PHYTOPHTHORA RAMORUM: Integrative Research and Management of an Emerging Pathogen in California and Oregon Forests

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■ **Abstract** *Phytophthoraramorum*, causal agent of sudden oak death, is an emerging plant pathogen first observed in North America associated with mortality of tanoak (*Lithocarpus densiflorus*) and coast live oak (*Quercus agrifolia*) in coastal forests of California during the mid-1990s. The pathogen is now known to occur North America and Europe and have a host range of over 40 plant genera. Sudden oak death has become an example of unintended linkages between the horticultural industry and potential impacts on forest ecosystems. This paper examines the biology and ecology of *P. ramorum* in California and Oregon forests as well discussing research on the pathogen in a broader management context.

INTRODUCTION

Sudden oak death is the latest in a long line of exotic plant diseases that threaten the integrity and biodiversity of native forests in North America. First observed causing bleeding stem cankers and mortality of tanoak (*Lithocarpus densiflorus*) and coast live oak (*Quercus agrifolia*) in coastal forests in California during the mid-1990s, this emerging disease has mobilized practitioners, researchers, and regulatory agencies (24, 34, 83, 92). Since the disease was linked to *Phytophthora ramorum* in 2000, the scope of sudden oak death research and management has changed dramatically (83, 104). The pathogen has been found in both North

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TABLE 1 Plant genera known to have species associated
with *Phytophthora ramorum* in forests or nurseries[1]

Abies	*Nothofagus*[§]
Acer[*]	Pieris
Aesculus[*§]	*Pittosporum*
Arbutus[*]	*Pseudotsuga*[*]
Arctostaphylos[*]	*Pyracantha*
Calluna	*Quercus*[*§]
Camellia	*Rhamnus*[*]
Castanea	*Rhododendron*[*]
Clintonia	*Rosa*[*]
Corylus[*]	*Rubus*
Drimys	*Salix*
Dryopteris[*]	*Sequoia*[*]
Fagus[§]	*Smilacina*[*]
Fraxinus[§]	*Syringa*
Hamamelis	*Taxus*[*]
Heteromeles[*]	*Toxicodendron*[*]
Kalmia	*Trientalis*[*]
Laurus	*Umbellularia*[*]
Leucothoe	*Vaccinium*[*]
Lithocarpus[*#]	*Viburnum*
Lonicera[*]	

[1]Symbols: *, found in California or Oregon forests; #, canker host in North
America; §, canker host in Europe. Canker hosts refer to species in which in-
fection by *P. ramorum* results in bleeding cankers on the main trunk of the tree;
this is the symptom usually associated with sudden oak death. On all other hosts
P. ramorum causes foliar or twig blight [based on (2, 10, 18, 63)].

America and Europe and its worldwide host range expanded to over 40 plant gen-
era (Table 1). Although extensive mortality associated with *P. ramorum* appears to
be restricted to certain members of the Fagaceae, in most circumstances foliar and
twig infection of other hosts appears to be critical for natural and human-mediated
spread of the pathogen (18, 19, 30). Recent findings of *P. ramorum* in North Amer-
ican nurseries and in European trees have increased anxiety about potential threats
to forest resources (2, 10, 11). A January 2005 symposium on sudden oak death
was attended by 350 people from 11 countries, and over 120 papers were presented
on wide-ranging topics concerning the biology and management of *P. ramorum* in
nurseries and forests (P.J. Shea, USDA Forest Service, personal communication).

The general biology and ecology of *P. ramorum* have been previously reviewed
(30, 82). Other reviews have covered the diagnosis of *P. ramorum*-associated

diseases on native and ornamental hosts (18, 75, 100). We focus this review on research and management of *P. ramorum* in the forests of California and Oregon, but it is not intended to be a comprehensive guide to management of *P. ramorum* in forest landscapes. Best management practices for prevention of spread and restoration are still being formulated (14, 93). Instead, we aim to put forest management in context with current and future research directions.

LEVELS AND COMPLEXITY OF MANAGEMENT

Although research and management on *P. ramorum* has proceeded quickly at multiple spatial and temporal scales (34, 55), our knowledge of the diseases that the pathogen causes is inevitably limited given its recent discovery. Management needs have been focused at three nonexclusive levels: the individual tree, the landscape (or forest stand), and the regional to international scale. To be effective, these management levels must constantly provide feedback to each other. Disease prevention and mitigation at the individual tree level have focused on chemical control or other programs designed to maintain health of trees and to lower inoculum pressure (33). These programs have been aimed mostly at homeowners and arborists who are attempting disease management at the urban-wildland interface where individual trees are important components of the landscape and may have historic value. Registration of phosphite treatments for *P. ramorum* on oaks was obtained (13) after a series of successful trunk injection treatments performed as described in the Australian literature (40, 45). Treatments were most effective if preventively administered, but also had some therapeutic value if administered soon after infection (27, 33). In addition to trunk injections, a mixture of the phosphite and an organosilicate surfactant applied directly on the outside of the trunk was found to slowly penetrate the bark, eventually reaching the cambium without the need for complex, wound-causing injections (27).

The broadest scale for management, regional to international, is driven by regulations and management practices designed to prevent further spread of *P. ramorum*. The enactment of quarantine regulations has affected the ornamental nursery industries in both North America and Europe. Despite regulations in place since 2001, large numbers of infected camellias (*Camellia* spp.) and rhododendrons (*Rhododendron* spp.) were shipped across the United States from California and Oregon nurseries during 2004, increasing fears of endangering the oak resource in the eastern United States (23). These events led to a broadening of national and international quarantines designed to prevent movement of the pathogen and to a renewed impetus to manage Phytophthora diseases in nursery settings. In all the attempts to impose quarantine restrictions on fungal pathogens, *P. ramorum* probably has the widest host range . In addition, *P. ramorum* is one of the most dramatic examples of unintended linkages between the horticultural industry and the health of forest ecosystems. The emergence of this pathogen has brought to the forefront the complexities of balancing ecological and economic well-being. This

complexity has often made it difficult to incorporate new scientific information into policy (23, 24).

The most difficult scale, however, for designing management strategies is the forest landscape. Disease management in the forest requires an understanding of the pathogen's biology and ecology as well as the ecology of the invaded forest. Landscape-scale management has been attempted for several exotic forest pathogens. In the early 1900s, large-scale clear-cutting was attempted to remove potential host material, but was ultimately unsuccessful in stopping the spread of chestnut blight (*Cryphonectria parasitica*) across the northeastern United States (1). Removal of alternate host material was tried on a very large scale for control of white pine blister rust (*Cronartium ribicola*) in both eastern and western North America (58). Various sanitation programs have been implemented against Dutch elm disease (*Ophiostoma ulmi*, *O. novo-ulmi*) in North America and Europe, *Phytophthora cinnamomi* in Australia, and *P. lateralis* in Oregon and California (42, 46, 98). Finally, fungicides were applied widely in Australia to reduce the spread on *P. cinnamomi* (46). The effectiveness of these programs has been variable; important successes were balanced by continuing tree mortality in many areas.

Decision making requires estimation of the potential impacts of the disease (i.e., setting priorities for management targets) and the ability to fit the disease into the context of other management goals within the broader landscape. Coastal forests and *P. ramorum* occur in an incredibly varied setting. The San Francisco Bay area is a major conurbation with over 6 million people in several major cities and extensive suburban areas. Intermixed within this area are many local and county parks, open space districts, conservation easements, State Parks, National Parks (e.g., Muir Woods National Monument, Point Reyes National Seashore) as well as undeveloped land under private ownership. Major state and federal highways intersect much of the area. Visitation rates are very high at many of the parks in the area. As one moves out of the Bay Area, the forests become intermixed with agriculture, particularly vineyards, and scattered urbanized areas. Urban areas become much less prevalent and overall population density decreases as one moves north through California into Oregon. In northern California, there are numerous state parks and Redwood National Park as well as several large Native American reservations. Timber production is much more common in northern California and southern Oregon and large forest areas are under management of private timber companies. These forests include naturally developed forests as well as areas converted to plantations (74).

Each of these ownership groups brings different management goals and desired outcomes to the table. Before decisions can be made on *P. ramorum* management, whether it be direct treatment or restoration, a key question to be answered is: How were, or how should, these forests be managed in the absence of *P. ramorum*? In forests around the Bay Area where timber production is not a major goal, management of the forests has been geared toward watershed management, fuel loads, wildlife, and aesthetics. There are also problems with exotic plant and animal species. Similar goals will be in place in public and private reserves outside of

the Bay Area (99). Private companies have goals for production of timber, primarily redwood and Douglas-fir. Timber management plans must also consider and incorporate other considerations such as riparian preservation and endangered species. Managers must formulate the management directions that are consistent with goals and/or regulations of regional land management (93). Ultimately, landscape management strategies for *P. ramorum* must incorporate prevention, treatment, restoration, and conservation into an overall program.

THE INVADED FORESTS OF CALIFORNIA AND OREGON

Coastal forests consist of a mosaic of vegetation types in various successional states (4, 20, 25, 87, 89, 103). *P. ramorum* is currently associated with a range of forest types that include oak (primarily coast live oak) or tanoak as a main structural component (83). There are a number of different forest types classified as mixed-evergreen; these forests can generally be divided into those with and without a significant component of Douglas-fir (*Pseudotsuga menziesii*) (4, 25, 87, 89). Many of the associated plant species, including coast live oak and tanoak, are evergreen broad-leaved sclerophyllous trees and shrubs (87, 89). Major hardwood associates of oaks and tanoak in mixed-evergreen forests include California bay laurel (*Umbellularia californica*), Pacific madrone (*Arbutus menziesii*), and bigleaf maple (Acer macrophyllum). Many of these same hardwood tree species also occur in coast redwood forests and include additional shrub species such as rhododendron and evergreen huckleberry (89).

This patchwork of plant communities is a result of edaphic and microclimate conditions as well as a legacy of past landscape disturbances. Physical disturbances, such as fire, flooding, and wind, can be locally important in the ecological dynamics of coastal forests (86). For example, in northern mixed-evergreen forests dominated by Douglas-fir, some have suggested a periodic crown fire regime that results in stand replacement with tanoak followed by Douglas-fir domination (4, 50). Tanoak, in particular, sprouts prolifically following fire or logging, and is eventually overtopped and or replaced by Douglas-fir or coast redwood. But tanoak can remain locally dominant through the effects of periodic fire. Coast live oak also sprouts from roots, and the structure of many forest stands is the past result of sprouting following tree felling (77). Over long periods of time, plant communities will shift between grasslands, shrubland, and woodland, depending on climate trends (e.g., wet El Niño years, periodic droughts) and associated fire frequencies (4). Humans (both Native Americans and Euroamericans) have altered much of the forest currently affected by *P. ramorum* (5, 54, 77, 87). Fire suppression, human-ignited fires, introduced plant and animal species, and logging have influenced forest structure and composition as well as the makeup of surrounding nonforest plant communities. Heterogeneity of plant communities in coastal areas can occur on very fine spatial scales; in many places, plant communities grade from conifer forest (e.g., coast redwood, Douglas-fir) to hardwood forest to grassland

and chaparral over a distance of only a few hundred meters (Figure 1, see color insert).

RECENT ADVANCES IN PATHOGEN CHARACTERIZATION

The original diagnosis of *P. ramorum* has generated considerable research effort to understand its population biology and evolutionary relationships within the genus *Phytophthora* (52, 60, 65, 83, 104). This encompasses research at multiple spatial and temporal scales that has been applied to all levels of management including disease diagnosis, host-pathogen interactions, epidemiology, and international policy. Of particular interest have been comparisons of the North American and European populations of *P. ramorum* (9, 52, 61). Research has proceeded quickly; the *P. ramorum* genome was sequenced in 2004, only four years after the discovery of the pathogen in North America (102).

The geographic origin of *P. ramorum* is unknown. There are no reports of this species in the United States or Europe prior to the mid-1990s. Extensive genetic evidence supports the hypothesis that this pathogen is exotic to both Europe and North America. Based on three DNA regions (*ITS*, *cox I*, *cox II*, and *nadh* 5), all European and North American isolates form a monophyletic group and share nearly identical sequences (52, 60, 65, 83, 104). However, AFLP (amplified fragment-length polymorphisms) banding patterns and microsatellites indicate that European and U.S. isolates of *P. ramorum* form two distinct lineages within this monophyletic group (32, 44, 52, 78, 104). In addition, all European isolates to date (with a single exception) have been of the A1 mating type, whereas North American forest isolates have been of A2 (12, 44, 104, 105).

AFLP profiles of *P. ramorum* indicate that a single clonal lineage dominates the North American population accompanied by rare clones isolated only once (52). In contrast, the genetic structure of the European lineage consists of an array of mainly unique, closely related AFLP types clustered in a few subclades (52). The uniqueness of many closely related genotypes, combined with our current understanding of the segregation of the mating types on two continents, suggest that groups of closely related clones do not represent a population of sexually generated individuals. Instead, unique genotypes are likely to have been generated through mitotic recombination or mutation from the original genotype. Recent reports indicate all U.S. AFLP types may be collapsed in a single genotype based on microsatellite analyses (31, 51, 78). Garbelotto et al. (31) reported that 50 individual AFLP types from Europe could be collapsed into 5 genotypes based on microsatellite analyses. New work with more complex microsatellite repeats, however, suggests the possibility of fine-scale population structure within the North American population (79). It will be interesting to see the extent to which the microsatellite genetic structure parallels the fine-scale diversity first suggested by AFLPs.

Variation in virulence has been found among U.S. isolates of *P. ramorum*. Inoculation studies on coast live oak seedlings and bay laurel leaves have resulted

in 8- and 40-fold variations, respectively, between the most and least virulent host-pathogen combinations when both variation in the host and in the pathogen are taken into account (49). Significant variation in virulence was found between and within AFLP types, indicating a high level of phenotypic plasticity. Virulence level of *P. ramorum* isolates on bay laurel was highly correlated with that on oak trees, indicating that individual isolates are likely to be aggressive, or not, on a broad range of hosts rather than host specific (49). When A1 isolates from Washington nurseries were inoculated on coast live oak seedlings, their virulence was comparable to the virulence of A2 isolates from nurseries and from the wild. Interestingly, the most aggressive isolates on coast live oak were always A2 isolates from the wild (D. Hüberli & M. Garbelotto, unpublished data). Other phenotypic differences between the European and North American populations, such as culture morphology and growth rate, have also been noted (9).

Resistance of *P. ramorum* to metalaxyl has been recently reported in Europe (S. Werres, personal communication). This may be the result of exposure to chemical treatments in nursery settings, where until recently *P. ramorum* was mostly found in Europe. Resistance to chemicals registered to control *Phytophthora* species has not been detected in the United States. However, in laboratory tests significant differences among isolates have been found at recommended rates for copper hydroxide, copper sulfate, phosphonates, maneb, and metalaxyl (29).

Recent findings of A1 isolates in North American nurseries (44), and their clear homology with the European genotype, based on AFLP and microsatellite analyses (44, 52, 78), raise the probability that plant material is still being exchanged between the two continents. Because the two lineages represent significant phenotypic and genotypic differences, further introductions should be avoided in order to keep the diversity of the pathogen genetic pool at a minimum. The occurrence of A1 and A2 isolates within the same nursery on several occasions in Oregon, Washington, and British Columbia is worrisome. However, in vitro mating between the two *P. ramorum* lineages is possible, but crosses result in a high proportion of aborted oospores (9, 12, 31).

PHYTOPHTHORA RAMORUM IN FORESTS

Establishment and spread of *P. ramorum* in forests will be dependent on environmental conditions (physical and biological), host resistance, and pathogen attributes. Remote sensing and plot-based research have begun to discern patterns of establishment and spread over large geographic areas, while finer spatial- and temporal-scale studies on host susceptibility and pathogen biology in the field and laboratory provide mechanisms for coarser-scale patterns (48).

Spatial Distribution

The occurrence of *P. ramorum* in California and Oregon on the landscape is patchy at many spatial scales (38, 56, 95, 96; P.E. Maloney, S.C. Lynch, S. Kane, C. Jensen

& D.M. Rizzo, manuscript submitted). At the largest scale, *P. ramorum* is found
discontinuously in coastal forests from the Big Sur area (Monterey County) in
central California to Curry County, Oregon, a distance of approximately 750 km
(Figure 2, see color insert). Most sites where *P. ramorum* has been collected are
within 30 km of the Pacific Coast or San Francisco Bay along an approximately
450-km stretch. But even within geographic areas with the greatest amount of
P. ramorum-associated mortality, many areas with susceptible host species are
apparently free of disease. Often forest stands with very high incidence of disease
may be spatially juxtaposed with forest stands with little or no disease even though
there are apparently few environmental differences between the sites (P.E. Maloney
et al., manuscript submitted). This suggests that the absence of *P. ramorum* in many
places is historical rather than strictly environmental or biological.

Regardless of forest type, the key correlative association that predicts the pres-
ence of *P. ramorum* has consistently been the presence of bay laurel (56, 72a,
95, 96; P. E. Maloney et al., manuscript submitted). Bay laurel has been shown
to play a key role in the epidemiology of *P. ramorum* in forests (16, 17, 19; see
below). This is especially true in mixed-evergreen forests dominated by coast
live oak (19). Ongoing surveys have also found that foliar host infection by *P.
ramorum* precedes infection of oak and tanoak on individual forest sites (72a; P.E.
Maloney unpublished data; A. Wickland, unpublished data). Because tanoak twigs
and leaves support sporulation, establishment and spread of *P. ramorum* in tanoak
forests does not require the presence of additional foliar hosts (P.E. Maloney et al.,
manuscript submitted). In Oregon, tanoak forests have relatively small numbers
of bay laurel present, and it is seldom found infected (38). Initial infection and
inoculum increase in Oregon tanoak stands appears to occur on leaves and twigs
in the upper crowns of tanoak trees (E.M. Hansen, unpublished data).

Many stand-level variables (e.g., stand density, basal area, slope, aspect) do not
appear to be good predictors for the presence of the pathogen in all situations, but
canopy exposure and edge effects seem to be associated with increased infection
(56, 72a, 95, 96). The data of Swiecki & Bernhardt (95, 96) indicate that trees
on sites that are relatively dry due to soil and topographic factors have a lower
risk of infection by *P. ramorum*. A significant positive correlation has been found
between stem water potential and diseased oaks, suggesting that sudden oak death
is not more common on water-stressed oaks and that water stress may not be as
important a factor as with many other plant diseases (95, 96).

Host Resistance

Individuals of coast live oak and bay laurel have been found to display different
levels of susceptibility to the disease (21, 30, 84; D. Hüberli & M. Garbelotto, un-
published data). Several field studies have commonly noted the slow development
or callusing over of cankers on coast live oak trees (96). Inoculation studies with
P. ramorum have confirmed significant variation among individuals of coast live
oak (21). The spatial structure of variation in native host populations may be an

important influence on the establishment and spread of *P. ramorum*. The variation in susceptibility of coast live oak occurs at all spatial scales with as much variation occurring within individual oak populations as between distantly separated populations (21). The pressing question is whether these less susceptible plant individuals are truly resistant, capable of surviving when conditions are highly favorable to the pathogen.

With bay laurel, differences in susceptibility have been found both within and between populations (49). Thus, the spatial distribution of resistance appears to be different between bay laurel and coast live oak. A better understanding of such distribution patterns among host populations may allow for the development of predictive epidemiological models.

Sporulation and Survival Patterns

Foliar infections of nonoak hosts play a key role in the epidemiology of *P. ramorum* in forests by serving as a source of inoculum (16, 17, 19). Sporangia are readily produced on foliage of a number of hosts. In particular, bay laurel leaves have been shown to produce large numbers of sporangia following rainfall (16, 17, 19). There is no evidence that the pathogen sporulates directly from bleeding cankers on the main stem of oak or tanoak (19; J.M. Davidson unpublished data). Therefore, in the field, oak (*Quercus* spp.) appears to be an epidemiological dead-end because it does not support foliage or twig infection (19, 30, 83). In contrast, the foliage and small twigs of tanoak are infected by *P. ramorum* and sporangia are produced on these substrates (J.M. Davidson, unpublished data).

California and Oregon have a Mediterranean climate with distinct wet and dry seasons. The current geographic range of *P. ramorum* includes a wide range of microclimates within a climate of predominantly winter-spring rainfall. Despite the frequent occurrence of summer fogs, sporulation and subsequent plant infection appear to be primarily restricted to the rainy season (19; J.M. Davidson, unpublished data). Although moisture levels and temperature clearly influence sporulation and infection by *P. ramorum* (30), their relationships to patterns observed in the field is complex (19). For example, sporangial production from foliar hosts in redwood forests commences soon after the start of the rainy season (P.E. Maloney, unpublished data). In mixed-evergreen forests, however, production of sporangia shows a distinct lag period early in the season despite favorable moisture and temperature conditions (19). This lag may be due to a decrease in the inoculum reservoir during the hot, dry summer months through abscission of infected leaves from the canopy and/or death of *P. ramorum* in most of the remaining attached, infected leaves (19; J. Davidson & E. Fichtner, unpublished data). The onset of detectable inoculum production in mixed-evergreen forest may depend on breaking dormancy for isolates that oversummered in bay laurel leaves or soil, followed by a subsequent build-up of new infection on leaves (19, 22).

Yearly variation in rainfall patterns also influences sporulation patterns of *P. ramorum* (19). Late rains in spring 2003 resulted in a 20-fold increase in

sporangial production, as collected in rainwater, over the previous two springs in a mixed-evergreen forest (19). Such data point to the potential importance of longer-term climatic events, such as El Niño, in influencing the establishment and spread of *P. ramorum*.

A key question is the scale at which treated sites are at most risk of reinvasion from untreated forests. Recent data suggest consistent aerial movement of *P. ramorum* propagules (sporangia, zoospores) in wind-driven rain 10 m from stand edges (19). Propagules have also been found in rainwater collected up to 25 m in the crowns of emergent redwood trees (P.E. Maloney, unpublished data). Distances up to 8 m have been detected between infected coast live oak and the nearest symptomatic bay laurel tree, the presumed source of inoculum (96). Several field studies have found that more open stands tend to be associated with infected oaks than very dense stands (95, 96). Dispersal may be somewhat more limited in dense forest where wind speed is reduced (P.E. Maloney, unpublished data). In Oregon, twig and foliar infections are commonly present in the upper canopies of still living tanoaks with bole cankers. Understory infection is found primarily on tanoak, rhododendron, and evergreen huckleberry, but only on plants growing in immediate proximity to overstory tanoak. There is no evidence of lateral spread within the understory (E.M. Hansen, unpublished data). Lateral dispersal appears to be above the canopy, from inoculum produced on tanoak leaf and twig infections, with local intensification from inoculum dripping, splashing, and running down stems from the initial high infections.

The Oregon portion of the *P. ramorum* epidemic demonstrates the spread of the pathogen across a recently invaded landscape. The epidemic appears to have started in rural residential areas on the outskirts of the coastal town of Brookings. The presence of *P. ramorum* was first confirmed in 2001, in 9 scattered locations within a 21-km^2 area. Examination of earlier aerial photographs revealed the first dead tree on these sites in 1998. The pattern of subsequent new infections across the landscape conforms to the classical dispersal gradient of a contagious disease spreading by aerial transport from distinct sources of inoculum. About half of the new infections each year occur within 100 m of trees killed the previous year. There is a long tail to the distribution of newly killed trees, suggesting occasional long-distance (up to 3 km) dispersal in storm winds, but most new infection occurs close to previously infected trees. There is no evidence to suggest pathogen transport along roads or in streams, or that the disease has been present for a long time and is widely spread in these forests (E.M. Hansen, unpublished data).

DETERMINATION OF IMPACTS

To set management priorities in coastal forests, we must understand the current and potential effects of *P. ramorum* on forest ecology. 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. However, any impacts caused by *P. ramorum* must be put into context with other agents that may influence the dynamics of these forests (82). Because many tree species that may serve as hosts for *P. ramorum* are not commercially important, the effects of biotic (pathogens, insects) and abiotic agents on individual tree species have not been extensively characterized (95). Background levels and rates of mortality are generally not known in many coastal forest types, but as in any forest, can be locally high because of many different biotic and abiotic factors.

Numerous research plots have been established in coastal forests since 2000 to understand disease progression at various spatial scales, ranging from individual trees to the landscape (e.g., 68, 72a, 95, 96; Maloney et al. manuscript submitted). Swiecki & Bernhardt (95, 96) have recently compared mortality associated with other diseases (mostly canker rot fungi) to that 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. Maloney et al. (manuscript submitted) found mortality levels of tanoak were significantly lower in coast redwood forests in which *P. ramorum* had not yet invaded. Cumulative mortality of tanoak was much higher in *P. ramorum*–associated plots in Santa Cruz (9.6%) and Monterey (15%) counties than in plots with no *P. ramorum* in those locations (1.4% and 0.57% for Santa Cruz and Monterey, respectively). Hunter (1997) reported the annual mortality rate of tanoak to be ~2.1% in a Douglas-fir–tanoak forest in northern California compared with the 6.0% year^{-1} documented in *P. ramorum*–infested forests (Maloney et al., manuscript submitted).

Once established in a stand, disease increase can be explosive (Figure 3, see color insert). For example, early disease development in 5 tanoak stands in Oregon was reconstructed from resource aerial photography. In 2001, *P. ramorum* was first isolated from dead trees at each of these sites. The first mortality at four of these locations was visible on year 2000 photographs. One dead tree was visible on the 1998 photographs at one of the sites. By August 2001, 110 trees had been killed by *P. ramorum* at this site. Aerial surveys in the Los Padre National Forest at the southern end of the current range of *P. ramorum* in California, have estimated 119,000 dead overstory tanoaks across approximately 3200 ha (J. Mai, USDA Forest Service, personal communication).

Cascading ecological effects due to invasion by exotic plant pathogens have been documented in a number of forests ecosystems (81). In California, *P. ramorum* has already begun to change forest stand structure through mortality of oaks and tanoak. Sublethal infections of nonoak 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 coexisting plant species (e.g., bay laurel versus oak), *P. ramorum*–mediated competition may influence future successional patterns in these forests (82). Additional ecosystem effects may come through the influence of tree mortality on other plant species, wildlife, and microorganisms (7, 82). Interactions between

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P. ramorum and native plant pathogenic fungi and insects may also lead to changes in ecosystem dynamics. For example, synergistic interactions between *P. ramorum* and the canker fungus *Botryosphaeria dothidea* have been demonstrated in the laboratory on madrone (64). Canker rot fungi (e.g., *Inonotus* spp., *Phellinus* spp.) are probably the most important endemic mortality-causing agents of oaks in these forests (95–97). Infected oaks typically died over many years due the relatively slow effects of these decay fungi. Snapping of *P. ramorum*-infected, but still green, trees has been shown to increase dramatically in areas with *P. ramorum* (97). This is primarily due the increased role of opportunistic decay fungi (e.g., *Hypoxylon thouarsianum*, *Phellinus gilvus*) and to a lesser extent wood-boring beetles (e.g., ambrosia beetles, *Monarthrum* spp.) (97). Rapid death of oaks and tanoaks due to infection by *P. ramorum* may lead to increased inputs of coarse woody debris into these forests with subsequent changes in soil nutrient status and impacts (positive or negative) on regeneration. Cascading impacts in these forests, if they occur at all, will likely become more apparent over the coming decades. However, although some important results can currently be documented in the field, many others are still hypothetical. Continued monitoring of coastal forests will be necessary to establish baseline data and document changes over time.

MANAGEMENT ACTIONS

Scientists and managers must continue to work to translate research findings into management actions to control *P. ramorum* and sudden oak death. Table 2 provides examples of recent research results and potential management actions that may be outcomes of this research. Below we summarize the necessary components of a management program for *P. ramorum* in forests of California and Oregon.

Monitoring

Continued monitoring for *P. ramorum* is essential for understanding spatial and temporal changes in the distribution of *P. ramorum* and quickly translating this into management action. This includes monitoring both of existing infested areas for changes in fine-scale distribution of *P. ramorum* and its entire potential geographic range for changes in coarse-scale distribution. Approaches used for early detection of the pathogen across the landscape include aerial surveys, ground-based surveys, stream monitoring, and community-based monitoring via the internet (37, 39b, 55, 57, 69, 72b). Risk models have been developed for California to target areas currently without the pathogen but that might be at high risk for invasion (41, 69). These models demonstrate the integration of data on host range, transmission biology, and plant community structure to develop rule-based models of *P. ramorum* establishment and spread risk in plant communities (69). Models must eventually incorporate the effects of spatial and temporal variability of multiple environmental variables on pathogen persistence (35, 80). The models developed to date

TABLE 2   Examples of research findings concerning *Phytophthora ramorum* and links to potential management actions

Research finding	Potential management applications
P. ramorum determined as causal agent of sudden oak death, but also with a wide host range. Symptoms differ significantly both in aspect, severity, and epidemiological role across plant species (18, 83, 104)	1. Chemical controls aimed at *Phytophthora* species 2. Development of specific diagnostic tests, both based on traditional culturing and DNA-based, to identify presence of pathogen, even without full understanding of symptoms type
P. ramorum spreads aerially via sporulation on infected leaves or twigs of plants (16, 17, 19)	1. Early detection of disease based on recognition of symptoms on leaves and twigs of such hosts 2. Reduction of inoculum potential through reduction of infection of foliar hosts (e.g., via composting or burns) 3. Control of spread of the disease by restricting trade in infected ornamentals
P. Ramorum consistentally disperses aerially 10 to 15 m from stand edges but up to 3 km in rare events (19; E. Hansen, unpublished data)	Dispersal distances used to set buffer zone distances in eradication areas and to develop survey and monitoring guidelines
Oaks are epidemiological dead-ends for the pathogen, i.e., pathogen sporulation does not occur on oaks. Sporulation can be induced by debarking and exposure to high moisture levels (19)	In the presence of infected oaks, inoculum reduction by chipping and drying of chips; avoidance of actions such as tarping in which debarking may be coupled by increase in moisture levels
Inoculum accumulates in soil and water; the pathogen is active year round in stream water; it becomes dormant but will survive in soil during dry Mediterranean summers. Some soil-to-plant and water-to-plant contagion documented through artificial experiments (19, 22)	1. Avoidance of movement of soil and of water from infested to uninfested areas 2. Avoidance of movement of water from infested drainage to other drainages (e.g., during dust abatement activities). Recycled water or water pumped from natural water bodies to be considered infectious
Smaller oaks rarely infected by the pathogen; there is a significant correlation between infected oaks and symptomatic bay laurel trees (56, 95, 96)	1. Focus of prevention on medium to large-size oaks within 10 m from infected bay laurel trees 2. No risk of infection to coast live oaks in open savannah 3. At the individual oak tree level, elimination of inoculum sources from the immediate vicinity of susceptible oaks

(*Continued*)

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TABLE 2   (*Continued*)

Research finding	Potential management applications
Sporulation is seasonal and positively correlated with rainy events and warm temperatures (19)	1. Limitation of activities in infested areas during periods (rainy and warm) of highest infection potential. Potential land closures in effect during these periods 2. Extra vigilance in nurseries when contagion conditions are optimal
P. ramorum is heterothallic; European isolates mostly bear the A1 mating type, while North American isolates bear the A2 mating type. Sexual reproduction is potentially possible between the two types. Significant genetic differences reported between the US and EU populations, better defined as independent lineages (12, 44, 52, 104)	1. Independent regulation of A1 and A2 isolates as they are genetically and phenotypically different even though part of the same species 2. Special vigilance where both types have been introduced
P. ramorum isolates display a phenotypic variability even when genetically identical based on selected molecular markers (9; D. Hüberli, unpublished data)	Limitation of movement of isolates within the known zone of infestation
Chemical treatments work best if preventively administered; several weeks are necessary to achieve maximum efficacy (27, 33)	Treatment of oaks and tanoaks that are not infected, but at high risk (i.e., near infected bays or tanoaks) at least three weeks before the rainy and warm season
Pathogen in infected green waste can sustain high temperatures for relatively long periods of time (26)	Flash heat treatment may be questionable; recommended treatments during which 55°C is reached within the infected plant tissue for an hour
Presence of varying resistance to a number of chemicals present in Europe, but also noticed in the United States (29; S. Werres, unpublished data)	No indiscriminate use of chemical treatments
Coast live oak populations across the entire geographic range in California, seem to have a percentage of individuals that are less susceptible to the pathogen (21)	Collection of germplasm from less susceptible oaks at each site for natural and human-assisted restorations, unless conditions become extremely favorable to the pathogen
Besides within-population variation in susceptibility of bay laurel trees to the pathogen, variation exists between some populations (D. Hüberli & M. Garbelotto, unpublished data)	Use of within- and between-population variation in susceptibility of bay laurel trees in predicting disease spread, especially in the absence of tanoaks

(*Continued*)

TABLE 2   (*Continued*)

Research finding	Potential management applications
Genetic variability within EU and US lineages is extremely limited and in agreement with exotic nature of *P. ramorum* in both continents (31, 44, 52, 79)	Implementation of action to stop or slow spread of *P. ramorum*, exotic aggressive pathogen, across potential new landscapes

have identified a number of currently uninfested forest ecosystems in California at considerable risk of invasion by *P. ramorum* (41, 69).

Diagnosis

Typically, diagnosis and confirmation of the presence of *P. ramorum* has been culture based. Such an approach is highly dependent on environmental conditions and may lead to false negative diagnoses, depending on the time of year at which samples are collected. Various molecular diagnostic assays have recently been developed to identify *P. ramorum* (28, 47, 59, 66, 106). RFLPs (61), lineage-specific primers (8), and microsatellites (31, 44, 51, 78) have also been successfully used to differentiate between the North American and European lineages of *P. ramorum*. In a comparison between successful diagnoses in California forests using a nested PCR approach and traditional isolation techniques, molecular diagnosis showed the greater sensitivity (47). However, PCR was not always successful in detecting the pathogen and was highly dependent on which plant part (e.g., wood versus leaves) or plant species was tested (47). The most promising results in forest surveys were obtained when combining traditional isolation and PCR-based assays (47). The development of newer diagnostic assays based on serological approaches or protein structure and composition may potentially reduce the rate of false negative results. The rate of false positive diagnoses using methods other than isolations needs to be monitored carefully by including negative controls at each step of the assay (28). Newly developed quantitative PCR assays have been (47, 66) used successfully (47) to provide information on the effects of substrate on the success of PCR-based assays and on viability of the pathogen.

Eradication

At the early stages of an invasion, eradication may be a viable option for disease management. Researchers and government agencies in Oregon are attempting an eradication program to eliminate *P. ramorum* from the small infested area in southwestern Oregon (39a, 53b). This program was established in 2001 because the disease was initially found to be concentrated in nine small patches of 5 to 40 diseased trees, each scattered over 21 km^2 of forest land, and located over 300 km from the nearest known infections in California. The disease was detected in

Oregon by aerial survey, and repeated surveys over the range of tanoak in the state verified its confinementto this small area. Existing state laws and administrative rules were invoked by the Oregon Department of Agriculture, and a quarantine area was delimited and landowners notified. Movement of soil and host plant material out of the regulated area is prohibited. Individual infected trees are confirmed by culture and PCR. Eradication areas are delimited after intensive ground checking to include all symptomatic host plants and host plants growing within 15 to 35 m of symptomatic plants. Host plants within each eradication area are cut and burned (Figure 4, see color insert), and stumps are treated to prevent sprouting. Sites are monitored for disease recurrence until they have been disease free for two years.

Three years into the eradication program, *P. ramorum* continues to kill tanoaks within the regulated area (53b). Fewer newly infected trees are found with each survey, however, and so far disease has not appeared outside of the watershed where it was initially identified. Although under-story rhododendron and evergreen huckleberry were commonly found infected in the first year, they are seldom infected several years following treatment. Infected bay laurel trees are rare. The program appears to have dramatically reduced inoculum levels in the area (53b).

The eradication prescription is a good example of adaptive management. New information about host range is regularly incorporated, and exceptions are allowed depending on individual circumstance. The prescribed buffer area around symptomatic trees is clearly inadequate to eliminate all new infections. Owners are encouraged to preemptively remove tanoak over a larger area once a new infection is found. It is usually not practical to increase the buffer in the portion of the quarantine area that is rural residential, but in areas of larger parcels, owners often find it better to expand the initial treatment spot than to face yearly treatments for the new spots of infection that may show around the initial perimeter.

Stand Manipulation

Eradication is being attempted in Oregon where the next nearest infestations are several hundred km away. However, within California such a strategy has not been implemented because the disease is considered to be too well entrenched. Silvicultural treatments in areas where *P. ramorum* is already well established will be similar to the procedures outlined above for eradication. Removal of infected plants (e.g., bay laurel, tanoak) within a forest stand could reduce inoculum levels. In addition, increasing the distance between plants may reduce tree-to-tree spread, as seen with other tree diseases. On the other hand, opening up a forest stand may actually lead to increased dispersal of the pathogen by allowing greater airflow within the canopy. A correlation has been found between disease presence and stand openness and edges in field plots (56, 95, 96). In addition, complete removal of one species (e.g., bay laurel) to save another may also cause unwanted changes in forest structure and composition. A safer approach, at least on smaller spatial scales, may be pruning the lower branches of bay laurel trees. This operation

may lessen inoculum loads without causing major shifts in forest communities. Current research is testing the effects of pruning or small bay tree removal on 0.25 hectare plots in the absence and presence of secondary carriers of *P. ramorum* such as tanoaks (M. Garbelotto, unpublished data). A pilot program is also under way to reduce inoculum densities in isolated infestations in Humboldt County in northern California through removal of bay laurel and tanoak (Y. Valachovic, personal communication).

Fire

Prescribed fire is an important consideration for any management program in coastal California and Oregon plant communities regardless of the presence of *P. ramorum*. Therefore, any use of fire as a management tool of *P. ramorum* must be put in context with its historical occurrence. Fire is long thought to have played an important role in coastal forests, although details remain scant (4). The severity and spatial distribution of fires varies considerably across the landscape in these areas (74). In coast redwood forests, fire return intervals appear to vary dramatically from 6 years to over 600 years, depending on the distance from the coast (85, 86). The historical role of fire in mixed-evergreen forests has not been extensively characterized (4). Fire regimes have changed since European Americans became part of the landscape, although Native Americans actively burned the landscape in many areas and certainly contributed to the heterogeneity of plant communities (54). Recent relationships of humans to fire in coastal forest are far from simple. Although fire suppression is generally considered to be the norm, in some areas (e.g., Klamath region of northern California), fire frequency is increasing (74). Even in some plant communities (e.g., shrublands) near the southern end of the range of *P. ramorum*, fire frequency also appears to have increased in recent years (70). Increased fire frequency through prescribed burns may actually be in conflict with natural fire regimes in some areas (70, 74).

How would fire be expected to influence the establishment, spread, and control of *P. ramorum*? The relationships of fire and *P. ramorum* are likely to be complex (70, 71). Fire suppression has undoubtedly influenced species composition and other environmental conditions of coastal forests that could potentially lead to increased establishment of *P. ramorum* (71). For example, lack of periodic fire may lead to increases in understory growth of highly susceptible species such as tanoak or bay laurel. In field studies in coast redwood forests, understory tanoak and bay laurel saplings have the highest infection rates (Maloney et al., manuscript submitted). On the other hand, severe stand-replacing fires can have the same effect by encouraging prolific regeneration of tanoak. This type of fire may be considered the natural fire regime in many northern California Douglas-fir—tanoak–mixed-evergreen forests (4, 50). Such a scenario has played out in northern Sonoma county where a stand-replacing wildfire approximately 25 years ago led to a nearly pure stand of tanoak that was invaded by *P. ramorum* (G. Slaughter, personal communication). Dieback in tanoak sprouts was noted following initial burning

treatments in the Oregon eradication zone (39a, 53b). Prescribed fires in areas already with extensive infection may lead to increased wounding of oak stems and possibly raise the susceptibility of infection. However, smoke from fires may lead to reduced survival of *P. ramorum* in bay leaves, as has been suggested for other pathogens (76, 88a).

Fungicides

Phosphite fungicides introduced into trees via direct injection or the use of bark organosilicate surfactant-penetrants have proven to be effective in preventing infection by *P. ramorum* on individual oaks and tanoak (33, 53a). These chemicals are now registered for use in California (27). Foliar applications have shown moderate success, whereas soil drenches have proven ineffective in preventing infection (33). Such chemical applications will be important in protecting highly valuable trees at the urban-wildland interface.

Landscape use of phosphite fungicides has proven useful in slowing the spread of the root pathogen *P. cinnamomi* in western Australia (6, 45, 46, 90). Treating at-risk forest areas greater than several hectares has required aerial application of the fungicide (90). The lack of significant side effects on the beneficial microbial flora or on invertebrates makes these treatments a viable tool to manage forests affected by *Phytophthora* species.

Translating such a landscape level control program to *P. ramorum* in California and Oregon could face many obstacles. Blanket treatments of entire stands are currently uneconomic. Large-scale aerial application of fungicides over densely populated areas would almost certainly provoke political opposition. The aerial nature of dispersal of *P. ramorum* mitigates against precise prediction of spread. The patchy distribution of *P. ramorum* on the landscape makes it difficult to target specific areas; a much wider broadcasting would be required. The ineffectiveness of soil drenches and the need for direct application to tree trunks are further drawbacks to the conduct of large-scale treatments. Landscape treatment may also exert a significant selection pressure toward resistant isolates in pathogen populations.

Perfecting dosages, timing of treatments, and selection of good candidate trees for treatments all require research attention. Recent reports have indicated that better delivery can significantly enhance the efficacy of fungicidal treatments (29), but still undetermined is the optimal number of trees to be treated per hectare to protect a population (obviously the number will change in different types of ecosystems) or whether naturally more resistant trees are the best candidates.

Prevention of Human Spread

Prevention of human-mediated spread of *P. ramorum* has been largely focused at the national and international levels through quarantines on nursery plants and other plant products (2). Relatively little attention has been paid to movement of the pathogen by people within the zone of infestation (15). This pathogen

is not ubiquitous, as evidenced by its patchy distribution on the landscape, and therefore measures designed to prevent its further spread could be useful. Anecdotal reports point to new pockets of disease associated with the planting of ornamental rhododendrons at the urban-wildland interface in California (83; D.M. Rizzo, personal observation). The important role of ornamental plants in the spread and establishment of *P. ramorum* is clearly seen in Europe where rhododendron plants have served as a pathway into forest trees (10). Therefore there should be no let up in surveillance of ornamentals even in areas where *P. ramorum* is already established in wild lands. This caution should extend to planting of infected stock at the urban-wildland interface or during restoration activities.

Other pathways for human-mediated transport include soil, green waste, and stream water (15, 19, 26, 101). Although the epidemiological significance of these latter pathways is not known and is probably low, there is still some risk, based on field and laboratory experiments (19, 26). These pathways must therefore be included in any management scheme. Treatments of green waste (26) and stream water (e.g., during removal for dust abatement) (42) are available. Prevention of soil movement will require educating the general public and professionals working in forests (14).

Restoration

After *P. ramorum*–impacted areas have been treated by interventions such as thinning or fire, decisions must be made on revegetation. Restoration of the forest or plant community where high tree mortality has been related to the presence of *P. ramorum* or other agents may perhaps require specific management strategies (93, 94). In addition, habitat restoration is being conducted in areas degraded by other problems (e.g., overgrazing, invasive plants), but that are in the vicinity of *P. ramorum*-affected areas (5, 99). Most existing oak forests have been manipulated by humans, so recent stand conditions, densities, species composition, and other factors may have changed subtantially from their original state. Many stand characteristics will change over time in both managed and unmanaged stands. Although the effects of *P. ramorum* may appear to be immediate and short-term, restoration (either natural or human-assisted) will typically proceed over at least several decades (93, 94).

Knowledge of host resistance will be useful to identify less susceptible genotypes for use in restoration programs. Trees with reduced susceptibility have been identified in all sites investigated, and are the most likely candidates to survive the predicted cyclic epidemic waves of *P. ramorum* (21). These less susceptible individuals may provide seed for the regeneration of *Q. agrifolia* populations that have been ravaged by *P. ramorum*. If reduced susceptibility is genetically inherited in significant proportions among offspring, seed is ecologically viable and genetically inheritable in significant proportions of the offspring, it may also be used to produce less susceptible planting stocks for reforestation programs, assuming there are no adverse genetically correlated traits.

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CONCLUSIONS

Phytophthora ramorum is one among many threats to the coastal forests of California and Oregon. Each year thousands of hectares of coastal forests are lost or modified by development, agriculture, timber harvesting, wildfire, or exotic species (4, 36, 77). Numerous initiatives by nongovernment and government agencies are under way to conserve oak forests and redwood forests: preservation of landscapes, reintroduction of historical landscape processes (e.g., fire), and development of long-term conservation policies. For example, managing the redwood resource in California requires integration of conservation issues with those of timber harvesting (99). The losses from *P. ramorum* have made the development of plans for conservation and landscape management even more imperative (48, 62). However, only recently has conservation strategy been geared toward protecting nontimber oaks (77).

It is tempting to decide that nothing can be done about this disease in coastal forests and simply let nature run its course. But the experience of past occurrences of other exotic diseases, such as chestnut blight and Dutch elm disease, confirms that inaction at the early stages of the epidemic is inappropriate. Ultimately, solutions will come through scientific knowledge combined with political intervention and public information. Task forces in both California and Oregon have brought together divergent interest groups to discuss the ramifications of sudden oak death and to develop strategies that will, through the setting of priorities and public education reach consensus (24). For example, eradication measures were implemented rapidly at sites in Oregon because the state was already committed to action if the disease was found. Cooperation between affected individuals and the forest industry has been very positive. Oregon state law has no provision to compensate landowners for eradication, but federal funds were secured to pay the costs of tree removal and burning. Public education, training sessions for professionals, open meetings, and media coverage continue to be important components in the wider strategy to manage the disease in California and Oregon.

Taking a broader look, many other forests throughout the world may potentially be susceptible to invasion by *P. ramorum*. The geographic range of many susceptible hosts on the west coast extends north and south from the main epidemic area. Combinations of tanoak and bay laurel extend into coastal areas of central Oregon. In British Columbia, the main known potential hosts include many ericaceous plants (madrone, *Vaccinium* spp.) as well as Douglas-fir (43). Highly susceptible hosts are also known to exist in other parts of the world (e.g., several oak species in eastern North America, beech in Europe), and foliar hosts of epidemiological importance, such as *Rhododendron* and *Kalmia*, are common genera in many forests (10, 82, 84). The availability of susceptible foliage and repeated rainy periods combined with mild temperatures seem to be keys to predicting risk and disease progression. Understanding survival rates of the pathogen from one year into the next is essential when attempting predictions of disease expression over multiple years. Whereas the pathogen has to survive through

hot dry summers in California, elsewhere it may have to survive through cold winters or through both summer and winter. These alternative scenarios exemplify the potential epidemiological differences that may be observed when disease progression is studied across different forest types and climate regimes, and highlight the risks of predictions based uniquely on the California and Oregon experience.

Ultimately, it may become clear that the presence of *P. ramorum* does not necessarily equate to presence of the disease in the way that it has been experienced in California and Oregon. Sudden oak death is likely to remain a disease characteristic of western coastal forests; different types of disease may be caused by *P. ramorum* in different parts of the world. These diseases may be equally, more, or less destructive than sudden oak death. Until we better understand the final effects of the pathogen in different ecosystems, a strict vigilance must monitor further introductions of *P. ramorum* around the world. The Oregon and European experiences have taught us that the spread of *P. ramorum* can be slowed with early detection. The California experience shows that although *P. ramorum* is dispersed aerially, it will not easily become ubiquitous like other aerially dispersed fungal pathogens. Even now, many areas in the zone of infestation in California remain free of the pathogen. This observation is a further reminder that continued monitoring is critical for early pathogen detection and for developing plans of action aimed at preventing further spread of *P. ramorum* in case of accidental introduction into wild ecosystems in different parts of the world.

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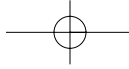
Figure 1 Aerial view of a typical landscape in Coastal Range of California showing the heterogeneity of plant communities (Marin Co.). Vegetation types include mixed-evergreen and redwood forests as well as grassland and chapparal plant communities. Mortality of coast live oak due to *Phytophthora ramorum* is visible in mixed-evergreen forest type. (Photo K. Parker)
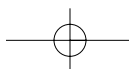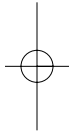
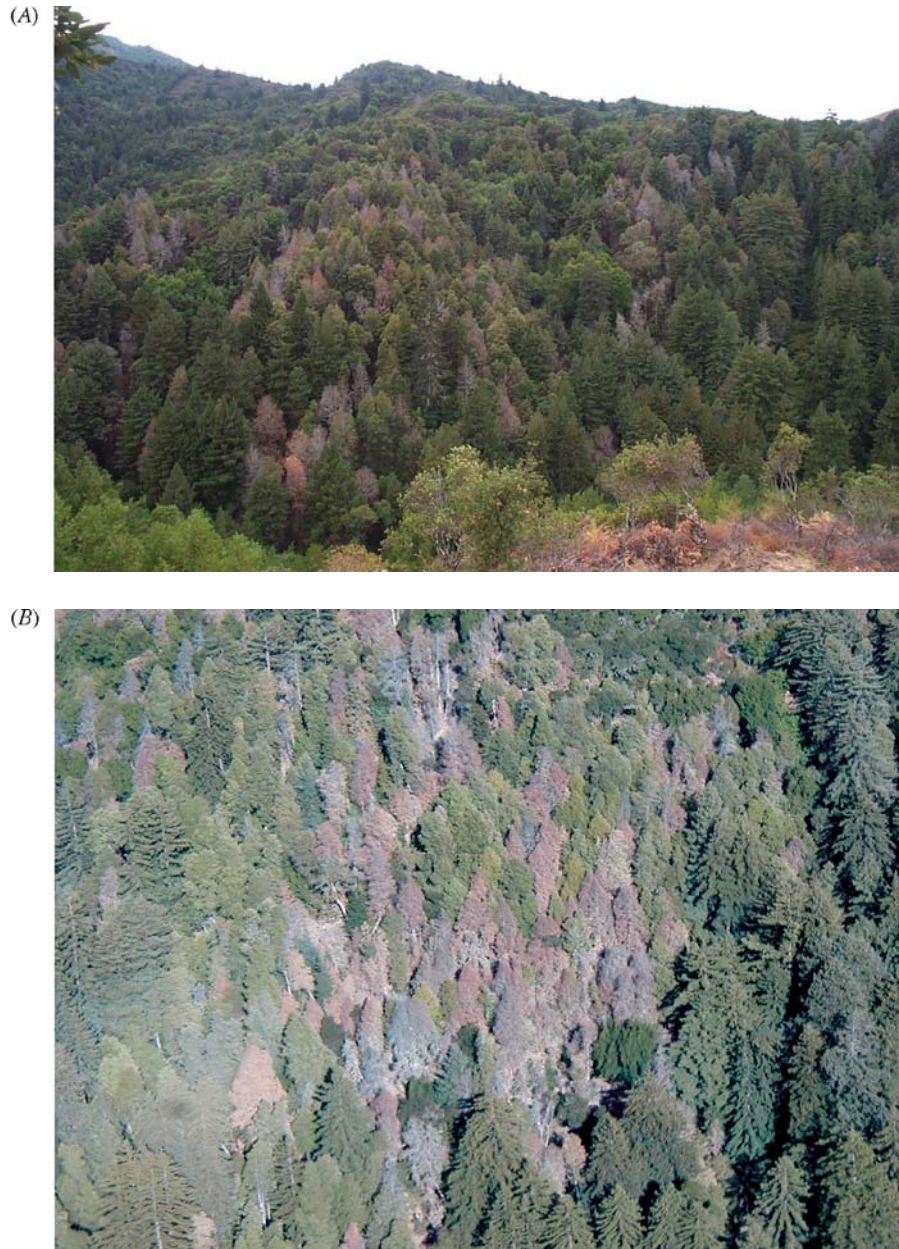**Figure 2** Distribution of *Phytophthora ramorum* in California and Oregon forests as of January 2005.
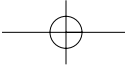
Figure 3 Overstory mortality of tanoak (*Lithocarpus densiflorus*) caused by *Phytophthora ramorum* in (*A*) Marin Co., CA and (*B*) Monterey Co., CA. (Photos by D. Rizzo, S. Frankel)
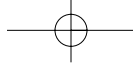
Figure 4 Host removal and burning as part of *Phytophthora ramorum* eradication efforts in southwestern Oregon. (Photos by A. Kanaskie, C. Brasier).