and infected plant material
- Very little is known about the ecology of this pathogen, but its long-term negative effects on coastal forests could be considerable

One of the most recent examples of an emerging forest disease is “sudden oak death”. The symptoms that define it were first recognized during 1994–95, and over the next few years it reached epidemic proportions in oak forests along approximately 300 km of the central California coast (Garbelotto *et al.* 2001). The most visibly affected hosts included tanoak (*Lithocarpus densiflora*), coast live oak (*Quercus agrifolia*), California black oak (*Quercus kelloggii*), and Shreve’s oak (*Quercus parvula* var *shrevei*) (Rizzo *et al.* 2002a). Eventually it was shown that the pathogen *Phytophthora ramorum*, recently described from rhododendron (*Rhododendron* spp) and viburnum (*Viburnum* spp) in Europe (Werres *et al.* 2001; Rizzo *et al.* 2002a), was causing the disease. Research soon revealed that *P ramorum* could infect other plant species as well. Over the past year and a half, over 20 additional species from 12 plant families have been identified as potential hosts (Rizzo *et al.* 2002b; Garbelotto *et al.* 2003; Table 1). Additional hosts are likely to be found, as many other plant species are susceptible to infection under laboratory conditions (Hansen and Sutton 2002; Linderman *et al.* 2002; Parke *et al.* 2002; Tooley and Englander 2002).

What is sudden oak death, and what are the ecological implications of this new disease? Here we review what is known about the biology, ecological implications, and management of *P ramorum* in the coastal forests of California and Oregon.

■ Disease characteristics

Across the range of hosts, we can distinguish two different diseases caused by *P ramorum*: lethal branch or stem infections, and non-lethal foliar and twig infections. The pathogen causes large cankers on the main stem of oaks and tanoak, and often kills the tree (Figure 1). The name “sudden oak death” comes from the fact that large groups

¹Department of Plant Pathology, University of California, 1 Shields Ave, Davis, CA 95616 (dmrizzo@ucdavis.edu); ²Department of Environmental Science, Policy and Management, Ecosystem Science Division, 151 Hilgard Hall, University of California, Berkeley, CA 94720

Table 1. Known hosts infected by *P ramorum* (compiled from Rizzo *et al.* 2002a, 2002b; Garbelotto *et al.* 2003)

<i>Quercus agrifolia</i>	<i>Rubus spectabilis</i>
<i>Quercus kelloggii</i>	<i>Aesculus californica</i>
<i>Quercus parvula</i> var <i>shrevei</i>	<i>Rhamnus californica</i>
<i>Quercus chrysolepis</i>	<i>Rhamnus purshiana</i>
<i>Lithocarpus densiflorus</i>	<i>Corylus cornuta</i>
<i>Arbutus menziesii</i>	<i>Lonicera hispidula</i>
<i>Vaccinium ovatum</i>	<i>Viburnum</i> spp ³
<i>Arctostaphylos</i> spp ¹	<i>Toxicodendron diversilobum</i>
<i>Rhododendron</i> spp ²	<i>Trientalis latifolia</i>
<i>Umbellularia californica</i>	<i>Sequoia sempervirens</i>
<i>Acer macrophyllum</i>	<i>Pseudotsuga menziesii</i>
<i>Heteromeles arbutifolia</i>	

¹Probably multiple species infected. Known definitively from *A manzanita*

²Multiple species infected including *R macrophyllum* and *R catawbiense*

³Multiple species infected including *V bodnantense*, *V fragans*, *V plicatum*, and *V tinus*; this host is only known from Europe

of infected oaks and tanoaks often appear to die rapidly, with their foliage turning brown within several weeks. However, the time from initial infection to tree death can range from many months to several years. Controlled inoculation studies suggest that environmental factors and host genotype will affect the length of the overall process, and there are many observations of trees in which disease lesions are still small after a few years. Tanoak appears to be the most susceptible species. All size classes of tanoak, from seedlings to mature trees, may be infected and killed (Rizzo *et al.* 2002a; Figure 2). Tanoak infections occur on stems, branches, and leaves. In contrast, *P ramorum* does not appear to infect small stems, branches, or leaves on true oaks; larger trees therefore appear to be more prone to infection and mortality than seedlings and saplings.

A number of opportunistic organisms are commonly observed on oak and tanoak trees with advanced *P ramorum* infections, including ambrosia beetles (*Monarthrum scutellare* and *Monarthrum dentiger*), bark beetles (*Pseudopityophthorus pubipennis*), and *Hypoxylon thouarsianum*, a sapwood rotting fungus, (McPherson *et al.* 2002). These organisms may hasten the death of trees infected with *P ramorum*.

Researchers have found that only oaks in the sections Lobatae (red oaks) and Protobalanus (intermediate oaks) are infected in the field. Despite the wide host range of *P ramorum*, oaks in the section *Quercus* (the white oaks) still appear to be unaffected by *P ramorum* in the field (Rizzo *et al.* 2002a, 2002b). Blue oak (*Q douglasii*) and valley oak (*Q lobata*) – the major oak species of the inner coastal ranges, central valley, and Sierra Nevada foothills of California – fall into the white oak group, as does Oregon white oak (*Q garryana*) and Engelmann oak

(*Q engelmannii*) (Pavlik *et al.* 1991).

The progression of the disease and the extent of damage to most non-oak hosts are not well characterized yet. One of the few consistent features of *P ramorum* infections is that the pathogen is only recovered from aboveground plant parts such as leaves, branches, or stems. On several species in the rhododendron family (Ericaceae), *P ramorum* causes considerable leaf blight and branch dieback (Figure 3). One researcher has observed the death of madrone (*Arbutus menziesii*) saplings in less than 4 months in the field (P Maloney unpublished), and we suspect that the pathogen can kill mature madrone trees. Mature native rhododendrons have been killed in Oregon (Goheen *et al.* 2002a), and dieback has also been noted on redwood (*Sequoia sempervirens*) sprouts and Douglas-fir (*Pseudotsuga menziesii*) saplings (Davidson *et al.* 2002a; Maloney *et al.* 2002). However, on other hosts, such as California bay laurel (*Umbellularia californica*) and bigleaf maple (*Acer macrophyllum*), *P ramorum* appears to be primarily a leaf pathogen with very limited stem infection. Although they do not lead directly to tree death, these foliar infections may influence tree physiology through such effects as premature leaf abscission.

■ Locations of infestations

Phytophthora ramorum is known from two main geographic locations: the western US and Europe. (See the website www.suddenoakdeath.org for the most up-to-date range.) In the US, the disease is found on oaks from the Big Sur coastline in Monterey County, California to Curry County, Oregon, a distance of approximately 650 km (Figure 4). Most sites where *P ramorum* has been collected are within 30 km of the Pacific coastline or San Francisco Bay. The most extensive areas of tree mortality in California are in the canyons of Big Sur, the hills above the city of Santa Cruz, and in Marin County north of San Francisco, especially around Mt Tamalpais.

In 1993, the pathogen was recovered in dead and dying ornamental rhododendron and viburnum in gardens and nurseries in Germany and the Netherlands (Werres *et al.* 2001; Werres and Marwitz 1997). It has since been confirmed from nurseries and gardens in the UK, Spain, Poland, France, Sweden, and Belgium. Although surveys are ongoing, *P ramorum* has not been found to infect native plants in those countries (C Brasier pers comm). To date, no mortality of overstory trees in Europe has been associated with *P ramorum*.

■ Origin of the pathogen

Phytophthora ramorum's geographic origin is unknown. There are no reports of this species in the US or Europe before the mid 1990s. Its aggressiveness on tanoaks of all ages, and its limited geographic range in relation to the distribution of its hosts, suggest that it may have been introduced only recently to the US. Based on amplified

fragment length polymorphisms (AFLPs), *P ramorum* appears to have a clonal population structure, with one dominant genotype which has been found from Big Sur, California to Oregon (Garbelotto *et al.* 2002a; K Ivors and M Garbelotto unpublished). This population structure also supports the hypothesis of an exotic origin. The pathogen's origin in Europe is also unknown, but AFLP analyses suggest that the European and North American populations are distinct (Ivors and Garbelotto unpublished). In addition, all European isolates of *P ramorum* tested to date appear to have an A1 mating type, while all North American isolates have the A2 type (Werres and Zielke in press). These data suggest that the microorganism was not exchanged between the two continents. If it is indeed exotic in both locations, then we hypothesize that it was transported from a third, unknown location.

We cannot completely dismiss the hypothesis that *P ramorum* has been in California for many years, and that changes in the environment, such as climate warming, fire suppression, or other modifications in land use patterns, have led to an increase in its aggressiveness and the prevalence of a single genotype. Another hypothesis is that a native *Phytophthora* species underwent a change in host specificity, host preference, or virulence. *Phytophthora* hybrids occur in nature, and may show a marked change in host range (Brasier *et al.* 1999). In England and other parts of Europe, a new *Phytophthora* species that appears to be a hybrid between *P cambivora* (an oak pathogen) and *P fragariae*-like isolates (a strawberry pathogen) has killed thousands of alder trees (*Alnus* spp) (Brasier *et al.* 1999). In this instance, neither of the parent species of *Phytophthora* can infect alder. Many *Phytophthora* species occur in agricultural and ornamental settings in California, so a hybrid origin for *P ramorum* is possible.

■ Epidemics and forest diversity

Over its range in coastal California and Oregon, *P ramorum* is associated with several different forest types, found at elevations ranging from sea level to over 800 m (Rizzo *et al.* 2002a; Figures 5 and 6). Oaks and tanoak occur in closed-canopy, mixed evergreen forests that can be divided into those with and those without a substantial component of Douglas-fir (Shuford and Timossi 1989; Sawyer *et al.* 1988). Other major hardwood associates in these mixed evergreen forests include California bay laurel and madrone. In coast redwood forests, an understory of tanoak mixes with a number of shrub species, including rhododendron and evergreen huckleberry (*Vaccinium*



Figure 1. Canker at the base of a coast live oak caused by *P ramorum*. The pathogen can be found in the phloem, cambium, and part of the xylem. The pathogen is only found on aboveground stem tissue; it does not colonize the roots.

ovatum) (Shuford and Timossi 1989). Nearly all of the woody plant species in these forest types are known to be susceptible to *P ramorum*.

The evidence collected to date suggests that *P ramorum*



Figure 2. Tanoak saplings, some killed by *P ramorum* infection. Understory trees typically die as a result of multiple cankers on the main stem and branches.



Courtesy of P. Maloney

Figure 3. Stem and leaf lesions on madrone caused by *P. ramorum*.



Courtesy of M. Kelly

Figure 4. Current distribution of confirmed cases of *P. ramorum* in California and Oregon, based on data published in Rizzo et al. 2002a and ongoing surveys by the University of California, California Polytechnic State University, Oregon State University, the California Department of Food and Agriculture, the California Department of Forestry, and the USDA Forest Service.

epidemics in California forests may be driven by the presence of associated susceptible plant species, not the oaks themselves. In this sense, *P. ramorum* acts like a zoonotic disease, such as bubonic plague or Lyme disease, in which humans serve as dead-end hosts (Keeling and Gilligan 2000; Woolhouse et al. 2001). Foliar infections of non-oak hosts may play a key role in the epidemiology of *P. ramorum* by serving as a source of inoculum (Davidson et al. 2002c). The most likely dispersal propagules of *P. ramorum*, sporangia and chlamydospores, are readily produced on foliage, particularly bay laurel (Figure 7), but we have yet to find these propagules on infected oak bark (Davidson et al. 2002c). Even hosts with relatively small lesions may be important in the transmission biology of *P. ramorum*, because such lesions do not kill leaves, and may support the abundant production and release of spores (sporulation).

Forests with a diversity of plant hosts may therefore be more susceptible to invasion by *P. ramorum*. Two recent studies in California have found a clear association between the presence of bay laurel trees and *P. ramorum* infection on oak (Kelly and Meentemeyer 2002; Swiecki and Bernhardt 2002). Ongoing surveys have found that foliar host infection may precede the infection of oak and tanoak on individual sites. Because tanoak leaves may be infected and potentially support sporulation, epidemics in tanoak forests may be different from those in coast live oak forests, in not requiring the presence of additional hosts.

As with most plant diseases, environmental conditions will play a major role in the spread and intensification of sudden oak death. The current geographic range of the pathogen in California and Oregon includes a wide range of microclimates within a climate of predominantly winter rainfall. The mean annual rainfall of the different locations where *P. ramorum* has been collected ranges from 85 to 200 cm. Our recent field data indicate that warm rains result in very high levels of sporulation (Davidson et al. 2002c; J. Davidson and P. Maloney unpublished). Inoculation experiments on bay laurel leaves have indicated 9–12 hours of leaf wetness and temperatures of ~18–22°C are necessary to obtain significant infection (Garbelotto et al. 2003). We hypothesize that heavy rains during several years in the mid-1990s, including the El Niño periods of 1993 and 1998, resulted in widespread infection and increased mortality of oaks 2 to 3 years later.

Little is known about how variation in site conditions might predispose hosts to infection or influence the severity of a local epidemic. Many stand level variables, such as stand density, basal area, slope, and aspect, do not appear to be good predictors of the presence of the pathogen (Swiecki and Bernhardt 2002), but canopy exposure and edge effects do seem to be associated with increased infection (Swiecki and Bernhardt 2002; Kelly and Meentemeyer 2002), which may reflect an increased interception of inoculum in wind-driven rain. A significant positive correlation between stem water potential and diseased oaks suggests that sudden oak death is not

more common on water-stressed oaks, and that water stress may not be as important a factor as it is with many other plant diseases (Swiecki and Bernhardt 2002). Genetic resistance in host populations will also affect spread of the disease. Preliminary field and laboratory experiments have indicated that individual coast live oak and bay laurel plants display different levels of susceptibility to the disease (Rizzo *et al.* 2002a; Garbelotto *et al.* 2003). Further studies have been initiated to follow up on these observations in both species (R Dodd and Garbelotto unpublished; D Hüberli and M Garbelotto unpublished). If resistance is found to occur, determining its distribution among host populations may allow us to develop predictive epidemiological models.

■ Ecological implications for western forests

Many studies to evaluate the short- and long-term impacts of *P ramorum* on coastal forests are currently underway. To do this, we need to understand the ecology of these forests, including the expected rates and causes of mortality. Coastal range forests consist of a mosaic of vegetation types in various successional states (Sawyer *et al.* 1988; Shuford and Timossi 1989). Humans have been altering much of the forestland currently affected by the pathogen for many years already (Barbour *et al.* 1993; Pavlik *et al.* 1991; Sawyer *et al.* 1988; Keeley 2002). Fire suppression, human-ignited fires, introduced plant and animal species, and logging have shaped the forest structure and composition. The dynamics of many of these forests are poorly understood, even in the absence of *P ramorum*.

On small spatial scales (0.2–1 ha), infection rates of *P ramorum* range from 20–70% for tanoak and 4–30% for coast live oak (Swiecki and Bernhardt 2002; P Maloney unpublished; J Davidson unpublished). Mortality measured in 2000–01 reached up to 22% for tanoak and 15% for coast live oak (Swiecki and Bernhardt 2002). Unfortunately, mortality rates of oaks and tanoak under historic conditions have not been well characterized for these forests (Swiecki and Bernhardt 2002; Hunter 1997). Swiecki and Bernhardt (2002) have recently compared mortality associated with other diseases (mostly canker rot fungi) to those caused by *P ramorum*. They concluded that the pathogen has doubled the mortality of coast live oak on their plots and increased the amount of tanoak mortality at least fourfold over the past decade.

The distribution of *P ramorum*-caused mortality across the landscape has not been well quantified, but it is clearly patchy (Kelly and Meentemeyer 2002; Swiecki and Bernhardt 2002). Even within the areas with the greatest amount of tree mortality, there are large areas with susceptible host species that are apparently free of disease. Clustering of dead and diseased trees has been found on scales of 100–300 m (Kelly and Meentemeyer 2002; Swiecki and Bernhardt 2002). Extensive aerial and ground surveys are currently underway to gain a better

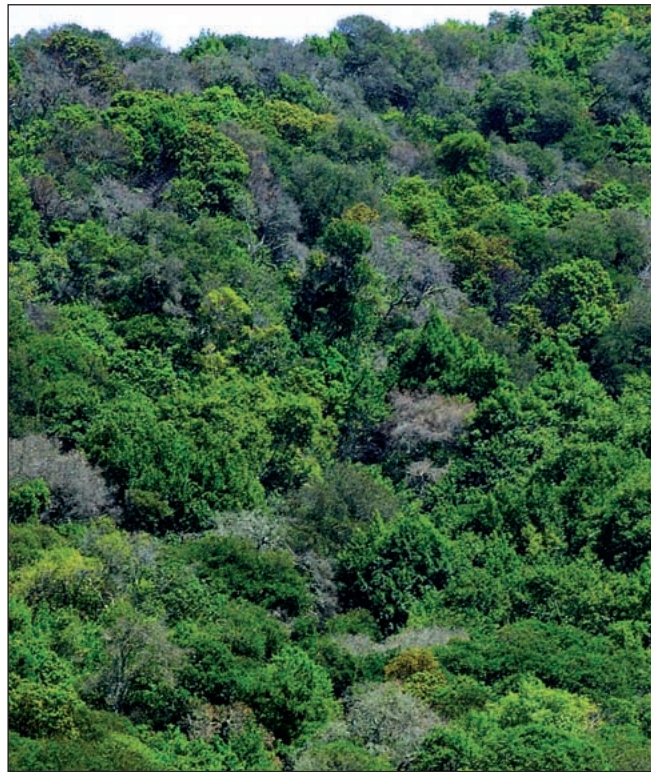


Figure 5. Mortality of overstory coast live oak in a typical mixed-evergreen forest in Marin County, CA.

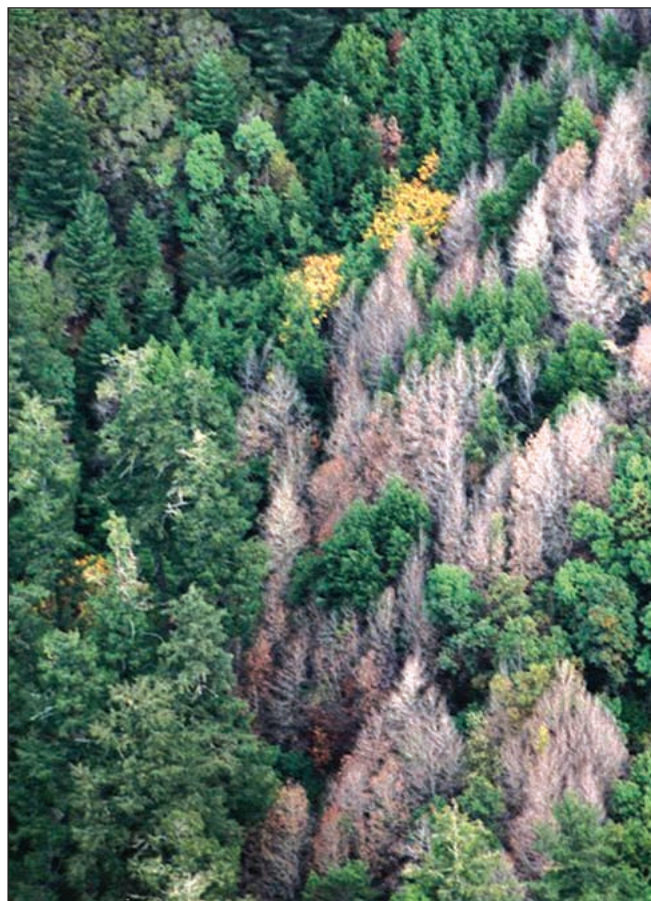


Figure 6. Mortality of overstory tanoak in a redwood-tanoak forest in Marin County, CA.

Courtesy of K Parker

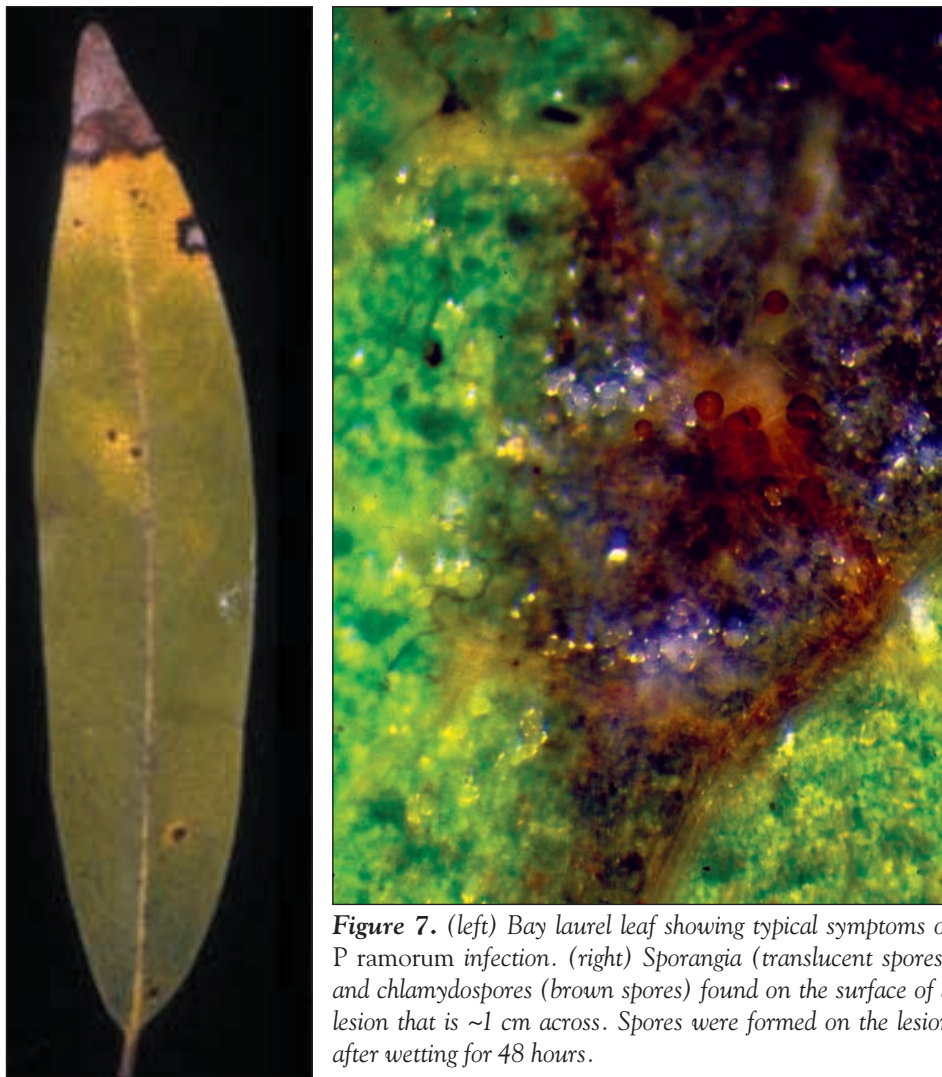


Figure 7. (left) Bay laurel leaf showing typical symptoms of *P. ramorum* infection. (right) Sporangia (translucent spores) and chlamydospores (brown spores) found on the surface of a lesion that is ~1 cm across. Spores were formed on the lesion after wetting for 48 hours.

understanding of mortality levels across the pathogen's complete geographic range (Kelly and McPherson 2001).

The broad host range of *P. ramorum*, the variability of symptoms between different hosts, and the pathogen's aerial dispersal suggest that it has the potential to cause a cascade of long-term landscape changes. Hosts include canopy trees, understory shrubs, and at least one herbaceous plant. Sublethal infections of non-oak hosts may allow *P. ramorum* to persist indefinitely in infested forests, alter the regeneration of infected species, and affect the success of future restoration efforts. Because of the range of susceptibility of co-existing plant species (for example, bay laurel and oak), pathogen-mediated competition may influence future successional patterns in these forests (Hudson and Greenman 1998). Selection pressure driven by resistance to *P. ramorum* may also be large enough to cause a directional shift in the genetic structure of oaks and tanoaks. Even in the case of the foliar hosts, the pathogen's subtle effects on plant physiology may result in a selection force.

Coastal oak and tanoak forests provide food and shelter for hundreds of vertebrate and invertebrate species (Pavlik *et al.* 1991), so the diseases caused by *P. ramorum* have the

potential to affect wildlife by changing or completely removing these resources. For example, reductions in insect populations associated with oaks and other hosts could affect insectivorous birds, by changing prey type or increasing foraging time and ultimately affecting nesting success. Reductions of acorn production by oaks and tanoaks could influence vertebrate populations that rely heavily on acorn mast as a food source. Research is currently underway by several research groups to test these possibilities (D Dahlsten and K Fischer pers comm).

Microbial community dynamics may also be affected. In many redwood forests, tanoaks are the dominant (or only) species with ectomycorrhizal fungi on their roots. If tanoak is removed from a site as a result of disease, does this eliminate or reduce these mycorrhizal species, or will mycorrhizal communities shift to other host species (eg ericaceous plants)? How will future plant generations be affected if significant mycorrhizal changes occur? As a leaf pathogen, *P. ramorum* interacts with other

foliar fungi species. Other foliar *Phytophthora* species are present, some of which appear to be native (Rizzo *et al.* 2002a; Davidson *et al.* 2002b). Does *P. ramorum* directly compete with these other species? If so, will it replace them in areas where they coexist? A similar instance of one pathogen causing the local extinction of a related but less aggressive pathogen occupying the same ecological niche has been documented for the two causal agents of Dutch elm disease (Brasier 2001). Given the apparently identical niches of these *Phytophthora* species, what is the potential for hybridization and horizontal gene transfer? Could this lead to the formation of unique, and potentially more virulent, genotypes?

Of great concern is the potential for the spread of *P. ramorum* to forests outside California and Oregon. Laboratory inoculations have found two eastern North American oaks, northern red oak (*Quercus rubra*) and pin oak (*Quercus palustris*), to be susceptible to *P. ramorum* infection (Rizzo *et al.* 2002b). One must be cautious when extrapolating results from seedling experiments to potential effects on mature trees. However, because lesion sizes in red oak and pin oak seedlings were much larger than in coast live oak seedlings (a species in which adults are very

susceptible), it is probable that mature trees of northern red oak and pin oak will also be susceptible to *P ramorum* infection. *Rhododendron catawbiense*, native to the eastern US, has been infected by *P ramorum* in nurseries and gardens in Germany (Werres *et al.* 2001). Because of our limited knowledge of the ecology of *P ramorum*, the ultimate impact of this pathogen in new regions is difficult to predict. Critical parameters to examine will include microclimate and the interactions between carrier hosts and those hosts that may die from the pathogen.

■ Responding to sudden oak death

California, Oregon, and the federal government have established task forces to bring to together various agencies in developing strategies and methodologies to manage this new disease. Many researchers at universities, NGOs, and government agencies in both the US and Europe are involved in learning more about *P ramorum* and its ecological ramifications.

Management of the pathogen is occurring at multiple scales, ranging from protecting individual trees to preventing the spread of the pathogen to other geographic locations. Various chemical compounds are being tested to protect high-value, individual oak trees at the urban–wildland interface (Garbelotto *et al.* 2002b). The combined use of chemical compounds and natural resistance in oaks may eventually have broader application on the landscape scale (Garbelotto *et al.* 2003; Hardy *et al.* 2001). However, landscape-level control of most forest diseases includes a silvicultural approach that incorporates knowledge of pathogen and host ecology. An understanding of pathogen spread, forest succession, and the role of fire in host ecology will be necessary to develop management protocols in California and Oregon. In addition, management goals will depend on forest type and geographic location. Researchers and government agencies in Oregon are attempting an eradication program (Goheen *et al.* 2002b), which was established after the disease was found to be concentrated in nine small patches (5–40 diseased trees each) scattered over 23 km² and located over 300 km from the nearest known infection sites in California. Even if the complete local eradication of *P ramorum* in Oregon is unsuccessful, the effort may slow the spread of the pathogen to new locations in the state.

In most areas of California, in contrast, the disease is too well established to be eliminated. In the forests around the San Francisco Bay, where timber production is not a major goal, management of the disease will be geared towards watershed management, fuel loads, wildlife, and aesthetics. On the northern coast of California, leading into Oregon, the disease occurs in areas that are currently managed for timber. In these forests, plans to combat *P ramorum* will need to be incorporated into already established forest management plans, for example through quarantine inspections, or washing vehicles in work areas to prevent the movement of infested soil.

Prevention of the spread of *P ramorum* to areas outside the known zone of infestation is considered a high priority. This requires coordination between government agencies, private industries, and the public. Because of the uncertainty concerning the origin, distribution, and host range of *P ramorum*, the US, Canada, the EU, Australia, and South Korea have implemented quarantines for plant material and/or soil from California and Oregon. In addition, the US has placed restrictions on rhododendron and viburnum imported from Europe, since the pathogen has clearly moved throughout western Europe on infected rhododendron and viburnum stock. Stopping the exchange of potentially infected plant material between Europe and the US is critical, because of the possibility of uniting the two mating types of *P ramorum*, leading to increased genetic variability. Such a situation occurred recently with *Phytophthora infestans*, cause of potato late blight, with devastating results (Fry and Goodwin 1997). The discovery that even small foliar lesions may be epidemiologically important for *P ramorum*, combined with the difficulties of culturing the pathogen, is proving to be a challenge for regulatory and monitoring agencies.

We must also keep this epidemic in context. Thousands of hectares of California oak woodland are already lost each year to urbanization and agriculture (Pavlik *et al.* 1991). Responses to sudden oak death cannot, and should not, divert attention from these considerable threats to the integrity of coastal forests.

■ Acknowledgements

We thank J Davidson, D Hüberli, K Ivors, and P Maloney for use of unpublished data, M Kelly for the map, and K Julin, P Maloney, and K Parker for use of their photographs. The input of Everett Hansen, Clive Braiser, Susan Frankel, and Keyt Fisher is particularly appreciated. This research has been supported by grants from the Gordon and Betty Moore Foundation, the USDA Forest Service Pacific Southwest Research Station, USDA Forest Service Forest Health Management, and the California Department of Forestry.

■ References

- Anagnostakis S. 1987. Chestnut blight: the classical problem of an introduced pathogen. *Mycologia* **79**: 23–37.
- Barbour M, Pavlik B, Drysdale F, and Lindstrom S. 1993. California's changing landscapes. Sacramento, CA: California Native Plant Society.
- Baskin Y. 2002. A plague of rats and rubber-vines: the growing threat of species invasions. Washington, DC: Island Press.
- Brasier CM. 2001. Rapid evolution of introduced plant pathogens via interspecific hybridization. *BioScience* **51**: 123–33.
- Brasier CM, Cooke DEL, and Duncan JM. 1999. Origin of a new *Phytophthora* pathogen through interspecific hybridization. *P Natl Acad Sci USA* **96**: 5978–83.
- Campbell F and Schlarbaum SE. 2002. Fading forests II: trading away North America's natural heritage. Knoxville, TN: Healing Stones Foundation.
- Davidson JM, Garbelotto M, Koike ST, and Rizzo DM. 2002a. First

- report of *Phytophthora ramorum* on Douglas-fir in California. *Plant Dis* **86**: 1274.
- Davidson JM, Hansen EM, Garbelotto M, et al. 2002b. Another canker-causing *Phytophthora* on oaks and tanoak in forests of California and Oregon. *Phytopathology* **92**: S18.
- Davidson JM, Rizzo DM, Garbelotto M, et al. 2002c. *Phytophthora ramorum* and sudden oak death in California: II. Pathogen transmission and survival. In: Standiford R and McCreary D (Eds). 5th Symposium on California oak woodlands. USDA Forest Service, Gen. Tech. PSW-GTR-184. p 741–49.
- Erwin DC and Ribeiro OK. 1996. *Phytophthora* diseases worldwide. St Paul, MN: APS Press, American Phytopathological Society.
- Fry WE and Goodwin SB. 1997. Resurgence of the Irish potato famine fungus. *BioScience* **47**: 363–71.
- Garbelotto M, Davidson JM, Ivors K, et al. 2003. Non-oak native plants are the main hosts for the sudden oak death pathogen in California. *Calif Agr* **57**: 18–23.
- Garbelotto M, Rizzo DM, Hayden K, et al. 2002a. *Phytophthora ramorum* and sudden oak death in California: III. Pathogen genetics. In: Standiford R and McCreary D (Eds). 5th Symposium on California oak woodlands. USDA Forest Service, Gen Tech PSW-GTR-184. p 765–74.
- Garbelotto M, Rizzo DM, and Marais L. 2002b. *Phytophthora ramorum* and sudden oak death in California: IV. Chemical control. In: Standiford R and McCreary D (Eds). 5th Symposium on California oak woodlands. USDA Forest Service, Gen Tech PSW-GTR-184. p 811–18.
- Garbelotto M, Svihra P, and Rizzo DM. 2001. Sudden oak death syndrome fells three oak species. *Calif Agr* **55**: 9–19.
- Goheen EM, Hansen EM, Kanaskie A, et al. 2002a. Sudden oak death caused by *Phytophthora ramorum* in Oregon. *Plant Dis* **86**: 441.
- Goheen EM, Hansen EM, Kanaskie A, et al. 2002b. Eradication of sudden oak death in Oregon. *Phytopathology* **92**: S30.
- Hansen EM and Sutton W. 2002. Log inoculations to assess tree susceptibility to sudden oak death. *Phytopathology* **92**: S33.
- Hardy GE, Barrett S, and Shearer BL. 2001. The future of phosphite as a fungicide to control the soil-borne plant pathogen *Phytophthora cinnamomi* in natural ecosystems. *Australas Plant Path* **30**: 133–39.
- Hudson P and Greenman J. 1998. Competition mediated by parasites: biological and theoretical progress. *Trends Ecol Evol* **13**: 387–90.
- Hunter JC. 1997. Fourteen years of change in two old-growth *Pseudotsuga–Lithocarpus* forests in northern California. *J Torrey Bot Soc* **124**: 273–79.
- Keeley JE. 2002. Native American impacts on fire regimes of the California coastal ranges. *J Biogeogr* **29**: 303–20.
- Keeling MJ and Gilligan CA. 2000. Metapopulation dynamics of bubonic plague. *Nature* **407**: 903–05.
- Kelly NM and McPherson BA. 2001. Multi-scale approaches taken to SOD monitoring. *Calif Agr* **55**: 15–16.
- Kelly NM and Meentemeyer R. 2002. Landscape dynamics of the spread of sudden oak death. *Photogram Eng Rem S* **68**: 1001–09.
- Linderman RG, Parke JL, and Hansen EM. 2002. Relative virulence of *Phytophthora* species, including the sudden oak death pathogen *P. ramorum*, on several ornamental species. *Phytopathology* **92**: S47.
- Maloney PE, Rizzo DM, Koike ST, et al. 2002. First report of *Phytophthora ramorum* on coast redwood in California. *Plant Dis* **86**: 1274.
- McPherson BA, Wood DL, Storer AJ, et al. 2002. Sudden oak death: disease trends in Marin County plots after one year. In: Standiford R and McCreary D (Eds). 5th Symposium on California oak woodlands. USDA Forest Service, Gen Tech PSW-GTR-184. p 751–64.
- Parke JL, Linderman RG, and Hansen EM. 2002. Susceptibility of *Vaccinium* to *Phytophthora ramorum*, cause of the sudden oak death pathogen. *Phytopathology* **92**: S63.
- Pavlik BM, Muick PC, Johnson SG, and Popper M. 1991. Oaks of California. Los Olivos, CA: Cachuma Press.
- Rizzo DM, Garbelotto M, Davidson JM, et al. 2002a. *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp and *Lithocarpus densiflorus* in California. *Plant Dis* **86**: 205–14.
- Rizzo DM, Garbelotto M, Davidson JM, et al. 2002b. *Phytophthora ramorum* and sudden oak death in California: I. Host relationships. In: Standiford R and McCreary D (Eds). 5th Symposium on California oak woodlands. USDA Forest Service, Gen Tech PSW-GTR-184. p 733–40.
- Sawyer JO, Thornburgh DA, and Griffin JR. 1988. Mixed evergreen forest. In: Barbour MG and Major J (Eds). Terrestrial vegetation of California. Sacramento, CA: California Native Plant Society. p 359–81.
- Shuford WD and Timossi IC. 1989. Plant communities of Marin County. Sacramento, CA: California Native Plant Society.
- Swiecki TJ and Bernhardt E. 2002. Evaluation of stem water potential and other tree and stand variables as risk factors for *Phytophthora ramorum* canker development in coast live oak. In: Standiford R and McCreary D (Eds). 5th Symposium on California Oak Woodlands. USDA Forest Service, Gen Tech PSW-GTR-184. p 787–98.
- Tooley PW and Englander L. 2002. Infectivity of *Phytophthora ramorum* on selected Ericaceous host species. *Phytopathology* **92**: S81.
- Werres S and Marwitz R. 1997. Triebsterben an rhododendron: unbekannte *Phytophthora*. *Deutscher Gartenbau* **21**: 1166–68.
- Werres S, Marwitz R, Man In'T Veld WA, et al. 2001. *Phytophthora ramorum* sp nov: a new pathogen on *Rhododendron* and *Viburnum*. *Mycol Res* **105**: 1155–64.
- Werres S and Zielke B. 2003. First studies on the pairing of *Phytophthora ramorum*. *J Plant Dis Protect*. In press.
- Weste G and Marks GC. 1987. The biology of *Phytophthora cinnamomi* in Australasian forests. *Annu Rev Phytopathol* **25**: 207–29.
- Woolhouse MEJ, Taylor LH, and Haydon DT. 2001. Population biology of multihost pathogens. *Science* **292**: 1109–12.